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Research
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SSAJM

THE SCHOLARLY SOCIETY OF AMERICA
JOURNAL OF MEDICINE

RESEARCH IS THE FUTURE

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Dr. Peter Iskander

Meet an Editor

- Where did you complete your education?
  - I completed my undergrad degree at the University of Toronto where I received an Honors Bachelor of Science majoring in Human Biology and Environmental Science. I went on to further pursue a medical degree at the American University of Antigua.

- Where do you see yourself in the next few years?
  - I am currently completing my PGY-3 year of Internal Medicine at the Wright Center for Graduate Medical Education where I serve as the Chief of Scholarly Activity. In the future I'm hoping to pursue a fellowship in Gastroenterology!

- What got you into research?
  - Initially, the idea of dedicating extensive hours to reading and dissecting lengthy journal articles didn't appeal to me. However, during my early years in medical school, a graduate (who had later become my mentor) approached me with an offer to collaborate on a research project focused on stroke patient rehabilitation. I began to realize that my thought process was naturally analytical, and this endeavor provided a thrilling experience. Deconstructing research papers and data into manageable fragments and piecing them together felt like solving a puzzle, much like a detective gathering evidence to reach a conclusion.

- Favorite hobbies?
  - Baking! Medical school was where I found my unexpected joy in the kitchen. It was a sort of therapy. Every little while I'd look up a fun recipe and experiment with each one giving it my own touch. It really came in handy for my classmates and friends during exam season; we soon came to realize that there were few stressors that a good brownie cheesecake couldn't solve! Otherwise, I can rarely say no to a good game of volleyball!

- Any career advice for our readers?
  - It might sound cliché, but my advice is to follow your passion. Choosing a career that genuinely resonates with you can significantly impact your work ethic and dedication on a daily basis. It's essential to select something you can see yourself doing every day because, trust me, burnout is a real challenge. I received one of the best pieces of advice from an attending physician in Baltimore during my Internal Medicine training. He emphasized that a patient interaction should not feel like an interview. If a patient has not felt better after you have left the room than before you had entered, then you have failed that encounter.

Acknowledgements

The Scholarly Society of America Journal of Medicine; a staple of innovation, a path to research. A rapidly evolving medical journal created by the community for the community; one that I am proud to be a part of. As one of the co-founders and the chief editor, it is with great pride I was given the honor of introducing our first issue.

I have only our readers and supporters to thank. Our team would not be here and flourishing today if it weren't for your persistence. With that, I would like to acknowledge our deep appreciation and thanks to Drs. Simin Nasr and Douglas Klamp. Two phenomenal physicians who have gone above and beyond supporting our cause, in terms of establishing the journal and providing constructive feedback along the way! To the rest of the editorial board, each one of you helped provide a crucial building block to the breathtaking infrastructure we are celebrating here. Thank you to Dr. Edwin Mogaka for your endless knowledge in designing and programming the online platform. To Dr. Amninder Singh for your expertise in online databases. To Dr. Anand Maligireddy for your proactive work ethic. Finally, to Nathan Cardona for helping be the glue to hold us all together and coordinating the seemingly never-ending meetings.

Research is the future. With medicine and technology exponentially growing, new advancements and therapies evolve daily. Let us continue to celebrate our achievements, push the boundaries of our knowledge, and embrace intellectual curiosity.

A great man once said, "with great knowledge comes great responsibility". It is our responsibility to use this knowledge to be able to provide the best possible care for our communities. Let us use this platform to bind our collective potential to make a difference. I have no doubt that we will continue to shine brightly in our respective fields.

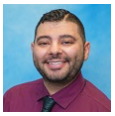
Thank you all once again, and congratulations to my fellow scholars.
Together, let us continue to inspire, innovate, and leave a lasting legacy.

Sincerely,

Peter Iskander, MD

Peter Iskander, MD
Co-Founder & Chief
Editor

Executive Board



Peter Iskander, MD:
Born and raised in Toronto, Ontario, Dr. Iskander completed his medical degree at AUA before joining The Wright Center for GME for his Internal Medicine residency. As the current Chief of Scholarly Activity of his program, he helped establish the SSAJM with his team to promote research and publications for all residents. He currently serves as our Chief Editor and Co-founder. In his free time, he loves to cook/bake and rarely passes up the opportunity for a game of volleyball!

Edwin Mogaka, MD, PhD:
Dr. Mogaka is the current Resident Leader for Scholarly Activity for Family Medicine at The Wright Center for GME. Dr. Mogaka has a Ph.D. in Psychology with a concentration in Educational Psychology from the Harold Abel School of Social Sciences at Capella University in Minneapolis, Minnesota. He also has a Doctor of Medicine degree from the Medical University of the Americas in the West Indies. He is a founding member of the Editorial Board at SSAJM.

Simin Nasr, MD:
Dr. Simin Nasr is a board-certified Family Medicine physician and a board-certified Geriatrician at The Wright Center for GME & CH in Scranton. Born and raised in Iran, she is a graduate of the Belarusian State Medical University in Minsk, Belarus. She completed an obstetrics and gynecology residency at Gilan University of Medical Sciences in Iran, then stayed in her native country for several years while providing OB-GYN services in both community-based and hospital settings. After immigrating to the United States, Nasr joined the Medical College of Wisconsin's Family Medicine – All Saints Residency Program and developed a keen appreciation for working with older adult patients. She subsequently completed a Geriatric Medicine Fellowship at UPMC in Pittsburgh. She is a founding member of the Editorial Board at SSAJM and a faculty mentor for the board.

Anand Maligireddy, MD:
Dr. Anand Maligireddy is an Internal Medicine resident physician at The Wright Center for GME. He serves as their Resident Leader for Scholarly Activity. With a rich background in research, including his previous role as a Research Fellow at Mayo Clinic and his current position as a Research Collaborator at the same institution, Anand brings a wealth of knowledge and dedication to advancing medical understanding. He is unwavering in his commitment to patient care and his passion for academic excellence. He is a founding member of the Editorial Board at SSAJM.

Tony Abdelmaseeh, MD:
Dr. Tony Abdelmaseeh is a board-certified pediatrician and an Family Medicine resident physician at The Wright Center for GME. Dr. Abdelmaseeh completed his Pediatrics training at Lincoln Hospital in the Bronx, New York. Interested in practicing full-scope family medicine, he is a founding member of the Editorial Board at SSAJM.

Amninder Singh, MD:
Dr. Amninder Singh is an Internal Medicine resident at The Wright Center for GME. He brings with him a wealth of experience, having served in the Indian military and acting as a reviewer for various medical journals. Dr. Singh is passionate about clinical research, particularly in Cardiology, and actively encourages fellow residents to get involved. He is a founding member of the Editorial Board at SSAJM.

Nathan Cardona, MS:
Nathan Cardona is a graduate of the University of Scranton with a background in Occupational Therapy. Nathan serves as the Director of Scholarly Activity, Institutional Research, and IRB Administration at The Wright Center for GME and has a passion for a wide range of medical research activities.

Editorial Team

Editorial Team

Navigating the Research Landscape: A Roadmap for Residents

Author: Edwin Mogaka, MD, PhD

In general, resident physicians often encounter the formidable challenge of embarking on their initial research projects, particularly when devoid of prior research skills. The intricacies of formulating a coherent and answerable question loom large, setting the stage for an undertaking that can be both intimidating and complex. Recognizing this, the American College of Physicians in the USA has ardently championed the cause by actively promoting resident abstract and poster sessions, serving as pivotal platforms for skill development and knowledge dissemination. These sessions embrace a diverse array of categories, encompassing traditional biomedical research, quality improvement projects, evidence-based medicine (EBM) initiatives, and clinical case studies.

For individuals new to the world of research, engaging in quality improvement projects and evidence-based medicine (EBM) initiatives represents a valuable initiation into the intricacies of scientific inquiry. These initiatives serve not only as educational endeavors but also as pivotal stepping stones, endowing residents with fundamental skills and fortifying them for the challenges inherent in more advanced research pursuits.

Embarking on a quality improvement project offers residents a hands-on learning experience, guiding them through a multifaceted process. They navigate the intricacies of identifying pressing problems, formulating plans for improvement, conducting a meticulous examination of intervention outcomes, and brainstorming innovative ideas for further enhancements. This comprehensive approach not only addresses the practical aspects of intervention but also cultivates a nuanced understanding of the entire research lifecycle.

Moreover, quality improvement projects immerse residents in the complexities of abstract writing, poster preparation, and the articulate defense of their work. These high-level skills, surprisingly, are often perceived as less daunting than the administrative challenges associated with managing Institutional Review Board (IRB) approvals, overseeing laboratory procedures, or navigating the intricacies of clinical data collection. The acquisition of skills through hands-on projects thus establishes a solid foundation, easing the transition for residents as they progress towards more intricate and demanding biomedical research endeavors.

By delving into the practicalities of research methodologies, residents not only cultivate a proficiency in addressing immediate healthcare challenges but also develop the acumen to navigate the broader landscape of scientific investigation. This experiential learning approach, grounded in the intricacies of quality improvement projects, positions residents to seamlessly transition from foundational research experiences to the complexities of advanced biomedical research, marking a transformative journey in their research proficiency and contributing to the collective advancement of medical knowledge.

Embarking on an Evidence-Based Medicine (EBM) project offers novice residents a straightforward yet highly educational avenue into the realm of research. This particular approach provides an accessible framework for residents to hone their research skills while delving into the intricacies of clinical inquiry. The journey begins with residents tasked with identifying a clinical question, firmly rooted in a specific case. Employing established EBM techniques, such as the widely used PICO framework (Patient, Intervention, Comparison, Outcome), residents systematically formulate their inquiries. This structured approach ensures a methodical exploration of the essential components, guiding residents in the precise delineation of their research questions.

Following the formulation stage, residents engage in a comprehensive literature search. This phase serves as a crucial aspect of the project, requiring residents to navigate through a vast expanse of scholarly literature to gather relevant information and evidence. The literature search, a skill in itself, allows residents to synthesize findings and distill pertinent information that contributes to a more profound understanding of their chosen clinical question.

The culmination of their efforts finds expression in a poster presentation, wherein residents articulate their research question, methodology, and key findings in a concise and visually engaging format. This presentation format not only facilitates effective communication of their work but also offers residents an opportunity to

showcase their ability to distill complex information into a clear and accessible format. This EBM project, with its structured approach and tangible outcomes, proves to be an excellent vehicle for residents eager to refine their abilities in answering clinical questions. Beyond that, it serves as a platform for mastering the intricacies of literature searches, allowing residents to navigate and critically evaluate existing knowledge, thus contributing to the establishment of the current landscape surrounding their inquiries.

In essence, the EBM project not only empowers residents with hands-on research experience but also equips them with essential skills in formulating clinical questions, conducting systematic literature reviews, and presenting their findings—an invaluable foundation for their evolving roles as practitioners engaged in evidence-based healthcare practices. For residents interested in biomedical research, a well-structured and deliberate curriculum emerges as a vital compass to navigate this intricate landscape. The city's medical community recognizes the significance of offering tailored guidance and has curated a wealth of online resources within the AAMC's MedEdPORTAL to empower these aspiring researchers.

These invaluable resources, ranging from tutorials on "How to Write an Abstract of a Research Project" to comprehensive guides on "How to Formulate a Clinical Question," form an indispensable toolkit. Each component serves as a beacon, illuminating the path for residents as they embark on their research journey. This curated curriculum ensures that residents have access to a well-rounded education, covering essential aspects of research methodology, project formulation, and effective communication of findings.

A structured timeline acts as a scaffolding, providing residents with a clear roadmap for their research endeavors. This temporal framework not only fosters discipline but also ensures a steady progression, allowing residents to navigate the various stages of their projects with a sense of purpose and direction. Mandatory mentorship meetings further enriches this educational experience. The guidance and insights provided by experienced mentors become invaluable, offering residents a compass to navigate challenges, refine their research objectives, and gain a more nuanced understanding of the research landscape.

Crucially, the overarching message is clear: residents need not be daunted by the prospect of immersing themselves in awe-inspiring bench work to derive meaningful insights from a research project. Programs encourage residents to opt for more manageable, smaller-scale projects. This strategic approach allows them to grapple with the entire research process—from the conception of ideas through the intricacies of execution to the articulate presentation of findings. By engaging in this comprehensive process, residents not only acquire profound insights into research methodologies but also cultivate a holistic understanding of the scientific inquiry.

The Author is a PGY3 and Leader of Scholarly Activities for the Family Medicine Residency of The Wright Center for Graduate Medical Education in Scranton, Pennsylvania.

Reduction of Musculoskeletal Pain in First Postgraduate Year (PGY1) Medicine Residents: A Randomized Controlled Trial on Improving Work Life with Laptop Ergonomics

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Abstract

Background: Work-related musculoskeletal disorders (WRMSDs) are a widely recognized problem among healthcare professionals. First postgraduate year (PGY1) students are particularly at risk of developing WRMSDs due to the demanding nature of their work. This study aimed to evaluate the effectiveness of an ergonomic intervention in reducing musculoskeletal pain among PGY1 and improving their work life.

Methods: This randomized control trial included participants from incoming first-year residents for the academic year 2022-2023. Exclusion criteria included daily medications for headaches, having a history of neck/spine pain injections, history of chronic pain, or already using laptop stands. Data collection utilized a Google Survey form incorporating pain scales at baseline, 3, and 6 months.

Results: Out of 57 potential candidates, 41 residents were eligible. Four subjects were lost due to dropout, transfer, or leaving the residency program, leaving 37 residents by the end. Significant differences ($p<0.05$) among control and intervention are shown in shoulder pain, lower back pain, taller subjects with wrist/hand pain, and males with upper back pain. Trends ($p<0.10$) existed with heavier subjects, males with upper back and wrist pain and taller subjects with headaches. The other differences in average pain scoring among different treatments, genders, weights, and heights was not statistically significant.

Conclusion: The study findings suggest that the intervention was effective in reducing shoulder, wrist, and lower back pain among residents. However, further research is needed to explore these outcomes.

Keywords: *Medical residents, Neck pain, Shoulder pain, Lower back pain, Wrist/hand pain, Upper back pain*

Introduction

Work-related musculoskeletal disorders (WRMSDs) have emerged as a significant concern for occupational morbidity in modern societies [1]. The adverse consequences of these conditions include absenteeism, increased costs, and reduced productivity. WRMSDs can be described as a wide array of degenerative conditions that impact supporting muscles, ligaments, blood vessels, tendons, and joints [2]. The most common manifestation of these conditions includes pain that is usually experienced in the neck and upper limbs which can lead to functional impairment. Neck pain is common and the fourth greatest contributor to global disability [3]. Based on the data from the Global Burden of Disease Study, a systematic analysis revealed that the incidence of neck pain per 100 000 population was 806.6, and for years lived with disability from neck pain per 100 000 population was 352.0 [4]. Although several factors can contribute to WRMSDs, biomechanical factors are considered to be major contributors [5]. Repetitive movements, mechanical vibration, forceful exertion, awkward postures, and compressions are common kinetic factors that can lead to MSDs. Apart from physical load factors, individual (age, gender, attitude) and organizational factors such as psychological, social, and work-related stress factors (work intensity, long working hours, management behavior, and lack of job satisfaction) can also contribute to MSDs [6,7]. During an injury, psychological risk factors like job stress, and depression increase the risk of disability in affected individuals and can promote the transition from acute to chronic pain [8].

