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The Development of an Educational and Screening Instrument for Attention Deficit Hyperactivity Disorder in a Pediatric Residency Program

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Abstract

Background. Numerous indices determine the presence of ADHD, but no screening instrument exists which would direct a more detailed evaluation that is designed specifically for pediatric residents. This article presents the development and assessment of a screening instrument for the assessment of Attention Deficit Hyperactivity Disorder (ADHD) in a pediatric residency program.

Methods. Pediatric resident physicians were assessed by survey regarding their comfort levels in taking an ADHD assessment before and after the introduction of a screening instrument. The Pediatric Residency Checklist (PRC)/ADHD was developed specifically for educational use. Its reliability and validity was assessed for its use by residents as a screening tool for ADHD.

Results. At a PRC/ADHD score of 10 or higher, 88.9% of patients were classified correctly as having ADHD or not having ADHD. The sensitivity for ADHD diagnosis was 94.4% and the specificity was 81.5%. The positive likelihood ratio using was 5.1. The negative likelihood ratio was 0.07. The odds ratio of predicting an ADHD diagnosis was 40.4, controlling for age and gender. Residents were more comfortable in their assessments and treatment of ADHD after instruction in the application and use of the Pediatric Resident Checklist/ADHD.

Conclusions. The results showed the viability of the PRC/ADHD as a screening device for ADHD, especially in the day-to-day operations of a pediatric residency clinic. The addition of the Pediatric Residency Checklist/ADHD benefitted residents in terms of increased comfort levels in the assessment and treatment of ADHD. *KJM 2008; 1(4):70-80.*

Introduction

Approximately 15% to 18% of children in the United States have developmental or behavioral disabilities.[1] Of these disorders, Attention Deficit Hyperactivity Disorder (ADHD) is one of the most commonly diagnosed, accounting for 30%-40% of all referrals to child guidance clinics.[2] Additionally, the different presentations of ADHD make it difficult for even experts in the field to define let alone diagnose. In the past, clinicians have characterized the disorder as "organic driven-ness", "minimal brain dysfunction", and more recently Attention Deficit Hyperactive Disorder.[3]

Although actual prevalence of the disorder is debated, most current research suggests that 2-5% of school-aged children have well defined and pervasive symptoms.[4] ADHD may be both under- and over-diagnosed, leading to concern with reference to how exactly we approach its diagnosis in the medical field.[5] Finally, there may be gender issues that result in both under- and over-diagnosis for girls and boys respectively.[6] Clearly, there is an emerging need in pediatric medical education for positive guidelines in making and responding to an ADHD diagnosis.[1]

Generally, pediatricians are very familiar with the process of screening in the course of their work with children and adolescents. However, most of the comfort level for such

screens remains at the biomedical level as opposed to the behavioral level. Although multiple behavioral screening instruments with adequate reliability and validity statistics exist, systematic evaluation for behavioral health issues is not common considering its importance to the child and family for overall health.[7]

Many ADHD evaluation instruments are available. However, few are designed to allow resident physicians to recognize common developmental presentations of ADHD, determine the need for additional assessment, and know what to do after an diagnosis has been made. Further, resident physicians often arrive at their respective residency programs ill equipped to deal with the demands of an ADHD assessment.

Special care needs to be given concerning extended assessment of ADHD. Resident physicians are expected to respond quickly and efficiently but may have little or no real experience with an ADHD child in their exam room. More intensive evaluation of ADHD typically involves behavior rating scales utilizing educational personnel. A diagnosis of ADHD may result in prescribed medicines. A pre-set screening instrument might well assist in that process.

This study presents the reliability and validity data for the Pediatric Residency Checklist/ADHD (PCR/ADHD), a brief screening instrument designed to assist pediatric resident physicians in diagnostic history-taking and decision-making with regard ADHD. In spite of numerous indices to determine the presence of ADHD,[8-10] no screening instrument which would direct a more detailed evaluation, designed specifically for pediatric residents, exists.

The Pediatric Residency Checklist/ ADHD (see Appendix A) contains a series of questions for pediatric residents to ask in conversation with both parents and children who present to pediatric clinics. It focuses awareness on specific behaviors required for diagnosis and prompts the resident physician to insure the behavior in question occurs across multiple settings, which is also a diagnostic requirement. In addition, the PRC/ADHD aids the resident in obtaining the necessary genetic history by providing a framework for taking a family genogram. Finally, the instrument allows residents to check on common presentations of children with ADHD across multiple age ranges. As such, it cues resident physicians to ask questions, which will shed light on specific behavioral questions required for a firm and meaningful diagnosis of ADHD. Also, common diagnoses to be ruled out are highlighted. Finally, the PRC/ADHD also contains standardized instructions for the administration of medical interventions should that be required.

In addition, the Pediatric Residency Comfort Questionnaire (PRCQ) (see Appendix B) was developed to survey resident comfort levels and understanding of the ADHD diagnostic process. It was developed as a pre- and post-test measuring device to determine the PRC/ADHD's value to residents. As such, this study assessed the value of the screening instrument by surveying residents before and after its introduction.

Methods

Each pediatric resident from first to fourth year was asked to complete the Pediatric Residency Comfort Questionnaire. Then, each resident was trained in the use of the Pediatric Residency Checklist/ADHD prior to its introduction into the residency program. The instruction included specific scoring, history taking, and the use of the interview in observation of the child in clinic.

The items of the PRC/ADHD were derived from DSM-IV[11] criteria for both inattention and hyperactivity/impulsivity. Positive scores were achieved when the item

was scored as "Very Often" and in "more than one setting". In addition, each resident was trained in steps occurring after the initial evaluation. These steps included additional assessment in more advanced and previously-validated assessment instruments as well as follow-up at the Pediatric Clinic. The additional assessment instruments included:

- The Conners' Continuous Performance Test II[12]
- The Conners' Teacher Rating Scale (CTRS-R)[13]
- The Conners' Parent Rating Scale (CPRS-R)[14]
- The Child Behavior Checklist (CBCL)[15]
- Teacher Rating Form (TRF)[15]
- The Attention Deficit Disorders Evaluation Scale-Home Version[16]
- The Attention Deficit Disorders Evaluation Scale-School Version[17]

All families with patients presenting at the Wesley Pediatric Clinic over a nine-month period with concerns regarding school-related behavioral problems were asked to be part of the study. Those agreeing (n = 63) were given each of the assessment instruments including the Pediatric Residency Checklist/ADHD. The combination of scores on all of these assessment instruments, plus clinical judgment, resulted in assignment of children to either the ADHD or Non-ADHD groups.

