The primary purpose of this peer-reviewed journal is to provide a formal publication option for research completed by MSUCOM students, residents and faculty. SMRJ's mission is to advance medicine and medical education through the timely publication of peer-reviewed clinically-oriented research, clinically-relevant basic science research, healthcare quality research, and medical education research from MSUCOM and the osteopathic medicine community, with the ultimate goal of improving patient care and the education of patients and care providers. SMRJ is the official scholarly publication of the Statewide Campus System (SCS) of MSUCOM. It provides a forum for communicating research findings, clinical practice observations, philosophic concepts, and other biomedical and medical education advances to MSUCOM medical students, residents, fellows and faculty, and any other interested readers.

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Welcome to Our Second Issue of 2016

I’d like to welcome you to the second issue of Volume One of The Spartan Medical Research Journal (SMRJ)! With the retirement of Dr. Eric Zemper at the Statewide Campus System (SCS), I now have the distinct pleasure of serving as your new Chief Editor of your SMRJ starting with this issue.

As noted inside the cover of this issue, the purpose of our online peer-reviewed journal is to provide a convenient, formal publication option for research/quality improvement project and clinical case study results from Michigan State University COM students, residents, fellows and faculty. We continue to receive submissions from both SCS-affiliated and non-affiliated researchers from other states. I’m also happy to announce the addition of Mr. Sam Wisniewski, MS as our biostatistician at the SCS; Sam is now also serving as our Editorial Assistant on the SMRJ.

Those of us at the SCS continue to be dependent on a large number of colleagues to create these journal issues, especially from our growing number of expert reviewers. Our editorial office team offer many thanks to those many individuals who reviewed these Issue 2 submissions. We are still recruiting expert reviewers from most all medical specialty areas to be members of our SMRJ Editorial Board. If you have an interest in participating as a reviewer, please contact me.

If you have comments or suggestions, please also feel free to contact me at any time. Please remember that we also accept Letter to the Editor and Systematic Review submissions. I hope that you enjoy reading this second issue!

Sincerely,

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Chief Editor
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Medical Marijuana and the Treatment of Post Traumatic Stress Disorder: A Survey of Michigan Psychiatrists’ Opinions

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ABSTRACT

CUSTODIA J, MAGEN J, and CORSER W. Medical Marijuana and the Treatment of Post-Traumatic Stress Disorder: A Survey of Michigan Psychiatrists’ Opinions. Spartan Med. Res. J. Vol. 1, No. 2, pp. 1-18, 2017. CONTEXT: Although recent studies have indicated a potential mechanism of action through which medical marijuana (MM) and its derivatives may treat Posttraumatic Stress Disorder (PTSD) symptoms, definitive evidence is still lacking. Few studies concerning physician attitudes regarding MM and/or marijuana-derived medications for PTSD are found in the psychiatric literature. METHODS: A non-probability convenience sample of psychiatric physicians in Michigan was surveyed during 2016. The 12-item survey questionnaire asked respondents a series of questions about their personal characteristics, prior experiences of treating PTSD and opinions concerning the use/potential use of MM for treatment of PTSD. RESULTS: A total of 83 psychiatrists (11.7% of total invited) responded to the survey. Several statistically significant correlations between respondent characteristics and other key measures (e.g., Age Category, Gender, Years of Psychiatric Practice, Psychiatric Practice Role (i.e., resident vs. attending), Number of Psychiatric Subspecialties, and Number of PTSD Patients Diagnosed and/or Treated to date) were found. A composite summary score was also formulated from questions related to opinion regarding the use of MM for PTSD and categorized into three comparison groups. The final stepwise multinomial logistic model demonstrated three statistically significant factors influencing what response category respondents fell into regarding MM use for PTSD: a) how often respondents had been exposed to recommendations concerning the use of MM for PTSD (p < 0.001), b) Age Category (p = 0.001) and how frequently respondents had recommended MM for treatment of PTSD (p < 0.001). CONCLUSIONS: The results from this smaller sample indicate that the majority of psychiatrist respondents did not support MM for the treatment of PTSD. Judging from these results, Michigan psychiatrists may be extremely conservative regard the prospective use of MM for PTSD. Few sample respondents indicated that they had been exposed to professional literature detailing MM and derivatives as a treatment for PTSD. Most respondents also indicated that they were Unsure/There is Not Enough Research concerning the scientific evidence for the use of MM for PTSD. Based on these findings from a smaller sample, the use of MM and its derivatives for treatment of PTSD may not currently be supported by the majority of Michigan psychiatrists. Keywords: medical marijuana, post-traumatic stress disorder, symptom relief
INTRODUCTION

Post-traumatic Stress Disorder (PTSD) is a psychiatric condition resulting from having experienced or witnessed a traumatic event, with a characteristic constellation of symptoms including event re-experiencing, avoidance of event reminders, and hyper-arousal.\(^1\) Although the great majority of people who experience a traumatic event never develop PTSD, some may develop long-lasting symptoms which can greatly affect their daily functioning and overall quality of life.

According to the U.S. National Institute of Mental Health’s most recent statistics, PTSD has a lifetime prevalence of 6.8%, with a 12-month prevalence of 3.5%. Of those cases, over a third are classified as severe.\(^2\) Although PTSD is among the most debilitating and prevalent of psychiatric conditions, it remains among the most recalcitrant conditions to treat with highly variable prognoses.\(^3\) Indeed, some patients suffer from reemergence of symptoms in spite of prolonged treatment.\(^3\)

Clinicians’ typical difficulty in ameliorating the symptoms of PTSD has driven a search for potential therapeutic agents that may provide relief.\(^3\) For many patients afflicted with PTSD, the only agent they feel can effectively palliate their symptoms may be medical marijuana (MM).\(^4\) Although marijuana has been listed as a Schedule I controlled substance since the inception of the Controlled Substances Act of 1970,\(^5\) there has been a growing movement to approve its use as a specific therapeutic agent for PTSD. In seven states and the District of Columbia, PTSD is now an approved condition for the prescribed use of MM.\(^6\)

In 2014, the State of Michigan legislature approved the therapeutic use of MM for PTSD after a panel of non-physician experts heard patients’ testimony.\(^7\) However, these MM use policy changes came with little input from physicians, and virtually none from psychiatrists, those clinicians most likely to treat PTSD. Currently, national psychiatric PTSD treatment guidelines do not universally advocate including the therapeutic use of marijuana or any marijuana-derived medications.\(^3,8\) In fact, the most recent practice 2009 guideline update from the American Psychiatric Association makes no mention of marijuana as a possible treatment option for PTSD.\(^8\)

Due to MM’s conflicting legal status (i.e., being legal at the state level but illegal at the federal level), very few, if any, physicians now appear to prescribe marijuana in...
any form to patients.⁴ Although studies concerning the effects of marijuana and its derivatives have steadily increased during the past decade, there are still only a handful of published studies concerning the effect of MM on PTSD symptoms.⁸

**PTSD and Marijuana Use**

There has long been an anecdotal association between the use of marijuana and PTSD symptoms.⁴ Most studies to date have focused on the well-known negative consequences of cannabis use, including an increased risk for several other substance use disorders.⁴ However, other groups have sought to specifically examine the relationship between PTSD symptoms and MM use.

For example, a study in 2011 by Cougle et. al., determined that having a diagnosis of PTSD significantly increased the odds of a lifetime patient history of cannabis use, with 50% of sample subjects stating that their PTSD symptoms occurred prior to, or around, the same time as their first cannabis use.⁹ A similar follow-on study demonstrated that cannabis users with higher PTSD scores were significantly more likely to use cannabis to improve their sleep and coping than subjects with lower PTSD scores.¹⁰ The results from these initial studies have suggested that MM may exert some neurobiological effect resulting in perceived relief of PTSD symptoms.

**Neurobiological basis for the use of marijuana in PTSD**

Within the past five years, more critical reviews of the medical literature regarding the use of marijuana for PTSD symptoms have been published. These reviews have cited many of the findings already described in this paper, but have also provided some notable new insights. Two separate reviews detailed marijuana’s possible mechanism of action through the effects of delta-9-tetrahydrocannabinol (THC), the main psychoactive cannabinoid in marijuana, on endogenous cannabinoid receptors of the brain’s endocannabinoid system (ECS).¹¹,¹² The ECS appears to be involved in memory formation, fear, and emotion or executive functioning, areas of the brain likely active during memory formation/extinction in PTSD.¹¹,¹²

Other studies have focused on the effects of marijuana-derived medications on the ECS, including the FDA-approved medication Dronabinol ¹³ (i.e., indicated for appetite stimulation in cancer patients and anorexia), the synthetic cannabinoid Nabilone ¹⁴ (i.e., primarily used as an antiemetic for cancer), and the endogenous...
cannabinoid neurotransmitter Anandamide (for anxiety/depression). Some research has focused on imaging studies using positron emission tomography (PET) to examine the ECS, showing an increase in the concentration of cannabinoid receptor availability in subjects with PTSD versus healthy controls.

However, there is still a dearth of information about psychiatrists’ opinions on this topic. Only one survey project was found by the authors during the past 20 years that attempted to gauge physicians’ opinions on the use of MM for any indication. The results from this project indicated that physicians were, in general, less supportive than the general public regarding the use of MM.

In summary, research has indicated that the ECS appears to be a viable therapeutic target to treat PTSD, and that cannabinoids (both synthetic and endogenous) may be potential therapeutic agents. However, many of these studies used smaller samples and have not been replicated. Furthermore, it remains unknown whether these results have been routinely disseminated to practicing psychiatrists.

In light of the increasing number of states legally approving MM for PTSD, it is imperative to examine the opinions of behavioral health professionals who treat PTSD about this newer treatment modality. Since psychiatrists most frequently treat PTSD patients, their expertise is extremely important in helping shape the discourse concerning this potential PTSD treatment.

**Project Purpose**

This exploratory pilot study was conducted to investigate the perspectives of psychiatrists on the evolving topic of MM use for treatment of PTSD. The authors conducted a survey of attending and resident psychiatrists in Michigan to ask respondents a number of questions related to the potential use of MM and/or its derivatives for the treatment of PTSD. A secondary goal of this study was to clarify psychiatrists’ current opinions concerning the use of MM versus marijuana-derived medications for the treatment of PTSD symptoms.

Before the study, the authors had generally hypothesized that younger (i.e., resident) psychiatrists would be more receptive to prescribing MM for PTSD. In addition, the authors had speculated that those respondents more familiar with guarded recommendations from groups (e.g., American Academy of Neurology, Federation of
State Medical Boards\textsuperscript{19}) for the use of MM to palliate PTSD symptoms would prove more supportive.

**METHODS**

A cross sectional email-based survey was distributed to a total of 723 licensed psychiatrists in the state of Michigan between February 11, 2016 and March 11, 2016 using the \textit{Survey Monkey} internet-based survey program.\textsuperscript{20} The survey developed by the first two authors asked respondents a series of 12 questions regarding their professional opinions about MM and its derivatives as choices for the treatment of PTSD symptoms. Most opinion questions used a Likert-type scale, including No Response/Unsure response option. An additional open-ended comment item was also added at the end of the survey to further gauge psychiatrists’ opinions on the study topic (see Figure 1).

**Study Population**

Subjects were drawn from an email database of psychiatrists currently practicing in the state of Michigan. The email database had been obtained from the state branch of the American Psychiatric Association, with study approval obtained through the Michigan State University institutional review board.

**Data Analyses**

All data analyses were conducted using \textit{S.P.S.S. Version 22}.\textsuperscript{21} A series of descriptive statistics were first generated with cross-tabulations completed to examine for statistically significant bivariate correlations across major study measures to inform the conservative selection of later multivariate modeling procedure model terms. Key correlations between respondent characteristics (i.e., Age Category, Gender, Years of Psychiatric Practice, Psychiatric Practice Role (i.e., resident vs. attending psychiatrist), Number of Psychiatric Subspecialties, and Number of PTSD Patients Diagnosed and/or Treated to Date) were initially examined.

Finally, a forward stepwise Main Effects multinomial logistic regression (MLR) model comprised of potentially-significant model terms was conducted. Such a modeling procedure was more appropriate for this non-normally distributed sample to predict the probability of use of MM for PTSD category membership based on major
In this MLR model, each model term was entered individually (i.e., *stepwise*) to gauge their significance for the three composite score categories (i.e., *Generally Against Use of MM for PTSD, Neither Against of Supportive of Use of MM for PTSD* and *Generally Supportive of Use of MM of PTSD*), with those variables with non-significant test statistics (i.e., greater than p value of 0.100) removed from the final predictive model.

**RESULTS**

Of the 723 email surveys sent, 16 (2.2%) were not successfully delivered due to invalid email addresses. Of the 707 remaining surveys sent to active email accounts, 83 (11.7% of total invited) subjects responded to the survey. A total of 34 (41%) respondents reported being younger than 35 years of age, with the seven age categories of respondents quite diverse. A total of 44 (53.0%) respondents were males (See Table 1).

A total of 42 respondents (50.6%) reported having practiced in psychiatry for less than five years at time of survey. The average number of psychiatric subspecialties reportedly held by respondents was 1.05 (SD 0.825). Forty-eight (57.8%) sample respondents reported having diagnosed or treated a broad range of between 1 and 100 PTSD patients at time of survey, with 26 (31.3%) additional respondents indicating that they had treated more than 100 PTSD patients (See Table 1).

Notably, only one respondent (1.2%) indicated that they *Sometimes* recommended the use of MM for PTSD, with the great majority of the remaining sample answering *Never* (90.4%). Only 11 (13.3%) of total respondents indicated that they had *Sometimes* or *Often* been exposed to the notion of MM being an appropriate PTSD treatment in the professional literature or at conferences.

Thirty-one respondents (37.4%) stated they would *Never* prescribe MM, even if it became FDA approved and/or removed from the federal *Schedule of Controlled Substances*. Twenty-five other respondents (30%) said they would *Rarely* prescribe MM for PTSD, with 17 of remaining respondents (20.5%) surveying that they were *Unsure/No Comment*. In the survey items related to respondents’ opinions on the actual scientific evidence of MM for the treatment of PTSD (Questions 9-12 in Figure 1), a
majority of the respondents (n = 44) selected the answer *Unsure/There is Not Enough Research*.

**Key (significant and non-significant) bivariate correlations included:**

1. Age Category with a) Gender (p = 0.030, with women respondents tending to be younger), and b) Category of Number of PTSD Patients Diagnosed/treated to date (p < 0.001, with older respondents having treated more PTSD patients);
2. Years of Psychiatric Practice with likelihood category of having recommended MM to patients more frequently in the past (p = < 0.001), with more experienced respondents more likely to recommend MM for PTSD); and
3. More frequent exposures to recommendations concerning use of MM and composite MM use belief score category (p = 0.001, with more frequently-exposed respondents more likely to prescribe).

The survey included three different related questions related to respondents' opinions regarding use of MM to treat PTSD which were conservatively categorized into three overall groups: 1. *Neither Against or Supportive of MM use for PTSD*, (n = 11, 13.3%) 2. *Generally Against MM use for PTSD*, (n = 19, 22.9%) and 3. *Generally Supportive of MM use for PTSD* (n = 9, 10.8%). It should be acknowledged that 44 respondents (53.0%) opted to not answer at least one of these three survey items. This overall opinion categorical measure was treated as a de facto *outcome measure* for most subsequent analyses.

In the final MLR stepwise model for outcome, the following model terms remained statistically significant after controlling for other non-significant model terms: a) How Often Respondent had been Exposed to Recommendations concerning use of MM for PTSD patients (p < 0.001), b) Age Category (p = 0.001), and c) How often Respondent had Previously Prescribed MM for PTSD (p < 0.001) (see Figures 2, 3, and 4 for general depiction of key results). The *goodness of fit* and other model fitting information from the final MLM model were each quite adequate, although the number of respondents with entirely complete survey data that could be included into this stepwise model was especially low (n = 27). Still, the authors were able to identify these
statistically significant predictors of respondents’ opinions regarding MM use for PTSD in spite of their limited sample size.

**Limitations**

This study did have several constraints related to the inherent limitations of smaller cross-sectional survey studies. The results may have also been skewed by other unmeasured factors, including a *self-selected* group of psychiatrists actually responding to the survey. For instance, respondents in this sample were predominantly young and less experienced, with approximately 40% in the 25-34 years age group category and over half of the respondents in psychiatric practice less than five years.

Based on fairly loose national estimates, it is certainly possible that these respondents comprised a somewhat non-representative (i.e., younger and less-experienced than average) group of Michigan psychiatrists. As for the survey tool itself, the questionnaire had not been psychometrically tested before the study, having only been previously examined by several psychiatric experts for overall validity. During these initial survey reviews, the clarity of some of the questions wording and use of terms and response options had been edited.

**CONCLUSIONS**

The results of this initial pilot study suggest that while a majority of the psychiatrists in the sample were treating PTSD patients at time of survey, they remained quite conservative in regard to using MM to alleviate patients’ PTSD symptoms. This is evidenced by that fact that only one (1.2%) of the 83 respondents stated they had actually recommended the use of MM for the PTSD treatment in the past. Also, a large majority of psychiatrist respondents stated that they would not prescribe MM even if it were to be taken off the FDA’s list of controlled substances. The authors’ hypotheses that both younger respondents and those more familiar with established guidelines for MM use would be more supportive of MM use were not generally supported by results.