A person suffering from MSD can manifest symptoms such as discomfort, paresthesia, and tiredness. A variety of professions are at risk of MSDs owing to their unique requirements of movements, body loading, and load management. However, healthcare professionals are particularly at risk of developing WRMSDs due to demanding work environments and prolonged working hours. A study by Freimann et al. reported that almost 70% of nurses experienced musculoskeletal pain in the past year. Among those, 57% had back pain whereas 56% had neck pain. Furthermore, higher work demands, low respect in the workplace, and inter-professional conflicts were significant contributors to musculoskeletal pain ($p<0.05$) [9]. Another study showed that more than 80% of surgeons suffer from MSDs across the world [10]. These findings show that healthcare professionals are at heightened risk of WRMSDs. The use of tablets and laptops are a common practice by healthcare professionals to assist in patient care. Healthcare professionals often use computers to seek medical information, guideline

adherence, and clinical decision-making [11]. While the extent of computer usage in the medical field may not be as widespread as in other professions, it is nonetheless employed by medical professionals as required [12]. Chronic deconditioning due to occupational stressors from screen usage and bad cervical ergonomics may lead to prominent exostosis projecting from the occipital squama [13]. Furthermore, cervical, thoracic, and upper extremity stress also affects lumbar and pelvic structures and stability during sitting [14]. A significant amount of data has shown that the increased use of computers in inappropriate posture can lead to significant MSDs [15].

This is particularly important in Postgraduate Year 1 (PGY1) residents who are already prone to MSDs due to high work demands and long working hours. This can hinder their ability to perform optimally and decrease their quality of life. For such individuals, ergonomic interventions can improve work efficiency and reduce musculoskeletal pain. Currently, there is a scarcity of data that has assessed the role of ergonomic intervention to improve work performance in PGY1. This leaves a significant gap in the literature. This study aimed to evaluate if intervention of modern computer ergonomics can reduce headache, musculoskeletal strain/pain, and injuries by decreasing head protraction angle over a 6-month period.

Method

Study design

A randomized controlled trial was undertaken to gather both quantitative and qualitative data utilizing a pain scale [16]. Following the initial measurement of the research variables, the participants were subsequently allocated into two distinct groups: the intervention group and the control group. The intervention was allocated randomly to 50% of the residency cohort. Before commencing the study, informed consent forms and a concise exclusionary survey were disseminated to participants through email. Prior to the implementation of the intervention, pain scales were integrated into a Google Survey form and distributed to the residents. This was done to establish a baseline measurement. Subsequently, follow-up assessments were conducted at 3 and 6 months to evaluate the efficacy of the intervention in terms of reducing headache, musculoskeletal strain/pain, and injuries.

Participants

The participants in the research were residents of the Wright Center who were going to be starting their first year there in the academic year 2022-2023.

Inclusion Criteria

All Family Medicine, Internal Medicine, Psychiatry and Physical Medicine and Rehab residents, PGY-1 during the 2022-2023 academic year were eligible for participation in the study.

Exclusion Criteria

Exclusion criteria included using daily medications for headaches, having a history of pain injections on the neck or spine, having chronic pain issues involving the head/neck/spine, or were already using laptop stands.

Intervention and Data Collection

The investigators of the study collected primary data. The data was collected via a survey conducted on the Google platform. The subjects who provided consent were randomly assigned to either a control group or a study group. Prior to the commencement of the study period, the study group was provided with laptop stands, keyboards, and mice. The researchers collected data by administering a Nordic Musculoskeletal Questionnaire in conjunction with a Visual Analogue Scale, which was incorporated into an online survey form hosted on the Google platform. This data collection process was conducted at three-time points: baseline, 3 months, and 6 months. The data that was gathered was subsequently transferred to an EXCEL file that was password encrypted. The identifying information of the patients was redacted, and subsequently, unique identification numbers were assigned to each patient. Additionally, the data were subjected to a coding process.

Statistical Analysis

Statistical analysis was conducted using IBM SPSS (Statistical Package for Social Sciences (SPSS), Version 22, Chicago, IL). The present study used a mixed-method approach that incorporated both quantitative and qualitative assessments. The quantitative analysis involved propensity score analysis and multivariable regression. Descriptive statistics were calculated for continuous variables. Pain scales were recorded using a Nordic Musculoskeletal Questionnaire and Visual Analogue Scale. Correlations between outcomes of interest were examined through univariate and multivariate analyses. The qualitative assessment consisted of questions related to work habits, physical pain, external influences, workload, and mental-emotional changes, incorporating content and narrative analysis to explore participants' experiences. A p-value of less than 0.05 was considered significant.

Results

Out of 57 possible candidates only 41 joined the study. A total of four subjects were lost during the study from dropout, transfer, or leaving the residency program. The final analysis included data from 37 participants only. Shoulder pain scoring responses among control and intervention is given in Table 1. Among control and intervention, 63 (56.8%) and 48 (43.2%) responses were observed for different severity of shoulder pain (Pain scoring: 1-9) including no pain responses. However, the shoulder pain scoring responses were not different among control and intervention except for 6 months. In 6th month, among control and intervention, 9 (42.9%) and 8 (50%) responded for no pain, 0 (0%) and 5 (31.3%) responded for pain severity score 1, 0 (0%) and 2 (12.5%) responded for pain severity score 2, 3 (14.3%) and 1 (6.3%) responded for pain severity score 3, 3 (14.3%) and 0 (0%) responded for pain severity score 4, 2 (9.5%) and 0 (0%) responded for pain severity score 5 and 6, 1 (4.8%) and 0 (0%) responded for pain severity score 7 and 9, 0 (0%) responded for pain severity score 8 ($p<0.05$).

Table 1. Shoulders pain scoring responses among control and intervention.

Shoulders	**No pain**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**Total**	**p-value**
Baseline												
Cont	10	2	3	0	3	0	3	0	0	0	21	0.55

rol	47.6%	9.5%	14.3 %	0%	14.3%	0%	14.3%	0%	0%	0%		
Inter venti on	6 37.5%	1 6.3%	3 18.8 %	2 12.5%	2 12.5%	1 6.3%	1 6.3%	0 0%	0 0%	0 0%	16	
3 months												
Cont rol	9 42.9%	2 9.5%	2 9.5%	3 14.3%	2 9.5%	1 4.8%	0 0%	1 4.8 %	1 4.8%	0 0%	21	0.89
Inter venti on	9 56.3%	2 12.5%	2 12.5 %	2 12.5%	1 6.3%	0 0%	0 0%	0 0%	0 0%	0 0%	16	
6 months												
Cont rol	9 42.9%	0 0%	0 0%	3 14.3%	3 14.3%	2 9.5%	2 9.5%	1 4.8 %	0 0%	1 4.8%	21	0.03
Inter venti on	8 50%	5 31.3%	2 12.5 %	1 6.3%	0 0%	0 0%	0 0%	0 0%	0 0%	0 0%	16	
Total												
Cont rol	28 44.4%	4 6.3%	5 7.9%	6 9.5%	8 12.7%	3 4.8%	5 7.9%	2 3.2 %	1 1.6%	1 1.6%	63	0.32
Inter venti on	23 47.9%	8 16.7%	7 14.6 %	5 10.4%	3 6.3%	1 2.1%	1 2.1%	0 0%	0 0%	0 0%	48	
Total	51 45.9%	12 10.8%	12 10.8 %	11 9.9%	11 9.9%	4 3.6%	6 5.4%	2 1.8 %	1 0.9%	1 0.9%	111	

The lower back pain scoring responses among control and intervention are given in Table 2. Among control and intervention, 63 (56.8%) and 48 (43.2%) responses were observed for different severity of lower back pain (Pain scoring: 1-9) including no pain responses. In comparison, 20 (31.7%) and 32 (66.7%) responded with no lower back pain in control and intervention, respectively. Among control and intervention, 4 (6.3%) and 5 (10.4%) responded for pain severity score 1, 9 (14.3%) and 2 (4.2%) responded for pain severity score 2, 4 (6.3%) and 4 (8.3%) responded for pain severity score 3, 5 (7.1%) and 1 (2.1%) responded for pain severity score 4, 8 (12.7%) and 2 (4.2%) responded for pain severity score 5, 9 (14.3%) and 2 (4.2%) responded for pain severity score 6 and 2 (3.2%), 1 (1.6%), 1 (1.6%) responded for pain severity score 7, 8 and 9, respectively for control group ($p<0.01$).

Table 2. Lower back pain scoring responses among control and intervention.

Low Back	**No pain**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**Total**	**p-value**
Baseline												
Control	6 28.6%	1 4.8%	3 14.3%	2 9.5%	2 9.5%	3 14.3%	3 14.3%	1 4.8%	0 0%	0 0%	21	0.26
Intervention	10 62.5%	0 0%	0 0%	3 18.8%	0 0%	2 12.5%	1 6.3%	0 0%	0 0%	0 0%	16	
3 months												
Control	7 33.3%	2 9.5%	2 9.5%	2 9.5%	0 0%	4 19%	3 14.3%	0 0%	1 4.8%	0 0%	21	0.16
Inter venti	12 75%	2 12.5%	1 6.3%	0 0%	0 0%	0 0%	1 6.3%	0 0%	0 0%	0 0%	16	

on												
6 months												
Control	7 33.3%	1 4.8%	4 19%	0 0%	3 14.3 %	1 4.8%	3 14.3%	1 4.8%	0 0%	1 4.8%	21	0.21
Intervention	10 62.5%	3 18.8%	1 6.3%	1 6.3%	1 6.3%	0 0%	0 0%	0 0%	0 0%	0 0%	16	
Total												
Control	20 31.7%	4 6.3%	9 14.3%	4 6.3%	5 7.9%	8 12.7%	9 14.3%	2 3.2%	1 1.6%	1 1.6%	63	0.01
Intervention	32 66.7%	5 10.4%	2 4.2%	4 8.3%	1 2.1%	2 4.2%	2 4.2%	0 0%	0 0%	0 0%	48	
Total	52 46.8%	9 8.1%	11 9.9%	8 7.2%	6 5.4%	10 9%	11 9.9%	2 1.8%	1 0.9%	1 0.9%	111	

The wrist/hand pain scoring responses among males and females are given in Table 3. Among males and females, 75 (67.6%) and 36(32.4%) responses were observed for different severity of wrist/hand pain (Pain scoring: 1-8) including no pain responses. However, overall wrist/hand pain scoring responses showed an increasing trend in males as compared to females (p=0.07). For pain scoring 2 and 3, only 2 (40%) and 1 (12.5%) responses were observed from females.

Table 3. Wrist/hands pain scoring responses among male and female.

Wrist/Hands	No pain	1	2	3	4	6	7	8	Total	p-value
Baseline										
Male	15 60%	2 8%	1 4%	3 12%	0 0%	3 12%	1 4%	0 0%	25	0.33
Female	11 91.7%	0 0%	1 8.3%	0 0%	0 0%	0 0%	0 0%	0 0%	12	
3 months										
Male	16 64%	2 8%	0 0%	3 12%	3 12%	0 0%	1 4%	0 0%	25	0.35
Female	10 83.3%	0 0%	1 8.3%	1 8.3%	0 0%	0 0%	0 0%	0 0%	12	
6 months										
Male	16 64%	0 0%	2 8%	1 4%	2 8%	3 12%	0 0%	1 4%	25	0.33
Female	12 100%	0 0%	0 0%	0 0%	0 0%	0 0%	0 0%	0 0%	12	
Total										
Male	47 62.7%	4 5.3%	3 4%	7 9.3%	5 6.7%	6 8%	2 2.7%	1 1.3%	75	0.07
Female	33 91.7%	0 0%	2 5.6%	1 2.8%	0 0%	0 0%	0 0%	0 0%	36	
Total	80 72.1%	4 3.6%	5 4.5%	8 7.2%	5 4.5%	6 5.4%	2 1.8%	1 0.9%	111	

The upper back pain scoring responses among males and females are given in Table 4. Among males and females, 75 (67.6%) and 36 (32.4%) responses were observed for different severity of upper back pain (Pain scoring: 1-7) including no pain responses. In the 6th month, upper back pain scoring responses were on trend and higher in males as compared to females (P=0.09). In comparison, 32 (42.7%) and 26 (72.2%) responded with no upper back pain in males and females, respectively. Among males and females, 10 (13.3%) males responded for pain severity score 1, 15 (20%) and 2 (5.6%) responded for pain severity score 2, 8 (10.7%) and 3 (8.3%) responded for pain severity score 3, 4 (5.3%) and 2 (5.6%) responded for pain severity score 4, 2 (2.7%) and 1 (2.8%) responded for pain severity score 6, 4 (5.3%) and 1 (2.8%) responded for pain severity score 7 and 1 (2.8%) female responded for pain severity score 8 (p<0.05).

Table 4. Upper back pain scoring responses among male and female.

Upper back	No pain	1	2	3	4	5	6	7	Total	p-value
Baseline										
Male	12 48%	4 16%	3 12%	4 16%	0 0%	1 4%	1 4%	0 0%	25	0.17
Female	7 58.3%	0 0%	2 16.7%	0 0%	2 16.7%	0 0%	1 8.3%	0 0%	12	
3 months										
Male	11 44%	3 12%	6 24%	3 12%	1 4%	0 0%	1 4%	0 0%	25	0.17
Female	9 75%	0 0%	0 0%	2 16.7%	0 0%	0 0%	0 0%	1 8.3%	12	
6 months										
Male	9 36%	3 12%	6 24%	1 4%	3 12%	1 4%	2 8%	0 0%	25	0.09
Female	10 83.3%	0 0%	0 0%	1 8.3%	0 0%	1 8.3%	0 0%	0 0%	12	
Total										
Male	32 42.7%	10 13.3%	15 20%	8 10.7%	4 5.3%	2 2.7%	4 5.3%	0 0%	75	0.04
Female	26 72.2%	0 0%	2 5.6%	3 8.3%	2 5.6%	1 2.8%	1 2.8%	1 2.8%	36	
Total	58 52.3%	10 9%	17 15.3%	11 9.9%	6 5.4%	3 2.7%	5 4.5%	1 0.9%	111	

The wrist/hands pain scoring responses among individuals of 58-67 inches and above 68 inches are given in Table 5. Among both, 51 (45.9%) and 60 (54.1%) responses were observed for different severity of wrist/hand pain (Pain scoring: 1-8) including no pain responses. At baseline, wrist/hands pain scoring responses were higher at the trend among 68-up inches people as compared to 58-67 inches people (p=0.06). Among individuals of height between 58 to 67 inches, 4 (7.8%) responses were observed for pain score 1. Among individuals of 58-67 inches and above 68, 3 (5.9%) and 2 (3.3%) responded for pain score 2, 2 (3.9%) and 6 (10%) responded for pain score 3. Among individuals of height above 68 inches, 5 (8.3%), 6 (10%), 2 (3.3%), and 1 (1.7%) responded to pain scores 4, 6, 7, and 8, respectively (p<0.01).