The Receiver Operating Characteristic (ROC) was utilized to assess the accuracy of the PRC/ADHD score in differentiating patients with and without the diagnosis of ADHD and to determine a cut-point for the PRC/ADHD score.[18] The PRC/ADHD cut-point score was incorporated into a logistic regression model with the binomial variable of ADHD diagnosis as the dependent variable.[19] In addition, the binomial variables, age and gender, were included as independent variables in the logistic regression model to control for possible confounding of results.

A sub-sample (n = 25) of the original respondents to the study was re-tested after six weeks with the PRC/ADHD for test/re-test reliability analysis using Pearson correlation coefficients.[20] Finally, scores on the Pediatric Residency Comfort Questionnaire were compared before and after introduction to pediatric residents and analyzed using t-test statistics.[20] All statistical analysis was performed using STATA version 8 software for Macintosh.[21]

Results

The Receiver Operating Characteristic (ROC) analysis is detailed in Table 1 and Figure 1 for the PRC/ADHD diagnosis. At a PRC/ADHD score of 10 or higher, 88.9% of patients were classified correctly as having ADHD or not having ADHD. The sensitivity for ADHD diagnosis was 94.4% and the specificity was 81.5%. The positive likelihood ratio using a PRC/ADHD cut-point score of 10 was 5.1. Patients with ADHD were 5.1 times more likely to have a PRC/ADHD score of 10 or higher as compared to patients without ADHD. Similarly, the negative likelihood ratio of 0.07 signified that subjects with ADHD were 0.07 times as likely to have a PRC/ADHD score less than 10 as compared to subjects without ADHD.

With the binary variable of ADHD diagnosis as the dependent variable (i.e., PRC/ADHD score of 10 or higher), a multivariate logistic regression model was constructed with gender, age, and PRC/ADHD score as independent variables. The odds of having a diagnosis of ADHD were 404 times the odds of not having

Table 1. Results of receiver operating curve analysis.

Cut Point	Sensitivity	Specificity	Correctly Classified
>=2	100.00%	0.00%	57.14%
>=4	97.22%	3.70%	57.14%
>=5	97.22%	14.81%	61.90%
>=6	97.22%	25.93%	66.67%
>=7	94.44%	37.04%	69.84%
>=8	94.44%	44.44%	73.02%
>=9	94.44%	66.67%	82.54%
>=10	94.44%	81.48%	88.89%
>=11	86.11%	85.19%	85.71%
>=12	75.00%	85.19%	79.37%
>=13	58.33%	85.19%	69.84%
>=14	36.11%	92.59%	60.32%
>=15	22.22%	100.00%	55.56%
>=16	16.67%	100.00%	52.38%
>=17	2.78%	100.00%	44.44%
>17	0.00%	100.00%	42.86%

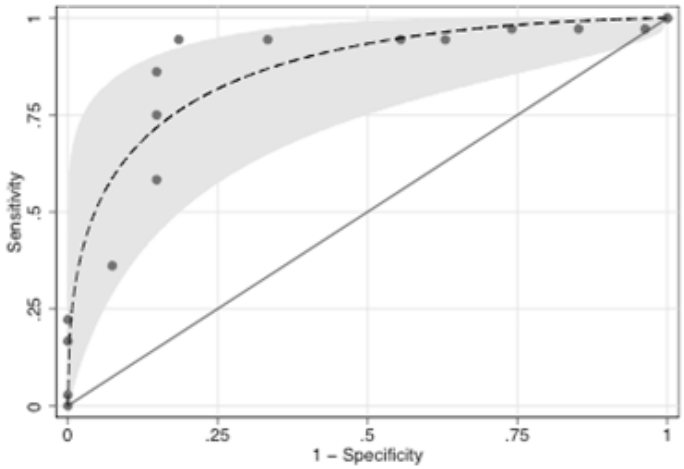

Figure 1. Fitted ROC curve with 95% confidence band for PRQ score and ADHD diagnosis.

statistically significant ADHD in those subjects with a PRC/ADHD score of 10 or higher while controlling for age and gender (95% CI 38-21388). Gender and age were not variables in the model (p values were 0.54 and 0.14 respectively).

Using Pearson product moment correlations (n = 25), the PRC/ADHD had six-week test-retest correlations of 0.89 (p < .0001) for Inattention, 0.91 (p < .0001) for Hyperactive/Impulsive, and 0.94 (p < >0001) for the combined total.

Comfort level scores before and after the introduction of the PRC/ADHD reflected that residents were more comfortable in their assessments and treatment of ADHD after

having had some instruction in the application and use of the Pediatric Resident Checklist/ADHD. Comfort level mean scores of the pre-test by all residents at each pediatric level were 28.5 while post-test comfort level means were 46.6 ($p < .001$).

Discussion

The results suggested the viability of the PRC/ADHD as a screening device for ADHD, especially in the day-to-day operations of a medical school pediatric clinic. The addition of the Pediatric Residency Checklist/ADHD benefitted residents in terms of increased comfort levels in the assessment and treatment of ADHD. However, this assessment device is only one in the growing arsenal of such instruments and possibly should be considered for use only in relation to medical residency programs as an educational as well as assessment tool. The PRC/ADHD offers a practical instrument for helping resident physicians offer the best care to this specific population of patients.

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Keywords: attention deficit hyperactivity disorder, medical residency, pediatrics, screening, diagnosis

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APPENDIX A

KUSM-W PEDIATRIC RESIDENCY CHECKLIST/ ADHD

Form completed by: ***Patient:***

Date:

Instructions: Make filling out the form a conversation. Include parent and child responses. "P" = parent response. "C" = child response. Score only "OFTEN TRUE" responses.

