COMMENTS AND RESPONSES

The Value of Physician Assessment

TO THE EDITOR: In response to Lipner and colleagues’ research (1), I would like to point out that approximately 6000 first-time candidates annually participate in the American Board of Internal Medicine (ABIM) certification process at about $1000 per candidate; curiously, about half as many candidates enroll in the Board’s Maintenance of Certification process. Revenues are nearly $9 million annually (2), and review and exam preparation materials provide additional income for the ABIM and other organizations. Studies about ABIM processes, which are often funded by the ABIM, have been conducted for decades. Consequently, evidence for the ABIM’s processes should be clear and widely understood, especially in academia (3).

The rate-limiting step of ABIM certification is the written examination. As an idea born of academia, the examination would ideally be valued in academia. However, my survey of 1 academic group of general internists revealed that the use of the ABIM examination as a measure of clinical quality is far from universally endorsed (2). The internists expressed sentiments that were similar to those reported by Lipner and colleagues (1). It remains to be shown whether other academic groups perceive the examination similarly.

Without concrete evidence, many employers and payers often mandate ABIM certification as a marker of high clinical quality. In an era of evidence-based medicine, such a policy is clearly unwarranted.

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Table. Survey of Full-Time Faculty in General Internal Medicine*

<table>
<thead>
<tr>
<th>Variable</th>
<th>Respondents (n = 21), n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Passing the ABIM written examination is a marker indicating that the internist provides higher-quality care than one who did not pass the examination</strong></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>11 (52.4)</td>
</tr>
<tr>
<td>No</td>
<td>10 (47.6)</td>
</tr>
<tr>
<td><strong>Fees for the ABIM certification and recertification process are worthwhile to ensure the practice of high-quality internal medicine</strong></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>6 (28.6)</td>
</tr>
<tr>
<td>No</td>
<td>15 (71.4)</td>
</tr>
<tr>
<td><strong>Periodic written ABIM examinations are necessary to providing high-quality care to patients</strong></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>8 (38.1)</td>
</tr>
<tr>
<td>No</td>
<td>13 (61.9)</td>
</tr>
<tr>
<td><strong>Respondent ABIM certification status†</strong></td>
<td></td>
</tr>
<tr>
<td>Time-limited certificate</td>
<td>12 (57.1)</td>
</tr>
<tr>
<td>Unlimited certificate</td>
<td>7 (33.3)</td>
</tr>
<tr>
<td>Not certified</td>
<td>2 (9.5)</td>
</tr>
</tbody>
</table>

* Response rate was 78%. ABIM = American Board of Internal Medicine.
† Percentages total < 100% because of rounding.

Potential Financial Conflicts of Interest: None disclosed.

References

IN RESPONSE: Nobody likes to be evaluated on his or her performance, but the public must have a way to ensure that physicians are providing high-quality care. The American Board of Medical Specialties established a standard approach called Maintenance of Certification with the goal of assuring the public that physicians do so. The program requires that physicians possess an unrestricted license to practice medicine and that they demonstrate lifelong learning, practice improvement, current medical knowledge, good clinical judgment, and diagnostic skill.

This approach, based on Miller’s 4-stage pyramid for assessing clinical competence (1), requires physicians to demonstrate that they know the material, know how to apply the knowledge, show how they apply it, and show that they actually do apply it. The secure examination is used to demonstrate knowledge and clinical decision-making skills and is a highly reliable and valid high-stakes assessment (2). Considerable research establishes that certified physicians have better patient outcomes than those who are not certified (2, 3). Demonstrating knowledge is not sufficient for proving adequate day-to-day performance, but it is necessary.

The ABIM Maintenance of Certification program includes tools that measure practice performance, survey patients and peers, and assess knowledge of recent advances in the field; in the future, we hope to assess skills using simulation methods. The fee for this broad-based evaluation is about $1000 every 10 years (that is, about $100 per year). This fee provides virtually unlimited access to a wide spectrum of self-assessment tools and permits choices that reduce redundancy for the individual physician. For example, ABIM coordinates with organizations that provide continuing medical education to give credit for medical society self-assessment products (for example, the American College of Physicians Medical Knowledge Self-Assessment Program) and to ensure that physicians earn an American Medical Association Physician Recognition Award for completing a practice improvement chart audit. These offer additional value to physicians and, of course, require additional organizational resources.