Table 5. Wrist/hands pain scoring responses among different heights.

Wrist/Hands	No pain	1	2	3	4	6	7	8	Total	p-value
Baseline										
58-67 inches	13 76.5%	2 11.8%	2 11.8%	0 0%	0 0%	0 0%	0 0%	0 0%	17	0.06
68-up inches	13 65%	0 0%	0 0%	3 15%	0 0%	3 15%	1 5%	0 0%	20	
3 months										
58-67 inches	13 76.5%	2 11.8%	0 0%	2 11.8%	0 0%	0 0%	0 0%	0 0%	17	0.23
68-up inches	13 65%	0 0%	1 5%	2 10%	3 15%	0 0%	1 5%	0 0%	20	
6 months										
58-67 inches	16 94.1%	0 0%	1 5.9%	0 0%	0 0%	0 0%	0 0%	0 0%	17	0.19
68-up inches	12 60%	0 0%	1 5%	1 5%	2 10%	3 15%	0 0%	1 5%	20	
Total										
58-67 inches	42	4	3	2	0	0	0	0	51	<0.01

	82.4%	7.8%	5.9%	3.9%	0%	0%	0%	0%		
68-up inches	38 63.3%	0 0%	2 3.3%	6 10%	5 8.3%	6 10%	2 3.3%	1 1.7%	60	
Total	80 72.1%	4 3.6%	5 4.5%	8 7.2%	5 4.5%	6 5.4%	2 1.8%	1 0.9%	111	

Head pain scoring responses among individuals of 100-159 pounds and more than 160 pounds are given in Table 6. Among both, 60 (54.1%) and 51 (45.9%) responses were observed for different severity of head pain (Pain scoring: 1-6) including no pain responses. However, at 3 months and overall head pain scoring responses were trending among individuals of 100-159 pounds and more than 160 pounds (p=0.07).

Table 6. Head pain scoring responses among different weights.

Head	No pain	1	2	3	4	5	6	Total	p-value
Baseline									
100-159 pounds	12 60%	1 5%	2 10%	2 10%	1 5%	1 5%	1 5%	20	0.81
160-up pounds	10 58.8%	1 5.9%	1 5.9%	4 23.5%	0 0%	0 0%	1 5.9%	17	
3 months									
100-159 pounds	13 65%	3 15%	2 10%	0 0%	2 10%	0 0%	0 0%	20	0.07
160-up pounds	9 52.9%	0 0%	2 11.8%	2 11.8%	0 0%	3 17.6%	1 5.9%	17	
6 months									
100-159 pounds	13 65%	2 10%	2 10%	1 5%	2 10%	0 0%	0 0%	20	0.47
160-up pounds	9 52.9%	0 0%	2 11.8%	1 5.9%	2 11.8%	1 5.9%	2 11.8%	17	
Total									
100-159 pounds	38 63.3%	6 10%	6 10%	3 5%	5 8.3%	1 1.7%	1 1.7%	60	0.08
160-up pounds	28 54.9%	1 2%	5 9.8%	7 13.7%	2 3.9%	4 7.8%	4 7.8%	51	
Total	66 59.5%	7 6.3%	11 9.9%	10 9%	7 6.3%	5 4.5%	5 4.5%	111	

Head pain scoring responses among individuals of 58-67 inches and above 68 inches are given in Table 7. Among both, 51 (45.9%) and 60 (54.1%) responses were observed for different severity of head pain (Pain scoring: 1-6) including no pain responses. However, head pain scoring responses were not different among individuals of 58-67 inches and above 68 inches.

Table 7. Head pain scoring responses among different heights.

Head	Height (58-67 in.)	Height (>68 in.)	Total
No pain	32 (48.5%)	34 (51.5%)	66
1	6 (85.7%)	1 (14.3%)	7
2	4 (36.4%)	7 (63.6%)	11
3	3 (30%)	7 (70%)	10
4	4 (57.1%)	3 (42.9%)	7
5	1 (20%)	4 (80%)	5
6	1 (20%)	4 (80%)	5
Total	51 (45.9%)	60 (54.1%)	111
Pearson Chi-Square = 9.123, df = 6, p-value = 0.16			

Average pain scoring among different treatments, genders, heights and weights are given in Table 8 and Figure 1. Average pain scoring was higher for head, neck, shoulders, upper and lower back for the control as compared to intervention ($p<0.05$). The wrist/hand pain and the lower back average pain scoring was higher among males as compared to females ($p<0.05$). For height above 68 inches, the wrist/hand pain and lower back average pain scoring was higher ($p<0.05$). However, the upper back average pain scoring also showed increasing trend for the individuals having height above 68 inches ($p=0.06$). The head, neck and lower back average pain scoring was higher for the individuals having more than 160 pounds body weight ($p<0.05$).

However, average pain scoring of shoulders, wrist/hand and upper back also showed increasing trend among individuals of above 160 pounds body weight (p=0.06-0.08).

Table 8. Average pain scoring (Mean±SE) among different treatments, genders, heights and weights.

Variables	Head	Neck	Shoulders	Wrist/Hand	Upper Back	Lower Back
Treatment						
Control	1.6±0.2	2.4±0.3	2.2±0.3	1.3±0.3	1.8±0.3	2.8±0.3
Intervention	0.8±0.2	1.4±0.3	1.3±0.2	0.8±0.3	0.8±0.2	1±0.2
p-value	0.02	0.02	0.01	0.16	<0.01	<0.01
Gender						
Male	1.4±0.2	1.9±0.3	1.7±0.2	1.5±0.3	1.5±0.2	2.3±0.3
Female	1±0.3	2.1±0.4	2±0.4	0.2±0.1	1.1±0.3	1.4±0.4
p-value	0.31	0.71	0.57	<0.01	0.24	0.05
Height						
58-67 in.	1±0.2	1.7±0.3	1.6±0.3	0.3±0.1	1±0.2	1.5±0.3
>68 in.	1.5±0.3	2.2±0.3	2±0.3	1.7±0.3	1.7±0.2	2.5±0.3
p-value	0.11	0.26	0.34	<0.01	0.06	0.04
Weight						
100-159 lb.	1±0.2	1.5±0.3	1.5±0.3	0.8±0.2	1.1±0.2	1.5±0.3
>160 lb.	1.6±0.3	2.5±0.4	2.2±0.3	1.4±0.3	1.7±0.3	2.7±0.4
p-value	0.05	0.05	0.08	0.08	0.06	0.01

Overall average pain scoring among different treatments (Figure 1), genders (Figure 2), weights (Figure 3), and heights (Figure 4). Average pain scoring among different treatments, genders, weights and heights was not different.

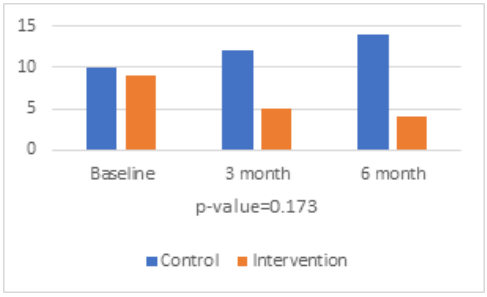

Figure 1. Pain scoring comparison between control and intervention groups.

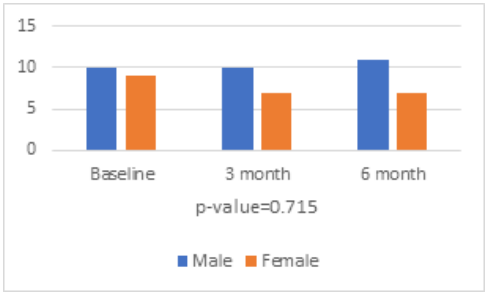

Figure 2. Pain scoring comparison between male and female participants.

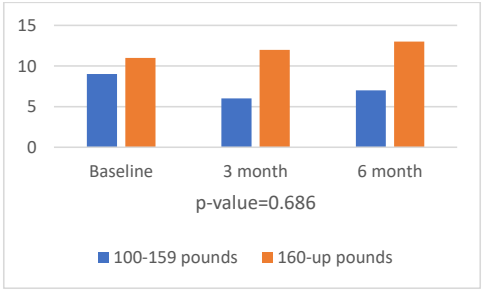

Figure 3. Pain scoring comparison between 100-159 pounds and 160-up pounds.

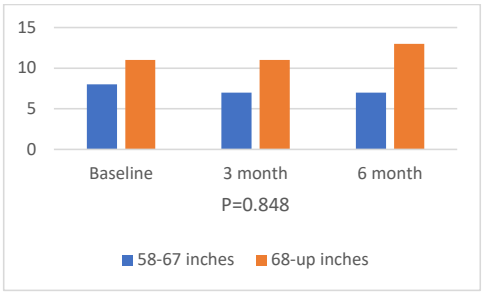

Figure 4. Pain scoring comparison between 58-67 inches and 68-up individual height.

Discussion

The lower back, neck, and shoulders are the most prone areas for the development of musculoskeletal symptoms among computer users [17,18]. Pain in the neck, shoulder region, and lower back is often manifested due to the static loading of the spine by prolonged sitting or standing [19]. Office workers who exhibit higher neck flexion angles tend to experience heightened activity in the upper trapezius muscle, which is also linked to neck and shoulder discomfort [20]. A study by Calick et al. reported that the most painful areas associated with higher desktop computer use were the upper back, neck, lower back, and shoulder respectively [21]. The "Laptop Sniffing" Position (face down forward head posture) or "Text Neck" may compromise the resting spinal column's double "S-shape" affecting the entire spine and neck. This cervical/upper extremity stress also affects lumbar and sacral/pelvic structures which allow stability during sitting [22]. Similarly, a 3D motion analysis showed that tablet and laptop use was associated with greater neck and upper trunk flexion compared to desktop computers [23]. However, pain can be decreased by balancing the spine through physical activity or using ergonomic equipment [24]. Factors that may play into laptop-related musculoskeletal symptoms and disorders include the lack of adjustability of the keyboard and screen (unlike the desktop) which leads to more pronounced neck and trunk flexion when typing inversely related to the size of the device [25].

The present study investigated the reduction in musculoskeletal pain by using ergonomic interventions (laptop stand and wireless mouse/keyboard) while using laptops in clinical situations. Our findings showed that these ergonomic interventions significantly improved shoulder pain ($p=0.03$), lower back pain ($p=0.01$), wrist/hand pain for tall subjects >68 inches ($p<0.01$) and upper back pain in males ($p=0.04$).

The present results align with previous studies that demonstrated the effectiveness of workstation adjustments in reducing musculoskeletal pain [26,27]. A study by Shariat et al. reported that ergonomic adjustments in the workplace resulted in reduced pain in the neck, right shoulder, left shoulder, and lower back compared to the control group [27].

When averaging pain scores and comparing between control and intervention, control groups had significantly higher scores consistently for head, neck, shoulders, upper and lower back ($p<0.05$). Males had significantly more pain that females in wrists ($p<0.05$) and lower back ($p<0.05$), taller subjects had significantly more wrist ($p<0.05$) and lower back pain ($p<0.05$) and heavier subjects had significantly higher head ($p<0.05$), neck ($p<0.05$) and lower back pain ($p<0.05$).

Trends (p-values between 0.05 to 0.10) existed with males with upper back ($p=0.09$) and wrist pain ($p=0.07$) and headaches in both taller (>68 inches, $p=0.06$) and heavier subjects (>160 lb, $p=0.07$ at 3 months and $p=0.08$ total) with headaches. The other differences in average pain scoring among different treatments, genders, weights, and heights was not statistically significant. Our results deviate from a previous study by Lee et al. that used ergonomic intervention involving furniture modifications [28]. Their findings showed that the lower back pain did not reduce with ergonomic intervention; however, the intervention was successful in reducing shoulder, wrist, neck, and upper back pain ($p<0.05$). Our results can be explained by the fact that physical ergonomics reduce physiological and physical stress on the body while working.

Furthermore, previous research has shown that the positioning of the scapula at rest can be significantly influenced by ergonomic risk factors. Scapular protrusion, for instance, can lead to reductions in the subacromial space and rotator cuff strength, while also increasing tension in the anterior glenohumeral ligaments and scapular stabilizing muscles [29,30]. The present intervention including laptop stand, keyboard and mouse is effective in improving positioning of scapula that can reduce upper back pain. A previous study by Price et al. showed that there was a notable inclination observed among users who did not utilize an external monitor or keyboard to display a heightened degree of neck flexion or a more prominent downward gaze. The analysis of upper arm angle data revealed that participants exhibited a greater extent of reaching movements in conditions where external monitors or keyboards were not utilized [31]. Similarly, a study by Gold et al. showed that prone posture during laptop use resulted in a significantly higher level of perceived discomfort intensity. Prone posture can be characterized by neck extension and non-neutral positioning of the shoulders, elbows, and wrists [32].

Study Limitations

There are several limitations of the study that should be considered while interpreting the results of the study. Due to the absence of consistent monitoring, it is conceivable that certain employees may have made alterations to their respective workstations. The measurement of the sitting period and the frequency of adjustments from sitting to standing was not conducted. An objective analysis of pain may give more accurate results. However, pain monitoring devices would have been costly and impractical in a residency program. Finally, our study was limited by the number of residents available at the time of the study and may explain the shift within the trend when comparing weight and height. The power analysis revealed that the sample size should be 45 per group which was not achieved in our current study. This can explain the high p-value in our results. However, we intend to replicate this randomized controlled experiment for an additional two to four more years to increase the number of subjects and power of our study.

Conclusion

There was a significant indication that the implementation of ergonomic equipment resulted in a reduction of individual pain. This study presents empirical evidence to residency programs regarding the potential benefits of implementing ergonomic equipment to mitigate the occurrence and alleviate the discomfort associated with musculoskeletal injuries resulting from prolonged laptop usage among their PGY-1 workforce, who experience a demanding workload during their initial year of physician training. These findings may be considered for any profession where extended laptop usage is required and where preservation of workforce musculoskeletal health is important. The implementation of strategies aimed at mitigating work-related pain has the potential to enhance productivity and reduce the financial burden on residency programs by minimizing absenteeism and healthcare expenses. These results are significant for promoting the well-being and comfort of medical professionals during their training under stressful and demanding work conditions and may lay down good habits to be

incorporated into future practice both at the workplace and in their respective patient populations.