	FREQUENCY			SETTING			
	NOT TRUE	SOME-TIMES	OFTEN TRUE	HOME	SCHOOL	PEERS	SELF
1. INATTENTION (scoring: 6 out of 9=often true)							
a. Tends to make careless mistakes on schoolwork / household chores?							
b. Easily distracted in tasks and/or play activity?							
c. Adult needs to repeat himself or herself?							
d. Tends to quit a project prior to completion?							
e. Tends to have difficulty organizing prior to attempting task? (i.e. Schoolwork)							
f. Tends not to get involved with activities that require attention?							
g. Frequently misplaces things?							
h. Easily distracted when in the middle of something they enjoy?							
I. Forget important events / items?							
2. HYPERACTIVITY/IMPULSIVITY (scoring: 6 out of 9=often true)							
a. Tends to fidget in their seat?							
b. Tends to leave their seat without good reason?							
c. Tends to run from place to place?							
d. Not able to have a quiet time of appropriate time for age?							
e. Seem to be "on the go" at all times?							
f. Talk excessively?							
g. Blurt out phrases / answers inappropriately?							
h. Unable to play board games with other children?							
I. Interrupt other conversations?							

3. Age at onset _____?

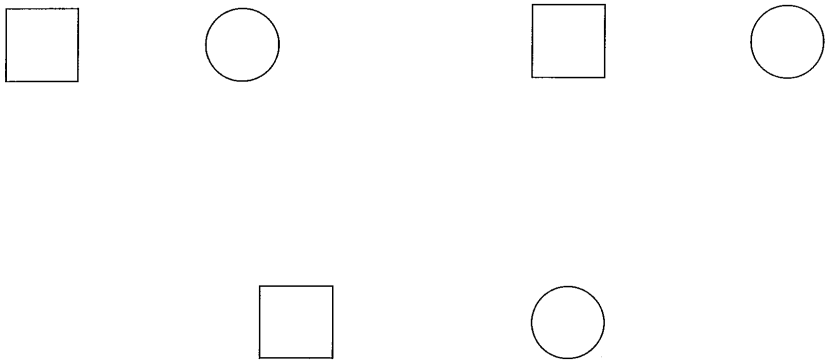
How long has this been a problem _____? (must be at least six months)

How severe _____ _____ _____ ?(must effect more than one setting)

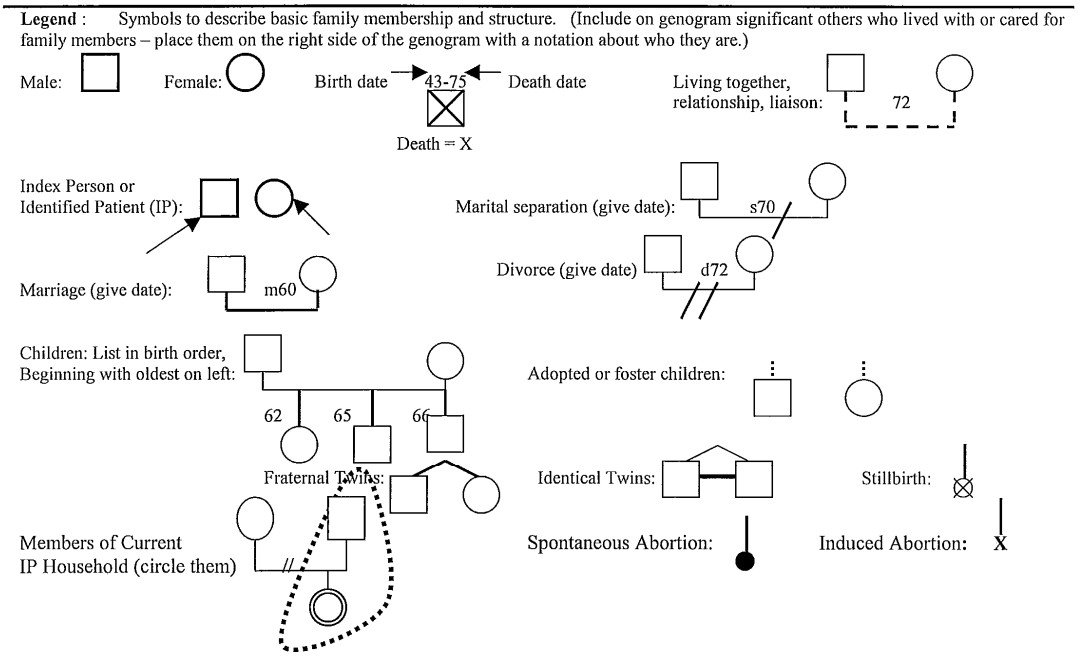
Affecting only 1 setting | Affecting 2 or more settings | Affecting everything

KUSM-W PEDIATRIC RESIDENCY / ADHD CHECKLIST

4. Genogram (Look for family history that may include ADHD, affective and anxiety disorders, learning disorders, conduct, oppositional and antisocial disorders, alcohol and substance abuse).



CHILD



KUSM-W PEDIATRIC RESIDENCY / ADHD CHECKLIST

Medical Notes:*

5. Rule out: Pervasive developmental disorder, hypoglycemia, anemia, diabetes, hyperthyroidism, sleep apnea, schizophrenia, anxiety disorder, depressive disorder, bipolar disorder, acute infections, seizure, hearing and/or vision problems, allergies, and genetic disorders.