These results also demonstrate that few psychiatrists may be getting exposed to recommendations in the professional literature or conferences regarding MM and/or its derivatives as a treatment modality for PTSD. Only 11 (13.3%) of the total sample
reported that they had *Sometimes* or *Often* been exposed to such recommendations. This is in line with responses made related to the actual perceived scientific evidence for the use of MM for PTSD, in which a majority of the respondents indicated *Unsure/There is Not Enough Research*. Even though the authors’ final MLR stepwise model showed three statistically significant model terms related to respondents' categorical opinions in the area, it is difficult to draw definitive conclusions from these study findings.

Perhaps the most revealing study finding came from the comments section in which respondents were allowed to provide open-ended statements concerning this practice issue. Many of these qualitative responses expressed ardent positions against the future use of MM for PTSD symptoms, but also against marijuana in general. As one psychiatrist stated, *I have seen enough functional deficits from marijuana in patients across all age spectrums as well as cognitive decline. I would never prescribe medical marijuana for any reason*. Many other respondents pointed out the negative effects of medical marijuana as a barrier to its therapeutic use.

Another respondent also questioned the validity of legislative processes for MM approval, stating, *Medical marijuana is a lie. It was passed in several states by vote or legislative [action] rather than scientific rigor like other medicines. There are many cannabinoids and some may be useful but we need more studies.*

In fact, the call for more research and literature on MM was a common theme amongst respondent comments. For example, one respondent stated, *Thank you for including me in this survey. Some of your questions are quite provocative and will cause me to pursue additional reading in this area. It would be helpful if you provided your respondents a few relevant citations on the subject.*

Future studies in this area would benefit from the use of psychometrically-tested surveys lending themselves to more detailed statistical analyses. It should be clearly acknowledged that this project team was very likely underpowered to detect meaningful sample subgroup differences that might be otherwise identified within larger future samples. Since marijuana is still illegal under federal law, doctors may not technically prescribe MM without violating the law, even in states where MM agents have been
approved. Therefore, it is not unreasonable to conclude this fact could have influenced
the responses from this study sample.

However, this cross-sectional survey study is still apparently one of the first to
systematically examine the opinions of psychiatrists on the use of MM for the treatment
of PTSD. Judging from these results, this remains an especially complex clinical care
issue, with therapeutic, social, and legal facets that have not yet been thoroughly
considered by practicing psychiatrists. As such, there is still a vital need for more
studies to further clarify clinician opinions concerning the potential use of MM and
marijuana-based medications for this disorder.

There remains a paucity of results from randomized controlled studies that could
be used to inform current clinical guidance in the use of MM for PTSD. Ideally, the
results from future research can inform the development of more evidenced-based
guidelines for practicing psychiatrists.6,8,9

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The authors declare no conflict of interest.
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5. Controlled Substances Act of 1970. Available at:
2016.


Figure 1: Survey Items of Psychiatrists’ Opinions on Medical Marijuana for Post-Traumatic Stress Disorder.

1. What is your age?
   - 18 or 24
   - 25 to 34
   - 35 to 44
   - 45 to 54
   - 55 to 64
   - 65 to 74
   - 75 or older

2. What is your sex?
   - Male
   - Female

3. How many years have you been in psychiatric practice?
   - Less than 5 years
   - 5-10 years
   - 11-20 years
   - 21-30 years
   - Greater than 30 years

4. What is/are your subspecialty(ies)? (select all that apply):
   - Adult Psychiatry
   - Child and Adolescent Psychiatry
   - Geriatric Psychiatry
   - Addiction Psychiatry
   - Forensic Psychiatry
   - Psychosomatic Medicine
   - Other

5. Estimate how many patients you have diagnosed and/or treated with PTSD:
   - None
   - 1-50
   - 51-100
   - 100-200
   - Greater than 200
   - Unsure/no comment

6. How often have you recommended to patients the use of medical marijuana for the treatment and/or management of PTSD?
   - Never
   - Rarely
   - Somewhat often
   - Very often
   - Always
   - Unsure/no comment

7. In your review of research literature and/or attendance of professional conferences, how often have you read or been told that medical marijuana may be an appropriate treatment for PTSD? (same response scale as #6)

8. How often would you prescribe medical marijuana for patients with PTSD if it became FDA approved and/or removed from the Schedule of Controlled Substances? (same response scale as #6)

9. What is your opinion of the scientific evidence concerning the use of medical marijuana for the specific treatment of PTSD?
   - Evidence is strongly against use
   - Evidence is somewhat against use
   - Evidence is neither for nor against use
   - Evidence is somewhat in favor of use
   - Evidence is strongly in favor of use
   - Unsure or There is not enough research
10. What is your opinion of the scientific evidence concerning the use of medical marijuana for the
treatment of symptoms related to PTSD (anxiety, hyper-vigilance, insomnia)? (same response scale as
#9)

11. What is your opinion of the scientific evidence concerning the use of marijuana derivatives (i.e.
Cannabidiol) for the specific treatment of PTSD? (same response scale as #9)

12. What is your opinion of the scientific evidence concerning the use of marijuana-derived medications
(i.e. Marinol) for the specific treatment of PTSD? (same response scale as #9)

13. Please leave any comments that you feel may be pertinent to this topic:
Table 1: Sample Respondent Characteristics (N = 83 Psychiatric Physicians)

<table>
<thead>
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<th>Category</th>
<th>n</th>
<th>% of category</th>
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<tr>
<td>• 35 to 54 Years Old</td>
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<td>• 55 Years and Older</td>
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<td>2. Gender *</td>
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<tr>
<td>• Female</td>
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* Data from One Respondent Missing
“How often have you recommended to patients the use of medical marijuana for treatment/management of PTSD?”
Figure 3: Composite Belief Category for use of Medical Marijuana for Post-Traumatic Stress Disorder by Age Category (n = 39)
"How often would you prescribe marijuana to treat PTSD if FDA approved and/or removed from ...
Original Contribution

Trauma Dictations in the Emergency Department: A Quality Improvement/Patient Safety Project

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ABSTRACT

TOTH EW, RICHARDSON K, BERRY C, and BUTKI N. Trauma Dictations in the Emergency Department: A Quality Improvement/Patient Safety Project. Spartan Med. Res. J. Vol. 1, No. 2, pp. 19-31, 2017. CONTEXT: Trauma patients frequently represent a unique and challenging patient population in emergency medicine care settings. The policy of the McLaren Oakland Emergency Department (ED) is to have the treatment of all Level 1 and Level 2 trauma activations dictated by the ED resident. This policy is intended to facilitate both patient safety through clear communication between multiple medical services and quality improvement through reporting trauma specific quality metrics to third party agencies. Despite this requirement, trauma dictations in this setting were often found to not be completed. The purpose of this quality improvement/patient safety project was to implement a trauma dictation template to increase the rate of completed ED trauma dictations to above 75% of all Level 1 and Level 2 trauma activations. METHODS: A trauma dictation template was created to aid ED residents while completing trauma dictations. It was thought by the authors that the implementation of a standardized dictation template would help residents specify the necessary components needed to improve both patient safety and quality reporting. The development of the template was a collaborative effort between the emergency medicine residents and faculty, the trauma coordinator and trauma surgeons. The project was evaluated using two separate measures. A "process measure" was first used to determine if the addition of the trauma template made dictating less burdensome for ED residents, and an "outcomes measure" helped the authors examine whether template implementation actually led to an increased rate in completed trauma dictation. RESULTS: Data were collected during a three-month period prior to template implementation and three months after implementation. From November, 2015 through April, 2016, a total of 132 Trauma Activations were reviewed. The rate of completed dictations on Level 1 trauma activations increased from 45.16% to 53.33%. However, the rate of dictations on Level 2 trauma activations decreased from 50% to 30.4%, suggesting that Level 1 trauma patient care may have derived greater improvements from the new dictation template. CONCLUSIONS: The results of the ED residents’ evaluative survey responses were generally positive, indicating that the trauma template was perceived by most residents as a useful tool to complete dictations.
Even though the outcome goal was not achieved, the project successfully achieved the goal of creating and implementing a usable trauma dictation template. Following the Plan-Do-Study-Act model for quality improvement/patient safety projects, the next project will examine additional barriers preventing users from utilizing this initial well-received tool. **Keywords:** emergency medicine, trauma, dictation, patient safety.

INTRODUCTION

Trauma patients often represent a uniquely challenging patient population in emergency medicine settings. Trauma patients frequently sustain injuries to multiple organ systems which necessitate consultation and treatment across multiple medical and surgical specialties. These consultations may occur across different locations quite distant from patients’ initial emergency department (ED) care setting. Therefore, patient safety principles necessitate accurate communications regarding patients’ initial presentation and ED care for all subsequent providers to convey correct and appropriate diagnostic and treatment plan information.¹

As a Level II trauma center, McLaren Oakland has observed a trauma activation protocol since 2006. Using objective criteria concerning patients’ mechanism of trauma, physiology (i.e., vital signs) and anatomic findings (e.g., depressed skull fracture), trauma patients are assigned by providers into one of three trauma activation categories. Based on these criteria, the most critical and unstable patients are activated at a Level 1. Seriously injured but currently stable patients are activated as a Level 2. Stable patients are activated as a Level 3. Activating each injured ED patient into a trauma level initiates a chain of events and notifications designed to meet their anticipated clinical needs.²,⁴

For example, Level 2 activations automatically mobilize radiology and laboratory technicians to immediately present to the ED to obtain emergency radiographs and blood specimens. A Level 1 activation does the same, but also activates operating room and anesthesia personnel to anticipate and prepare for emergent surgery.

The current documentation platform used before this project in the McLaren Oakland ED in Pontiac, Michigan was a typical physician ‘T sheet’ template. These available paper templates are designed to be specific to the most common types of patients’ presenting chief complaints. Each template has a series of questions and fields concerning physical exam findings that are likely to be relevant to that chief complaint.
The T sheet allows physicians to quickly document the key parts of an ED encounter by checking boxes and circling items for positive results, and crossing through negative finding fields.\textsuperscript{4,5}

T sheets have long been used in EDs, and are widely used as excellent tools to capture the necessary elements of an encounter for billing purposes.\textsuperscript{5} Examples of T-sheets used for trauma cases include “Motor Vehicle Collision,” and “Multiple Trauma.”

Although efficient, T sheets tend to impose several limitations on providers. For example, they often provide very little space for clinician progress notes and documentation to inform providers’ subsequent medical decision making. The extensive use of circled boxes and pre-determined phrases on most T sheets also typically means that the individual thoughts of the physician are not recorded. It is therefore difficult for other providers to glean the critical details of the case when simply reviewing a T sheet.\textsuperscript{4,5}

In addition, T sheets do not currently allow documentation of ATLS protocol elements for major trauma patients. There are generally no areas which designate primary and secondary survey information or a section for procedures, critical aspects of the management of most all trauma patients and outlined by both the American College of Surgeons (ACS), and the Advanced Trauma Life Support (ATLS).\textsuperscript{2,3} When it came to allowing providers to enter detailed documentation for their trauma patients, the standard physician T sheet is simply inadequate.

Since 2006, the McLaren Oakland Emergency Department had implemented a policy requiring that ED patient encounters leading to all Level 1 and Level 2 trauma activations be verbally dictated by providers in addition to their standard T sheet documentation. A verbal dictation to receiving providers was completed by the ED resident prior to the patient leaving the ED for the general medical floor. These dictations at the authors’ setting are transcribed by a medical transcription service with an average single business day turnaround time. These ED dictations are separate from the trauma History and Physical reports completed by the admitting medical services.

During this study, the purpose of the trauma dictation template was twofold: 1. To help facilitate patient safety by clearly communicating the patient presentation and ED treatment to multiple medical and surgical services subsequently caring for the patient,
and 2. To improve the accuracy of quality trauma service metrics reported to third party agencies.

**Purpose**

Despite the established trauma dictation policy, the authors noted that many Level 1 and Level 2 activations were not being verbally dictated. This quality improvement/patient safety (QIPS) project had two primary phases. The first phase was a systematic needs assessment to examine factors contributing to trauma dictations not being completed. The second phase included the creation and implementation of an easier streamlined template for trauma dictations. The overall goal of the project was to increase the percentage of completed Level 1 and Level 2 trauma activation dictation to over 75% during the six months after template implementation.

**METHODS**

Since no patients were being studied during this project, a request for Determination of Non-Human Subject Research was completed in January, 2016 and approved by the McLaren Human Research institutional review board. During the design of the project, the authors had made extensive use of the Plan, Do, Study, Act (PDSA) sequential model for quality improvement as advocated by the Institute of Healthcare Improvement.6,7

The first step in the project was to identify barriers currently influencing the rates of complete trauma dictations made by EM residents. At the time of the study, almost 100% of all trauma patients were staffed by ED residents, making ED resident physicians the principal documenters and target group for the intervention. During September and October, 2015, three discussion groups were conducted, during which a group of over ten EM residents were asked why they thought trauma dictations were not being performed. The cause-and-effect or ‘fishbone’ diagram was used as a tool during these sessions to help determine the root causes of this problem.8 During the discussion groups, the three following primary barriers emerged: 1. Many EM residents were not aware that a dictation was required for all Level 1 and Level 2 trauma activations; 2. The dictation process was viewed by many participating residents
as being prohibitively arduous and time consuming; and 3. It was unclear to some participants what information was expected to be included in the dictation.

In response to these perceived barriers, the next step in the project was to design a trauma dictation template based on published best practice examples.9,10 The template was designed through collaboration of the McLaren Oakland trauma surgeons, senior EM residents, the administrative trauma coordinator at McLaren Oakland and EM program faculty physicians.

The template (see Table 1) listed all of the information that the participating parties felt was necessary to achieve adequate levels of patient safety and included fields for information that the trauma coordinator needed for reporting quality metrics to payers. The template fit neatly onto a two-sided page and had been presented and approved at the Trauma Committee meeting in January 2016. After approval, the template was placed as a pdf file on the desktop of every computer in the ED department where verbal trauma dictations occurred.

The final step in the project was an educational intervention designed to train the users of the new template. An educational lecture was delivered in January, 2016 as a 15-minute EM resident didactic session. The lecture was intended to educate the ED residents about the policy for completing dictations for all Level 1 and Level 2 trauma activation patients and how proper documentation of medical decision making was expected to impact ED patient care and safety. Short “booster” reminders of the dictation policy were provided later in the months following implementation during three subsequent three didactic sessions.

The impact of the project was evaluated using two separate measures. A “process measure” was first used to determine whether the availability and design of the dictation template was perceived to minimize perceived barriers and make dictating less burdensome for the EM residents. An “outcomes measure” also determined if the template actually led to an increase in the rates of completed trauma dictations.

To evaluate the process measure, a June, 2016 post-implementation survey was distributed to the EM resident users of the trauma dictation template. Answers were anonymous, with the intent to gauge the perceived usefulness of the dictation template among the residents. The residents were asked four questions regarding their
awareness of the dictation template policy, existence and location of the template, whether they found the template useful, and whether they felt that dictating trauma cases affected the quality of the ED care they provided.

For the outcomes measure, a retrospective chart audit was completed to compare the percentage of completed Level 1 and Level 2 trauma activations with verbal dictations from the three months prior to the introduction of the template with the three post-implementation months.

**RESULTS**

Project data concerning the outcome measure were collected by the authors’ healthcare system Trauma Department charged with quality control measurement. For this project, the primary metric was the rate of Level 1 and Level 2 trauma activations that had a dictation completed by the ED resident. No attempt was made to determine the quality of the dictation or evaluate any content in individual template dictations. Pre-implementation data were collected from November 2015 through January 2016, with post-implementation data extracted from February 2016 through April 2016. Overall, 132 trauma activations were reviewed during the total six-month project period.

During the three months preceding the template implementation, the complete dictation rate for Level 1 trauma activations were 14 out of 31, or 45.16%. Three months after the dictation template was introduced, the rate of complete Level 1 trauma activations had increased to 11 out of 21, or 53.33%. This 18.09% increase represented a minimal change from the pre-implementation rate, far below the authors’ goal rate of 75%. Of course, the number of trauma activations during this time period was largely out of the direct control of the investigators and the authors may certainly have been underpowered to demonstrate statistically significant pre-post differences using inferential statistical analyses.

The results for the Level 2 trauma activations demonstrated a decreased dictation rate after the introduction of the dictation template. In the three months before the dictation template implementation, the completed dictation rate was 17 out of 34, or 50%. After the introduction of the dictation template, the dictation rate actually
decreased to 14 out of 46, or 30.4% (See Figure 1). This change represented a 39% decrease from the pre-implementation rate.

To examine the selected process measure, a post-intervention survey was distributed to the EM residents regarding their experiences using the trauma dictation template. Overall, 13 (46.4%) of the 28 EM residents responded to the survey. Survey results indicated that although 8 (61%) of the 13 respondents were aware of the trauma dictation policy and that a dictation template was available, a large number 5 (39%) of 13 residents indicated that they were still unaware of the trauma dictation policy. Of those who had used the dictation template, 9 (81%) of 11 indicated that it was useful and made their trauma dictations easier. A majority 9 (69%) of 13 respondents expressed a belief that the dictation of trauma patients was important for patient safety and quality measurement and improvement (see Figure 2).

Anecdotally, the authors believe that the addition of the dictation template has had positive impact on the quality of their documentation of trauma patients. While not a primary measure of this project, several resident survey comments indicated that their trauma dictations were more thorough and organized after the implementation of the trauma dictation template. For example, one core EM faculty member commented that, “Now every injury and diagnosis is nicely documented, where before the diagnosis was often simply status/post Motor Vehicle Collision.” The McLaren Oakland Assistant Trauma Coordinator commented that, “When a trauma case has an ED dictated note, it is so much easier to tell what actually happened at two in the morning instead of just having to guess.”