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Statements and Declarations

I declare that this thesis has been composed solely by our team and that it has not been submitted, in whole or in part, in any previous application for a degree. Except where stated otherwise by reference or acknowledgment, the work presented is entirely my own.

Ethics Approval

This study was performed in line with the principles of the Declaration of Helsinki. Approval was granted by the Ethics Committee of The Wright Center for GME (June 4, 2022/1927081-1).

Competing Interests

The authors have no relevant financial or non-financial interests to disclose. The authors have no conflicts of interest to declare that are relevant to the content of this article.

Consent to participate

Informed consent was obtained from all individual participants included in the study.

A Case of Calcium Resistant Pancreatitis

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Abstract

Gallstones and alcohol abuse are two of the most common causes of acute pancreatitis in the US. Aggressive fluid hydration and pain management are the mainstays of management. In some cases, however, symptoms and electrolyte derangements cannot be fixed until the underlying triggering cause is addressed. We present a case of a 39-year-old male with a significant alcohol abuse history presenting with an episode of acute pancreatitis and elevated triglyceride and low calcium levels. Curiously, despite aggressive calcium repletion, levels did not respond. It was only when the triglyceride levels began to normalize did the calcium levels begin to respond.

Introduction

Gallstones and alcohol abuse are two of the most common causes of acute pancreatitis (AP) in the US. Other common causes include hypertriglyceridemia, autoimmune pancreatitis, post-endoscopic retrograde cholangiopancreatography, gain of function mutations, pancreatic duct injury, and medication-induced [1]. Normal serum Calcium (Ca) ranges between 8.5 to 10.2 mg/dL and both hypercalcemia and hypocalcemia can lead to certain clinical symptoms. Acute hypocalcemia can cause papilledema, long QT interval, and neuromuscular irritation, whereas chronic forms can manifest as ectopic calcification, extrapyramidal symptoms, parkinsonism, and dementia. Symptoms of hypercalcemia, on the other hand, depend on the severity of hypercalcemia; mild forms (<12 mg/dL) may be asymptomatic, or have nonspecific symptoms like constipation, fatigue, and depression. Chronic and slowly progressive elevation may be well tolerated, but an acute increase can result in polyuria, polydipsia, and sensorium changes. Up to 88% of hospitalized patients with AP have concurrent hypocalcemia (prevalence 15–88%), which correlates with the severity of their illness [2]. Magnesium deficiency may contribute to the pathogenesis of hypocalcemia in patients with AP. Despite normal serum magnesium concentrations, patients with AP and hypocalcemia frequently have magnesium deficiency [3]. One possible mechanism of hypocalcemia seen in AP includes the autodigestion of mesenteric fat by pancreatic enzymes, causing the release of free fatty acids, ultimately leading to the formation of calcium salts [4]. Elevated levels of urinary and salivary amylase are important markers of disease severity [5].

Clinical Course

A 39-year-old male with a past medical history of significant alcohol abuse (up to 15 beers and 10-15 shots of vodka daily) and seizure disorder (on lamotrigine) presented to the hospital for worsening abdominal pain associated with nausea and non- bloody / non-bilious vomiting. Diagnostics on arrival were pertinent for hypertriglyceridemia >4,425 mg/dL, hypocalcemia 5.3 mg/dL, hypokalemia 3.1 mmol/L, hyponatremia 128 mmol/L and elevated lipase of 839 U/L. Abdominal ultrasound was significant for hepatomegaly and cholelithiasis without biliary or pancreatic duct dilation. CT and MR imaging, however, did show evidence of peripancreatic fat stranding and fluid (without drainable collection) (Figure 1).

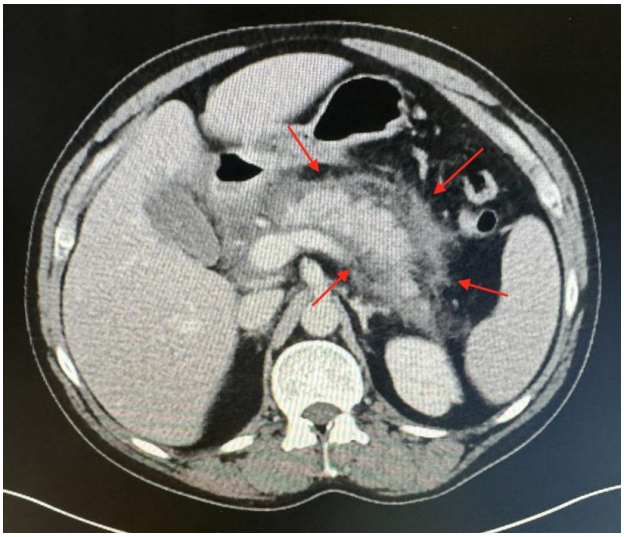

FIGURE 1: CT imaging of the abdomen with arrows depicting peripancreatic fat stranding and free fluid; concerning for AP.

Patient received aggressive fluid resuscitation and was placed on an insulin drip for the first 48 hours for persistent hypertriglyceridemia. He was replenished with up to 5000mg of IV calcium gluconate over the first 24 hours with minimal changes in serum levels. It was only after triglyceride levels began to normalize did Ca begin to rise. He was given approximately 20,000 mg of IV calcium gluconate over his hospital stay (roughly 96 hours). Basic metabolic panels done every four hours showed consistent signs of treatment-resistant hypocalcemia with calcium levels less than 8.0 mg/dL as seen in Table 1 and Figure 2.

TABLE 1
Table depicting the relationship over time (from admission to discharge) of TG to Ca levels.

	Triglycerides (TG) (mg/dL)	Calcium (Ca) (mg/dL)
0hr (admission)	> 4425	5.2
24hr	1133	6.2
48hr	588	6.6
72hr	278	8.1
96hr (discharge)	232	8.7

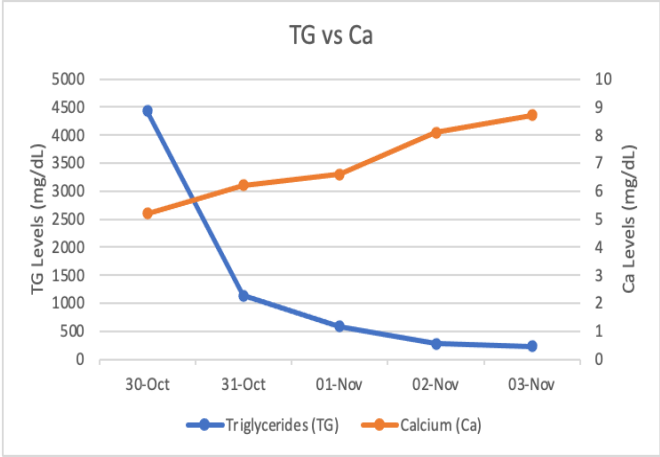

FIGURE 2: Graph depicting the gradual rise of Ca as TG levels (mg/dL) decrease over hospital stay

Discussion

Diagnostic Criteria

AP is one of the most common causes of gastrointestinal-associated hospitalizations in the United States. Hypertriglyceridemia is the third most frequent cause of AP, accounting for approximately 5% of all cases. As per a study conducted in China, the annual admission rate of hypertriglyceridemia-induced pancreatitis (HTGP) had increased from 14.3% to 35.5% [6]. A TG level of above 1000 mg/dL is often used to define hypertriglyceridemia as the source of AP; for every 100 mg/dL rise in serum TG level above 1000 mg/dL, there is approximately 4% increase in the incidence of AP. Nevertheless, there is a greater but lower risk of HTGP at lower levels of triglycerides (TG), making it important to check the levels of TG on admission to identify hypertriglyceridemia as either the sole reason or a cofactor. This is because HTGP is often more severe as compared to other causes [7]. As per the diagnostic criterion established in Japan, two of the following three manifestations are to be met for a diagnosis of AP: characteristic upper abdominal pain, elevated levels of pancreatic enzymes, and findings of ultrasound, computed tomography, or magnetic resonance imaging suggesting AP. Due to high specificity and sensitivity, measurement of blood lipase levels is usually recommended and is considered superior to other pancreatic enzymes. Blood amylase levels can be elevated in diseases other than pancreatitis and because of its low specificity should be used with caution. The cut-off level of pancreatic enzymes for the detection of AP has not yet been determined due to a lack of adequate evidence and consensus to date [8]. The diagnosis of AP continues to evolve. A few patients can develop severe AP resulting in major complications and increased morbidity and mortality, hence signifying the importance of increased recognition of such patients.

Scoring Systems

Several scoring systems specific to pancreatitis have been described that correlate with subsequent morbidity and mortality. Ranson score was introduced in 1974 by Dr John Ranson and includes variables on admission and within 48 hours. These variables include: age older than 55, white blood cell count more than 16000/microL, blood glucose greater than 200 mg/dL, lactate dehydrogenase level greater than 350 IU/dL, and aspartate aminotransferase level more than 250 IU/dL. The variables during initial 48 hours include: hematocrit decrease more than 10% points, serum blood urea nitrogen increase greater than 5 mg/dL, Ca levels less than 8 mg/dL, PaO2 less than 60 mmHg, base deficit greater than 4 mEq/L, and fluid sequestration less than 6 L. As per the original study of Ranson criteria, the presence of 3 or more variables was associated with 62% mortality [9]. Other commonly used scoring systems used in AP include Acute Physiology and Chronic Health Evaluation (APACHE II), Bedside Index of Severity in AP (BISAP), and Modified computed tomography severity index (MCTSI) [10]. The APACHE score was developed in 1981 and is confined to critically ill patients admitted to the ICU. The three key actors making up this scoring system are acute physiology scores, age scores, and chronic health scores, with point scores from 0-71 [11].

Pathophysiology

Hypocalcemia is used to evaluate the severity of pancreatitis as per Ranson's criteria. Various animal studies have indicated that hypocalcemia can be a negative prognostic indicator in pancreatitis patients. The exact cause of this remains unclear, however. Several theories have been proposed, including reduced basal levels of parathyroid hormone [12], normal parathyroid hormone levels but an inadequate response to hypocalcemia [13] [14], unresponsiveness of parathyroid hormone receptors [15] [16], hypomagnesemia [17] [18], and formation of extravascular calcium soaps [19] [20]. The theory of calcium soap formation, although widely accepted, has been challenged due to the insufficient quantities found in patients with severe pancreatitis [19]. Other potential causes such as abnormal levels of glucagon [21] [22], calcitonin [23] [24], or other hormones have been suggested, but these have not been consistently observed [16]. Hypocalcemia has also been linked to a decrease in protein-bound calcium due to hypoalbuminemia, but ionized calcium levels also decline [13] [15].

It is being observed that severe hypocalcemia in acute pancreatitis is more likely in patients with extremely high levels of TG in their blood [25]. The association between high TG levels and AP is reported to be around 20-30% [26] [27]. These patients may also have very high levels of free fatty acids (FFAs) in their blood, and in some cases, measurements exceeding 4 mEq/L, the highest being ever recorded in humans.

FFAs can bind to Ca, leading to the theory that FFA-calcium complexes could cause hypocalcemia. This idea is based on anecdotal human observations and in vitro experiments. One study showed that high plasma FFA levels caused a decrease in serum calcium concentration. However, it is still unclear whether the calcium bound to FFAs is effectively removed from the active ionized calcium pool or if it remains available in a complex form. High levels of Ca can also promote pancreatic injury; bile acids and ethanol can lead to the pathologic release of Ca from the endoplasmic reticulum. Persistent hypercalcemia can lead to continuous trypsinogen activation resulting in the vacuolization and death of acinar cells [4].

Imaging

In terms of imaging, abdominal ultrasound is recommended for patients with first presentation, and suspicion of AP, to look for the presence of calculi, gas, biliary dilatation, and fluid collection. CT scan allows for analysis of pancreatic morphology and to determine the extent and severity of the disease. MRI is used in cases of limitations or contraindications to CT scans and in patients with presentation of AP but negative CT results. CT scan reveals an enlarged pancreas with normal relative enhancement and regular peripancreatic fat, or ground glass opacity due to an inflammatory process. The presence or absence of necrotic tissue differentiates between acute edematous and necrotic pancreatitis [28]. The role of imaging in AP has substantially increased. The 2012 revision of the Atlanta classification signifies accurate characterization of collections that complicate AP. As per the Atlanta classification, the disease has been classified into two phases, an early phase lasting for a week and a late phase respectively. The late phase can extend for months and it is during this phase that the assessment of local complications with imaging may be crucial for management. Although a CT scan is the first choice in acutely ill patients with AP, an MRI is more appropriate for discovering necrotic debris, which may change the management [29].

Management

The management of AP is divided into three major areas which are hydration, pain control, and nutrition. The initial treatment for AP, as per ACG guidelines, consists of aggressive hydration, especially in the first 12-24 hours as it may have little benefit after that. Unless there are any renal or cardiovascular comorbidities, 250-500mL per hour of an isotonic crystalloid solution, preferably Lactated Ringer's, needs to be administered. Although there is conflicting evidence of the type of fluid to be used, Lactated Ringer's vs normal saline, limited evidence does suggest Lactated Ringer's reduces intensive care unit admissions and hospital stays [30]. Abdominal pain is the most common symptom in AP and opioids are considered effective and safe in controlling pain [31]. Previously it was believed that keeping the patient nil per os was the best way forward when dealing with AP in an attempt to decrease the stimulation of an already inflamed pancreas. Current evidence, however, demonstrates that early feeding may be the best approach. Oral feeding can be initiated within 24 hours if the pain is decreasing and there is no nausea, vomiting, or ileus [32]. Multiple varieties of diets have been proven efficacious including low fat, normal fat, and soft or solid consistency; starting with a clear liquid diet is not required [33]. In patients who cannot tolerate oral feeds, enteral instead of parenteral is recommended in moderately severe and severe AP, nasogastric and naso-enteral enteral feeding are comparable in safety and efficacy [34] [35]. The use of antibiotics when suspecting an extra pancreatic infection is highly recommended as these infections are associated with an increase in mortality.