6. Medical and developmental risk factors:
    a. Prenatal and post-natal difficulties
    b. Maternal substance abuse
    c. Poor maternal health
    d. Vitamin and mineral deficiencies
    e. Multiple ear infections
    f. Headaches
    g. Frequent illnesses
    h. Poor eye-hand coordination
    i. Accident prone

7. Characteristics:
    a. ADHD presentation in infancy:
        i. Crying frequently
        ii. Unable to soothe
        iii. Sleep disturbance
        iv. Feeding difficulties

    b. ADHD presentation in preschool age:
        i. Motor restlessness
        ii. Insatiable curiosity
        iii. Vigorous > destructive play
        iv. Demanding
        v. Non-compliance
        vi. Excessive temper tantrums
        vii. Difficulty completing developmental tasks
        viii. Decreased/restless sleep
        ix. Delays in motor/language development
        x. Family difficulties

    c. ADHD presentation in middle childhood:
        i. Distractibility
        ii. Over-engagement in off task activities
        iii. Inattentive
        iv. Lack of social skills
        v. Aggressive
        vi. "Class clown"
        vii. Problems with peers
        viii. Labeled "difficult" or "lazy"
        ix. Behavioral impulsivity
        x. Cognitive impulsivity

    d. ADHD presentation in adolescence
        i. Discipline problems
        ii. Family conflict
        iii. Emotional lability
        iv. Lags in academic performance
        v. Poor peer relationships
        vi. Poor self-esteem
        vii. Helplessness ("Given up" Syndrome)
        viii. Lack of motivation
        ix. Driving mishaps – accident prone

*Adapted from *Attention Deficit Hyperactivity Disorder (in Adults and Children)* by C. Keith Conners, PhD and Juliet L. Jett, PhD, Compact Clinicals, Kansas City, MO, 1999.

KUSM-W PEDIATRIC RESIDENCY / ADHD CHECKLIST

ADHD Med Check Instructions:

Once diagnosis of ADHD has been made, you may want to utilize one of the following strategies with reference to medical interventions involving medications:

1. Use Parent/Teacher questionnaires to assess the medicine's effectiveness(for example the ASQ-T and ASQ-P by Conners or the Pediatric Department's own Parent and Teacher evaluation forms which are located at the nurses station):
    a. Ten forms are given to each i.e. one set of 10 for parents and one set of 10 for the teacher.
    b. Parents and teachers fill out forms on a daily basis.
    c. On the third or fourth day, medicines are taken (teacher is "blind" to whether medicines are "on board").
    d. Evaluations are returned to the Pediatrician.
    e. The evaluations should reflect positive change. If not, a change in medicines and/or dosage may be indicated.

2. Alternative strategy:
    a. Ten forms are given to each i.e. one set of 10 for parents and one set of 10 for the teacher.
    b. Start child, first week, with the lowest dose of medicine and have both parents and teacher fill out forms randomly two days out of each week.
    c. For the next week, repeat with next highest dose.
    d. Repeat for total of four weeks.
    e. Assess results of both parent and teacher evaluations to determine best medication dosage or need for medication change and repeat a-b.

NOTES:

APPENDIX B

PEDIATRIC RESIDENCY COMFORT QUESTIONNAIRE

1. PGY level_____
2. Approximately how many ADHD assessments have you participated in prior to residency training?_____
3. Approximately how many ADHD assessments have you participated in since your residency experience began?_____
4. What evaluation instruments do you use?_____
   _____
   _____

Please use this scale on the following questions:

not at all						very
1	2	3	4	5	6	7

5. How comfortable are you with the evaluation instruments you use?_____
6. How comfortable are you with performing ADHD assessments generally?_____
7. How comfortable are you with your knowledge of DSMIV criteria for ADHD?__
8. Do you routinely take a family genogram when evaluating for ADHD?_____
9. How comfortable are you generating a family genogram in the assessment of ADHD?_____
10. How would you rate your understanding of common developmental presentations of ADHD?__
11. How comfortable do you feel in beginning evaluations for treatment (meds dosage etc.) of ADHD?_____
12. How would you rate your understanding of medical and developmental risk factors for ADHD?_____
13. How much understanding do you believe you have concerning disorders that must be ruled out before an ADHD diagnosis can be made?_____

The Danger of Diagnostic Error: Community-Acquired MRSA or a Spider Bite?

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Introduction

In today's world of medicine, the state of the art for diagnosis has reached levels of accuracy never dreamed. Nevertheless, diagnostic error still is encountered frequently. Regardless of whether the diagnosis is made based on clinical evaluation, imaging, or laboratory studies, when the diagnosis is wrong, patient injury can result. This report describes a case of methemoglobinemia after starting dapsone for a presumed spider bite.

Case Report

A previously healthy 23-year-old female presented to her primary care physician for evaluation of a progressively enlarging lesion on the pre-tibial space of her right lower leg (see Figure 1). She noticed the lesion five days prior to presentation. The lesion steadily enlarged and began to ooze blood and frank pus over the prior two days.

The patient was started on dapsone, the combination of sulfamethoxazole and trimethoprim, and prednisone for a presumed spider bite. Two days after starting the treatment, the patient returned to the clinic complaining of shortness of breath during routine daily activities and significant bi-frontal headaches. Lip cyanosis and a draining carbuncle over the right pre-tibial space was noted on physical exam. Her vitals revealed a pulse oximetry of 88% on room air. Otherwise, the vital signs and physical findings were normal. A complete blood count also was normal. The patient was transferred to the hospital for further evaluation of the hypoxia.

On admission, the patient had a temperature of 98.2F, a blood pressure of 134/78, a pulse of 82, a respiration rate of 24, and an oxygen saturation of 89% on room air. The physical findings were similar to those in the office exam.

There was no scleral icterus and lungs were clear to auscultation. Arterial blood gas on room air, done simultaneously with pulse oximetry, showed a pH of 7.47, a partial pressure of carbon dioxide of 29, a partial pressure of oxygen of 107, and an oxygen saturation of 99%. The methemoglobin level was 16.3% with normal being less than 1.5%.

The wound culture from the lesion grew methicillin-resistant S*taphylococcus aureus* (MRSA) with a susceptibility pattern consistent with community-acquired MRSA. The patient was treated with methylene blue one mg/kg, intranasal mupirocin calcium ointment, and chlorhexidine gluconate showers. Recovery was uneventful.