Certification is valued, by physicians and by others. Our survey of a nationally representative sample of certified internists found that, in general, physicians value certification as a way of improving their professional image, updating knowledge, and improving the quality of patient care. Furthermore, both physicians (4) and patients (5) consider board certification of major importance when choosing a physician for patient referrals or to see themselves.

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Potential Financial Conflicts of Interest: Dr. Lipner is employed by the American Board of Internal Medicine.

References

CLINICAL OBSERVATIONS

Quicksilver Cholecystitis

Background: Mercury is a highly toxic heavy metal that may cause accidental poisoning through industrial and environmental exposure. In 1923, Umber (1) was the first to report the rare occurrence of deliberate injection of elemental mercury. In this case, a young woman attempted suicide by self-injecting the substance, which caused diffuse abdominal pain, renal insufficiency, and pulmonary embolization of mercury particles. Since then, there have been approximately 20 sporadic case reports of attempted suicide by parenteral injection of elemental mercury (2). When injected intravenously, mercury usually becomes trapped in the pulmonary capillary bed; however, mercury can pass to the systemic circulation through various pulmonary shunts and form deposits in other organs, such as the kidney or liver. Elemental mercury can remain in the body for extraordinary lengths of time because its elimination depends on a slow oxidation process to convert it to a soluble salt that can be excreted through urine or feces.

Objective: To report a case of mercury deposition in the gallbladder after self-injection.

Case Report: A 53-year-old man was admitted for elective open cholecystectomy for suspected gallbladder cancer. Results of previous laboratory studies were positive for antibodies to hepatitis B and C viruses. Magnetic resonance imaging showed a focal area of thickening of the gallbladder wall, which could not be differentiated from surrounding liver tissue. Surgical exploration revealed a mass in the fundus of the gallbladder with infiltration of surrounding liver tissue. The gross appearance was that of gallbladder carcinoma. A cholecystectomy and partial liver resection were performed. A biopsy revealed droplets of elemental mercury in chronically inflamed gallbladder and liver tissues (Figure). After surgery, the patient admitted that he had attempted suicide at 18 years of age by injecting elemental mercury.

Discussion: Although rare instances of foreign bodies in the gallbladder have occasionally been reported (3), this is, to our knowledge, the first reported finding of elemental mercury deposits in the gallbladder that mimicked gallbladder cancer. The toxicity of mercury depends on its chemical composition as well as the route and quantity of administration. Elemental mercury is far less toxic than organic or inorganic mercury salts and can persist in an inert state for many years if the acute phase of foreign-body embolization is tolerated. We can only speculate that a right-to-left shunt resulted in arterial seeding of mercury droplets, which made their way to the liver through hepatic artery circulation. The mercury was then excreted into the hepatobiliary tree and deposited in the gallbladder, where it remained as a chronic irritant for 35 years.

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References

Chagasic Encephalitis as the Initial Manifestation of AIDS

Background: Late presentation to care remains common in patients with HIV infection, even in developed countries (1). A patient presenting with neurologic symptoms who has multiple ring-enhancing lesions, a low CD4 cell count, and positive results on serologic tests for Toxoplasma gondii would be presumptively treated for toxoplasmosis encephalitis.

Objective: To describe a case of chagasic meningoencephalitis in a patient with previously undiagnosed AIDS.

Figure. Droplets of elemental mercury in resected liver tissue.
Case Report: A previously healthy, 56-year-old heterosexual man presented to the emergency department for progressive leg weakness, malaise, and urinary retention that began 3 weeks earlier. He was originally from southern Mexico but had lived in North Carolina for the past 15 years. Initial physical examination revealed a temperature of 38.5 °C, tachycardia, and distended bladder. The neurologic examination showed flaccid lower extremities, patellar hyperreflexia, and ankle clonus. The Babinski sign was present bilaterally.

Magnetic resonance imaging of the brain (Figure, left) and spinal cord showed multiple enhancing lesions with mild edema. Brain biopsy showed encephalitis with hemorrhagic necrosis, consistent with toxoplasmosis encephalitis. Results of serologic tests for HIV were positive, and CD4 cell count was 0.037 \( \times 10^9 \) cells/L. Serum \( T. \text{gondii} \) IgG antibody test results were positive. Therapy with pyrimethamine and sulfadiazine was initiated.