Amongst the causes of AP, hypertriglyceridemia is one of the main culprits behind pancreatitis accounting for 1-35% of all cases of AP [36]. General measures to tackle hypertriglyceridemia as a cause of AP is to treat the pancreatitis with supportive measures and pain control, diet should be restricted in terms of fat until the TG levels are < 1000mg/dL, and discontinuing any medication that might be increasing TG levels [37]. Additional options in treating hypertriglyceridemia in patients with AP and signs of hypocalcemia, as in our patient, lactic acidosis, or worsening systemic inflammation include plasmapheresis and IV insulin administration. Insulin has several beneficial effects on TG metabolism and has been shown to help lower serum TG levels. This is achieved by increasing the activity of lipoprotein lipase (LPL). This enzyme accelerates the breakdown of chylomicrons and very low-density lipoproteins (VLDL) into glycerol and free fatty acids (FFAs). Additionally, insulin inhibits hormone-sensitive lipase in adipocytes, the enzyme responsible for releasing fatty acids from adipose tissue into the bloodstream. In cases of severe AP with high TG levels, the primary goal of insulin therapy is to counteract the stress-induced release of fatty acids from adipocytes, encourage the storage of TG within adipocytes, enhance fatty acid metabolism in insulin-sensitive cells, reduce peripheral insulin resistance, and primarily correct hyperglycemia. In studies involving mice, insulin has also been shown to reduce the severity of AP and improve recovery [38] [39]. For individuals with hypertriglyceridemia-induced pancreatitis, keeping TG levels below 500 mg/dL (5.6 mmol/L) can potentially speed up the process of clinical recovery [40]. In an experiment by Rattner et al., fatty acids were injected into rats to induce necrotizing pancreatitis; a time-dependent decrease in calcium was observed. The severity of pancreatitis correlated to the degree of hypocalcemia [41].

Hypocalcemia is a component of Ranson's scoring system used for evaluating the severity of pancreatitis. Multiple studies on animal models have demonstrated that low levels of calcium in the blood serve as an unfavorable indicator for the prognosis of individuals with pancreatitis [42]. There has not been any research conducted on the impact of correcting hypocalcemia in patients with pancreatitis, but studies performed in other groups of patients with hypocalcemia correction have not yielded beneficial results in favor of correcting hypocalcemia with parenteral calcium infusion. One study with liver transplant patients demonstrated that the likelihood of experiencing biochemical AP following transplantation was elevated in relation to the quantity of intravenous CaCl given during the pre-anhepatic phase and the surge in serum calcium within the first 2 hours after reperfusion of the liver graft [43]. Another study looking at septic patients in the ICU noted that the use of

IV Ca was linked to an elevated risk of death and a deterioration in organ function [44]. Calcium infusion may result in skin tissue death. When administering it intravenously, it should be exclusively through a central venous catheter. Severe hypocalcemia can have many associated life-threatening complications including, but not limited to, arrhythmias, seizures, tetany, and other neurological/psychological disorders. Patients with hyperphosphatemia should not receive calcium, as it can lead to the formation and buildup of calcium phosphate deposits in different tissues [4].

Conclusion

Various factors can affect the development and progression of acute pancreatitis. Aggressive fluid hydration, pain management, and early feeding when tolerable are current recommendations for management. Electrolyte derangements may not be able to be corrected unless the triggering mechanism is addressed. As with our case, the patient's hypocalcemia remained resistant to treatment until his TG levels had begun to normalize. Other electrolytes, such as magnesium and phosphorus, should also be monitored and managed as they may also be deranged in patients with a history of alcohol abuse. Currently, the administration of insulin to normalize TG levels is not part of current guidelines, but in our case, had a substantial improvement in his clinical course and recovery. Further investigations should be performed into the efficacy of treating hypertriglyceridemia-induced pancreatitis with such therapy to help prevent life-threatening complications.

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A Novel ASXL1 Variant in a Case with Bohring-Opitz Syndrome

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Abstract

We report a case of a child born with features consistent with Bohring-Opitz syndrome, an extremely rare genetic disorder, confirmed by genetic analysis. As with other previously documented cases, the patient ultimately required tracheostomy and percutaneous endoscopic gastrostomy for support. Whole exome sequencing was performed on himself and his parents; a novel de novo variant, NM_015338.6:c.1180dup (p.R394Pfs*16), in the Additional Sex Combs-Like (*ASXL1*) gene was identified in the patient.

Introduction

Bohring-Opitz syndrome (BOS) is an extremely rare and severe genetic disorder. Up until recently, only roughly 20 cases have been genetically confirmed worldwide.[1] Reported cases had several clinical features in common; these included a prominent metopic suture, hypertelorism, exophthalmos, cleft lip and palate, limb anomalies, difficulty feeding, and severe developmental delays. In almost 50% of cases meeting the clinical criteria of this syndrome, de novo pathogenic variants have been detected in the Additional Sex Combs-Like (*ASXL1*) gene.[2] The *ASXL1* gene encodes a protein belonging to the polycomb group and trithorax complexes family, suggesting that it plays a role in overall gene silencing and transcription regulation.[3] Whole Exome Sequencing (WES) has proven invaluable in identifying de novo germline pathogenic variants as the underlying cause of rare diseases.[4] We present a case of BOS, confirmed through the detection of a novel frameshift variant in the ASXL1 gene using WES. This discovery not only contributes to the understanding of BOS but also highlights the critical role of genetic analysis in diagnosing and managing this exceptionally rare genetic disorder.

Case

Proband is a seven-week-old boy, who was born via induced vaginal delivery at 37 weeks and 3 days of gestation due to severe fetal growth retardation. He was noted to have a low birth weight of 1.8 Kg with Apgar score of 3-7-9. He was the third child born to a 23-year-old Caucasian female; the other children were within normal limits. After delivery, the patient developed apneic events with persistent desaturations requiring continuous positive airway pressure (CPAP) placement. Chest X-ray and arterial blood gas were unremarkable. The patient was then admitted to the neonatal intensive care unit for close monitoring and further evaluation by otolaryngology with concern for obstructive sleep apnea.

On physical examination, the child was noted to have exophthalmos, nevus flammeus, posteriorly rotated ears, abundant hair on the body and forehead, prominent lips, retrognathia, elongated fingers, bilateral cryptorchidism (confirmed via scrotal ultrasound), bilateral overlapping third toes, a tuft of hair on sacral dimple, and was small for gestational age.

Figure 1: Photo depicting physical features of the patient that can coincide with BOS; exophthalmos, nevus flammeus, forehead hair, prominent lips, retrognathia.

Because of these phenotypical features, a genetics consultant recommended chromosomal microarray testing; results were within normal limits. WES of the patient and his parents showed that the patient had a de novo heterozygous frameshift variant, NM_015338.6:c.1180dup (p.R394Pfs*16), in *ASXL1*. According to the guideline for the interpretation of sequence variants,[5] this frameshift variant appeared to be classified as pathogenic due to a predicted null variant (PVS1), a variant absent in population databases (PM2), and a de novo variant (PM6), resulting in the diagnosis of BOS. Further questioning with the family revealed no obvious signs or evidence of other genetic disorders as well for at least 2 generations (Figure 2).

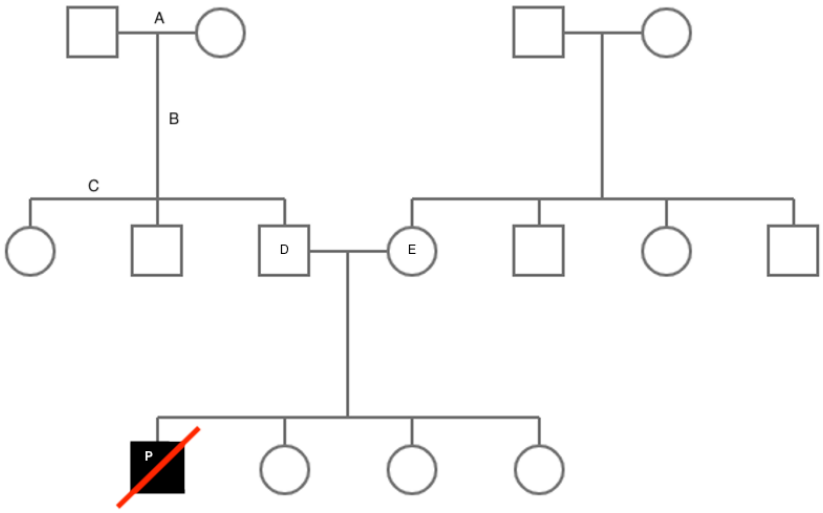

Figure 2: Figure depicting pedigree of deceased proband spanning two generations. Proband is indicated by the letter "P" (black box) with a red strike-through indicating that the patient is deceased. Line "A" indicates a relationship between 2 individuals. Line "B" indicates descent. Line "C" indicates siblingship. Male gender is represented as squares. Female gender is represented with circles. The letter "D" represents unaffected father. The letter "E" represents unaffected mother.

For further screening, an echocardiogram and renal ultrasound were performed, but the acute findings were negative. A brain MRI was unremarkable, and a spinal canal and spinal cord ultrasound was negative for spinal deformity. The patient continued to require mechanical ventilation ultimately requiring tracheostomy due to developing tracheomalacia. Because the patient had difficulty feeding, a percutaneous endoscopic gastrostomy (PEG) tube was placed to provide nutritional support (Figure 3). He was eventually discharged to a long-term care facility with a follow-up with genetics, urology, nephrology, and otolaryngology teams as well as surveillance renal imaging due to increased risk for Wilms tumor. Due to worsening oxygen requirements, he returned to the hospital shortly after. Imaging showed significant worsening of pneumonia. Tracheostomy suctioning and bag-valve oxygenation were initially attempted. Respiratory status continued to diminish and bradycardia worsened; cardiopulmonary resuscitation was initiated following an advanced cardiovascular life support protocol. Despite best efforts, the patient remained in pulseless electrical activity on the monitor and was pronounced dead approximately 25 minutes later.

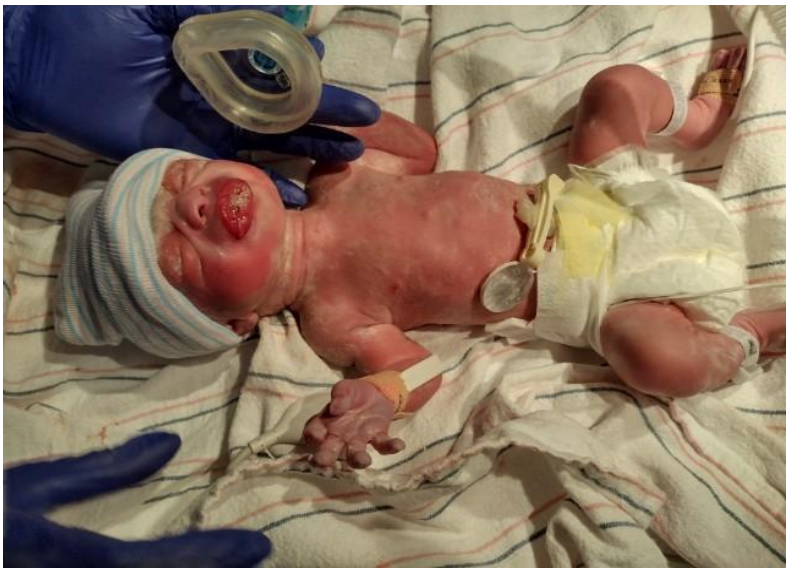

Figure 3: Figure depicting small for gestational age patient status post PEG tube placement for nutritional support.

Discussion

Clinical features of BOS are vast since the *ASXL1* gene provides instructions for proteins involved in regulating gene activity.[6] Craniofacial abnormalities can be one of the most prominent and obvious features present at birth.[7-8] They are associated with feeding difficulties; issues with latching causing poor oral intake can further exacerbate growth delays. Impedance in swallowing can also lead to complications such as gastroesophageal reflux disease and aspiration. Evaluation for possible interventions via fundoplication or gastric/gastro-duodenal tubes can also be made. These may help in decreasing reflux and aspiration risk as well as improve nutritional delivery. These interventions can help in the interim to provide the patient with adequate feeding until they are stable/strong enough for any kind of reconstructive surgery (if

warranted/indicated). As with our patient, difficulties in respiration and feeding can lead to the decision for placement of tracheostomy and PEG tube.

Intrauterine growth restriction is also an important complication affecting many of these infants and can be worsened by inadequate feeding and oxygenation. Respiratory issues can also arise secondary to the facial/pharyngeal deformities. Issues with oxygenation can lead to apneic events resulting in OSA. With this, interventions can be implicated to help temporize and circumvent specific problems. Sleep studies can be performed for further assessment for which appropriate supplemental oxygen can be indicated (i.e. CPAP).

Those with BOS often develop seizure-like activity varying from tonic-clonic to absence. Neurological work-up would be warranted pending the severity; this can include electroencephalogram monitoring and medical intervention. Brain imaging can be done to look for intracranial malformations as well. Hypoplasia or absence of the corpus callosum is one of the most common neurological defects; others, such as Dandy-Walker malformations, have also been noted.[1] Patients can be commonly started on anti-epileptic medications if there is concern for seizure-like activity. Intellectual disabilities are also associated for which there is no cure.[2] Close educational and language therapy can however help with development. Deformities to the jaw can also bring forth speech impediments and difficulties for which continued speech therapy can be of great benefit.[9]

In terms of cardiovascular complications, structural deformities such as defects and hypertrophy have been documented; these can include atrial septal defects, patent foramen ovale, dysplastic valves, etc.[10] These can lead to worsening of cardiac function. Roughly 1/3rd of reported cardiovascular deaths have been associated with apneic and bradycardic events.[1] An echocardiogram can be done following initial diagnosis to help better delineate the underlying structural pathology.[11] Ophthalmologic complications are common as well. These can range from exophthalmos, strabismus, and hypertelorism. Severe myopia has also been documented as well for which individuals would require corrective lenses at an early age.[1]

Musculoskeletal anomalies can result in the typical “BOS posture”. This presents as elbow flexion and ulnar deviation/flexion of the metacarpophalangeal joints and wrists.[12] Both hypo and hypertonia can both be observed in extremities with contractures of various joints (i.e. arms, fingers, knees, etc.). Physical deformities and muscle tone discrepancies can also be seen. These posturing complications would require routine exercises and stretching; continuous work with physical and occupational therapists would be ideal. For those who still find difficulty in ambulation and movement, support devices such as canes, special footwear, and wheelchairs may be required.

The associated genetic mutations can lead to a downstream effect of DNA methylation. The variations in hyper and hypomethylation at certain promoter sites of DNA show association with an acceleration of epigenetic age. In other words, an affected individual's genetic age may not correspond to their chronological age.[13] Hypermethylation of certain tumor suppressors is also of concern; the associated hypermethylation of the HOXA5 CpG site, for example, has been related to an increased risk of developing Wilm’s Tumor.[14] In one study, it was noted that there was roughly a 6.9% increased risk of Wilm’s Tumor with a median age of occurrence around 24 months.[15] Further imaging, such as renal ultrasounds, can be useful tools for evaluation.

Conclusion

BOS is devastating and current management is to address complications on an individual basis. As seen with our patient, pneumonia seems to be one of the most common causes of death in infants with a morbidity rate of up to 40%.[11] Other common causes include unexplained bradycardia and OSA.[16] With rapid advances in genetic sequencing, more definite etiologies for this disorder may surface which can lead to the development of better treatment options. At this time, a combination of surgical interventions, various therapy modalities, and adequate nutrition are the mainstay of treatment. Prenatal and genetic counseling are recommended to help inform families of the possible risks and outcomes of their newborns.