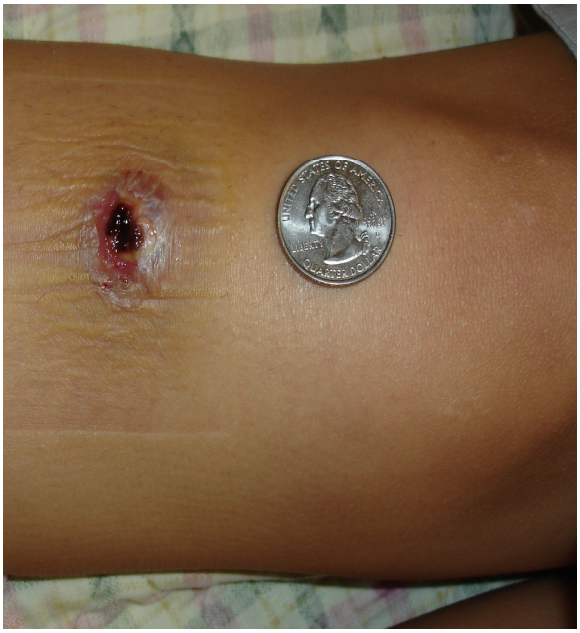

Figure 1. Lesion on the pre-tibial space of the right lower leg of the patient case.

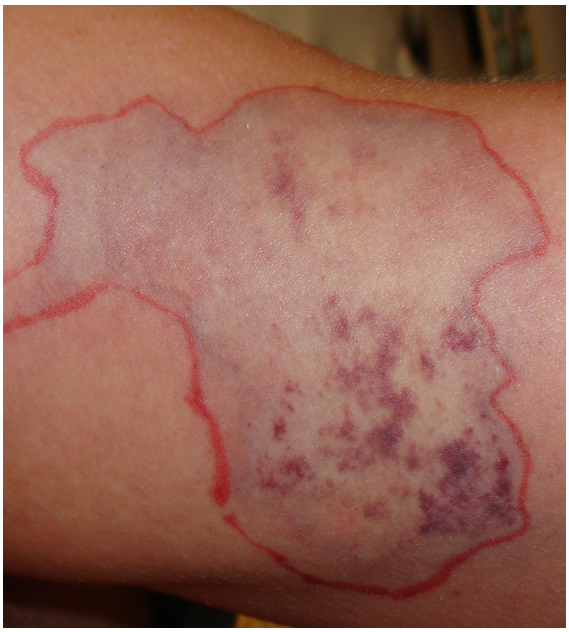

Figure 2. Lesion of a brown recluse spider bite.

Discussion

Spider bites occur, but they are the exception, not the rule. Over-diagnosis of brown recluse spider (*Loxosceles recluse)* bites has led to harmful sequelae and misdiagnosis of other common and uncommon dermonecrotic wounds.[1-2] Skin lesions resembling bites of brown recluse spiders can have many different etiologies. They can be caused by infections (bacterial, fungal, viral), inflammatory and metabolic diseases (diabetic ulcer, pyoderma gangrenosum, erythema multiforme), or arthropods either directly (ticks, fleas) or as vectors (Lyme borreliosis, flea-born diseases).[3-4] Improper diagnoses of spider bites have been given to patients with cutaneous anthrax, lymphoma, basal cell carcinoma, Lyme borreliosis, pyoderma gangrenosum, and other serious and potentially debilitating or deadly conditions.[4]

Spiders frequently are blamed for causing skin disease incidents based completely on speculative, unsubstantiated associations and historical prejudice. In almost every case, no spider is seen biting or is collected in the incident.[5-6] Many patients present with a "spider bite", assumed because of "how bad it looks", but on investigation they have community-acquired MRSA (CA-MRSA). When spiders are blamed, medical and entomological personnel divert their efforts onto the wrong remedial pathway and delay the correct assessment of the situation.[7]

In the United States, brown recluse spiders are endemic only in the southwest and midwest.[1] Brown recluse spiders are not present in vast regions of the country, such as the Pacific Northwest. In such locales, it also is difficult to find a black widow spider, thus making the diagnosis of a spider bite highly unlikely.[8]

In North America, brown recluse spiders are the only spiders that are proven to cause dermonecrotic lesions.[4] These spider bites manifest as single lesions in a given patient (see Figure 2). Most bites heal well with no or minimal medical intervention. Most bites heal without noteworthy scarring.[7] In a diagnostic situation, a caregiver almost always can

rule out a spider bite when there are multiple contemporaneous lesions on one person, multiple consecutive lesions on one person, or multiple persons with lesions.[9]

The infamy of this spider is exaggerated in part due to the tendency of the medical community to emphasize lesions with severe necrosis, which are rare manifestations of venom insult.[4] In addition, there is an epidemic of skin lesions infected with CA-MRSA; many may have originated in pruritic bites and stings, or in other puncture wounds that eventually necrose and can mimic necrotic arachnidism.[10] Most CA-MRSA infections are mild, but some advance to more serious systemic infection, bacteremia, and death.[11]

CA-MRSA infections with secondary familial transmission have been described in some reports.[12] Obtaining the proper diagnosis of a CA-MRSA infection is important because misdiagnosis and delay of proper treatment can have serious consequences for both the patient and the medical community.

Conclusion

The diagnosis of brown recluse bites is overused. This case demonstrated the ease with which patients and clinicians can confuse spider bites with other necrotic skin lesions, especially MRSA skin lesions. A diagnosis of a brown recluse spider bite should be made after careful consideration is given to other possible diagnoses, especially if the patient is not within the region endemic to the brown recluse spider.

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Keywords: community-acquired infections, methemoglobinemia, methicillin resistance, Staphylococcus aureus, spider venoms, diagnosis

Sinusitis and Orbital Cellulitis Due to Community-Associated Methicillin-Resistant Staphylococcus Aureus

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Introduction

Community-acquired methicillin-resistant *Staphylococcus aureus* (CA-MRSA) has a predilection to cause severe skin and soft tissue infection in both immunocompetent and immunocompromised adults.[1] Other serious invasive infections, such as necrotizing pneumonia, sepsis, bacteremia, sinusitis, and urinary tract infections, are on the rise.[2] We report a case of bacterial sinusitis and orbital cellulitis due to CA-MRSA.

Case Report

A 56-year-old woman developed mild pain around her left eye six days prior to hospital admission. Although over-the-counter analgesics were helpful initially, the pain increased in intensity prompting the patient to seek emergent care.

Her physical exam was within normal limits, except for left maxillary sinus tenderness. Pansinusitis was found on a CT scan of the sinuses (see Figure 1). The patient was prescribed amoxicillin/clavulanate and released. She returned for evaluation in the clinic two days later with worsening pain and swelling around the left eye.