DISCUSSION

Judging from these results, the creation and implementation of the trauma dictation template may have alleviated some of the primary barriers perceived by ED residents to prevent them from completing trauma dictations. The survey results, however, also demonstrate that nearly 40% of respondents still remained ignorant of the trauma dictation policy requiring verbal dictation for Level 1 and Level 2 trauma activations after implementation. This finding suggests that lack of awareness remained a major barrier to meeting the goal dictation rate of 75%.
The authors learned several lessons during this QIPS project that could be used as next steps in the PDSA process. It appears that merely making a trauma dictation template available is not sufficient to increase the utilization of such a tool. Since the EM resident physicians in this setting were the principal documenters of trauma cases, increasing EM resident awareness of the template will prove critical to increasing its future use.

Providing a single educational session with subsequent ‘boosters’ during the same month was apparently insufficient for increasing some residents’ awareness of the new trauma dictation policy. The impetus for the next step in the PDSA cycle is that many residents were not at every didactic session every week. Many were either off service or absent from didactic sessions so as to comply with duty hour restrictions. Multi-month educational sessions may therefore be needed to capture more resident users.

The answers provided by those EM residents who did not favorably review the dictation template were also revealing. Several residents commented that they did not use the template because they had never before dictated a trauma despite being aware of the requirement. Most negative responses seemed to be directed toward questioning the perceived validity of dictation policies, not necessarily the elements of the process. Motivational techniques will also be considered as the next steps in the PDSA cycle to increase EM resident utilization of the dictation template.

**Limitations**

A significant limitation of the study is the authors’ smaller sample size. With only 15-20 monthly Level 1 and Level 2 trauma activations occurring in this setting, the authors’ calculated dictation rate changes could have been inflated. The shorter period of intervention rollout for the project may have also limited the authors’ capability to note the full improvement derived from their dictation template. The post-intervention results demonstrating that 40% of the ED residents still denied having any knowledge of the dictation requirement should be considered during review of these results. It would also have been interesting to see what rate changes might have been over the course of an entire year. The authors believe the rate of completed trauma dictations would have likely increased as EM residents’ awareness of the template had risen.
One possible explanation for the decrease in Level 2 dictation rates was the time of year during which the project was conducted. The post-intervention months occurred near the end of the academic year, when the ED was increasingly staffed by more junior residents and off-service rotating residents. It is during this time of year when many of the junior ED residents now ATLS certified begin to staff trauma cases on their own. The senior residents are more likely to have been aware of the dictation requirement as they have more time to absorb the policies and procedures of the McLaren ED. Further education among new classes of EM residents will therefore need to be a permanent part of the annual Intern Orientation in July.

CONCLUSION

Although the project did not achieve its outcome goal to increase Level 1 and Level 2 trauma dictation rates, the project was successful in that a trauma dictation template was developed and well-received by most users. There was also some anecdotal evidence that the quality of documentation for trauma patient care has been positively affected since the introduction of the template. The template also appears to have eased the perceived burden of documenting complex trauma cases with multiple injuries and consultants. By doing so, cross-provider communication has been improved. The next step of the PDSA cycle will be to further increase awareness and motivation to use the dictation template to ultimately increase rates of completed trauma dictations.

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Table 1: Trauma Dictation Template

Trauma Dictation Template

Subjective
- Age/ Gender
- Describe Mechanism: “GSW to head”
- CC: “C/o of Headache”
- HCC:
- PMH:
- PSH:
- Meds:
- Allergy:
- Social:

Physical Exam
Vitals: BP: RR: Pulse: Temp: O2Sat:

Primary Survey (Relevant Negatives)
- A: Airway Patent, Patient in C- Collar placed by EMS
- B: Breath sounds equal B/L, breathing unlabored
- C: No active hemorrhage, Carotid, Radial, Femoral, Pedal pulses 2+ B/l.
- D: Pupils, GCS
- E: Full exposure

Resuscitation – What was done to correct anything in the primary survey
- Intubated by…., Needle Decompression, Direct Pressure placed on hemorrhage, etc..
- Patient Placed on telemetry and continuous pulse ox.
- Two Large Bore IV placed
- Initial Fluid Bolus, Blood Products…

Secondary Survey (Relevant Negatives)
- Head: No signs or trauma, laceration, abrasion
- ENT: No hemotympanum, No septal hematomata, Trachea Midline
- Chest: No crepitus, abrasion, bruising. RRR. Breath sounds clear B/L
- Pelvis: Stable to rock. Not tender to palpation
- Back: Patient was log rolled. No deformity step-off or point tenderness
- Extremities: No obvious deformity, abrasion, laceration.
- Neuro: No focal lateralizing deficit. Cranial Nerves 2-12 grossly intact. Normal strength B/L. Normal sensation B/L.
- GU: No blood at urethral meatus, No scrotal hematoma, no perineal bruising. Rectal tone normal. No gross blood
Table 1: Trauma Dictation Template (contd.)

Medical Decision Making

Trauma Activation: What level, what criteria, by radio call or by presentation in ED

Radiology: Include what studies were ordered, why, and their results.
- eFAST EXAM and who performed it.
- Chest and Pelvis XR
- CT Head, C-Spine, Chest/Abdominal/Pelvis
- Other XR: Ankle, knee, wrist, etc...

Procedures: Central Line, Intubation, Chest tube, etc...Include who did it, and why it was done.
Procedure note done separately

Labs:

EKG:

Consultants:
- Who did you talk to and why.
- Did they come in or by phone (very important)
- What were their recommendations
- This includes the ICU resident.

Progress Notes: When did the surgical team arrived, when did the patient go to CT scan.
When was a consultant paged. Describe the sequence of events as they occurred.

Assessment
- Include ALL diagnosis
- Laterality
- Don't forget small fractures, contusions, lab abnormalities, etc...

Plan
- Disposition (Home, Admit, etc.)
- What Floor. Who are they admitted to.
- Pain Control
- PT/O T
- DVT/GI PPx
- Consultants for the floor
- C-Collar Cleared or not, and by whom
- Who did you transition care to (Surgical team, ICU team)
Figure 1: Changes in Complete Trauma Dictations after Template Implementation

![Graph showing changes in rate of dictation by month](image)

Figure 2: Survey of ED Residents regarding Template Use

![Survey bar chart](image)
Delayed Intracranial Hemorrhage in Patients Taking Warfarin with Head Trauma

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ABSTRACT


CONTEXT: Patients presenting to the Emergency Department after a minor head injury have been shown to have a higher incidence of delayed spontaneous intracranial bleed, even with an initial negative CT. Some institutions have initiated a protocol of 24-hour observation followed by repeat head CT scan the next day. At the authors' institution, a 24-hour observation protocol was adopted in January 2012. The authors sought to evaluate the utility of this protocol given the apparently limited amount of data to support such a practice. METHODS: A 12-month prospective observational study conducted at a community hospital evaluating a 24-month observation protocol for patients presenting after a minor head injury. During this study period, a sample of minor head injury patients were identified and followed through their hospital course. CT scan results were also compiled, and patient charts were reviewed at 30 days for any return visits or post-injury complications. RESULTS: A total of 51 patients were enrolled during the study period. Of those patients receiving a repeat head CT, none showed any evidence of a new or worsening intracranial bleed, with all patients discharged safely from the hospital. At 30 days, patients’ charts were reviewed to examine whether any patients had returned to the hospital for any reason. No sample patients returned to the hospital within the 30-day review period. CONCLUSION: Similar to the results of the retrospective chart review, no sample patients with minor head injuries showed any evidence of a subsequent delayed intracranial bleed during the 12-month study period. Our study results suggest that 24-hour observation protocols with costly repeat CT head scans may not be useful in the management of most head trauma patients. Patients may be better served to be discharged home with education including signs of symptoms of a worsening bleed (i.e., confusion, worsening headaches and not feeling right) that actually warrant a return to the hospital. Keywords: delayed intracranial hemorrhage, minor head trauma, warfarin.

INTRODUCTION

Since its approval for use in the 1950s, Warfarin (also known as Coumadin) has become one of the most widespread used anticoagulant medications with a fairly low side effect profile, and well understood mechanisms of absorption, metabolism, and
elimination.\textsuperscript{1} After decades of studies concerning possible complications of systemic anticoagulation, Warfarin remains the anticoagulant by which others are generally measured.\textsuperscript{1} One of the more recent complications of Warfarin therapy cited in the literature is that of the delayed intracranial bleed, after even fairly minor head trauma.\textsuperscript{2,3,4} Minor head trauma has been defined as a Glasgow Coma Scale (GCS) score of 14 or 15 (possible 3 (maximum number of deficits) to 15 (no deficits) range) and a negative CT scan of the head.\textsuperscript{2}

During the 1990s, delayed intracranial bleed after head injury in patients on Warfarin therapy was first described in case reports.\textsuperscript{3,4} These reports noted that minor head trauma patients on Warfarin with initially clear CT scans after were at increased risk of a spontaneous intracranial bleed, even without repeat trauma.\textsuperscript{2} Following this finding, the recommendation to admit all patients on Warfarin for 24-hour observation with a repeat head CT scan after admission became part of the 2002 European Federation of Neurological Society guidelines for minor head injury patients on Warfarin therapy.\textsuperscript{4} Complicating this issue, the exact time frame for risk of increased intracranial bleed remains unknown, with some studies suggesting such a bleed may occur days or even weeks after the initial injury.\textsuperscript{2,5,6}

This recommendation has been adopted by some emergency departments (ED) in the United States, despite lack of definitive evidence that such policies would lead to increased detection or prevention of delayed intracranial bleeds.\textsuperscript{2,5,6,7} Widespread adaptation of this policy has led to considerable cost and time spent by physicians, patients, and hospitals in managing these patients, as well as additional radiation exposure. In this prospective observational study, the authors evaluated the utility of a 24-hour observation protocol including a repeat head CT scan.

**Purpose**

This study was conducted to evaluate the efficacy and utility of admission and repeat head CT scan protocol for patients who had sustained minor head injuries after a ground level fall, presented with a normal or near-normal GCS and were on Warfarin therapy. The primary goal of the study was to evaluate the utility of a 24-hour observation protocol for potential complications from the fall that could be attributed to a
delayed intracranial bleed. Most importantly, the study sought out to define the clinical importance and subsequent management of such events.

Although the phenomenon of the delayed bleeds after minor head trauma may indeed exist, the authors examined whether such an observation protocol and repeat imaging would add to, or change, the overall management of these patients. Based on the authors' experiences, they had hypothesized that such a 24-hour observation and reimaging protocol would prove unnecessarily costly and ultimately unhelpful in the management of these patients.

METHODS

This study was designed as a prospective observational study specifically examining those minor head trauma patients who reported to the emergency room after a ground level fall while on Warfarin therapy. The study was conducted at a community hospital in Southern Michigan with an annual ED census of roughly 100,000 visits. Before data collection, approval for this study had been obtained from the institutional review board.

Study Sample

Physicians in the ED were asked to enroll and consent any minor head trauma patient aged 18 years or older who were on Warfarin and reported to the ED after a ground level fall. The study excluded patients younger than 18 years of age or who were not currently taking Warfarin.

Measures and Timeline

The study was conducted from February 2014 through January 2015. On initial evaluation, the data collected included presenting GCS scores, results of head CT scans, and presenting international normalized ratio (INR) values. INR values serve as an important marker of current state of anticoagulation, allowing for verification of adherence to Warfarin medication regimen and providing a quantitative measurement of the patients' anticoagulation status. Some studies have suggested that a higher INR increases the risk of both an immediate and delayed intracranial bleed and can be used as a marker of degree and severity of patients' anticoagulation status.7
As a general hospital protocol initiated several years prior to this study, anticoagulated patients with an isolated head injury and no other reason for admission were typically admitted to the hospital’s overnight observation unit for 24-hour observation and a repeat head CT. Patients received a follow up CT the next day, with their disposition determined depending on the condition of the patient and head CT results. Patients who were not admitted to the observation unit but otherwise eligible for the study were also examined for follow-up imaging and 30-day readmission rates. These sample patients were found to possess comorbid conditions requiring admission for stabilization, were discharged home at the discretion of the attending physician, and/or signed out against medical advice.

The study hospital was a 475-bed facility without a major competing hospital within a 30-mile proximity. As such, patients enrolled in the study were evaluated for readmission to the ED or the hospital for any reason at 30 days after initial presentation. It was an assumption of the authors that if any study participants had experienced a complication or were symptomatic from a delayed intracranial hemorrhage that the most available hospital with facilities capable of managing such complications would be the study hospital.

All head CT scans that were completed were non-contrast in nature and interpreted by staff radiologists. A positive result in the initial head CT was defined as any evidence of intracranial bleed. All positive head CT results were included in the study with repeat CTs evaluated for interval change in size or severity of bleeding.

**Outcome Measures**

The primary outcome measures of interest were related to progression of intracranial bleeds or new bleeds after an initial negative CT head scan. Any new intracranial findings with follow-up CT scans, whether clinically significant to patient outcomes or not, was considered a positive finding. A second outcome measured in this study was readmission to the ER or hospital for any reason within 30 days of initial head trauma.
RESULTS

During the 12-month study period, a total of 51 adult patients were identified by the authors as being eligible for the study. The final study sample demographics consisted of 30 (59% of total) females and 21 (41%) males, with their mean age averaging 71.8 years and ranging from 47 to 98 years (see Table 1). As indicated earlier, the GCS was used to evaluate all patients on initial presentation. Using eye movement, vocalizations, and motor function, a standardized score from 1 to 15 was calculated, with a GCS of 15 indicating someone without evidence of any deficit. One sample patient presented with a GCS of 12, with the remainder of the patients either starting with a GCS of 14 or 15. For the entire sample, the mean INR level on presentation to the ER was 2.38 (SD) 1.02 (see Table 1). INR is a lab value used to measure therapeutic effectiveness of Warfarin and to help guide medication adjustments. The therapeutic goal of most patients being a value of 2, with a value of 1 being a normal level.

Of the 51 sample patients, 21 (41%) were admitted for 24-hour observation, and 15 (29%) were discharged home from the ED with primary care follow up. An additional 14 (27%) patients were admitted to the hospital, and one (2%) patient left against medical advice. A total of 19 (38%) of study patients received a follow up CT the next day for evaluation of possible new onset bleed or progression of existing bleed (see Figure 1). One patient in the study received a follow up MRI in the place of a CT to evaluate for the possibility of an ischemic stroke as the cause of her fall. Of the 51 patients enrolled in the study, two (4%) had positive findings on the initial CT, and one (2%) person refused the initial CT. Finally, all of the patients who had initially enrolled in the study survived their hospital stay.

In evaluation of the 19 patients provided follow up imaging within 24 hours after initial head trauma, no patients were found to have developed a new intracranial bleed. Two of these 19 (11%) patients did have a positive finding on initial CT of intracranial hemorrhage, and follow up imaging done the next day for both of these patients failed to show any interval change (development of a new bleed) or progression of intracranial bleed. Of those 21 patients admitted for observation, six (29%) of this sample subgroup were determined to not need a follow up CT on Day Two of their stay.
In contrast, of those 14 admitted for inpatient services, only four (29%) of this group had any follow up imaging done to evaluate for a potential bleed based on managing provider discretion (see Table 2 and Figure 2). Presumably, those patients who were admitted were globally more unstable as a whole, yet only 29% of them were imaged on Day 2 compared to 71% of those brought in for observation. None of the enrolled patients were readmitted to our facility within 30 days of discharge.

**DISCUSSION**

These initial reports provide evidence regarding the low level of utility of a 24-hour observation and repeat head CT protocol for anticoagulated patients who have sustained minor head trauma. None of the authors’ sample patients suffered from a delayed intracranial hemorrhage. Our study results evaluated data from eligible patients presenting to one Midwestern ED for one year in total. Not one single case of delayed intracranial hemorrhage was detected during this entire analytic period. This finding calls in to question the true prevalence of delayed intracranial bleeds for minor head trauma cases and raises the question of whether or not concern over delayed post traumatic hemorrhage may be exaggerated.

It has been well documented in the literature that being on Warfarin therapy does present genuine immediate increased risk of intracranial bleed in patients with head injuries.6,7 Some studies suggest as many as 17% of such patients can present with an intracranial bleed 2,6,8 and American College of Emergency Physicians guidelines recommend an immediate initial head CT.7 Reviews of the literature on delayed bleed rates, however, reveal that this phenomenon is inconsistently tracked or studied.5,7,9,10 In fact, the results of previous studies have suggested that bleeds of this type may occur days to weeks after initial injury.7,10

As this and previous studies have demonstrated, delayed intracranial bleeding as a phenomenon has proven to be both unpredictable and fairly rare.5,8,10 The lack of any delayed bleed episodes in our sample further calls in to question the utility of a 24-hour observation and reimaging protocol. Such protocols very likely result in increased hospital costs and physician workload as well as greater unnecessary radiation exposure to patients.6 Until providers develop an improved evidence-based
understanding of the etiology and mechanism of delayed intracranial bleeds, the benefits derived from such costly observation and reimaging protocols should be questioned.