Magnetic resonance imaging was repeated 10 days later because the patient did not improve, and new lesions were seen in the temporal lobes and the conus medullaris. Brain biopsy showed encephalitis with hemorrhagic necrosis, consistent with toxoplasmosis encephalitis. Results of serologic tests for HIV were positive, and CD4 cell count was 0.037 \( \times 10^9 \) cells/L. Serum \( T. \text{gondii} \) IgG antibody test results were positive. Therapy with pyrimethamine and sulfadiazine was initiated.

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Discussion: Toxoplasmosis encephalitis remains a relatively common AIDS-defining illness in this era of highly active antiretroviral therapy. The differential diagnosis of central nervous system lesions in an immunocompromised HIV-infected patient is broad; however, when multiple ring-enhancing lesions are seen in the setting of \( T. \text{gondii} \) seropositivity and a low CD4 cell count, a presumptive diagnosis of toxoplasmosis encephalitis is made and therapy is initiated accordingly. The response to therapy should be prompt, with 80% of patients responding within 7 to 10 days (2). Failure to respond rapidly should prompt an investigation of alternative diagnoses.

Chagas disease, caused by the protozoan \( T. \text{cruzi} \), is endemic to Mexico and Central and South America. Reactivation of latent infection is a well-described phenomenon in immunocompromised patients, and the central nervous system is the predominant site of involvement. The typical clinical presentation includes delirium, fever, headache, vomiting, and focal neurologic deficits. The most common radiographic finding is single or multiple contrast-enhancing tumor-like lesions of the brain. Chagas disease of the central nervous system cannot be distinguished from toxoplasmosis encephalitis by radiographic or clinical features. However, fulminant disease with a relevant exposure history should raise clinical suspicion and lead to further diagnostic evaluation if the patient does not respond to therapy for toxoplasmosis (3).

Several strategies can be used to diagnose reactivation of Chagas disease, including brain biopsy and careful pathologic examination to distinguish \( T. \text{gondii} \) from \( T. \text{cruzi} \) amastigotes, direct microscopic visualization of parasites in blood and cerebrospinal fluid, polymerase

Figure. Diagnostic test findings in a patient with chagasic encephalitis.

Left. Contrast-enhanced, T1-weighted magnetic resonance imaging (axial cut) of the brain showing multiple ring-enhancing lesions. Right. Brain biopsy showing rod kinetoplasts indicative of the amastigote form of \( T. \text{cruzi} \).
chain reaction of cerebrospinal fluid, and indirect fluorescent antibody testing of the serum buffy coat for T. cruzi.

Conclusion: Our case of reactivation of Chagas disease in an HIV-infected individual is the fourth reported in the United States; however, the incidence is expected to increase as more persons immigrate to the United States from Latin American countries (4, 5). Early diagnosis, facilitated by a high index of suspicion for Chagas disease in certain populations, is critical to infected patients’ survival.

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Potential Financial Conflicts of Interest: None disclosed.

References

Correction: Report of Specific Cardiovascular Outcomes of the ADVANTAGE Trial

We recently published a letter by Egilman and Presler (1) that cited a letter from an official at Merck & Co., Inc. (Whitehouse Station, New Jersey) to the U.S. Food and Drug Administration (FDA) (2). The cited letter reported that, according to Merck’s adjudication of adverse events, 6 patients had a myocardial infarction and 1 patient died suddenly while receiving therapy with Vioxx (rofecoxib, Merck & Co., Inc., Whitehouse Station, New Jersey) during the ADVANTAGE (Assessment of Differences between Vioxx and Naproxen To Ascertain Gastrointestinal Tolerability and Effectiveness) trial (3). Dr. Braunstein, another Merck official, reviewed the cited letter at our request and informed us that it contained an error. The correct numbers of adjudicated events were 5 myocardial infarctions and 2 sudden deaths; Merck submitted materials to the FDA (along with the letter containing the error) that documented these statistics. The correct number of events was also reported in Table 2 of a previously published letter from Dr. Braunstein (4), which Egilman and Presler also cited (1).

References