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An Unusual Etiology of Celiac Duodenopathy

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Abstract

Immune checkpoint inhibitors (ICPI) are an evolving therapy for treating various malignancies. However, as their use increases in medical management, the associated side effects become more prominent. These immune-related adverse events can include gastrointestinal complications and exacerbation of autoimmune disorders. Diarrhea is a commonly reported side effect in the literature, and colitis or enterocolitis are also frequently noted. Although current management is mainly symptomatic treatment, discontinuing immunotherapy is necessary if symptoms become severe. This case report presents a patient who started on Pembrolizumab therapy and subsequently developed abdominal pain and nausea. Endoscopic biopsy findings suggest Celiac duodenopathy; treatment with steroids and dietary modifications helped improve symptoms. It is important to note that ICPIs can induce autoimmune phenomena, including those affecting the small bowel. Therefore, a high index of suspicion is necessary when patients present with gastrointestinal symptoms during or after ICPI therapy. Close monitoring of patients on ICPI therapy is crucial to identify and manage any potential side effects promptly.

Introduction

Cancer cells have unregulated division as a characteristic. One mechanism by which they achieve this is through the downregulation of inhibitory ligands/receptors such as Cytotoxic T-lymphocyte-associated protein 4 (CTLA-4), Programmed Cell Death Receptor 1 (PD-1), and Programmed Death Ligand 1 (PD-L1), leading to unsuppressed proliferation [1]. Immune Checkpoint Inhibitors (ICPI) are effective anti-cancer drugs that enhance T-cell mediated killing of cancer cells by blocking these checkpoints. ICPIs may cause immune-related adverse events (irAEs). As their popularity and efficacy increase, the incidence of these adverse events becomes more prominent [2]. Currently, seven ICPIs targeting three different checkpoints are available: Ipilimumab and Tremelimumab for CTLA-4; Pembrolizumab and Nivolumab for PD-1; Atezolizumab, Avelumab, and Durvalumab for PD-L1 [3]. Diarrhea is a commonly reported gastrointestinal (GI) side effect of immunotherapeutic drugs [4]. It is essential to closely monitor patients on ICPI therapy for potential irAEs. Timely recognition and management of these adverse events are crucial to ensure patient outcomes.

Case Presentation

A 79-year-old male with metastatic melanoma to the left distal humerus, on therapy with Pembrolizumab, presented with nausea, vomiting, and fatigue. The patient reported a sweet taste with a dry mouth, hesitancy to swallow solids, loss of appetite, and weight loss over a week. The symptoms began when he started Pembrolizumab. Initial labs were notable for TSH of 59 mIU/L, otherwise unremarkable. The anti-thyroid peroxidase antibody was negative. The patient was started on Synthroid 50 mcg daily. He eventually underwent an esophagogastroduodenoscopy (EGD) for persistent symptoms. EGD showed diffuse mucosal edema and scalloping-like erosions in the duodenum, with severe esophagitis and mild gastritis. Duodenal biopsy revealed lymphocytic infiltration of the small bowel mucosa associated with atrophy and blunting of the villous border at the lumen of the bowel mucosa, suggesting celiac enteropathy. TTG and IgA antibodies were negative. The patient was treated with steroids and a gluten-free diet for suspected Celiac disease on biopsy findings resulting in improved symptoms.

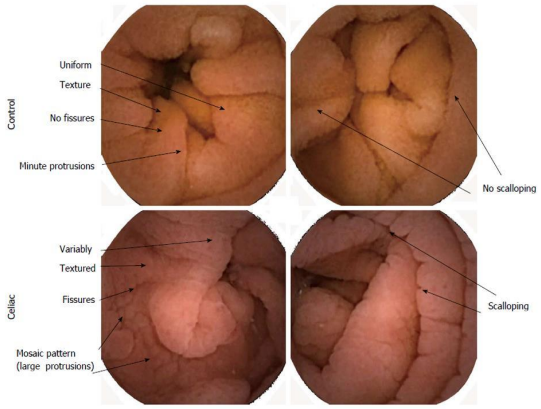

Figure 1: Figure depicting features of Celiac disease that can be observed on endoscopic visualization [5].

Discussion

Immunotherapeutic medication regimens have various indications, but one of great importance is treating malignancies. These drugs stimulate the body's immune system to attack cancer cells rather than directly targeting the tumor cells [4]. Unfortunately, as the body learns to attack these tumor cells, it may also attack healthy mucosa. Immune checkpoint receptors such as Cytotoxic T-lymphocyte antigen 4 (CTLA-4) and Programmed Cell Death 1 (PD-1) receptors inhibit T cell function. Monoclonal antibodies targeting these checkpoints can stimulate cell death in tumor cells, thus improving therapeutic outcomes. Targeted pharmacotherapy against these receptors has effectively treated various cancer subtypes [6].

Toxicities are related to autoimmune phenomena, termed "immune-related adverse events". Gastrointestinal (GI) side effects are well known to occur with the administration of ICPI. Colitis and enterocolitis have been found in roughly 15-20% of patients on these medications who underwent endoscopy [7]. Ipilimumab, for example, is a monoclonal antibody against CTLA-4. It is associated with diarrhea in 1/3rd of patients and colitis in 7-22%. By inhibiting the regulatory function of T-cells, local erythema and inflammation can result [4].

Celiac disease is another such noted symptom that has been reported in the literature. Whether the medication precipitates the disease or exaggerates a previously asymptomatic state, both CTLA-1 and PD-1 help regulate the disease's autoimmune nature; with their effects diminished, there is decreased disinhibition of the autoimmune antibodies leading to exacerbations of the disease [8]. One case in the literature reported a patient developing nausea, diarrhea, and weight loss after a week of Ipilimumab for metastatic renal cell carcinoma (RCC). Biopsies were positive for gastritis, duodenitis, erosions, neutrophilic cryptitis, and villous atrophy; these are some characteristic findings of Celiac disease [2].

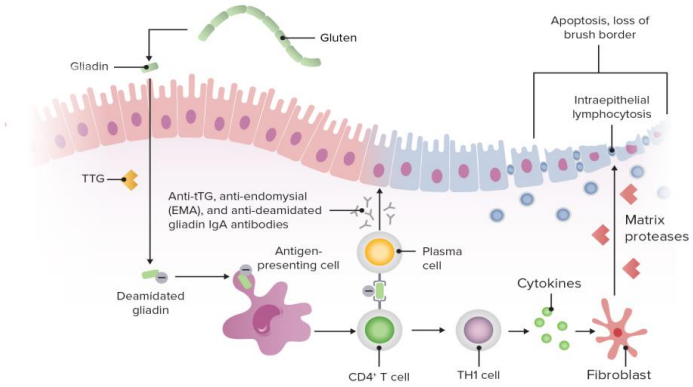

Figure 2: Figure depicting the autoimmune mechanism of Celiac disease resulting in brush border blunting [9].

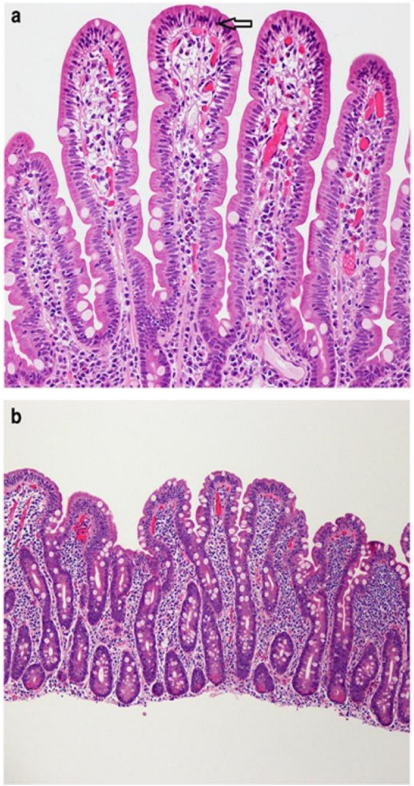

Figure 3: Typical histopathologic findings of villous atrophy and brush border blunting as associated with Celiac disease [10].

Pembrolizumab, a monoclonal antibody to PD-1 receptors, has been shown to potentiate antitumor responses in patients with advanced melanoma. To our knowledge, there have not been many specific associations between Pembrolizumab and Celiac disease, although T-cell and macrophage infiltration and antibody deposition have been hypothesized [11]. We suspect that using Pembrolizumab potentiated the clinical presentation of Celiac disease without positive serological evidence. One case was reported of a patient who developed abdominal pain and diarrhea after six months of Pembrolizumab therapy despite being concurrently treated with steroids. Biopsies were significant for erosions, lymphoplasmacytic inflammation, and elevation of intraepithelial lymphocyte count [12].

GI hemorrhage is another complication that could potentiate secondary to chronic inflammation. Very few cases of bleeding have been documented in the literature. One study noted a patient on Atezolizumab therapy for small-cell lung cancer who presented with hematemesis and abdominal pain. Emergent endoscopy revealed bleeding erosions and ulcers requiring clipping and cauterizations. Tissue biopsies showed eosinophils, lymphocyte infiltration, and plasma cells [13]. These patients should be closely monitored as the recurrent inflammation and bleeding may weaken the mucosa to the point of perforation [3].

Peripheral tolerance is part of the body's immune response to help prevent self-reactive T and B cell escape or activation. By inhibiting these checkpoints, there is the potential to exacerbate various autoimmune pathologies. Worsening hypothyroid symptoms, for example, have been noted after the immunomodulating regimen initiation [14].

Management

Interventions for patients who experience GI side effects may depend on the nature and location of the symptoms. Those who experience hematochezia and diarrhea, for example, may warrant colonoscopy. On the other hand, those who experience nausea, reflux, and dysphagia may benefit more from EGD. In both cases, biopsies are crucial in further evaluation. In one study, ~63% of patients who underwent colonoscopy for persistent diarrhea were found to have ulcerations and erythema [4].

Initial management can be as simple as targeting symptoms. If not severe, for example, diarrhea can be treated with antidiarrheals without having to discontinue the therapy. Medication discontinuation would be indicated when more severe, however, with episodes of greater than six watery bowel movements per day [4]. Gluten-free diets (GFD) have also decreased symptoms [2]. In one study, GFDs helped improve GI symptoms in patients with Celiac disease by up to 65.6% [15]. Further evaluation via fecal calprotectin and lactoferrin can help distinguish diarrhea and whether it has an infectious vs. inflammatory etiology, in which case appropriate therapy, for example, antibiotics vs. steroids, can be initiated [16] for those refractory to steroid therapy, Infliximab or fecal microbiota transplant may be used [3].

As various autoimmune disorders can be precipitated, cases have been known of worsening hypothyroid symptoms; treatment can be either initiation or appropriate dose adjustments of thyroid supplementation (as seen with our patient). Those with worsening acid reflux-type symptoms can also be treated with anti-emetics and Proton Pump Inhibitors (PPIs). Due to concern for ulcerations and bleeding, patients on immunomodulating medications should be counseled to minimize NSAID use.

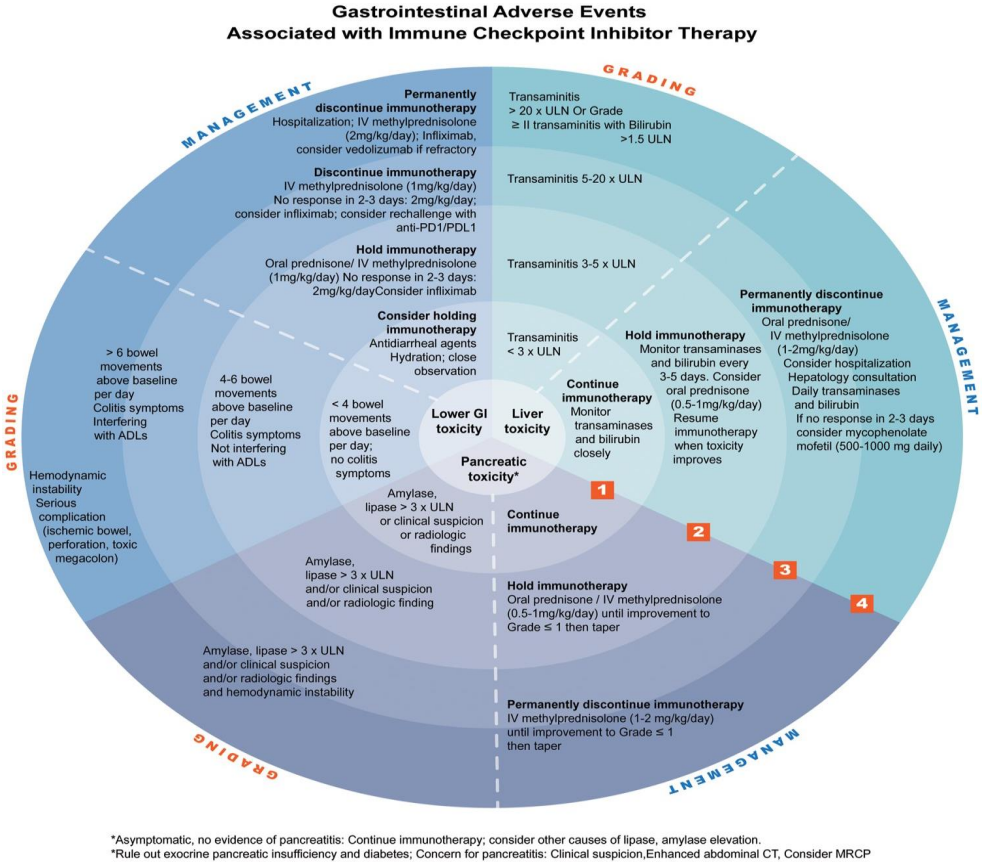

Figure 4: Figure depicting grading symptoms of various GI side effects with their associated recommended management strategy [16]

Conclusion

Immune checkpoint inhibitors have proven practical tools in managing patients with various malignancies. As their popularity increases, however, so does the occurrence of immune-related adverse events. Diarrhea is one of the most common side effects of ICPI treatment. The decreased cytotoxic regulation effect may exacerbate localized inflammation and autoimmune disorders complications. Management options for these adverse events include dietary modifications and symptomatic relief with antidiarrheals, while discontinuing immunotherapy is considered in severe cases. Celiac disease has been hypothesized to be an irAE; however, whether it is due to a new onset or worsening of a previously silent disease remains unclear. Although some cases have been overall associated, more studies need to be performed to confirm the correlation of these ICPIs with Celiac disease.