The second part of our study involved 30-day follow up and chart evaluation to assess for readmission to the hospital due to complications from a delayed intracranial bleed. Once again, the fact that none of the study subjects returned to the hospital is telling in this case. Previous studies in this area have demonstrated that the large majority of patients showing some evidence of delayed bleeds do not have a significant enough bleed to require more than medical observation.\textsuperscript{5,6,7,9,10} It should be noted that the patients in this study each presented with a low risk mechanism and a normal or near-normal GCS. Certainly, more data needs to be gathered to better understand the nature of these delayed bleeds. Until then, however, the author’s findings match earlier and concurrent studies indicating that the chance of a massive, sudden, and life threatening hemorrhage in a patient that generally looks well with negative initial head CT findings is very low.\textsuperscript{2,5,7,8,9,10}

\textbf{Limitations}

The generalizability of our study results to other settings is limited since these findings are from a single center study site with a smaller sample. A larger multicenter study would help to validate the results of this study. Additionally, some sample patients in this study did not receive a repeat head CT scan as per the protocol being evaluated. These protocol deviations were most likely due to treating physicians’ clinical judgment that a repeat CT scan was not warranted. Although we found no evidence of any missed cases of delayed intracranial bleeds, it may also be possible that some sample patients presented to other institutions after discharge with symptoms that warranted additional evaluation and treatment.

\textbf{CONCLUSION}

Despite these limitations, our study results provide additional evidence challenging the current protocol of keeping anticoagulated patients in the hospital after minor head trauma, even with an initial negative CT scan. Patients should certainly be made aware of the risks and signs of delayed intracranial bleeding, and providing them...
adequate follow-up care and sufficient social support. Given the uncertain nature and
timeline of delayed intracranial bleeds, these results indicate that effective patient and
family education may be of far greater utility than costly 24-hour observation protocols.
The study findings support previous works demonstrating that ongoing observations and
costly repeat imaging are unwarranted in many cases and ultimately consume valuable
hospital resources.\(^6\)

The authors report no external funding source for this study.
The authors declare no conflict of interest.
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   CT may not be Indicated in patients on anticoagulant/antiplatelet therapy following
   intracranial hemorrhage after blunt trauma: Are patients on pre-injury anticoagulants
Table 1: Demographics of Sample Patients and Summary of Hospital Course

<table>
<thead>
<tr>
<th>Total Population</th>
<th>51</th>
</tr>
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<tbody>
<tr>
<td>Median Age (range, SD)</td>
<td>71 (47-98)</td>
</tr>
<tr>
<td>Male (%)</td>
<td>21 (41%)</td>
</tr>
<tr>
<td>Presenting GCS (%)</td>
<td></td>
</tr>
<tr>
<td>15</td>
<td>48 (94%)</td>
</tr>
<tr>
<td>14</td>
<td>2 (4%)</td>
</tr>
<tr>
<td>&lt;13</td>
<td>1 (2%)</td>
</tr>
<tr>
<td>INR (mean, std deviation)</td>
<td>2.38 ± 1.02</td>
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<tr>
<td>&gt;2.0</td>
<td>23 (45%)</td>
</tr>
<tr>
<td>1.5 – 2.0</td>
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<tr>
<td>&lt;1.5</td>
<td>6 (12%)</td>
</tr>
<tr>
<td>Disposition</td>
<td></td>
</tr>
<tr>
<td>Observation</td>
<td>21 (41%)</td>
</tr>
<tr>
<td>Admission</td>
<td>14 (29%)</td>
</tr>
<tr>
<td>Discharge</td>
<td>15 (29%)</td>
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<tr>
<td>Left AMA</td>
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<td>Return to hospital at 30 days</td>
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<tr>
<td>Initial CT</td>
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</tr>
<tr>
<td>Positive</td>
<td>2 (4%)</td>
</tr>
<tr>
<td>Negative</td>
<td>48 (94%)</td>
</tr>
<tr>
<td>Refused</td>
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<td>Follow up CT</td>
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</tr>
<tr>
<td>Not done</td>
<td>29</td>
</tr>
<tr>
<td>Positive / changed</td>
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## Table 2: Patient Progression through Hospital Course and Subsequent Imaging Completed based on Disposition from ED

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<tr>
<th>Disposition and Hospital Course</th>
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<tr>
<td><strong>Observation</strong></td>
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<tr>
<td>Initial Head CT done</td>
<td>21</td>
<td>100%</td>
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<tr>
<td>Follow up head CT done</td>
<td>15</td>
<td>71%</td>
</tr>
<tr>
<td><strong>Admission</strong></td>
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<td></td>
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<tr>
<td>Initial Head CT done</td>
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<td>100%</td>
</tr>
<tr>
<td>Follow up head CT done</td>
<td>4</td>
<td>29%</td>
</tr>
<tr>
<td><strong>Discharged from ED</strong></td>
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<td></td>
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<tr>
<td>Initial Head CT done</td>
<td>16</td>
<td>94%</td>
</tr>
<tr>
<td>30 day return to hospital</td>
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<td>0%</td>
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<tr>
<td><strong>Discharged from hospital</strong></td>
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<td></td>
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<tr>
<td>30 day return to hospital</td>
<td>35</td>
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</tr>
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</table>
Figure 1: Hospital Course from Presentation in the ED through Discharge

Figure 2: Summary of Initial Head CT Findings and Subsequent Imaging
Case Reports

Transient Hyperammonemia Seen in Post Seizure Activity: A Series of Six Case Reports

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ABSTRACT

DAUGHTRY JT, BOEHM KM. Transient Hyperammonemia Seen in Post Seizure Activity: A Series of Six Case Reports. Spartan Med. Res. J. Vol. 1, No. 2, pp. 44-54, 2017. CONTEXT: Elevations in serum ammonia levels (i.e., hyperammonemia) have often been interpreted as signs of liver failure or errors in metabolism. The purpose of this series of case studies is to evaluate a trend in both the markedly elevated levels of ammonia, along with its rapid clearance in patients with seizures. METHODS: These patient cases each occurred in a community-based, academic emergency department in the metropolitan Detroit area that provided care to approximately 70,000 patients during a four-year period. RESULTS: These six patient cases had each been found with observed seizure activity in which an initial and repeat ammonia in the emergency department was ordered. In all six cases, there was an initial elevation in ammonia with rapid subsequent clearance of ammonia. CONCLUSION: As demonstrated in this series of case studies, transient hyperammonemia levels may occur within the clinical context of seizure activity. With further research, it may be confirmed to be a differential diagnostic marker to delineate new onset or recurrent seizures in the ED. Keywords: seizure, hyperammonemia, clinical markers, diagnostics

INTRODUCTION

A new onset seizure is often difficult to diagnosis by an emergency medicine (EM) clinician unless the seizure actually occurs in front of them until an electroencephalogram (EEG) has been taken. The information that the emergency department (ED) physician has to rely on comes from family or the patient themselves, physical exam (e.g., new intraoral lesions), and based on diagnostic tests to rule out other non-epileptic form of seizure like activity.1 Inpatient or outpatient EEGs can be arranged to confirm diagnosis of seizure, but clinicians have asked whether there a lab value that may aid in these diagnoses.

This series of case reports demonstrates the possible link between transient hyperammonemia and seizure activity. Although hyperammonemria is usually
Transient Hyperammonemia Seen in Post Seizure Activity

associated with hepatic encephalopathy, inborn errors of metabolism, and disruption in the urea cycle, the authors hope to demonstrate from this series of case reports that ammonia levels as a laboratory marker for seizure activity can potentially provide physicians with another tool to better diagnose new onset seizures in the ED.

Case Reports

Case 1

This patient was a 46-year-old male who per EMS had a witnessed grand-mal seizure prior to arrival to the ED. The patient’s history was obtained primarily from the EMS providers, who stated they suspected a possible overdose due to a medication bottle found near the patient near time of seizure. The patient’s vital signs were as follows: heart rate (HR) 118, blood pressure (BP) 136/64, respiratory rate (RR) 20, and oxygen saturation of 100% was measured on a non-rebreather oxygen mask. His initial Glasgow Coma Scale (GCS) score (on range from 3 (maximal neurological deficit) to 15 (no deficits) was 6 at arrival to the ED. During primary survey, however, his neurological status later improved to a GCS score of 9. The patient was found to be oriented to person only, and responded to pain.

Physical exam showed pupils 4mm and reactive bilaterally, a minor laceration on the outer lower lip, lungs were clear, and heart rate was tachycardic (i.e., elevated). The patient had pulses in all extremities with an external fixator on the right lower extremity from a recent orthopedic surgery at another hospital. The patient had a past medical history that included: seizure disorder, bipolar disorder, depression, suicidal attempt, schizophrenia, anxiety, hypertension, diabetes peripheral neuropathy, hyperlipidemia, osteoarthritis, chronic low back pain. His social history included past alcohol abuse and cocaine abuse.

The patient’s oral home medications included: aspirin 81 mg. daily, citalopram 20 mg. daily, pregabalin 75 mg. twice a day, felodipine 10 mg. daily, quetiapine 300 mg. twice a day, iron 325 mg. daily, clonidine 0.1 mg. twice a day, valproate 500 mg. in the morning and 1500 mg. at night, and folate 1 mg. daily.

After his initial assessment in the ED, the patient was given lorazepam 2mg. intravenously (IV) at 0744 hours. His labs studies demonstrated elevated lactic acid and creatinine levels with a low potassium and an anion gap of 33. His ammonia was
elevated to 145 umol/l with normal liver function testing (normal ammonia is under 50 umol/l). Other medications which were administered included potassium chloride 40 mEq. orally, and valproate 1500 mg. orally at 1302 hours. The patient continued to have a prolonged postictal state (i.e., altered state of consciousness after an epileptic seizure) and he was admitted to the hospital.

During his hospital stay, a computerized tomography (CT) of the brain was performed showing no acute pathology. An ultrasound of the abdomen was performed which showed gallstones without evidence for acute cholecystitis but poor views of the liver and pancreas obtained due to the patient’s physique. A hepatobiliary (HIDA) scan showed a normal hepatobiliary system with normal response of gallbladder to cholecystokinin (CCK) stimulation following administration of CCK with the ejection fraction to be 77%. An EEG was performed showing the presence of slow frequency in the theta range varying from 4-7 cycles per second. No epileptiform activity was seen. The conclusion of the consulting neurologist was that the EEG was mildly abnormal because of the presence of slow theta frequency with this abnormality suggestive of mild cerebral dysfunction. The patient stayed in the hospital for two days and was discharged with a diagnosis of seizure. This case showed the ammonia clearing to 23 umol/L in approximately 13 hours (see Table 1).

Case 2
This patient presented as a 47-year-old male with a past medical history of cardiac disease, including a myocardial infarction and untreated hypertension. His surgical included history of a hernia repair, and he had a social history of cocaine and alcohol abuse. The patient had been brought to the ED by several members of his family. Per family member reports, the patient usually drank between 12 and 24 beers each day. The patient had reportedly stopped drinking 24 hours prior to his arrival to the ED. Per the family, the patient had also been “shaking” all day and had seemed to have become increasingly confused.

During the ED triage process, the patient had a witnessed grand-mal seizure that lasted for approximately 60 seconds. During that time, an IV line was established and the patient was given 2mg. of lorazepam for the seizure. As the seizure subsided, he
Transient Hyperammonemia Seen in Post Seizure Activity

appeared postictal but eventually began experiencing hallucinations. His was slightly hypertensive with a BP of 148/103, a HR 136, RR of 18, temperature of 37°C and pulse oximetry of 95% on room air. On physical exam, the patient was diaphoretic, with no pain to palpation on the chest or abdomen. His lung sounds were clear bilaterally and he was able to move all four extremities to pain.

However, he was unable to follow commands, attempted to make incoherent speech, and seemed neither alert nor oriented. Patient was administered a two-liter IV fluid bolus of normal saline. The patient’s labs were then drawn and the results are listed in Table 1. These labs showed the patient to have an elevated anion gap, likely secondary to a lactic acidosis, elevated ammonia, elevated liver enzymes, and thrombocytopenia. A CT of the head was also performed with no acute findings.

The patient’s abnormal LFT’s and thrombocytopenia were thought to be secondary to his chronic alcoholism, and he was started on the hospital ethanol withdrawal pathway. This protocol included treatment with IV lorazepam, multivitamins, folic acid, and thiamine. The authors decided to treat the patient’s elevated ammonia with oral lactulose since his mental status had improved at the time they received the lab results. The lactic acidosis was treated with an initial IV fluid bolus of two liters with plans to repeat the lactate to see if it was clearing.

Approximately three hours after the first lab set was drawn, the patient’s electrolytes, ammonia, and lactic acid levels were rechecked along with a total bilirubin (Table 1). The ammonia level had returned to normal before the patient’s system having had enough time for the oral lactulose treatment to start working since it must first change the flora of the gut and cause the resulting diarrhea. In light of these circumstances, it was concluded that the seizure, and not the suspected liver disease, must be the cause of the patient’s transient hyperammonemia.

The patient continued to improve in the ED with normalization of vital signs and he was admitted to a hospital unit for further monitoring. During his hospital course, an infectious hepatitis panel blood test was performed that was negative for hepatitis A, B, and C. An ultrasound of the liver was performed which showed the liver to be mildly echogenic. During his hospital stay, the patient had no subsequent elevations of his ammonia level and no further seizure activity was noted.
Case 3

This patient was a 49-year-old male seen in the ED for witnessed seizure activity. Emergency medical service personnel had been called to the patient’s house for an impending seizure and actually witnessed the patient seizing in the hallway. The seizure had stopped prior to his arrival in the ED, where the patient had another witnessed grand-mal seizure once in the ED. The patient began foaming at the mouth, not protecting his airway, with a GCS score of 3. The decision was made to intubate the patient. The patient was given 4mg of midazolam and 80mg of rocuronium. After the intubation, the patient was placed on a propofol drip for sedation and administered IV hydromorphone 1mg. twice approximately one hour apart.

The patient’s past medical history included seizure disorder, alcohol abuse, pancreatitis, and a prior history of seizures during ethanol withdrawal. He had no surgical history and took no recorded home medications. His social history was positive for alcohol abuse. The physical exam showed a normal head-eyes-ears-nose throat, lungs and abdomen. The patient neurological exam prior to intubation showed an unresponsive patient with a GCS score of 3 and foaming at the mouth.

He was admitted to the intensive care unit (ICU) for respiratory failure and seizures. This patient was never started on any medications for the elevated ammonia, having no prior history of hepatic encephalopathy. With only IV fluids and monitoring, the patient’s ammonia was cleared to normal levels in a little over 24 hours.

The patient had an extended stay in the ICU and on the general hospital floor. Neurology service was consulted, although no EEG was performed since the etiology of the seizures was thought to be due to alcohol withdrawal. The patient was initially started on phenytoin for the seizures, but per neurology's recommendations the patient was switched to levetiracetam due to the elevation in his ammonia and liver function tests (LFT). During the patient’s hospital stay, he developed pneumonia and a GI bleed that were all successfully treated. Once stable, the patient was transferred to a patient rehabilitation unit with a psychology consult placed for the patient’s suspected psychosis. The patient was finally discharged home 2.5 months after his initial ED visit with a normal ammonia level.
Case 4

This patient was a 28-year-old male with a known seizure disorder. He was brought to the ED for evaluation after his brother had witnessed his seizure which he described as a grand–mal seizure at home. The patient only apparently experienced one seizure and was now presenting postictal. Per the family the patient had noncompliance with the medication carbamazepine, which he had been prescribed for twice a day use. The patient’s past medical history is positive only for seizures, with no surgical history. He had a past history of tobacco use with a total 5 pack year history, and smoked marijuana on a daily basis with the last use the day prior to arrival.

The patient’s exam showed a somewhat confused patient despite a GCS of 15. HEENT, cardiac, lung, and abdominal exam were negative for acute findings. Neurological exam showed no focal deficits. His labs showed an elevated ammonia level at 227 and elevated lactic acid. Patient also had a serum anion gap, indicating some form of electrolyte disturbance. During the patients ED stay, the patient only received a dose of carbamazepine 500mg orally and IVF. As shown in Table 1, the patient’s ammonia and lactic cleared in 3 hours. The patient was observed in the ED and with return to normal mental status before being discharged home.

Case 5

Patient was a 37-year-old man, who was non English speaking, with a past medical history for seizures and brought to the ED for a witnessed seizure by family. Most of the history was given by EMS. Upon arrival to the ED, this patient continued to have seizures. The patient was given 2mg lorazapam followed by 2 mg of midazolam. The seizures stopped and he gradually became more alert. Over the course of the ED visit, the patient's neurologic status continued to improve. The patient was examined with the help of a phone interpreter, although he remained unclear regarding the seizure medication he took. Valproic acid, phenytoin, and carbamazepine lab levels were obtained, with each failing to show such medications detected.

The results from a CT of the head were negative for acute pathology. The patient's labs showed an initial lactic acid at 15.8, an ammonia level of 96 with an anion gap of 23. Labs were repeated in approximately three hours showing resolution of lab
abnormalities. During that time, the patient was given 4 liters of Normal saline and 500mg of levetiracetam intravenously. The family eventually arrived in the ED to be at the patient’s bedside. The patient was completely alert and oriented to his baseline per family and was discharged home.

Case 6

The patient in this case was a 24-year-old male. According to his parents, the patient was a healthy individual with no past medical history or social history. He had been traveling with his family in the back seat of the car when he became unresponsive and “started shaking” per his parents. The emergency medicine personnel who were on scene indicated that the patient had experienced a witnessed generalized seizure and was unresponsive until arrival to the ED.

In the ED, the patient appeared to be postictal but slowly became more arousible, capable of answering questions for himself. As the patient's mental status improved, he became combative, requiring an initial dose of 2mg of lorazepam then a second dose of 6mg of lorazepam. The labs for this patient showed an elevated ammonia level at 86, lactic acid of 8.1 and an anion gap of 18. Urinary drug screen, alcohol, and CT of the head were also negative.

After discussion with the family, the patient was then admitted to the hospital due to the possibility of a first seizure. An MRI of the brain and EEG were both performed and returned with normal results. Patient was seen by neurology and was discharged home on phenytoin with a discharge diagnosis of generalized seizures.

