

Teachable Moment | LESS IS MORE

Blind Obedience and an Unnecessary Workup for Hypoglycemia

A Teachable Moment

Elizabeth Y. Wang, BA; Lauren Patrick, MD; Denise M. Connor, MD

Story From the Front Lines

A man in his 70s with alcoholic cardiomyopathy with an ejection fraction of 20%, an automatic implantable cardiac defibrillator, and atrial fibrillation was admitted for increasingly frequent episodes of lightheadedness. His fingerstick blood glucose levels (FSBGs) in the hospital were between 60 mg/dL and 70 mg/dL during periods of lightheadedness (to convert to mmol/L, multiply by 0.0555). He was neither diabetic nor taking medications known to cause hypoglycemia. His symptoms did not improve with glucose administration. Aside from lightheadedness, he had no sweating, nausea, or other typical symptoms of hypoglycemia. Repeatedly low FSBGs prompted evaluation for hyperinsulinemia.

While fasting overnight for his insulinoma evaluation, the patient had a FSBG of 45 mg/dL, decreasing to 30 mg/dL thirty minutes later, despite treatment with 8 oral glucose tablets. At the time of his lowest FSBG, a simultaneous venous plasma glucose level was 80 mg/dL. His FSBG increased to 85 mg/dL after warming his hands.

Given the discrepancy between the patient's FSBG and simultaneous venous glucose levels, systemic hypoglycemia was excluded as a cause of his lightheadedness, which was instead ascribed to end-stage heart failure. Low FSBGs were attributed to sluggish peripheral perfusion owing to advanced cardiomyopathy, leading to decreased blood flow with continued peripheral tissue glucose consumption. He was treated for fluid overload and discharged after improvement in his lightheadedness.

Teachable Moment

Internists frequently encounter patients with low FSBGs. In patients taking glucose-lowering medications, low FSBGs should be treated promptly to avoid neuroglycopenia. However, clinically important hypoglycemia is uncommon in nondiabetic patients,¹ and evaluation for a hypoglycemic disorder in these patients should only occur if the Whipple triad is met: symptoms are present that can be explained by hypoglycemia, the glucose concentration is low when symptoms are present, and symptoms are relieved by glucose or glucagon. Low FSBGs in nondiabetic patients should prompt consideration of the causes of hypoglycemia including critical illness, malnutrition, adrenal insufficiency, and endogenous hyperinsulinemia, as well as "pseudohypoglycemia" (also called "artifactual hypoglycemia"), which encompasses 2 phenomena.² In the form seen in this patient, FSBGs accurately reflect glucose levels in the microcirculation, but are considerably lower than the systemic plasma glucose owing to sluggish capillary blood flow. This form may occur with shock, peripheral vascular disease, cyanotic heart disease, acrocyanosis, Raynaud phenom-

enon, or scleroderma.² The other type can occur with leukemia, polycythemia, and Waldenström macroglobulinemia, owing to in vitro glycolysis in the collection tube.²

This patient's lack of improvement after glucose supplementation led us to check a simultaneous venous blood glucose, revealing pseudohypoglycemia. However, our reliance on repeatedly low FSBGs prompted an avoidable insulinoma work-up, requiring transfer to a higher level of care for supervised fasting and overnight glucose checks, increasing our elderly patient's risk for hospital-acquired delirium. This evaluation diverted attention from his end-stage heart failure, the true cause of his lightheadedness, delaying appropriate treatment.

In patients with inadequate tissue perfusion owing to trauma, sepsis, cardiogenic shock, or severe dehydration, FSBGs are frequently lower than venous measurements. One-third of hypotensive emergency department patients were incorrectly diagnosed as hypoglycemic via FSBG.³ Pseudohypoglycemia is uncommon in noncritically ill patients like this one; however, this case demonstrates the more generalizable risk of uncritically accepting laboratory results.

"Blind obedience" is a cognitive bias that occurs when clinicians overvalue the weight of an authoritative source, such as laboratory results or imaging, making it difficult to recognize or act on conflicting findings.⁴ Our patient's nurse remarked that his hands were always cold, a clue suggesting the mechanism for his pseudohypoglycemia, yet our overconfidence in his FSBGs prevented us from questioning the accuracy of these measurements. Just as overconfidence in abnormal laboratory results blinded us to clinical clues and led to an unnecessary evaluation, overreliance on normal results can falsely reassure clinicians, delaying accurate diagnoses.

Diagnostic errors are estimated to occur in 10% to 15% of encounters, with cognitive errors playing a role in many cases.⁵ Attention to base rates of disease may have lowered our risk of blind obedience. When insulinoma, a rare diagnosis, appeared on our differential, critical appraisal of the evidence pointing us toward it may have prompted us to consider the more common possibility of misleading test results. A critical mindset, triggered by the low pretest probability of an insulinoma, may also have called earlier attention to our patient's lack of improvement with glucose supplementation, a red flag indicating that Whipple's triad was not met.

While it is unrealistic to expect to have detailed knowledge of every test's range of performance, understanding the limitations of common laboratory tests may also reduce errors. Many factors can result in misleading laboratory results including inherent test characteristics (ie, poor sensitivity or specificity), patient charac-

teristics, assay performance at extremes, or sampling and storage. When inconsistent with clinical findings, even repeatedly abnormal laboratory results should be questioned. An apprecia-

tion of the fallibility of objective tests and of the risk of blind obedience may prevent unnecessary, costly, and potentially harmful treatment.

ARTICLE INFORMATION

Author Affiliations: School of Medicine, University of California San Francisco, San Francisco (Wang); Department of Neurology, University of California San Francisco, San Francisco (Patrick); Department of Medicine, University of California San Francisco, San Francisco (Connor); Division of Hospital Medicine, San Francisco VA Medical Center, San Francisco, California (Connor).

Corresponding Author: Denise M. Connor, MD, Associate Professor of Clinical Medicine, University of California San Francisco, San Francisco VA Medical Center, 4150 Clement St, San Francisco, CA 94121 (denise.connor@ucsf.edu).

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