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Anaplasmosis Presenting as Septic Shock of Unknown Origin

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Abstract

Human ehrlichiosis and anaplasmosis represent acute febrile tick-borne illnesses manifesting with varied symptoms, ranging from mild febrile episodes to severe multi-organ dysfunction. This case report highlights a 77-year-old Caucasian female residing in Northeast Pennsylvania, who initially sought medical attention for confusion and abdominal pain during the summer. She was initially diagnosed with a urinary tract infection and commenced on cefdinir. Subsequently, her condition deteriorated, necessitating hospitalization.

Laboratory investigations revealed elevated transaminases, acute kidney injury, troponemia, and thrombocytopenia. Despite initial interventions, including intravenous fluids, the patient progressed to acute respiratory failure, requiring positive pressure ventilation. Consequently, she was transferred to a tertiary care facility. Notably, a peripheral blood smear exhibited neutrophilic intracytoplasmic inclusions.

Empiric therapy with intravenous doxycycline, enteral azithromycin, and atovaquone via nasogastric tube was initiated, resulting in rapid clinical improvement. Peripheral blood PCR confirmed the diagnosis of anaplasmosis, ruling out babesiosis. Consequently, azithromycin and atovaquone were discontinued, and the patient completed a 10-day course of doxycycline. This case underscores the critical importance of promptly diagnosing tick-borne infections in individuals with nonspecific symptoms, particularly in geographically endemic regions.

Key words: *Tickborne, Transaminitis, Intracytoplasmic inclusions*

Introduction

The rise in human infections attributable to ehrlichiosis and anaplasmosis has become more pronounced, coinciding with the expansion of animal reservoirs and tick vectors in regions densely populated by humans. This case report delineates an instance of anaplasmosis, wherein the clinical presentation mimicked sepsis and manifested as multiorgan failure involving myocarditis, renal failure, respiratory failure, and encephalopathy. Vigilant management, incorporating broad-spectrum antimicrobial coverage with doxycycline, azithromycin, and atovaquone, yielded prompt and substantial clinical amelioration. This case underscores the escalating relevance of these tick-borne illnesses and emphasizes the efficacy of comprehensive therapeutic strategies in mitigating their clinical impact.

Case

A 77-year-old Caucasian female residing in Northeast Pennsylvania presented to her primary care physician in June with a three-day history of urinary frequency, urgency, chills, generalized body aches, anorexia, intermittent disorientation, altered balance, and fever that commenced on the day of presentation. Her medical history was notable for hypertension, type 2 diabetes mellitus, osteoarthritis, restless legs syndrome, and anxiety.

During the clinic visit, vital signs indicated a fever (102.5˚F) and tachycardia (119 beats per minute), while the rest were unremarkable. Clinic labs revealed transaminitis (AST 222 U/L, ALT 144 U/L, ALP 182 U/L), acute kidney injury (BUN 20, Cr 1.1, eGFR 53 ml/min), hyponatremia (Na 132 mmol/L), and thrombocytopenia (platelets 99 K/uL). Urinalysis showed 4+ ketones, 3+ protein, and 1+ leukocyte esterase. Blood culture and viral panel were negative, and the patient was initiated on Cefdinir 300 mg bid.

Two days later, the patient's son brought her to a local hospital due to worsening confusion, vague abdominal pain, and nausea. A head CT scan was negative, but labs indicated worsening transaminitis, direct hyperbilirubinemia, escalating AKI, hyponatremia, and thrombocytopenia. Following a fluid bolus, she developed pulmonary edema, leading to acute hypoxic respiratory failure. Broad-spectrum antibiotics, stress steroids, and furosemide were administered. Due to worsening condition, she was transferred to a higher-level care facility, where a pending peripheral blood smear revealed neutrophilic intracytoplasmic inclusions suggestive of anaplasmosis.

Upon transfer, vital signs indicated hypotension, tachycardia, tachypnea, and a GCS of 8. Labs showed metabolic acidosis and elevated lactate. Chest X-ray revealed pulmonary edema and left basilar opacity. Infectious disease department-initiated empiric treatment with IV doxycycline, enteral azithromycin, and atovaquone. The patient was admitted to the ICU, and hematology was consulted for elevated LDH and thrombocytopenia. Peripheral smear ruled out intravascular hemolysis. After

24 hours, peripheral blood PCR confirmed *Anaplasma phagocytophilum*. Azithromycin and atovaquone were discontinued, and doxycycline were continued.

During the hospital stay, cardiology and nephrology monitored the patient for acute systolic heart failure and stage 3 Acute Kidney Injury (AKI). Aspirin and beta blockers were initiated, diuretics administered for fluid overload, and lisinopril withheld due to kidney injury. Troponins trended down, and NT-Pro BNP remained elevated. On discharge, the patient was started on carvedilol, and lisinopril was restarted. Nine months later, a repeat TTE showed recovery of EF to 36%.

Discussion

The inaugural documentation of human anaplasmosis dates back to 1986, delineating a patient's clinical presentation marked by fever, hypotension, confusion, acute renal failure, coagulopathy, and gastrointestinal hemorrhage (1). Both anaplasmosis and ehrlichiosis are zoonotic tick-borne diseases caused by obligate intracellular gram-negative bacteria. Human granulocytic anaplasmosis (HGA) and human monocytic ehrlichiosis (HME) denote the white cell line involvement in each infection (2). Symptoms are typically nonspecific, encompassing myalgia, tendon pain, soft tissue tenderness, right upper quadrant pain, slumped posture, hepatomegaly, diaphoresis, muscle spasms, elevated liver enzymes (intrahepatic pattern), and, notably, a diminished white cell count (3,4).

A presumptive diagnosis of HME/HGA may be established in patients exhibiting a compatible systemic febrile illness with pertinent epidemiologic exposures and an absence of a clear alternative explanation. For instance, outdoor exposure in an endemic area during spring or summer, coupled with a febrile illness and isolated leukopenia and/or thrombocytopenia, provides substantial circumstantial evidence for anaplasmosis. The diagnosis should not be dismissed even in those with a history of HGA or HME, given that prior infection may not confer enduring immunity, as evidenced by cases with recurring episodes spaced years apart (5).

Recognizing the potential for swift progression to serious illness, the Centers for Disease Control and Prevention (CDC) advocates for initiating antimicrobial treatment promptly upon clinical suspicion, even before laboratory confirmation (6). When HME or HGA is suspected, blood samples should be subjected to polymerase chain reaction (PCR), serology, and peripheral blood smear examination. In endemic regions for both HME and HGA, separate PCR and serologic tests should be conducted for each organism.

PCR, widely employed, offers acute diagnostic results, though a positive result confirms infection, while a negative result does not definitively rule out disease. Although serology can provide a definitive diagnosis, its utility in the acute setting is limited. If PCR yields negative results with continued suspicion, a second serologic test should be administered two to four weeks later, with a fourfold rise in antibody titers between acute and convalescent tests confirming the diagnosis. Microscopic examination of a blood smear, though less sensitive than PCR, is highly specific and can offer rapid results. Buffy coat examination, while enhancing sensitivity, is labor-intensive and infrequently performed (6).

Hospitalization is required in over 50 percent of reported cases (7). The broad differential diagnosis for HME and HGA encompasses infectious and noninfectious etiologies. Coinfection with other tick-borne infections should also be considered. The clinical presentation aids in narrowing the differential diagnosis:

1. Individuals with severe sepsis or septic shock – Resembling severe HME and HGA infections, broad-spectrum antimicrobial therapy should be initiated until a definitive microbiologic diagnosis is established for bacterial infections such as bacteremia, acute cholangitis, community-acquired pneumonia, urosepsis, and meningoencephalitis.
2. Individuals with fever plus leukopenia, thrombocytopenia, and/or abnormal aminotransferases – Lab findings akin to HME and HGA may also be attributed to mononucleosis-like illnesses caused by Epstein-Barr virus, cytomegalovirus, and acute HIV. Other viral infections with similar syndromes include hepatitis A, B, and C.
3. Other arthropod infections – Consideration should be given to Rocky Mountain spotted fever (RMSF) and babesiosis, which may share symptoms and lab findings with HME and HGA.
4. Noninfectious illnesses – Conditions such as thrombotic thrombocytopenic purpura (TTP), hemolytic-uremic syndrome (HUS), *Hemophagocytic lymphohistiocytosis* (HLH), hematologic malignancy, and drug reactions (e.g., to trimethoprim-sulfamethoxazole and chemotherapeutic agents) may mimic the clinical presentation of HME and HGA.

In our patient, the initial indication of anaplasmosis was the discovery of neutrophilic intracytoplasmic inclusions in the peripheral smear conducted at the first facility, later confirmed by a positive PCR for *Anaplasma phagocytophilum* in the second facility. Empiric treatment with vancomycin and piperacillin/tazobactam failed to yield clinical improvement, and it was only after the initiation of doxycycline that the patient exhibited improvement, resolving the associated multisystem organ impairment.

Conclusion

Human granulocytic anaplasmosis (HGA) and human monocytic ehrlichiosis (HME) represent tick-borne maladies characterized by predominantly nonspecific symptoms. Severe cases may precipitate multiorgan system failure, rendering diagnosis challenging owing to the lack of distinct clinical manifestations. Sustaining a heightened clinical suspicion, particularly in endemic regions and during the spring and summer seasons, proves pivotal for expeditious diagnosis and intervention. The primary therapeutic modality remains doxycycline, supported by peripheral smear findings and a positive polymerase chain reaction (PCR). Failure to institute timely treatment may culminate in the progression of HGA/HME to multisystem organ failure, ultimately resulting in mortality.

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Unraveling the Dynamic Progression: Lyme Carditis Transforming from AVB Type 1 to Complete Heart Block

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Abstract

This case report describes the presentation of a 47-year-old female living in Pennsylvania who presented with a two-week history of palpitations associated with episodes of pre-syncope. Initially, the patient's electrocardiogram (EKG) showed a heart rate in the 90s with type 1 atrioventricular (AV) block; however, once admitted, her EKG revealed complete heart block. This case highlights the importance of considering vector borne diseases as a potential cause of heart block in patients with relevant exposure and the significance of vigilant history-taking, especially in regions endemic for tick-borne diseases.

Introduction

Lyme disease, resulting from Borrelia burgdorferi infection transmitted via tick bites, is the most common tick-borne ailment in the US. Typical symptoms encompass fever, headache, fatigue, and the distinctive *Erythema migrans* rash [1]. If left untreated, it can lead to serious complications, including Lyme carditis, where the bacteria invade heart tissues, disrupting the heart's electrical system. This invasion can cause symptoms like dizziness, fainting, shortness of breath, palpitations, or chest pain [2]. Lyme carditis occurs in 1% to 10% of Lyme disease cases [3], adding to the challenge of managing its fluctuating nature, requiring medical intervention to minimize disease duration and cardiovascular risks [4] [5] [6] [7]. Within cardiology, Lyme carditis manifests as an atrioventricular (AV) conduction abnormality [2] [8], disrupting the typical conduction sequence of a P wave followed by a QRS complex [8]. AV block, a delay in impulses from atria to ventricles, can stem from causes like coronary artery disease, congenital heart defects, inflammation, or certain medications [8] [9]. There are four types of AV blocks: first degree, Mobitz I, Mobitz II, and third-degree block, each varying in severity and conduction pattern [8] [9][12]. Proper identification and management of AV blocks, especially in Lyme carditis cases, are vital to prevent complications [10] [6] [7]. This study explores Lyme carditis intricacies, including clinical manifestations, diagnosis, and management, addressing challenges in cardiological contexts.

Case

A 47-year-old female residing in Pennsylvania, USA with no remarkable PMH presented to the emergency department with a two-week history of palpitations, described as a sensation of rapid and irregular heartbeats. These palpitations were associated with periods of feeling lightheaded and nearly fainting. She reported no chest pain, shortness of breath, or other symptoms. The episodes occurred at rest and were paradoxically better with physical activity. Over the two weeks, she noted a progressive increase in the frequency and severity of these episodes. On initial evaluation in the emergency department, the patient exhibited stable vital signs including a temperature of 98.2°F, respiratory rate of 18 breaths per minute, heart rate of 82 beats per minute, and blood pressure of 124/61 mmHg. The initial EKG indicated first degree AVB. Given the concerning EKG findings and recurrent pre-syncope, she was admitted to the telemetry unit for further evaluation and management. While in the hospital and within hours of admission, the heart rate dropped to 57 beats per minute, and complete AVB was observed on the EKG, accompanied by a decrease in blood pressure to 114/51 mmHg and later to 99/43 mmHg. Laboratory investigations, including troponins, electrolytes, and thyroid levels, remained within normal limits. An echocardiogram revealed moderate concentric left ventricular hypertrophy without valvular abnormalities or changes in ejection fraction.

Considering her history, exposure status to ticks and the negative tests for other possible contributors to her AV block [11], serologic testing for Lyme disease was performed and she was started on empiric intravenous antibiotics with ceftriaxone [2] [4] and received treatment in the ICU with dopamine infusion for symptomatic bradycardia which needed to be uptitrated from the start dose of 5mcg/kg/min to 15mcg/kg/min to achieve adequate heart rate control [12]. Transvenous pacemaker placed on standby [3] [11]. After a 3-hour infusion of dopamine and the initiation of ceftriaxone therapy, the heart rate improved to 60 beats per minute. Over the course of 7 hours with continuous therapy, the heart rate increased to 80 beats per minute, but persistent complete AVB with premature ventricular contractions (PVC) and a junctional escape rhythm was noted. Subsequently, after one day of antibiotic therapy, the heart rate stabilized at 75 beats per minute with a return to first-degree AVB on the EKG. On the second and third days, the heart rate further improved to 78 beats per minute with consistent first-degree AVB on the EKG. Serologic testing for Lyme returned positive for Borrelia antibodies via enzyme-linked immunosorbent assay findings (IgG: 4.69 (0.00–0.90) and IgM: 5.17 (0.00–0.79) confirming the diagnosis of Lyme disease-induced complete heart block [13]. Her clinical condition improved after 3 doses of Ceftriaxone [2] [3] [5] with follow-up EKG demonstrated reversal from complete heart block to baseline EKG of AV1 block [4]. Patient was subsequently transitioned to oral doxycycline to complete treatment course [2] as an outpatient.