Table 1 highlights the lab work that was performed for each of these patients and the specific times that the initial and subsequent labs were drawn. Please note that abnormal values are listed in bold font. As this was a retrospective case series, there were no criteria for timing of initial or repeat lab draws followed. Labs were drawn upon evaluation of the patients and repeated as indicated by clinicians' judgments.

As shown in Table 1, each of the patients had initially elevated ammonia levels. Even though repeat labs were drawn at various times, each showed decreased patient ammonia levels. Of note, Cases 2, 4, 5 showed ammonia levels of 290, 277, 96 respectfully, with each of these patients’ repeat labs performed around 3 hours later and
showing normal ammonia lab values (i.e., 19, 22, 47, respectively). Also of interest, Case 6, with an initial ammonia of 86, showed a repeat normal ammonia level approximately two hours later.

**DISCUSSION**

The differential for hyperammonemia can include many etiologies such as congenital deficiencies of the urea cycle enzymes, hepatic encephalopathy’s, Reye syndrome, as well as several other metabolic disorders. Each of these etiologies can result in sustained ammonia levels and require treatment for resolution. Transient hyperammonemia etiologies, and their effects, have been discussed far less often in the medical literature.

Transient hyperammonemia of the newborn (THAN) is primarily seen in premature infants, the exact cause unknown but potentially lethal. The treatment involves protein avoidance, adequate caloric support, arginine supplementation, and pharmacological priming of alternative pathways for nitrogen excretion. Once the serum ammonia level is normalized, there is no further risk of new episodes of hyperammonemia and normal intake of protein can occur.³

Transient Hyperammonemia has also been studied in patients on chronic valproate treatment for seizure prevention. One of the potential side effects of valproate treatment is elevated ammonia levels. The cause of this is thought to be related to a deficiency of the amino acid derivative carnitine. By depleting carnitine from the body, there is a subsequent decrease in the amount of free acetyl CoA, which is required to activate carbamyl phosphate synthetase I, the first enzyme in the urea cycle. However, elevated ammonia levels have still been shown in patients with high protein diets supplementation of oral carnitine to decrease serum ammonia in patients on valproate.⁴ A similar therapeutic effect of oral carnitine on ammonia levels was supported during a study of patients with hepatic encephalopathy.⁵ In patients with hepatic encephalopathy, neuronal activity improved after a single short course of IV L-carnitiine and acetyl-L-carnitine (ALCAR).⁵

During these cases, transient hyperammonemia was discovered in the context of witnessed seizure activity. In these cases, it was found that over half of these witnessed
seizure patients had measured hyperammonemia in the absence of hepatic disease. The patients’ hyperammonemia levels in these cases had a short clearance time of the ammonia of one to three hours, which is unexpected if the elevation had been caused by valproate use or hepatic encephalopathy.\textsuperscript{6}

The possible mechanisms of action causing hyperammonemia in some instances could also be from muscle.\textsuperscript{7} In a previous study of cyclists undergoing strenuous exercise elevated levels of ammonia could be detected.\textsuperscript{8} Most seizures involve a considerable amount of muscle activation, and may explain rises in ammonia levels.\textsuperscript{6} Although these types of transient elevations may seem to be harmless, one study showed that a long-lasting alteration in protein kinase A occurred after a single episode of hyperammonemia. These ammonia alterations were shown to affect signal transductions in the brain and the blood, altering levels of cGMP and possibly affecting platelet aggregation, portal hypertension and hypotension. Additionally, such changes may be involved in other manifestations of liver disease.\textsuperscript{6}

**CONCLUSION**

True seizure activity that is not witnessed by a treating physician can be difficult to diagnosis in absent of EEG testing. Using transient hyperammonemia lab values could, however, be used as a practicable solution to identifying true seizure activity in ED settings. Since few studies involving this phenomenon have been conducted, further investigations of the causes of elevated ammonia levels and the effect of these elevations is also needed. As demonstrated in this series of case studies, transient hyperammonemia levels may occur within the clinical context of seizure activity. With further research, it may be confirmed to be a diagnostic marker to delineate new onset or recurrent seizures in the ED.

The authors report no external funding source for this study.
The authors declare no conflict of interest.
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REFERENCES


Table 1: Highlighted lab work for the six cases presented that was performed and the approximate times the repeat labs were drawn*

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<tr>
<th></th>
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<th>Case 3</th>
<th>Case 4</th>
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<td>139</td>
<td>137</td>
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*Please note, abnormally high or low values are listed in **bold font**. (Normal lab value ranges are listed in brackets).
Developmental Consequences of Restrictive Eating Disorders after Childhood Obesity: Two Case Reports

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ABSTRACT

KENNEDY SF, and KRIVE K. Developmental Consequences of Restrictive Eating Disorders after Childhood Obesity: Two case reports. Spartan Med. Res. J. Vol. 1, No. 2, pp. 55-62, 2017. CONTEXT: Although childhood overweight has been associated with an increased risk for later development of eating disorders, there has been little research indicating whether previously overweight children and adolescents have an increased risk for restrictive eating disorders such as anorexia nervosa or atypical anorexia nervosa. The fifth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5) defines anorexia nervosa as the restriction of energy intake relative to requirements, leading to a significantly low body weight, an intense fear of gaining weight or becoming fat, and disturbance in the way in which one's body weight or shape is experienced. In atypical anorexia nervosa, the DSM-5 specifies that all criteria for anorexia nervosa can be met for this disorder, except that after despite significant weight loss the individual’s weight is currently within or above the normal range. In treatment of these disorders, the intense fear of weight gain may be further intensified in previously obese children because they have earlier experienced the significant consequences of obesity. METHODS: The purpose of this report is to present two cases of previously obese children who later developed restrictive eating disorders. RESULTS: These cases demonstrate how previously obese children may be at risk for restrictive eating disorders with treatment further complicated by an intensified fear of recurrent weight gain. In these cases, treatment was complicated by an intensified fear of weight gain due to the patients’ prior obesity. CONCLUSIONS: The mortality rate of restrictive eating disorders may be increased in previously obese children and adolescents due to delay in diagnosis and more complicated treatment, making clinician screening imperative. Keywords: Anorexia Nervosa, childhood obesity, eating disorders.

INTRODUCTION

Childhood overweight is often associated with an increased risk for later development of eating disorders. Overweight children frequently have symptoms of excessive shape and weight concerns, binge eating and pathogenic weight loss behaviors, such as calorie restriction, excessive exercise, self-induced vomiting and
misuse of laxatives or diuretics.\textsuperscript{1,2} Overweight children can display elevated levels of weight dissatisfaction compared to average-weight children and may utilize calorie restriction in an attempt to control their weight.\textsuperscript{3}

Research has indicated that children and adolescents who were previously overweight are at increased risk for bulimia nervosa and binge-eating disorders.\textsuperscript{1,2,4,5} However, there has been little research indicating whether previously overweight children and adolescents have an increased risk for restrictive eating disorders including anorexia nervosa or atypical anorexia nervosa.\textsuperscript{6,7}

The fifth edition of the \textit{Diagnostic and Statistical Manual of Mental Disorders} (DSM-5) \textsuperscript{8} defines anorexia nervosa as \textit{the restriction of energy intake relative to requirements, leading to a significantly low body weight in the context of age, sex, developmental trajectory, and physical health}. Additional signs can include intense fear of gaining weight or becoming fat, persistent behaviors that interfere with weight gain, disturbances in the way in which one’s body weight or shape is experienced, undue influence of body weight or shape on self-evaluation, or lack of recognition of the seriousness of one’s current low body weight.\textsuperscript{8}

Under the DSM-5 category of \textit{Other Specified Feeding or Eating Disorder}, atypical anorexia nervosa is defined as meeting all the criteria for anorexia nervosa, except that despite significant weight loss, the individual’s weight is within or above the normal range.\textsuperscript{8} Restrictive eating disorders have been associated with higher mortality rates than bulimia nervosa and binge-eating disorder and anorexia nervosa has been shown to have the highest mortality rate of all psychiatric illnesses.\textsuperscript{9}

The current literature regarding the treatment of restrictive eating disorders emphasizes the importance of full weight restoration through high-calorie diets.\textsuperscript{9} This goal may prove difficult for patients with anorexia nervosa due to their preoccupation with shape and weight and the fear of becoming obese again. This type of fear in previously obese children may also complicate treatment.

The purpose of this report is to present two cases of previously obese children who later developed restrictive eating disorders. Both patients presented to the Michigan State University Psychiatry Clinic in 2015 and were evaluated and treated by the psychiatry resident author (SFK). At the time of initial evaluation, the \textit{short form} of
the *Eating Attitudes Test* (EAT-26)\textsuperscript{10} was administered to both patients. The EAT-26 is a validated screening tool used to assess eating disorder risk. A score of 20 or greater on the EAT-26 indicates risk for an eating disorder.\textsuperscript{10}

**Description of Cases**

**Case A**

Case A, a 15-year-old female, lived at home with both parents and an older brother. Growing up, she was described as a “big eater.” In 8\textsuperscript{th} grade at age 12, she was obese with weight 66.2 kg. and height 152.4 cm. Her BMI at that time was 28.5 kg/m\textsuperscript{2}, placing her in the 96\textsuperscript{th} percentile for age and sex, placing her in the obese category. (For children and adolescents, BMI weight status categories are not determined as categories are in adults). The patient’s vital signs were within normal limits and she had no diagnosed medical problems. She began to slowly lose weight by cutting back her portion sizes. Toward the end of 8\textsuperscript{th} grade, she began to “hate” her body and developed pathogenic weight loss behaviors, including skipping lunches in order to achieve better results. This practice was soon followed by skipping breakfast as well.

With high school approaching and her concerns over her shape growing, she began purging after dinner seven nights per week. During the summer before high school, she attended a camp and was thus consistently provided higher calorie meals. In response, she began excessively exercising, once completing 1600 abdominal crunches in a single day, to counteract her “excessive” intake.

She began to explore online content regarding how to lose weight, deciding on a 500 calorie deficit as well as burning 500 calories per day through exercise. Initially, she limited herself to 1500 calories per day, but then decreased to 1000 calories per day. During a six-month period, she lost approximately 18.6 kg. and her menses ceased for three months. Due to online information, she believed she was losing weight in a healthy manner and asked to see a nutritionist who she believed would agree with her.

At that time, she was diagnosed with anorexia nervosa and was referred to the MSU Psychiatry Clinic for treatment. At the time of initial psychiatric evaluation at age 15, her weight was 45.8 kg. and height 157.5 cm. Her BMI was 17.6 kg/m\textsuperscript{2}, placing her in the 13\textsuperscript{th} percentile for age and sex. In children and adolescents, significantly low
weight is what is less than that minimally expected and determined by growth curves. Her EAT-26 score was 31, indicating risk for an eating disorder.

The patient’s treatment plan included weekly psychotherapy with the psychiatry resident, consisting of supportive psychotherapy and cognitive behavioral therapy (CBT), which focused on treating cognitive distortions related to her shape. Her vitals were taken weekly at these appointments, including weight which was taken with patient facing away from the scale. She saw a nutritionist once every two weeks for refeeding, which is the restoration of normal nutrition after a period of starvation. She was placed on a meal plan consisting of three meals and three daily snacks which were prepared by her parents. At time of presentation, her daily caloric intake increased gradually from her reported caloric intake to allow her body to adjust to the increased intake.

During refeeding, she experienced constipation which was successfully treated with Miralax. After four months of treatment, the patient achieved weight restoration to 54.4 kg. and height remained 157.5 cm. Her new BMI was 20.7 kg/m², placing her in the 55th percentile. Although the patient was able to maintain her weight, she continued to struggle with shape preoccupation and fixated on eating healthy due to her “terror” of becoming obese again. She frequently expressed concern regarding the number of calories she was required to consume. Following an additional three months of psychotherapy with an emphasis on CBT, she began to shift her value of self from shape to achievement in athletic activities and relationships with friends and family. She began to think of food as fuel for her activities, which helped to assuage her fear of becoming obese. Although her fear of becoming obese lessened, the fear remained to some extent. At time of submission, the patient continued in biweekly psychotherapy.

**Case B**

Case B, a 17-year-old female, lived at home with both parents and an older sister. In 8th grade at age 13, she was obese with weight 90.0 kg. and height 167.6 cm. BMI was 32.1 kg/m², placing her in the 98th percentile for age and sex. Her pediatrician recommended weight loss. She began to slowly lose weight by exercising and decreasing consumption of “junk” foods. Following menarche at age 15 during her sophomore year of high school, her weight loss was more difficult and she began
utilizing pathogenic weight loss behaviors, including calorie counting. She began to restrict certain foods that she considered calorie-dense or foods with high fat or sugar content from her diet. This resulted in weight loss, although she became more concerned about her shape. She expressed feeling terrified of gaining the weight back and becoming obese again.

During her senior year of high school at age 17, she began to severely restrict her caloric intake. Her mother noticed her “strange” eating habits, such as eating lettuce without dressing and consuming large amounts of egg whites. She was consuming only 500 calories per day and exercising for at least 30 minutes daily. Additionally, she was using laxatives and occasionally purging to further control her weight. Due to online information she had found regarding weight loss, she felt that her eating habits were healthy.

She was referred to the MSU Psychiatry Clinic for treatment and presented with weight 58.5 kg. and height 171.4 cm. Her BMI was 19.9 kg/m², placing her in the 30th percentile for age and sex. The patient’s EAT-26 score was 23, indicating a clear risk for an eating disorder. Despite significant weight loss, her weight was still within normal parameters. Since all other criteria for anorexia nervosa were met, she was diagnosed with atypical anorexia nervosa.

Her treatment plan consisted of weekly psychotherapy with the psychiatry resident consisting of supportive psychotherapy and CBT. CBT focused on treating her cognitive distortions regarding food and fear of being overweight. Coping techniques were developed to decrease her behaviors such as covering mirrors to decrease excessive mirror checking. Her mother was involved by monitoring her daughter for excessive exercise. No nutritionist was involved because her weight was within the normal range and she appeared to be highly motivated to increase her caloric intake on her own.

After two months of treatment, she was able to maintain her weight and increased her daily intake to 1500 calories. This increased caloric intake was initially very difficult for her, and her fear of becoming obese intensified. Following an additional two months of CBT focused on her fear of becoming overweight, she became less preoccupied with food and was able to occasionally enjoy eating dessert foods on
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special occasions. The patient began to shift her value of self away from shape and instead valued her achievements in other activities such as her relationships with family members and friends. Her preoccupation with food lessened, and she was able to go out with friends. However, she continued to experience fear of becoming obese. At the time of submission, she was meeting with her psychiatrist monthly for continued psychotherapy.

DISCUSSION

This report presents two cases in which previously obese adolescents developed restrictive eating disorders with their treatment complicated by fear of becoming obese again. Although previous research has correlated childhood obesity to later development of binge-eating disorder or bulimia nervosa, there have been few studies describing an increased risk for restrictive eating disorders.\(^1,4\) Overweight children are more likely to develop maladaptive body image concerns and low self-concept than average-weight peers.\(^9\) Additionally, overweight children are frequently teased because of their weight and may therefore be limited in typical activities of childhood, further isolating them from peers.\(^3\)

Adolescent patients with eating disorders generally possess an intense fear of gaining weight or becoming fat, which may be further increased when a patient has suffered prior consequences of obesity. In both of these reported cases, patients experienced intense fear of becoming obese again. Since they had experienced significant consequences of obesity, their fear of weight gain appeared to be intensified. In Case A, this intensified fear was complicated treatment because it interfered with their healthy weight restoration. In both cases, fears of subsequent weight gain persisted long after the resolution of other symptoms such as restriction of energy intake and undue influence of shape on self-evaluation.

Eating disorders are typically underdiagnosed in the pediatric population, resulting in many adolescents who do not recover, or may only partially recover.\(^9\) Studies have demonstrated that diagnosis of eating disorders can be delayed in patients with a history of obesity.\(^9,11\) Clinician diagnosis may also be missed in previously obese children and adolescents because they may be normal weight even
after significant weight loss. Missing or delaying the diagnosis of restrictive eating disorders may increase the potential for long-term complications in children and adolescents.

Since treatment is often more difficult in previously obese children, patients’ risk for life-threatening complications may increase even further. Eating disorders can result in a multitude of consequences for every organ system and anorexia nervosa has been shown to impose the highest mortality rate of all psychiatric illnesses. In fact, most deaths associated with anorexia nervosa are due to medical complications or suicide.

As the prevalence of obesity among children and adolescents continues to increase, health professionals must remain aware that obese children and adolescents may be at increased risk for development of restrictive eating disorders following weight loss. Obese or previously obese children and adolescents presenting with weight loss, restrictive eating behaviors, frequent vomiting, excessive or compensatory exercise, or concerns about body image should be assessed for eating disorder risk. Screening tools, such as the Children’s Eating Attitudes Test (ChEAT-26) or the EAT-26 for adolescents, can be used to assess patients’ eating disorder risks.

CONCLUSIONS

Although previously obese children may be at risk for development of restrictive eating disorders, there is currently little research on this potential risk. These children may be at risk for increased complications due to clinicians’ lack of suspicion resulting in delayed diagnosis. Additionally, treatment in previously obese children and adolescents may be further complicated by an intensified fear of weight gain due to the significant consequences of obesity they have previously experienced. For treatment to be effective to prevent long-term complications, early identification by clinicians is imperative.