Her left eye was swollen, erythematous, warm to touch, and tender to palpation. Her left pupil was normal in size and reactive. The left conjunctiva was erythematous. There was decreased ocular motility with pain elicited by eye movement.

Upon hospital admission, a CT scan of the brain and sinuses revealed extensive paranasal sinusitis and evidence of new inflammatory changes in the postseptal region of the left orbit as compared to the previous study done three days prior. These findings were consistent with left orbital cellulitis (see Figure 2).

The patient's past medical history was remarkable for hypertension and asthma. She had no history of smoking, alcohol intake, or drug abuse. Her medications included an albuterol inhaler as needed, hydrochlorothiazide 25 mg daily, and amoxicillin/clavulanate.

Ampicillin/sulbactam was started empirically. About eight hours later, the patient underwent endoscopic drainage of both the maxillary and left frontal and ethmoid sinuses. Gram's stain of material from surgery revealed moderate neutrophils and moderate gram positive cocci, a finding that prompted the addition of vancomycin 1g intravenously (IV) every 12 hours.

Ampicillin/sulbactam was changed to piperacillin/tazobactam 3.375g IV every six hours the following day. Cultures yielded a predominant growth of methicillin-resistant *Staphylococcus aureus* (MRSA) and a light growth of *Escherichia coli*. The MRSA exhibited a susceptibility profile typical for the USA300 strain (CA-MRSA). A follow-up sinus CT scan done two days after the

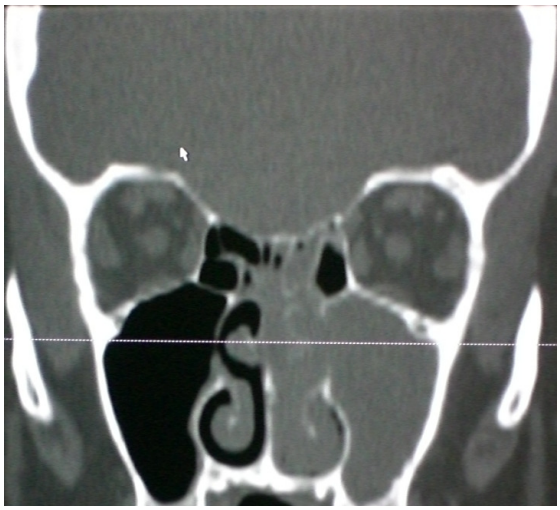

Figure 1. Pansinusitus on CT scan of the sinuses.

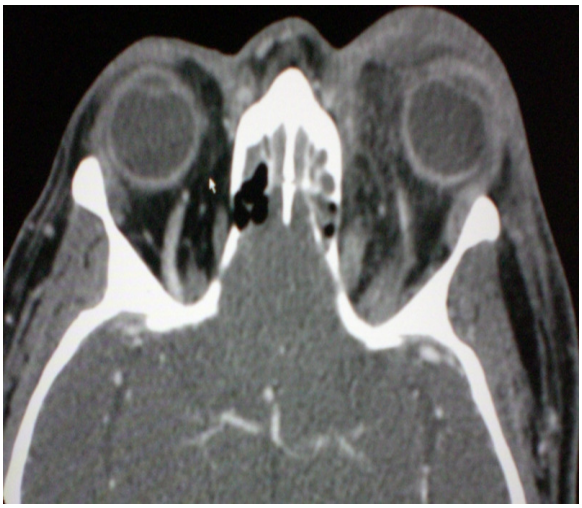

Figure 2. CT findings consistent with left orbital cellulitis**.**

surgery showed significant improvement of inflammatory changes (see Figure 3). The patient was dismissed to complete a 21-day course of vancomycin 1g IV every 12 hrs as an outpatient. Recovery was uneventful.

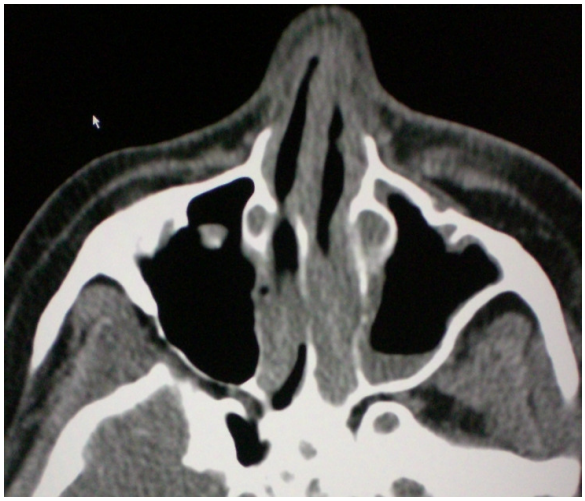

Figure 3. Significant post-surgical improvement of the inflammatory changes.

Discussion

Staphylococcus aureus is a common cause of disease, particularly in colonized persons. The prevalence of MRSA colonization is estimated at 0.8%.[3] Strains of MRSA were first detected in 1961, but occurred sporadically and were only resistant to ß-lactam antibiotics.[4,5] Resistant hospital-acquired strains appeared in Australia in the late 1970s and subsequently spread to hospitals worldwide.[6,7]

Hospital-acquired MRSA is one of the most common causes of bacterial healthcare-associated infection, responsible for 40 to 70% of *S. aureus* infections in intensive care units.[8,9] In the United States, CA-MRSA was first reported in 1982 in a large, urban Michigan hospital.[10] The infection was found in a cluster of 40 persons, including 24 who were injection drug users. While CA-MRSA primarily causes skin and soft tissue infections, other serious invasive infections are on the rise.[2]

Current recommendations for the diagnosis and treatment of acute bacterial rhinosinusitis are based on the expected prevalence, spontaneous resolution rate, and specific drug-resistance patterns of pathogens.[11] Recent literature has indicated an increasing prevalence of *S. aureus* in sinus cultures. Culture rates were 32.7% for *Streptococcus pneumoniae,* 31.6% for *Hemophilus influenzae,* 10.1% for *S. aureus,* and 8.8% for *Moraxella catarrhalis*.[12] CA-MRSA sinusitis has been reported in literature, however, there are no data about the prevalence.