(A) First Degree AVB with PR interval of 384 ms (day 1). **(B)** Complete Heart Block with dissociation of Atrial and Ventricular contractions (Day1). **(C)** Third-degree AVB with a junctional escape rhythm (day 1) **(D)** First Degree AVB with PR interval of 320 ms (Day2). **(E)** First Degree AVB with PR interval of 240 ms (Day 3). AVB = Atrioventricular Block

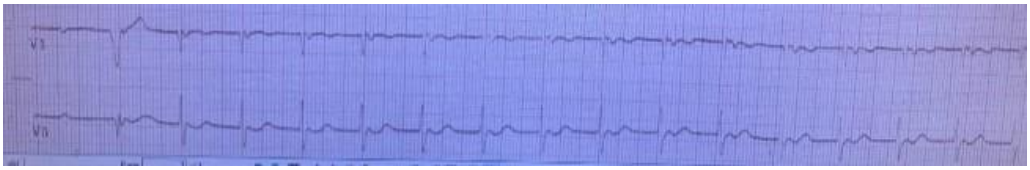
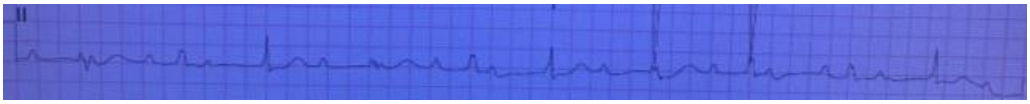


FIGURE 1:



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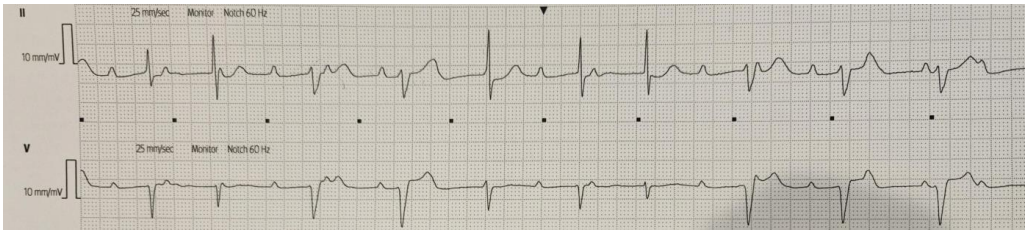

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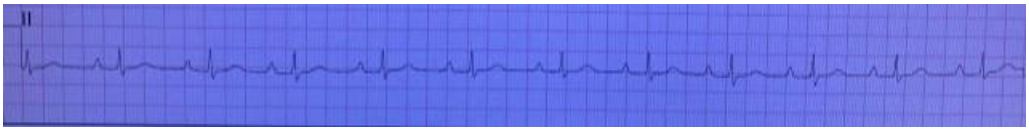

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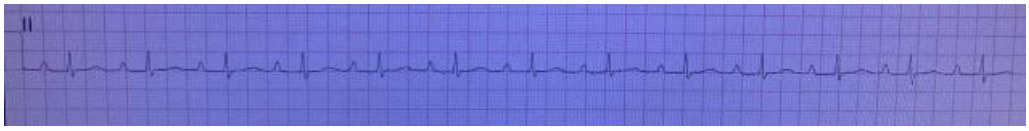

FIGURE 5:

Discussion

This case underscores the importance of considering tick borne diseases as a potential cause of complete heart block in patients with a history of tick exposure, even in the absence of other symptoms [3]; especially in the absence of other more obvious causes such as ischemia and known arrhythmia [6]. The primary identifiable sign of Lyme carditis is the atrioventricular (AV) conduction block. This block can swiftly shift in severity, ranging from first degree AV block to more severe forms and occasionally reverting back within minutes. [6] [14] When Lyme carditis is strongly suspected and other causes are ruled out, it is crucial to initiate antibiotic treatment promptly, preferably with ceftriaxone especially with high degree blocks [2]. If the patient shows symptoms, interventions like pacing, dopamine, or epinephrine, alongside antibiotics, should be considered. Early recognition and intervention are vital in managing Lyme carditis [14]. Lyme carditis diagnosis relies on serology to aid diagnosis and treatment, Besant and colleagues devised the Suspicious Index in Lyme Carditis (SILC) score, boasting 93% sensitivity. This score evaluates the likelihood of new-onset AVB being due to Lyme carditis based on CO-STAR factors: constitutional symptoms, outdoor activity in endemic areas, male sex, tick bite, age under 50, and rash (EM). Patients with high-degree AVB and SILC score > 2 should undergo Lyme serology and begin antibiotic treatment while awaiting results [10].

Conversely, those with SILC score ≤ 2 and high-degree AVB should receive standard AVB treatment without antibiotics [17]. While serology isn't usually needed for the latter, healthcare teams might opt for testing based on unique case circumstances [17]. Yeung and Baranchuk outlined a thorough cardiac testing protocol for Lyme carditis patients, encompassing both pre- and post-discharge assessments. After the restoration of 1:1 AV conduction, the temporary pacemaker can be removed. Prior to discharge, a stress test is recommended to assess the stability of AV conduction and decide on the necessity of a permanent pacemaker. Furthermore, all Lyme carditis patients should have an outpatient ECG performed 4 to 6 weeks after discharge to detect any rhythm or conduction abnormalities [17]. Finally, to minimize the risk of Lyme disease, it's crucial to adopt preventive measures such as wearing appropriate clothing and using insect repellent when in wooded areas or regions with high Lyme disease prevalence. Additionally, individuals at risk of Lyme disease should consider prophylactic treatment with a single dose of doxycycline (200mg) under specific conditions. All the following should be met in order to achieve the greatest benefit from prophylaxis- tick is identified as an engorged deer tick and has been attached for a minimum of 24 hours, individuals live in areas where Lyme disease prevalence among ticks exceeds 20%, treatment commencing within 72 hours of tick removal, and there are no contraindications to using doxycycline. Implementing this approach, even by primary care physicians, significantly reduces the risk of disseminated Lyme disease [9].

Conclusion

This case highlights the critical importance of considering tick borne vectors as a potential cause of palpitations and syncope, particularly in patients residing in regions endemic for tick-borne diseases like Pennsylvania. Lyme carditis, an uncommon but serious manifestation of Lyme disease, exhibits a propensity for rapid progression, with first-degree heart block transitioning to complete heart block in a matter of minutes to hours. Therefore, suspicion in at-risk population should prompt evaluation for Lyme disease and starting empiric antibiotic therapy in a timely manner. The management of Lyme carditis involves a two-fold approach: controlling the heart rate with temporary pacing and/or medications as necessary and initiating empiric antibiotics. Timely administration of antibiotics targeting Borrelia Burgdorferi can be curative, as it not only addresses the infection but can also reverse the heart block, preventing the need for long-term pacing. Consequently, maintaining a high index of suspicion for Lyme disease in patients with cardiac conduction abnormalities can significantly impact their prognosis and improve clinical outcomes.

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The Mandate of Nutritional Courses for Medical Students: A Consideration

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Abstract

Unhealthy lifestyle choices and eating habits are the primary factors contributing to chronic diseases. Many diseases are associated with obesity and an unhealthy lifestyle. An unhealthy lifestyle has contributed to nearly half of U.S. deaths from heart disease, stroke, and type 2 diabetes. Modifications in lifestyle choices can significantly decrease coronary artery disease (CAD), stroke, diabetes, and colon cancer. A recent statistic suggests that although 94 percent of physicians recognize the importance of diet, only 14 percent felt sufficiently trained in the topic [1].

Physicians possess a critical deficiency in nutrition knowledge, and there exists a gap in medical education pertaining to nutrition. A study reflected only 40 medical schools in the U.S. required classes on nutrition, and medical students on average received only 23.9 contact hours of nutrition instruction during medical school [2]. The same study reflected that 88 percent of instructors expressed the need for additional nutrition instruction at their institutions. This lack of knowledge does a disservice to physicians, medical students, and patients.

Nutritional education is noticeably insufficient, if it exists at all, both in medical schools and among practicing physicians [3]. Based on the studies, it is clear that knowledge in this area is insufficient. Therefore, the purpose of this literature review is to reveal the lack of nutrition education in medical schools, as well as review the importance of mandating it.

Keywords*: Nutrition, Medical school, Mandating courses*

Correlation between lifestyle modification and diseases

Maintaining a healthy lifestyle and adopting proper eating habits can offer protection against numerous chronic noncommunicable diseases and can reverse or even cure some chronic diseases. Requiring education on nutrition in medical school would ensure that every physician receives exposure to this topic before entering residency. It is crucial to initiate this education during medical school, prior to students starting their clinical rotations and becoming doctors.

Physicians have a critical deficiency in nutritional knowledge, and there exists a gap in medical education pertaining to nutrition. A study reflected only 40 medical schools in the U.S. required classes on nutrition, and medical students on average received 23.9 contact hours of nutrition instruction during medical school [2]. The same study reflected that 88 percent of instructors expressed the need for additional nutrition instruction at their institutions. Nutritional education is noticeably insufficient, if it exists at all, both in medical schools and among practicing physicians [3].

The prevalence of obesity in the United States was 42.4% in 2017 [4]. Obesity contributes to many diseases and disease processes. It is important to recognize that modifications in lifestyle choices can significantly decrease coronary artery disease (CAD), stroke, diabetes, and colon cancer [5]. Diet-related conditions have led to nearly half of U.S. deaths from heart disease, stroke, and type 2 diabetes [6]. As a result, obesity is indirectly one of the leading causes of death in the United States. Healthcare professionals lacking sufficient knowledge about nutrition's role in disease lose out on a treatment modality for addressing, preventing, and potentially reversing chronic conditions.

Can doctors help their patients with nutritional information if they do give the correct nutritional advice? It has been found that doctors can improve their patients' dietary habits by providing the proper nutritional advice and education [7][8]. Moreover, the food humans put into their bodies directly impacts their overall health. Doctors can provide successful nutritional advice without being a nutritionist. The major challenges and barriers for doctors providing nutritional advice are contested space and time constraints. On average, students received 23.9 contact hours of nutrition instruction during medical school [9]. Only 40 schools required the minimum 25 hours recommended by the National Academy of Sciences. Most instructors (88%) expressed the need for additional nutrition instruction at their institutions [9].

The amount of nutrition education in medical schools remains inadequate [9]. The limited nutritional information taught in medical schools and limited continuing medical education in nutrition does a disservice to patients. An unhealthy lifestyle and poor diet have been linked to numerous chronic diseases. A poor diet is either an insufficient intake of essential nutrients or an imbalanced distribution of these nutrients. Such diets often consist of high quantities of sugars, saturated fats, and heavily processed foods. Therefore, a proper diet could be used in conjunction with medications to improve patients' health. Unhealthy eating habits and lifestyle choices are associated with chronic diseases, yet physicians frequently lack the necessary knowledge and resources to offer patients accurate information and education on proper nutrition and lifestyle modifications.

Lack of nutritional education

Despite limited contact hours in medical school, it was found that doctors often overstated their knowledge regarding nutrition [10]. In a study done in 2011, Parker concluded that most physicians overstated what they knew and many did not know much at all [10]. The issue is there are no governing rules regarding the hours required or courses needed in nutrition for

a medical student or a physician. Medical schools dedicate less than 25 hours to this subject over five years, with some schools completing zero hours [9].

Although nutrition plays an important role in our health, education regarding this topic is notably insufficient. It is clear that physicians' knowledge in this area is lacking [1]. In a recent study, Canadian medical students agreed they would prefer if their undergraduate programs dedicated more education to nutrition [11]. Physicians may also be more inclined to eat healthier themselves if they knew more about nutrition. There is evidence that if doctors practice healthier habits themselves, they are more likely to counsel their patients about those habits [12]. Lastly, education leads to increased awareness. If doctors knew the real effects of poor nutrition, they may advocate more for lifestyle modifications rather than just prescribing medication. If doctors are not fully aware of the extent of diet-related diseases and preventative-based medicine, they cannot transfer this message effectively to their patients. 94 percent of physicians recognize the importance of diet, although only 14 percent felt trained and knowledgeable enough to offer this to their patients [1]. An evident gap exists within the healthcare system and medical school curriculum concerning nutrition education. There should be a heightened emphasis on promoting awareness of preventive medicine knowledge.

The deficiency in physicians' understanding of nutrition primarily arises from inadequate instruction in medical school and a lack of mandatory post-graduate education on the topic. Medical schools often do not offer mandatory courses on nutrition. The courses tend to focus on symptoms, diagnoses, and treatment of disorders and diseases, often disregarding aspects of prevention and proactive management. This is a probable explanation for physicians' limited knowledge in the field of nutrition.

Alternative perspective: Opposing Mandated Nutritional Education

Another question is, even if doctors did have adequate nutritional training, do they have time to deliver this information? If extra time is required, how would this impact physicians who already have limited time with their patients? It is important to always explore opposing views. It can be an argument that physicians are not nutritionists; hence why there is a separate field and degree for this altogether. On the contrary, physicians do have a duty to act in the best interest of the patient and a poor diet can directly impact patients' well-being [13]. A patient's unhealthy eating habits can directly influence their well-being [13]. While physicians may not have control over their patients' actions, it remains imperative that we educate patients about the impact of their diet choices on their health.

The task of distinguishing between "healthy" and "unhealthy" foods is challenging due to the constantly changing and evolving nature of nutritional information. The vast amount of nutrition-related knowledge can be overwhelming, making it challenging to design a course that remains current given the rapid evolution of this field. Nutrition is intricate, and simply advising patients to consume more fruits and vegetables falls short of addressing the complexity. Often, there is no single, universally applicable solution. Customizing nutrition plans for patients is a time-intensive process, typically requiring more time than the standard 15-minute duration of a general practice appointment [14].

80% of physicians across all specialties already report being fully occupied or overextended [15]. This situation underscores the potential benefit of referring patients to specialized nutritionists or dieticians. These professionals possess targeted expertise in this area. Obtaining a dietetics degree entails completing either a 4-year Bachelor's program or a 2-year Master's program, which cultivates proficiency and enables accreditation for delivering dietary counseling [14]. Consequently, it becomes comprehensible why doctors express reservations about their competence and knowledge in providing effective, person-centered nutrition counseling [16]. On the contrary, it has been shown that medical students want more education on nutrition and regardless of time constraints, it is a topic that needs to be discussed with patients.

Conclusion

The amount of nutritional education in medical schools can vary based on various factors, including curriculum design, available resources, and historical emphasis on other medical topics. Traditional medical education has historically been focused on treating and diagnosing diseases rather than preventative measures. Medical school is already packed with an immense number of topics that need to be covered. Therefore, nutritional education may not always be prioritized due to these constraints. As a counterargument, the field of nutrition can be complex, with evolving recommendations. Therefore, it may be difficult to mandate courses in residency, therefore it should be taught in medical school.

Nutrition plays a pivotal role in both a physician's and a patient's health. Health can be further defined as a state of "physical, mental, and social well-being". Physicians are defined as professionals who are concerned with "promoting, maintaining, and restoring health". Therefore, physicians should have more education courses on how a proper diet can promote and maintain health. In many countries, funding, time, and resources are skewed towards rewarding 'treatment' with little or no financial reward for 'prevention'.

In closing, medical schools should have mandated education regarding nutrition and the direct impact it has on our health. Medical students should receive courses on nutrition before being in a clinical setting. While formal education on nutrition in medical school may be limited, many medical professionals pursue additional nutrition education through continuing medical education courses.

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