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Case Report

A unique case of progressive hemi-facial atrophy successfully treated with methotrexate

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ABSTRACT


CONTEXT: The effects of many dermatologic syndromes are not exclusive to the skin. Disorders commonly involve a complex interplay between multiple organ systems, thus not relying solely on the dermatologist for proper work up, diagnosis, and treatment. Morphea is one such rare disease which involves progressive loss or atrophy of subcutaneous tissue, muscle, and bone with a relatively mysterious etiology. The initial lesion of morphea can be subtle and appear as a pink to red plaque without any additional symptomatology. A biopsy at this early stage is non-specific and will only show the presence of a T cell infiltrate, vascular swelling, and edema. This active or progressive stage will continue for years before “burning out,” or halting progression, although still affecting underlying tissues. Many times, the sclerosis becomes severe enough to cause deformity and secondary systemic symptoms. Five general subtypes of morphea exist, including: plaque-type, linear, deep, guttate, and nodular. METHODS: In this paper, the authors report a case report of a rare subtype of linear morphea called Parry Romberg syndrome, also known as progressive hemi-facial atrophy (PHA). PHA usually involves at least one branch of the trigeminal nerve unilaterally. CONCLUSIONS: The authors will emphasize the importance of a multidisciplinary approach to diagnose and treat this disorder while also considering the multiple theories surrounding its pathophysiology. Keywords: morphea, progressive hemi-facial atrophy, Parry-Romberg syndrome

INTRODUCTION

Parry-Romberg syndrome, or progressive hemifacial atrophy (PHA) is a sporadic, acquired neurocutaneous disorder characterized by progressive hemiatrophy of the skin, soft tissue, muscles, cartilage, and underlying bony structure.1-3 The disorder was first described by Parry in 1825 and Romberg in 1846.4 The onset of this syndrome typically develops in the first and second decades of life, and more commonly occurs in women.2,4,5
Clinically, the disease presents as atrophy of the skin, fat, and osteocartilagenous structures of the lower face. The atrophy typically follows the distribution of the trigeminal nerve. As the disease progresses, hyperpigmentation or depigmentation accompanies the overlying shiny, dry, atrophic skin, and induration ensues. The skin appears adherent or bound down to the underlying structures without the pliable subcutaneous tissue. The lack of underlying tissue generally creates a striking skeleton-like appearance to the affected side of the face. The overlying skin is also commonly either hypopigmented or hyperpigmented, furthering the patient’s cosmetic concerns. The amount of cutaneous deformation is dependent on duration and aggressiveness of the progressive phase.2-5

Cicatricial (i.e., sub-epithelial scarring) alopecia of the affected hair-bearing areas is common.2-5 PHA more commonly manifests on the left side of face with unclear causality.1 Early in the disease process, progression can be halted by topical steroids and/or systemic immune modulators.2 Unfortunately, patients commonly present after the disease has “burnt out” or halted in its progression to become more atrophic. Significant atrophy can affect the tongue, gingiva, teeth, and bone causing the face to pull toward the defect. Ipsilateral arm, trunk, and leg may also be involved.

Depending on the extent of the disease, dermatology, neurology, plastic surgery, ophthalmology and oral maxillofacial specialists may need to be involved in developing a plan of care. Through presentation of this unique case report, the authors’ objective is to demonstrate how a multispecialty approach is often necessary to optimally treat PHA. This paper will also include discussion of the etiology, pathophysiology and various treatment options aimed at slowing the progression of the disease and addressing associated cosmetic concerns.

**Case Description**

This patient was a Caucasian male in his 40s who presented to the dermatologist office in 2015 with a chief complaint of hypopigmentation, and atrophy of the skin overlying his left temple and lower jawline (see Figure 1). During the prior two years, the patient reported a gradual loss of subcutaneous tissue, growth of the hypopigmented area, discomfort with mastication, and intermittent “muscle spasms” in his left cheek.
In 2006, he had been initially examined for a patch of hypopigmentation on his left temple. A biopsy was performed, which was consistent with Lichen Sclerosis et Atrophicus or morphea. A thorough examination revealed no other skin or systemic manifestations at that time. Treatment was directed toward the hypopigmentation area with close monitoring for disease progression. The patient’s hypopigmentation improved with a topical steroid and pimecrolimus, and he was subsequently lost to follow-up. Approximately nine years later, the aforementioned skin changes and increasing neurologic symptoms prompted him to seek re-evaluation.

Pertinent review of systems was also positive for joint pain affecting the small joints of his hands. Family history included a mother with osteoarthritis and a father with psoriasis.

Physical exam revealed an approximately 5cm by 3.5cm hypo-pigmented, firm, tightly adherent, or bound down plaque extending from the left temple, to the angle of the mandible and medially to the mid cheek. Atrophy of temporalis and adjacent muscles of facial expression was present (see Figure 1). No fasciculations were observed and sensation was intact. Neurologic exam was otherwise negative and tongue was midline without deficit. Laboratory testing was negative, including markers for autoimmune disease. MRI of the brain with and without contrast revealed no gross mass, white matter changes, or adenopathy.

A 4mm. punch biopsy of the atrophic area on the left cheek was completed. Histology was consistent with late stage morphea, characterized by deep dermal sclerosis with thick, hyalinized collagen bundles (see Figure 2). Based on the clinical course and histologic findings, the patient was diagnosed with Parry Rhomberg syndrome, also known as PHA. The patient was referred to a rheumatologist and neurologist for further assessment of associated joint paint and muscle spasms of the jaw. A diagnosis of seronegative rheumatoid arthritis led to the addition of Celebrex for his joint pain. An MRI and EMG were recommended to rule out underlying musculoskeletal involvement. The patient elected to undergo systemic treatment with methotrexate, secondary to rapid advancement of the morphea and musculoskeletal symptoms. After one month of treatment, the hypopigmentation, jaw stiffness, and muscle spasms were subjectively improved.
DISCUSSION

Although the underlying etiology and pathogenesis of PHA is not well known, it has been hypothesized to be related to trauma, genetics, infection, vascular malformations, endocrine disturbances, increase in cervical sympathetic nerve activity, or auto-immune disorders.\(^4\)\(^-\)\(^6\) Studies report that 24-34\% of PHA patients had a history of trauma including: accidental trauma, operative trauma (thyroidectomy and dental extraction), and obstetric trauma (vacuum maneuvers or forceps).\(^2\)\(^-\)\(^4\) Some experts also believe that PHA may be inherited in an autosomal dominant manner with incomplete penetrance.\(^2\)\(^,\)\(^5\)

Viruses or bacteria may also play a role in the pathogenesis of some PHA cases.\(^2\) Herpes zoster, Lyme disease, syphilis, rubella, tuberculosis, otitis media, and diphtheria have all been implicated in the development of PHA. A disorder in the neural crest migration, from which cranial vessels take origin or a chronic cell mediated vascular injury, may also comprise the source of the dysfunction. Abnormal thyroid activity is the most common endocrine disturbance associated with PHA.\(^2\)\(^-\)\(^5\) Thyroid dysfunction may have a negative effect on adipose tissue, leading to atrophy.\(^2\)\(^-\)\(^5\),\(^7\)

Sympathetic hyperactivity caused by superior cervical ganglion inflammation and irritation has also been hypothesized as a potential pathogenic mechanism leading to development of PHA.\(^8\) Frequent associations with autoimmune conditions and the detection of autoimmune markers in the blood and cerebrospinal fluid, support the hypothesis that PHA can be triggered by an autoimmune disorder.\(^2\)\(^-\)\(^4\) Pregnancy, stress, and surgery have also been reported to accelerate the disease.\(^9\) The patient described in this case report denied having sustained any previous trauma to the area, and could not recall if he had developed an infection at the time of initial presentation. He denied having any family history of autoimmune conditions, and his labs (ANA, SCL70, anti-dsDNA, TSH) were negative excluding an underlying autoimmune condition. Therefore, the etiology in this patient’s case is most likely a combination of stress and genetic predisposition.

Systemic manifestations of the disease can include neurologic, psychological ophthalmologic, rheumatologic, cardioligic, endocrine, and maxillofacial abnormalities. Seizures and headaches are the most common neurologic manifestations.\(^5\)
Neurological referral and MRI evaluation are therefore imperative. Ipsilateral hyper-intense white matter lesions is a common MRI finding.\textsuperscript{2-5} Secondary trigeminal neuralgia, dysarthria and or aphasia can often be noted due to destruction of bony structures further impinging on local vasculature.\textsuperscript{4,8,10} Recurrent migraines, cerebral atrophy, hemiplegia, and cranial aneurysms can also occur.\textsuperscript{2,3} Often overlooked, about 46\% of patients with PHA experience psychosis and or depression.\textsuperscript{1,3} Therefore, it is important to screen office patients and refer them to appropriate mental health professionals whenever possible. When the authors discussed the diagnosis with this particular patient, he denied having had any feelings of depression or anxiety. It is still important to follow PHA patients closely through treatment and have an open conversation with them during each office visit encounter.

Enopthalomos, uveitis, glaucoma, oculomotor nerve palsies, eyelid atrophy, heterochromia, restrictive strabismus and even blindness are a few of the many ophthalmologic findings sometimes associated with PHA.\textsuperscript{1-5,11} Our patient had recently visited the ophthalmologist and denied any vision changes. Ocular findings are also more common with a V1 trigeminal distribution as the disease can affect the underlying orbital tissues.

A positive anti-nuclear antibody (ANA) is the most common rheumatologic lab abnormality, seen in about 25-52\% of PHA cases. Inflammatory bowel disease (5\%), rheumatoid arthritis (4\%), ankylosing spondylitis (2\%), and systemic lupus erythematosus (2\%) are the most common autoimmune disorders associated with PHA.\textsuperscript{4} Any suspected clinical indications of hypertrophic cardiomyopathy and/or congenital heart disease should trigger a cardiology consult.\textsuperscript{2,12}

Oral manifestations such as angular cheilitis, gingivitis, and absent parotid glands can be appreciated in PHA. Unilateral tongue atrophy, jaw hypoplasia, and a palate deficiency can further result in communication disorders and dysphonia. Tooth and mandibular involvement should be assessed by a general dentist or orthodontist.\textsuperscript{3,13}

Once a clinical diagnosis of PHA has been made, a multidisciplinary approach helps ensure that systemic manifestations are evaluated and treated appropriately. The diagnosis of PHA is primarily clinical. Still, biopsies are commonly performed for further diagnostic confirmation. Histopathologic analysis can reveal atrophy of the epidermis,
dermis, and adnexal structures, and hypertrophy of collagenous fibers in the superficial dermis is characteristic (see Figure 2).

As already stated, an MRI, ophthalmologic exam, and rheumatologic screening serology should be performed upon diagnosis. Photographical records from the time of diagnosis are recommended to follow disease progression and duration. Ultrasound tests can also be used to monitor disease activity and progression by measuring dermal thickness and echogenicity of affected areas. Increase in dermal blood flow can be seen on color doppler as a sign of active disease.

Treatment of PHA is challenging and aimed at impeding progression of the disease as there are no curative treatments. Initial treatment of lesions in the inflammatory or active stage can be accomplished using high potency topical corticosteroids alternating with a calcineurin inhibitor, which is a non-steroidal inhibitor of T-cells. Topical treatments may take months to show an effect, so close follow-up is recommended with transition to systemic therapy if the disease continues to progress. Standard treatment of late-stage disease is methotrexate with dose ranges from 0.3-1mg/kg/week with a maximum dose of 25 mg weekly either by oral or injection routes. Prednisone (1mg/kg/day) can be added to this regimen for two months with a taper in the third month, due to methotrexate’s delayed effect on inflammation and fibrosis.

Once disease progression has halted or “burned out,” aesthetic augmentation to restore facial symmetry and volume may be beneficial. Injectable fillers and fat grafting can be used to add volume to the face. If bone is involved, facial plastic surgeons as well as oral maxillofacial specialists work to restore the jaw line. Treatment will involve a multispecialty approach with multiple procedures to correct the appearance, function, and also minimize psychosocial effects. This unique case of a male with PHA highlights how a seemingly specific dermatologic disease truly involves a multidisciplinary approach to care, treatment, and follow-up.

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Figure 1: Clinical image demonstrating the atrophic, and hypo pigmented lower cheek/jaw.
Figure 2: Hematoxalin and eosin Histology image showing atrophy of the epidermis, dermis, and thickening of the collagen with little inflammation.
Case Report

A Novel Technique for the Removal of an Intramedullary Femoral Guidewire Lodged in the Femoral Canal

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ABSTRACT

Kasik C, Rosen M, and Sauchak J. A Novel Technique for the Removal of an Intramedullary Femoral Guidewire Lodged in the Femoral Canal. Spartan Med. Res. J. Vol. 1, No. 2, pp. 73-81, 2017. CONTEXT: Intramedullary nails are currently the most commonly used device for the fixation of intertrochanteric hip fractures. An initial threaded guidewire is used for localizing the site of entry and determining the final position of the fixation device. Hardware failure with guidewire breakage can complicate the procedure and lead to unplanned challenges for the surgeon. Predisposing mechanical properties of the hardware, along with technical surgical errors may lead to inadvertent breakage or migration of guidewires. METHODS: The authors report a case of initial threaded starting guidewire migration into the femoral intramedullary canal with subsequent impaction into the distal femoral subchondral bone after advancement of the proximal femoral canal reamer. RESULTS: A method for antegrade removal of a lodged intramedullary guidewire through the distal femoral condyles is described. A set of key technical points to avoid this complication are also provided. CONCLUSIONS: Although guidewire migration during hip fracture surgery is a rare occurrence, it is important to recognize the technical measures that can be used to prevent this potentially devastating complication. Surgeons should be familiar with several different techniques for extraction of such hardware surrounding the hip, as there is no universally successful method. Keywords: guide wires, fracture fixation, intramedullary hip fractures.

INTRODUCTION

Intramedullary fixation is the most commonly used construct for the current treatment of intertrochanteric hip fractures.¹ Short intramedullary nails have become increasingly popular for the fixation of stable intertrochanteric hip fractures due to a shorter operative time, lower intraoperative blood loss, and a decreased need for blood transfusions when compared to the use of long intramedullary nails.² These types of proximal femoral nailing systems utilize an initial threaded guidewire and cannulated reamer system to initially prepare the proximal canal.
During this procedure, a guidewire is used to determine the final intended position of the intramedullary nail. Failure to take preventative cautions along with technical surgical errors can lead to inadvertent hardware breakage or migration. Hardware failure with guidewire breakage can also complicate the procedure and lead to unplanned challenges to the surgeon. Many techniques have been previously described for extraction of broken Kirschner wires and guidewires, including extraction utilizing pituitary forceps, Kerrison rongeurs, and over-reaming the guidewire with a cannulated drill bit.\textsuperscript{3,4,6-8}

In this paper, the authors report on a case of inadvertent migration of the initial 3.2 mm. distally threaded guidewire into the femoral intramedullary canal after use of the cannulated opening reamer. The guidewire became inadvertently impacted into the subchondral bone of the patient’s distal femur and was resistant to initial retrograde extraction techniques. Specific technical challenges presented during this case included the surgeons’ inability to provide a firm grip of the guidewire with the ongoing objective to perform a less invasive method of extraction in order to conserve the patient’s bone stock.

A specific method will be described for antegrade removal of an intramedullary femoral guidewire that has migrated into the femoral canal and become lodged in the distal femur. After strategic positioning of the patient’s leg, this technique can be generally reproduced using standard operating equipment.

**Description of Case**

An 86-year-old female presented to the operating theatre with an Evan’s stable type intertrochanteric hip fracture after falling from a standing height. The decision was made to use a short trochanteric fixation nail (Synthes, West Chester, PA, USA).\textsuperscript{11} The patient was first placed supine on a standard radiolucent fracture table and initial closed reduction of the fracture was performed under fluoroscopic guidance.

A 3.2 millimeter (mm.) distally threaded guidewire that was 400 mm. in length was inserted using a standard greater trochanter starting point with entry slightly lateral to the tip of the greater trochanter. The guidewire was then advanced with a power drill down to the level of the lesser trochanter. The 17.0 mm. soft tissue protection sleeve
and 3.2 mm. trocar were inserted over the guidewire down to bone, and a 17.0 mm. cannulated drill bit was used to ream the proximal femur.

There was initial resistance felt by the authors upon full advancement of the cannulated drill bit to the distal extent at the level of the lesser trochanter. After failure to fully advance the drill bit, fluoroscopic images were taken proximally to reveal the entire guidewire to be advanced into the intramedullary femoral canal.

Multiple failed attempts were made to remove the guidewire using Kocher forceps, arthroscopic basket forceps, needle drivers, and Kerrison rongeurs (see Figure 1). Additional fluoroscopic images of the distal femur were subsequently obtained, revealing the guidewire to be inadvertently impacted into the subchondral bone of the distal femoral condyle (see Figure 2). Due to the inability to retrieve the guidewire using other extraction methods, a decision was made by the surgeons to remove the guidewire antegrade through the distal femoral condyle.

Traction on the operative leg was first released. The knee was then flexed to approximately 30° and held in the flexed position by a surgical assistant. A bone tamp was placed into the opening of the proximal femur and placed in direct contact with the guidewire (see Figure 3). A mallet was then used to gently tap the guidewire through the anterior aspect of the medial femoral condyle. The guidewire was advanced until tenting of the skin over the anterior knee was noted. A stab incision was made over the tip of the guidewire, and it was gently advanced further. Once enough guidewire was exposed, it was subsequently removed with a power drill.