The most feared complications of sinusitis are orbital and central nervous system (CNS) complications. Devastating outcomes, such as temporary or permanent loss of vision, diplopia, residual proptosis, optic neuritis, and epidural or subdural infection may develop if appropriate treatment is delayed.[13]

Two cases of MRSA sinusitis with orbital cellulitis has been reported in the English literature.[1,13] The patient reported by Mehra et al.[13] had a history of chronic intravenous drug use, an iatrogenic displacement of the tooth-root tip, and residual visual symptoms after completion of treatment. The case reported by Rutar et al.[1] resulted in bilateral blindness.

Patients with immotile cilia syndrome and cystic fibrosis and those with a history of IV drug use are prone to infections with resistant bacterial species, including MRSA. Our patient had none of these risk factors or those for CA-MRSA infection such as young age, incomplete development of the immune system, participation in contact sports, sharing towels or athletic equipment, having a weakened immune system, or living in crowded or unsanitary conditions.[14] She also did not have a prior history of skin and soft tissue infection with CA-MRSA.

CA-MRSA should be included in the differential diagnosis of progressive sinusitis not responding to standard antimicrobial coverage even in the absence of classic risk factors for MRSA. Early microbiologic diagnosis might be helpful in preventing severe complications such as orbital or CNS extension.

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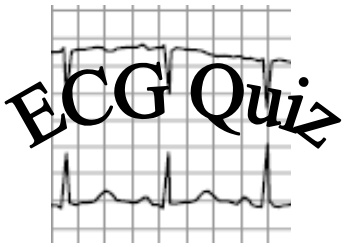
Keywords: sinusitis, orbital cellulitis, community-acquired infections, methicillin resistance, Staphylococcus aureus



Right Bundle Branch Block or Something Else?

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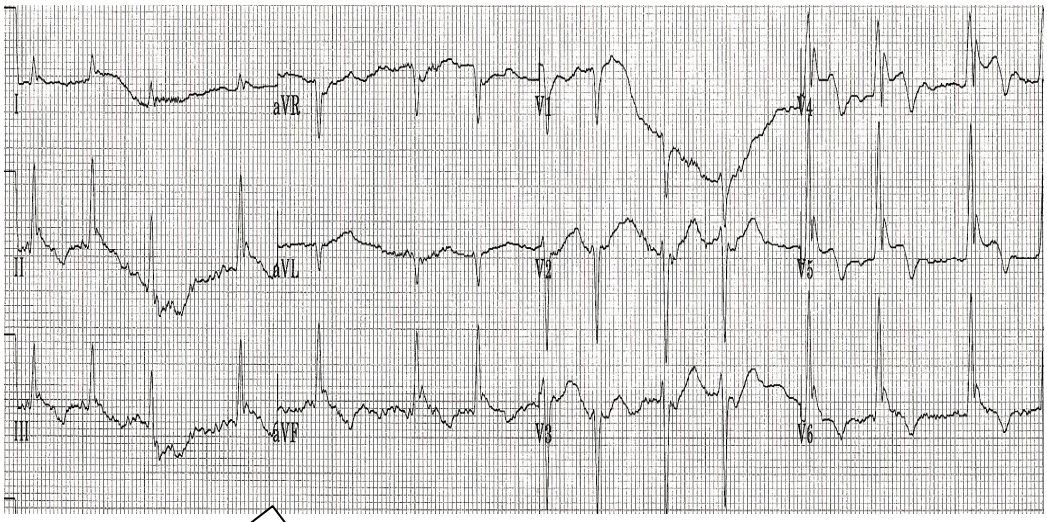
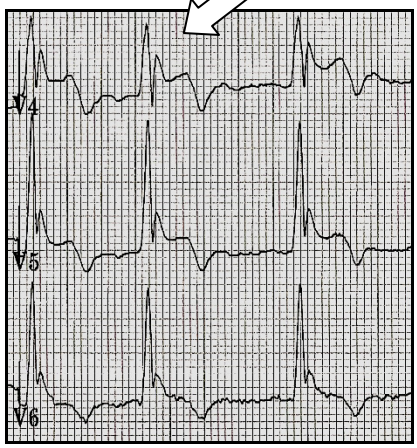
Case

A 28-year-old male patient with insulin-dependent diabetes mellitus was found lethargic on the garage floor. At the hospital, he was unresponsive to verbal stimuli, his breath sounds were coarse, and his heart sounds were irregular. The admitting diagnosis was diabetic ketoacidosis and sepsis secondary to aspiration pneumonia. A CT scan of the head did not reveal any acute abnormality. Calcium and magnesium levels were normal. His initial ECG is below:





What is the diagnosis?

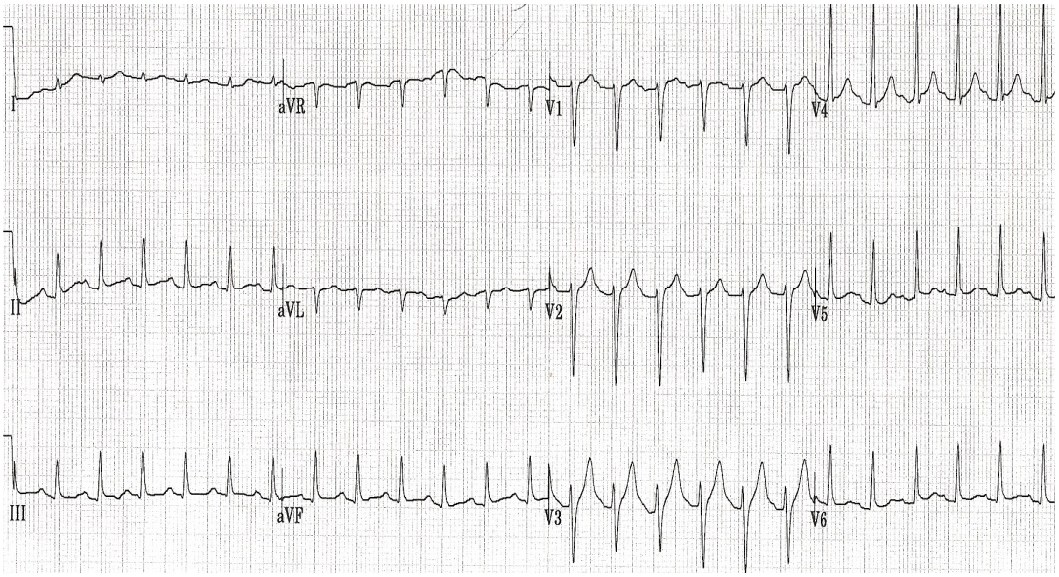
(A) Right Ventgricular Hypertrophy
(B) Right Bundle Branch Block
(C) Osborne Waves
(D) Epsilon Waves
(E) Wolf Parkinson White Syndrome
(F) Brugada Syndrome