**DISCUSSION**

Orthopaedic hardware breakage or migration is a rare occurrence that can still pose major surgical challenges and result in potentially devastating effects. Guidewire complications during intramedullary nailing have been previously described in the past, with multiple case reports of complications due to migration into the thoracic and abdominal viscera.\(^3\)\(^-\)\(^8\) Recent literature concerning rates of orthopaedic hardware breakage has revealed that drill bits and Kirschner wires are the most commonly broken surgical instruments.\(^9\) Both mechanical and technical aspects of guidewire handling have been found to contribute to this phenomenon.\(^3\)\(^,\)\(^4\)\(^,\)\(^6\)\(^-\)\(^8\)

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Spartan Med Res J
Repeated use of guidewires for different orthopaedic patients can lead to an increased rate of breakage and failure, with rates approaching 0.18%. Mechanical deformation of the guidewire can also occur with repeated usage of the device, leading to both decreased bending and torsional strength of the wire. The results of a questionnaire survey administered by Ashford et al. to 248 orthopaedic units led to the proposal that surgeons should ensure single use by bending the guidewires after each case to prevent their re-use.

Technical errors by the surgeon can also predispose to an increased risk for inadvertent guidewire failure and breakage. Careful attention should therefore be paid to avoid inadvertently bending the guidewire or making any changes in its direction during insertion. Inserting the cannulated reamer over the guidewire with a different direction results in excessive contact stress upon the guidewire, possibly leading to migration and breakage.

The cannulated drill bit should also be properly cleaned before reusing. Small cortical bony fragments may become incarcerated in the cannulated drill as it passes through the fracture site. The lumen of the drill bit may become blocked, leading to an increase in frictional force. This increased friction can result in bending or migration of the guidewire with forceful reaming. As the drill bit is in contact with the guidewire, an eccentric reaming point is created which can lead to guidewire breakage.

This type of bony debris on the drill bit effectively decreases the diameter of the reamer, and prevents smooth gliding over the guidewire. The reamer and guidewire then subsequently move as a single unit. With forceful undue reaming, the guidewire may be inadvertently propelled forward into the intramedullary canal.

Frequent fluoroscopic imaging utilizing both anteroposterior and lateral views should be employed when a cannulated instrument is not adequately progressing using powered instruments. Impediments to advancement of cannulated reamers include guidewire migration and eccentric bone reaming. Forceful reaming should therefore be avoided. Guidewire repositioning should be reassessed using fluoroscopic imaging if any resistance is felt during opening of the proximal femoral canal.

As indicated earlier in this paper, various techniques have been described to address removal of broken guidewires and Kirschner wires during hip fracture repair.
These techniques include over-reaming with a cannulated drill bit, extraction using pituitary forceps or Kerrison rongeurs, arthroscopically assisted removal, and open arthroscopy with hip dislocation.\textsuperscript{3,4,6-8}

The specific technical issues presented in this case included the inability to provide a firm grip of the guidewire, with the surgeons aiming to perform a lesser invasive method of extraction designed to conserve the patient’s bone stock. The method described by the authors in this paper involved use of a bone tamp and mallet to gently tap the guidewire through the distal femoral condyles.

Strategic positioning of the leg is critical in order to remove the guidewire through the femoral condyles. With traction on the foot released, the knee could be manipulated and flexed to approximately 30 degrees. This method provided an adequate exit angle for guidewire through the distal femoral condyles and can be performed with the use of standard operating room tools.

One disadvantage of this technique includes the potential damage to the articular cartilage of the knee. However, the small diameter footprint of the guidewire, in conjunction with its removal through the non-weightbearing aspect of the femoral condyles made these minor drawbacks that the authors were willing to accept to conserve this patient’s remaining bone stock.

Bone quality should also play a role in decision-making while utilizing this technique. The patient in the case had confirmed osteoporosis and decreased bone mineral density. The porosity of the patient’s bone made it feasible to tamp the terminally threaded guidewire out through the distal femur. Caution should be advised when employing this technique in younger patients, as their increased bone mass may prevent removal of the guidewire through the thickened bone of the femoral condyles.

CONCLUSION

Although guidewire migration into the femoral intramedullary canal has not been well documented, it is important to recognize the technical and preventative aspects that can lead to this intraoperative complication.\textsuperscript{5} As outlined in Table 1, there are several key points in guidewire management that should be considered. Both preventive and proper technical techniques should be followed to decrease rates of guidewire migration.
and hardware breakage. Since no single surgical technique is universally successful for extraction of broken or migrated hardware, surgeons should be familiar with several different techniques that they can consider employing in varied hip fracture repair situations.

ACKNOWLEDGEMENT

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Figure 1: Attempted retrieval of threaded guidewire with pituitary rongeur.

Figure 2: AP and lateral views of the right knee demonstrating distal subchondral impaction of the threaded guidewire.
Figure 3: Schematic illustration demonstrating bone tamp placement into the opening of the proximal femur and in direct contact with the guidewire.

Table 1. Key points in guidewire management
1. Use guidewires only once. Be familiar with your institutions current practice regarding re-use.
2. Avoid inadvertent changes in direction when reaming over a guidewire.
3. Consider frequent intra-operative cleaning of the lumen of the cannulated reamer.
4. Perform both AP and Lateral imaging when failing to progress with the cannulated reamer.
5. Avoid forceful reaming and do not push through resistance.
6. Be familiar with several different techniques to remove broken guidewires.
Case Report

First reported case of Lyme carditis in Southwest Michigan

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ABSTRACT

FRAZIER NM and DOUCE RW. First reported case of Lyme carditis in Southwest Michigan. Spartan Med. Res. J. Vol. 1, No. 2, pp. 82-90, 2017. CONTEXT: Lyme disease is the most common tick-borne infection found in the eastern United States. In recent years, it has become an emergent Michigan public health concern. Lyme carditis is a recognized rare complication which is classically characterized by rapidly fluctuating degrees of heart block. In severe cases, or if inappropriately treated, Lyme carditis can also result in profound bradycardia, perimyocarditis, and sudden cardiac death. This report describes the first documented case of third degree heart block associated with Lyme carditis to occur in Michigan. METHODS: This is a retrospective case report of a patient evaluated and treated for Lyme carditis in Southwest Michigan in July, 2016. All information was obtained from either the patient or his electronic medical record. RESULTS: Despite initial misdiagnosis and inappropriate management, this patient ultimately received more appropriate medical therapy within 24 hours of first presentation. After eight days of high dose intravenous Ceftriaxone and supportive care, and more than two weeks of oral Doxycycline, the patient’s symptoms resolved and the disease was treated to resolution. Neither permanent nor temporary pacing was needed during/after the course of treatment. CONCLUSIONS: When correctly identified, Lyme disease and Lyme carditis can be easily treated. Although this patient’s history was without reported tick bite or exposure to a known host for Lyme disease, the authors believe that the patient’s history and physical exam was definitive enough to warrant the start of IV therapy with telemetry monitoring upon first presentation. The fact that the condition was not first diagnosed by providers indicates a potential gap in medical knowledge and awareness that should be addressed in clinical practice. The authors consider this case a harbinger of the emerging disease of Lyme carditis. Physical exam and EKG findings should guide clinicians’ therapeutic approaches. Although treatment with appropriate antibiotics is typically curative, therapeutic delays can lead to deadly results. Keywords: Lyme carditis, Lyme disease, heart block, tick-borne pathogens.

INTRODUCTION

Lyme carditis is a rare complication of Lyme disease occurring in about 1% of cases.¹ The complication typically develops within the first two months of infection, and is characterized by rapidly fluctuating classes of atrioventricular (AV) block.² Typical
symptoms including erythema migrans (i.e., rash), fatigue, fever, myalgias (i.e., muscle pain) and arthralgias (i.e., joint pain), and are often present in some combination.

Despite purported underreporting of Lyme disease, there were over 251,000 confirmed cases reported to the Centers for Disease Control and Prevention from 2005-2014, making it the most common tick-borne disease in the United States. During this period, 96% of cases were confined to 14 states. The State of Michigan accounted for only 0.32% of all confirmed cases.

In recent years, there has been an increased incidence of Lyme disease in Michigan and nationally (see Figure 1), making this tick-borne pathogen an emergent public health concern. This increase has been confirmed in the western and northern portions of Michigan, where *Borrelia burgdorferi* is endemic. The etiology for this trend are multifactorial. However, the northward expansion of the primary vector (or organisms responsible for transmission) for Lyme disease, the tick *Ixodes scapularis*, as well as the possible emergence of a new tick species carrying Lyme, are also possible contributors. Despite this increased incidence, the authors believe this is the first published account of Lyme carditis in Michigan resulting in complete heart block.

**Case Presentation**

This patient was a 36-year-old Caucasian male who first presented to an emergency department (ED) at a small community hospital a week after visiting Michigan's upper peninsula. His chief complaint of three days was of low back and bilateral leg pain associated with intermittent night sweats and fever. Although the patient had been working outside as a concrete pourer, he had not spent time in the woods nor near wildlife. The patient’s past medical history was significant for dyslipidemia and tobacco use. His father and brother had significant cardiac disease, with his brother having sustained a myocardial infarction (heart attack) when just 28 years old.

Per the original ED records, initial physical examination was only pertinent for mildly diaphoretic skin with an egg-shaped erythematous rash on his anterior left hip and groin measuring 26 cm. with central clearing, or area void of red coloration (see Figure 2). The rash was neither tender nor warm to palpation. The patient had
intermittent bradycardia in the 50s, and his vital signs were otherwise within normal limits.

Due to the patient’s reported chest discomfort, an EKG (electrocardiogram) was completing, showing 2nd degree AV block (Mobitz I) with heart rate of 64. Three serial troponin labs were drawn to evaluate for possible heart muscle damage but found to be negative. A nuclear exercise stress test was then done but was non-diagnostic due to the patient not reaching target heart rate. The inflammatory markers Erythrocyte Sedimentation Rate (ESR) and C-Reactive Protein (CRP) were noted to be elevated, suggesting an inflammatory and possibly acute infective process. Therefore, a Lyme serology was ordered. The patient was prescribed oral Amoxicillin for possible Lyme disease and discharged home.

Within a few hours after leaving the ED, the patient developed substernal chest pains, nausea and vomiting and sought re-evaluation at Lakeland Regional Medical Center in St. Joseph, MI. Initial examination revealed an anxious but fit-looking man in mild distress. The patient’s exam demonstrated bradycardia with pulse in the 50s, a regular rhythm and without murmur. The aforementioned rash was present and unchanged.

A repeat EKG now showed 3rd degree heart block with new T-wave inversion in leads II, III, and aVF, suggestive of possible inferior ischemia. The patient reported improvement in his chest pain while in the ED, but had mildly elevated troponins of 0.05 ng/mL at three separate times, suggestive of some level of cardiac damage. ESR and CRP levels were elevated at 81mm/h and 7.1mg/dL respectively, again signifying an acute inflammatory process. The Cardiology service was consulted and therapy was initiated for possible acute coronary syndrome (ACS), with the patient receiving full dose Aspirin and IV heparin. The patient was ultimately admitted to the cardiac/telemetry floor for 3rd degree heart block, bradycardia and chest pain.

Overnight, the patient experienced significant subjective improvements of his myalgias and arthralgias without fever. Although the patient’s rash was somewhat improved, a new systolic murmur was appreciated on cardiac exam. Telemetry demonstrated fluctuations between 1st and 3rd degree AV block, with 3rd degree being dominant.
Due to the fluctuating heart block and the patient’s presenting symptoms, an intravenous (IV) Ceftriaxone 2g daily dose was started by the hospitalist team to treat presumptively for Lyme disease as clinicians awaited serology results. In view of the patient’s family history, labs values and EKG abnormalities, the Cardiology service felt that ACS could not be ruled out. An echocardiogram showed a normal left ventricular ejection fraction (i.e., percentage of blood leaving the heart each time it contracts) of 60-65% and moderate left atrial enlargement. A cardiac catheterization was scheduled and temporary pacing was discussed and made available if needed.

The cardiac catheterization was performed and demonstrated only mild coronary artery disease which required no further intervention. Shortly after catheterization, Western Blot testing from the patient’s original ED visit resulted demonstrating positive IgM (p41 and p23) and IgG (p18, p23, p39, p41, p45, and p66) bands. In light of these findings and a largely negative cardiac work-up, a diagnosis of Lyme carditis was made. In house serologic testing confirmed this diagnosis one day later.

Due to the patient’s worsening cardiac function with plans to transition to oral antibiotics on discharge, an additional Doxycycline 100mg every 12 hours order was added to the existing Ceftriaxone regimen. All eventual blood cultures results were negative. During the next six days, the patient's heart rate and conduction abnormalities gradually improved.

By hospital day eight, the patient remained bradycardic in the high 50s but was now in 1st degree AV block. The patient was discharged on oral Doxycycline 100mg twice daily for 14 to total 21 days of antibiotic therapy and scheduled for a cardiac follow-up appointment. A week later, the patient was asymptomatic and in normal sinus rhythm without any conduction abnormalities. The patient was instructed to finish the remaining course of oral antibiotics and make aggressive lifestyle changes to decrease his underlying cardiac risk factors.

DISCUSSION

This patient's presentation with erythema migrans, fever, chills, myalgias, and arthralgias as well as rapidly fluctuating AV block was classic for Lyme carditis. Although cases of severe conduction abnormalities have been documented in top endemic areas, this case represents the first published account of complete heart
block related to Lyme infection occurring in Michigan. As the incidence of Lyme disease continues to rise, so should the incidence of Lyme carditis.

Although Lyme carditis can be fatal, it generally carries a good prognosis, especially when appropriate antibiotic therapy is administered early. Based on the severity of conduction abnormalities, patients should be treated with either oral or IV antibiotics for a total of 21-28 days to minimize cardiac abnormalities and prevent future complications. For patients with mild disease (1st degree AV block with PR interval <300 milliseconds), oral agents such as Doxycycline 100mg twice daily, Amoxicillin 500mg three times daily, or Cefuroxime 500mg twice daily and outpatient follow-up can be appropriate. However, adults who are symptomatic, have 2nd or 3rd degree AV blocks, or have a PR interval >300 milliseconds on EKG should be hospitalized for telemetry monitoring and administered daily IV Ceftriaxone 2 grams until conduction abnormality improves.10

Typically, patients’ severe AV block improves to a lesser degree block within one week.11 In rare cases, Lyme carditis has been implicated in nonreversible AV block12,13 and even more infrequently with sudden cardiac death.14,15,16 Due to the reversible and typically short-lived nature of conduction abnormalities, permanent pacing is not generally needed unless advanced heart blocks fail to improve after six weeks of therapy.18 If the need for temporary pacing arises as seen in up to 40% of severe cases,2 percutaneous or modified temporary transvenous pacing should be used.17

Despite this patient presenting with classic features of Lyme carditis from a diagnosis of Lyme disease, optimal therapy was not initiated at his onset of care. Instead of being admitted and started on IV antibiotics due to his 2nd degree AV block, the patient was treated as an outpatient with oral Amoxicillin and symptom relief. Whether the patient would have transitioned from 2nd degree to complete heart block if he had been started on IV Ceftriaxone on first presentation is uncertain. Nevertheless, our hope is to prevent future delays in optimal management by increasing clinician awareness of the appropriate therapy for cases of this potentially fatal Lyme carditis condition.
CONCLUSION

Patients with significant new conduction abnormalities and suspected Lyme infection should be admitted for IV antibiotics and telemetry monitoring. This should be continued until either serology rules out Lyme, or in the case that Lyme is confirmed, until conduction abnormality improves to 1st degree AV block. The safe transition to oral antibiotics is indicated for the remainder of 21-28 days of antibiotic therapy. Considering the potentially fatal outcome of untreated Lyme disease, this approach is reasonable. In so doing, we as healthcare providers can work to minimize the unnecessary burden of a curable disease, avoid invasive procedures, and decrease the financial burdens of associated complications.

Primary care and hospital based clinicians generally need to initiate aggressive antibiotic therapy in patients presenting with possible Lyme carditis. Even when obvious or confirmed exposure to ticks is absent, it would behoove clinicians to treat patients aggressively until this disease is ruled out when symptoms are suggestive of Lyme disease. For this particular patient, it was fortunate that his misdiagnosed and mismanaged condition during both ED visits and initial hospital admission did not result in permanent comorbidity. This case report highlights a rare, albeit present, clinical knowledge gap which warrants special attention in view of the emerging public health concern of Lyme disease in Michigan.

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Figure 1: Michigan Lyme Disease Incidence Rates (2002 – 2015)

![Graph showing incidence rates](image)


Figure 2: Anterior left hip and groin, 26cm. erythematous rash with central clearing.
Accuracy of Patient Height, Weight and Ideal Body Weight Estimates in the Emergency Department

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ABSTRACT

BOEHM K, WELT C, and GRIMALDI J. Accuracy of Height, Weight and Ideal Body Weight in the Emergency Department. Spartan Med. Res. J. Vol. 1, No. 2, pp. 91-99, 2017. CONTEXT: The purpose of this study was to establish the accuracy of emergency medicine professionals’ estimates of a sample of patients’ heights, weights and ideal body weights (IBW). METHODS: This was a cross-sectional survey study with 69 emergency medicine professionals concerning estimates of five standardized patients. Board-certified emergency physicians, emergency medicine residents and emergency department nurses were asked to estimate patient height, weight and (IBW) by looking at a series of photographs of five standardized patients with varied body types. Repeated measure analysis of variance procedures were used to examine for estimates-to-actual measurement differences. RESULTS: Overall height, weight and IBW estimate differences did vary significantly for the majority of the five standardized patients. Respondents’ clinical position (i.e., attending physician, resident, or nurse) and years of clinical experience did show a significant level of influence on estimate-to-actual differences for some proportion of patient estimates. CONCLUSION: Our results support the common belief that the accuracy of overall weight and height estimates in emergency department settings is unacceptably low, and that a patient's stated weight and height is likely to be more accurate. Further work is required in this area of emergency medicine practice since these types of inaccuracies could potentially compromise the effectiveness of therapies and treatments during emergent situations. Keywords: estimated body weight, ideal body weight, clinical estimates

INTRODUCTION

The number of annual emergency department (ED) visits in the US is on the rise, reaching over 127 million visits in 2010.1 Patients frequently present in the ED with emergent scenarios requiring rapid administration of lifesaving medications before there is time for them to be accurately weighed. Critically ill patients may also present with an altered mental status or inability to bear weight due to their injuries. Since many
medications used in such emergent settings are dosed by either weight or ideal body weight (IBW), inaccurate weight estimates by ED professionals can lead to inappropriate medical therapies.\(^2\) Key medications requiring this type of information include heparin, thrombolytics, phenytoin, and rapid sequence inductions agents such as rocuronium and ketamine.\(^2\)

**Purpose**

The purpose of this cross-sectional study was to examine the accuracy of a sample of emergency medicine professionals’ estimates of a series of standardized patients’ height, weight and IBW compared to actual measurement readings.

**METHODS**

**Study Setting**

The ED of Henry Ford Wyandotte Hospital is an academic community-based hospital with over 70,000 annual visits. During a two-week period in 2014, a sample of board-certified emergency medicine attending physicians, residents and registered nurses practicing at the time of the study were surveyed.

**Study Design**

Before data collection, the study protocol was reviewed and approved by the Henry Ford Health System international review board. The authors administered a cross-sectional survey that they had created to ED staff at a community-based academic hospital as well as an affiliated standalone ED in the community. To generate photographs of the five standardized patients, five healthy adult volunteers with varied body types were recruited. After obtaining consent from each subject, a survey of 10 photographs of the five standardized patients was constructed and distributed to a convenience sample of all ED attending physicians, residents, and registered nurses. No specific inclusion or exclusion criteria were observed for enrollment of these standardized patients.

Each patient’s height and weight were documented using the same scale and measuring tape at the same time of day. Patient heights were calculated in inches and weight in pounds. Their IBW was then calculated from these data using the following formulae: Males: \(\text{IBW} = 50 \text{ kg.} + 2.3 \text{ kg. for each inch height over 5 feet}\). Females: \(\text{IBW}\)
= 45.5 kg. + 2.3 kg. for each inch height over 5 feet. The images of each patient were taken at two angles, lying on the same ED stretcher, and wearing the same type of patient gown.

The faces of standardized patients were obscured to maintain their anonymity. All photographic images were inserted onto a survey sheet and disseminated to ED attendings, residents and nurses during the weeks of 1/29/2014 through 2/7/2014. Participating ED professionals were asked to provide estimates of each standardized patient’s height, weight and IBW based on the photos. Participating respondents were also asked to report their position in the ED (i.e., attending, resident or nurse) and years in clinical practice. Years of practice were categorized into PGY 1-4 (if resident), and less than five years, between five and 10 years, or greater than 10 years (if attending physician or nurse).

A total of 69 surveys were collected, including 14 attending physicians, 18 ED residents, and 37 registered nurses. Survey data were entered into an electronic spreadsheet by two of the authors (CW, JG). All survey data were initially entered by one researcher and rechecked by a second researcher to ensure accuracy. The height, weight and IBW estimations were then compared to actual measured values to calculate estimate-to-actual (ETA) differences.

**Data Analysis**

ETA difference data were analyzed as a series of continuous outcome variables for height, weight, and IBW. A series of repeated measure analysis of variance (ANOVA) analytic procedures were used to assess the level of influence of respondents’ position in the ED and clinical experience category on overall standardized patient individual height, weight and IBW estimates as well as ETA differences.

**RESULTS**

The overall results for patient estimates and ETA values are depicted in Tables 1 through 3. The first table reports the overall and mean ETA height measurement differences. The second table depicts weight measurement patterns, and Table 3 depicts IBW data results. In each of these tables, statistically significant results are denoted by bolding of the p-values.
As Table 1 demonstrates for height, while there were statistically significant variations observed for overall height estimate differences in four of five patients, as well as the combined estimate from all five patients, the significance of height estimate variations was lost when controlling for position subgroups (i.e., EM attending, EM resident, and nurse), and by levels of clinical experience (i.e., PGY year, <5 years, 5-10 years, and > 10 years).

Table 2 summarizes the weight estimate results, with a statistically significant variation in ETA differences observed from three standardized patients. Similarly, statistically significant variations were observed for ETA weight estimates by position, although not by clinical experience levels. Table 3, which depicts IBW estimate patterns, demonstrates somewhat similar patterns of significance to Table 2 for overall, EM position and across clinical experience category estimate differences.

DISCUSSION

Many of the calculations and medications routinely used in the ED require that professionals possess accurate information concerning patients’ weight and height. Many times, rushed ED professionals may feel obliged to make such estimates due to patients’ presenting circumstances. Current research in the adult population has shown that patients are historically more accurate at estimating their own weight than ED staff.2

Earlier studies comparing attending physicians, resident physicians, nurses, paramedics and patients estimates to actual weight measurements have suggested that ED professional estimates are frequently quite inaccurate.3,4 In fact, one study group concluded that patients were almost nine times more likely to accurately estimate their own actual weight than providers.4

Two similar studies have reported that patients were generally more accurate in estimating their true weight, with healthcare workers showing only a moderate degree of accuracy when patients’ personal weight estimates were compared to providers.2,5 Specifically, it was specifically reported in another project that ED staff provided accurate weight in only 33% of estimates, with such estimates even less accurate for underweight and overweight patients.6
The results of the authors’ study in a typical community-based ED setting were somewhat mixed. As seen in Table 1 through 3, sample professionals’ overall height, weight, and IBW estimates varied at statistically significant levels for the majority of standardized patients. Results also indicated that estimates were significantly variable amongst staff for some patients based on position in the ED and amount of clinical experience.

Of course, there were several limitations to our study. First, our use of photographs offered only a two-dimensional image of the standardized patients. Obscuring the patients’ faces in photographs could have limited how accurately participating providers estimated patients’ weights and/or heights. Moreover, we have subsequently concluded that many ED employees actually prefer to interact directly with patients to make more accurate estimates. Finally, the generalizability of our results to other settings may be limited due to our smaller study sample. A larger sample may have enabled us to detect more statistically significant ETA sample subgroup differences.

CONCLUSION

In spite of these limitations, this study demonstrated the high variability and inaccuracy of weight and height estimations typically made by ED employees, regardless of their position or years of clinical practice. Our results and conclusions support our theory that overall estimates of weight and height in ED settings are unacceptably low, and that a patient’s stated weight and height may tend to be more accurate. Further work is required in this area of emergency medicine practice since these types of inaccuracies could potentially compromise effective therapies and treatments during emergent situations.

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REFERENCES

Table 1: Differences in Height Estimates (N = 69)

<table>
<thead>
<tr>
<th>Height Difference*</th>
<th>Patient A</th>
<th>Patient B</th>
<th>Patient C</th>
<th>Patient D</th>
<th>Patient E</th>
<th>Patients A-E</th>
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<tbody>
<tr>
<td>Mean ± SD</td>
<td>76.0</td>
<td>73.0</td>
<td>75.5</td>
<td>64.5</td>
<td>75.0</td>
<td></td>
</tr>
<tr>
<td>Overall Differences</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Height difference</td>
<td>-2.2 ± 1.4</td>
<td>-4.5 ± 2.9</td>
<td>-2.3 ± 1.8</td>
<td>0.3 ± 2.5</td>
<td>-2.6 ± 2.1</td>
<td>-2.3 ± 1.4</td>
</tr>
<tr>
<td>p-value (t-test)**</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
<td>0.325</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

Differences by Position

| RN | -2.2 ± 1.4 | -4.3 ± 2.8 | -2.6 ± 2.0 | 0.0 ± 2.5 | -2.3 ± 2.5 | -2.3 ± 1.5 |
| EM resident | -2.2 ± 1.5 | -4.8 ± 3.1 | -1.9 ± 1.8 | 0.2 ± 2.5 | -3.1 ± 1.3 | -2.4 ± 1.0 |
| EM attending | -2.8 ± 1.8 | -4.7 ± 3.2 | -1.9 ± 1.1 | 1.1 ± 2.4 | -2.4 ± 2.0 | -2.2 ± 1.4 |
| p-value (t-test)** | 0.289 | 0.549 | 0.285 | 0.280 | 0.302 | 0.968 |

Differences by Clinical Experience

| PGY | -2.2 ± 1.5 | -4.8 ± 3.1 | -1.9 ± 1.8 | 0.2 ± 2.5 | -3.1 ± 1.3 | -2.4 ± 1.0 |
| < 5 years | -2.5 ± 1.4 | -4.1 ± 2.7 | -2.6 ± 1.5 | 0.5 ± 2.6 | -3.0 ± 1.8 | -2.4 ± 1.4 |
| 5-10 years | -2.6 ± 1.4 | -4.5 ± 3.5 | -2.7 ± 1.9 | 0.0 ± 2.3 | -3.2 ± 3.0 | -2.6 ± 1.4 |
| > 10 years | -1.9 ± 1.8 | -4.8 ± 2.8 | -2.1 ± 2.2 | 0.4 ± 2.7 | -0.9 ± 1.9 | -1.9 ± 1.6 |
| p-value (t-test)** | 0.591 | 0.901 | 0.573 | 0.968 | **0.002** | 0.437 |

*refers to difference between estimated and actual height

**statistically significant values (bolded) suggest a low probability that result observed would have occurred if no true correlation, (i.e., \( \rho \) (rho)=0)
# Table 2: Differences in Weight Estimates (N = 69)

<table>
<thead>
<tr>
<th>Actual Measured Weight (lbs.)</th>
<th>Patient A</th>
<th>Patient B</th>
<th>Patient C</th>
<th>Patient D</th>
<th>Patient E</th>
<th>Patients A-E</th>
</tr>
</thead>
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<tr>
<td>Weight Difference* Mean ± SD</td>
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<tr>
<td>Overall Differences</td>
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<td></td>
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<td></td>
<td></td>
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<tr>
<td>Weight difference</td>
<td>-22.2 ± 25.2</td>
<td>-4.0 ± 19.0</td>
<td>-0.9 ± 22.7</td>
<td>19.0 ± 24.6</td>
<td>-51.8 ± 31.2</td>
<td>-12.0 ± 15.3</td>
</tr>
<tr>
<td>p-value (t-test)**</td>
<td>&lt;0.001</td>
<td>0.083</td>
<td>0.744</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
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<tr>
<td>Differences by Position</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>RN</td>
<td>-15.3 ± 23.5</td>
<td>-0.8 ± 19.9</td>
<td>-1.6 ± 24.3</td>
<td>21.8 ± 28.4</td>
<td>-43.6 ± 33.8</td>
<td>-7.9 ± 15.0</td>
</tr>
<tr>
<td>EM resident</td>
<td>-26.6 ± 28.0</td>
<td>-7.5 ± 15.4</td>
<td>-1.9 ± 20.2</td>
<td>12.8 ± 18.3</td>
<td>-61.1 ± 30.2</td>
<td>-16.9 ± 15.2</td>
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<tr>
<td>EM attending</td>
<td>-34.9 ± 20.4</td>
<td>-7.9 ± 20.3</td>
<td>2.2 ± 22.8</td>
<td>19.8 ± 20.2</td>
<td>-61.4 ± 18.1</td>
<td>-16.5 ± 13.9</td>
</tr>
<tr>
<td>p-value**</td>
<td>0.010</td>
<td>0.105</td>
<td>0.940</td>
<td>0.893</td>
<td>0.013</td>
<td>0.024</td>
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<tr>
<td>Differences by Clinical Experience:</td>
<td></td>
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<td></td>
</tr>
<tr>
<td>PGY</td>
<td>-26.6 ± 28.0</td>
<td>-7.5 ± 15.4</td>
<td>-1.9 ± 20.2</td>
<td>12.8 ± 18.3</td>
<td>-61.1 ± 30.2</td>
<td>-16.9 ± 15.2</td>
</tr>
<tr>
<td>&lt; 5 years</td>
<td>-24.5 ± 23.8</td>
<td>-2.4 ± 24.8</td>
<td>-1.6 ± 26.3</td>
<td>23.5 ± 28.2</td>
<td>-52.5 ± 22.8</td>
<td>-11.5 ± 15.0</td>
</tr>
<tr>
<td>5-10 years</td>
<td>-22.7 ± 23.5</td>
<td>4.6 ± 15.4</td>
<td>2.9 ± 27.1</td>
<td>20.8 ± 22.1</td>
<td>-38.3 ± 37.6</td>
<td>-6.6 ± 16.7</td>
</tr>
<tr>
<td>&gt; 10 years</td>
<td>-14.3 ± 25.2</td>
<td>-8.5 ± 14.4</td>
<td>-1.5 ± 18.3</td>
<td>18.6 ± 27.5</td>
<td>-50.6 ± 35.8</td>
<td>-11.3 ± 14.4</td>
</tr>
<tr>
<td>p-value**</td>
<td>0.247</td>
<td>0.632</td>
<td>0.729</td>
<td>0.844</td>
<td>0.190</td>
<td>0.241</td>
</tr>
</tbody>
</table>

*refers to difference between estimated and actual weight

**statistically significant values (bolded) suggest a low probability that result observed would have occurred if no true correlation, (i.e., ρ (rho)=0)
Table 3: Differences in Ideal Body Weight (IBW) Estimates (N = 69)

<table>
<thead>
<tr>
<th></th>
<th>Patient A</th>
<th>Patient B</th>
<th>Patient C</th>
<th>Patient D</th>
<th>Patient E</th>
<th>Patients A-E</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Actual</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td><strong>Calculated IBW</strong></td>
<td>189.0</td>
<td>165.0</td>
<td>188.0</td>
<td>126.0</td>
<td>186.0</td>
<td></td>
</tr>
<tr>
<td><strong>IBW Difference</strong></td>
<td>8.4 ± 20.6</td>
<td>-15.9 ± 17.6</td>
<td>-2.2 ± 21.3</td>
<td>9.8 ± 15.4</td>
<td>6.0 ± 22.4</td>
<td>1.2 ± 13.5</td>
</tr>
<tr>
<td><strong>Mean ± SD</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td><strong>Overall:</strong></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>IBW difference</td>
<td>8.4 ± 20.6</td>
<td>-15.9 ± 17.6</td>
<td>-2.2 ± 21.3</td>
<td>9.8 ± 15.4</td>
<td>6.0 ± 22.4</td>
<td>1.2 ± 13.5</td>
</tr>
<tr>
<td>p-value (t-test)**</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
<td>0.391</td>
<td>&lt;0.001</td>
<td><strong>0.031</strong></td>
<td>0.459</td>
</tr>
<tr>
<td><strong>By staff:</strong></td>
<td></td>
<td></td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>RN</td>
<td>15.1 ± 19.2</td>
<td>-14.0 ± 18.3</td>
<td>-2.5 ± 21.3</td>
<td>10.0 ± 15.4</td>
<td>14.1 ± 24.2</td>
<td>4.6 ± 13.4</td>
</tr>
<tr>
<td>EM resident</td>
<td>-3.7 ± 21.7</td>
<td>-20.3 ± 14.1</td>
<td>-5.5 ± 22.2</td>
<td>3.7 ± 13.0</td>
<td>-7.7 ± 15.9</td>
<td>-6.7 ± 10.8</td>
</tr>
<tr>
<td>EM attending</td>
<td>6.4 ± 15.7</td>
<td>-15.6 ± 19.7</td>
<td>2.6 ± 20.8</td>
<td>17.2 ± 16.2</td>
<td>1.9 ± 14.8</td>
<td>2.5 ± 13.6</td>
</tr>
<tr>
<td>p-value**</td>
<td><strong>0.031</strong></td>
<td>0.345</td>
<td>0.829</td>
<td>0.407</td>
<td><strong>0.009</strong></td>
<td>0.170</td>
</tr>
<tr>
<td><strong>By experience:</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>PGY &lt; 5 years</td>
<td>-13.0 ± 22.4</td>
<td>-14.9 ± 20.7</td>
<td>-0.1 ± 22.9</td>
<td>14.7 ± 18.5</td>
<td>3.5 ± 18.9</td>
<td>3.3 ± 15.8</td>
</tr>
<tr>
<td>5-10 years</td>
<td>10.2 ± 15.2</td>
<td>-10.0 ± 22.7</td>
<td>2.0 ± 24.4</td>
<td>8.4 ± 12.8</td>
<td>20.7 ± 34.2</td>
<td>6.3 ± 15.8</td>
</tr>
<tr>
<td>&gt; 10 years</td>
<td>14.1 ± 15.8</td>
<td>-16.9 ± 11.8</td>
<td>-4.5 ± 16.4</td>
<td>11.1 ± 14.0</td>
<td>13.1 ± 12.8</td>
<td>3.4 ± 7.5</td>
</tr>
<tr>
<td>p-value**</td>
<td><strong>0.021</strong></td>
<td>0.329</td>
<td>0.492</td>
<td>0.204</td>
<td>&lt;0.001</td>
<td><strong>0.008</strong></td>
</tr>
</tbody>
</table>

*refers to difference between estimated and actual measure of ideal body weight

**statistically significant values (bolded) indicate a low probability that result observed would have occurred if no true correlation, (i.e., p (rho)=0)