Correct Answer: C

The Osborn wave (also referred to as the J wave, or camel hump sign) was first described by Dr. John Osborn in 1953.[1] It is a distinctive deflection occurring at the QRS-ST junction of approximately 80% of hypothermic patients.[2] However, the presence of prominent J waves is not pathogonomic of hypothermia. They have been reported in the literature in normothermic individuals, and in patients with hypercalcemia, subarachnoid hemorrhage, cerebral injuries, and myocardial ischemia.[3] The presence of J waves occurs after resuscitation from cardiac arrest, especially in association with ventricular fibrillation.[2] Very large Osborn waves may mimic right bundle branch block.

Hypothermia increases the epicardial potassium current relative to the current in the endocardium during ventricular repolarization. This transmural voltage gradient is reflected on the surface electrocardiogram as a prominent Osborn wave.

Our case had a temperature of 84 degrees Fahrenheit (29°C) on admission. Active external re-warming using warm blankets was done. The Osborn waves diminished in amplitude and disappeared after 24 hours and spontaneous conversion to sinus rhythm occurred. His ECG after re-warming is shown below:



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Keywords: electrocardiography, bundle-branch block, coronary artery disease

Commentary

Quality and Patient Safety

Statins Not Beneficial in Most Chest Pain Admits

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There is current "statin hysteria" with people prescribing statins for everything from STEMI to stroke to Alzheimer's. And the push is to give as much of the drug (high dose) and as early in the course ("ER") as possible. There is a reason why atorvastatin is one of the hottest selling drugs in the world.

How much of this is "stuff we heard at a meeting" or worse "at a free dinner" – and how much is actually proven in good studies? While I am not arguing about the use of statins for coronary stents, I do worry about the national recommendations to get a lipid level in the emergency department for patients with ST Elevation Myocardial Infarction (STEMI) and Acute Coronary Syndrome (ACS). Why? We do not know if the patient is fasting which can skew the measured LDL level significantly. We know that periods of significant metabolic stress can affect the lipid levels.[1] Then the most obvious question, what are you going to do with the level for the admitted patient who does not have ACS and does not receive a stent? To which the makers of statins say "put them on a statin!"

So here is the question. In light of national groups asking for cholesterol and lipid panels in the emergency department for the possible ACS patient[2], do we have any evidenced-based data that supports this recommendation? Answer: "No."

We do have data from 12 trials involving over 13,000 patients that proves that giving statins (compared to placebo) to patients with proven acute coronary syndromes as a whole does not reduce death, does not reduce nonfatal MI, and does not reduce nonfatal stroke, when one looks at the first four months after hospitalization for ACS.[3] Furthermore, we have evidence that putting people on statins that do not have heart disease does not improve morbidity or mortality.[4]

So forget the ER "lipid level" (it's not accurate anyway) and say "no" to statins in the ER, at least until better data can prove to us otherwise.

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Keywords: statin, myocardial infarction, emergency service, commentary

Commentary

Quality and Patient Safety

In Response: Statins Not Beneficial in Most Chest Pain Admits

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While I share Dr. Mosley's skepticism at some of the recent hope for 3-hydroxy-3-methyl-glutaryl-CoA reductase inhibitors (statins) as treatment for non-vascular illnesses, I disagreed with his assertion that lipid levels should not be evaluated in the emergency department. Though Dr. Mosley's cynicism regarding pharmaceutical companies' influence on research findings may be well founded[1], it should be pointed out that large, independently funded studies showing the mortality benefit of lipid reduction[2] and lipid reduction specifically by statins[3] in high-risk populations have been in the literature since the 1980s.

When a statin-naïve patient presents with chest pain, his or her cardiovascular risk cannot be evaluated fully without knowledge of lipid status, a position endorsed by the National Cholesterol Education Program.[4] The point that low-risk patients may not benefit from statin therapy is moot until all the relevant data are known. Young people with heterozygous familial hypercholesterolemia, a common condition whose mortality can be reduced with cholesterol-lowering therapy, present to physicians every day complaining of chest discomfort.

In people already prescribed statins, compliance, even among high-risk patients, is poor.[5] Beside directly questioning the patient, a method whose poor reliability is highlighted by the recent national movement toward "medication reconciliation", adherence can be judged in only one way: calculation or direct measurement of the serum low-density lipoprotein (LDL) level. Calculation of the LDL can be problematic because, as Dr. Mosley points out, a non-fasting specimen may be incalculable secondary to high serum triglyceride content. If the patient is non-fasting, though, the "non-HDL" cholesterol level serves as a suitable alternative.[6] In the near future, measurement of the LDL level likely will be replaced by measurement of apolipoprotein B, which will eliminate the need for a fasting specimen.[7]

While it is true that the immediate post-myocardial infarction effect of statin therapy has not lived up to its initial promise, the long-term effect of statins on mortality in high-risk populations is so profound, roughly a 1% decrease in five-year mortality for every 1 mg/dl reduction in LDL in high risk groups[4], that adherence to therapy must be evaluated.

Hypercholesterolemia is one of the primary modifiable risk factors for cardiovascular death. A lipid panel, fasting or not, is a relatively inexpensive test that aids in risk-stratification of patients presenting with chest pain, helps evaluate compliance in people already prescribed statins, and is indispensable to the patient's primary care provider, hospitalist, cardiologist, or endocrinologist.

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Keywords: statin, myocardial infarction, emergency service, commentary