

We try to publish authors' responses in the same edition with readers' comments. Time constraints might prevent this in some cases. The problem is compounded in a bimonthly journal where continuity of comment and redress are difficult to achieve. When the redress appears 2 months after the comment, 4 months will have passed since the article was published. Therefore, we would suggest to our readers that their correspondence about published papers be submitted as soon as possible after the article appears.

Hyponatremic Seizures After Ultrasonic Imaging

To the Editor: Gopal and Blum¹ report a patient developing hyponatremic seizures after orally ingesting hypotonic fluid in preparation for an ultrasound examination. The authors state: "We doubt that our patient had chronic underlying SIADH [syndrome of inappropriate antidiuretic hormone secretion] because her serum electrolytes were normal when measured in the office 4 months earlier." In other words, they must postulate that a hypotonic fluid load can somehow trigger either central secretion of antidiuretic hormone (ADH) or impair the diluting ability in the kidney.

The patient's laboratory studies are clearly compatible with SIADH at the time she was admitted after the fluid load. Her urine osmolality was 382 mOsm/kg with a concurrent serum osmolality of 242 mOsm/kg, while her kidneys were not avidly holding onto sodium and while she was clinically euvolic.

A fluid load, in and of itself, could not be expected to cause this sort of defect in renal concentrating ability or an inappropriate secretion of ADH. In fact, 5 days later, when the patient's serum osmolality was approaching the normal range (284 mOsm/kg), her urine osmolality was 338 mOsm/kg, which still fails to show that she could appropriately dilute her urine. These latter results remain completely consistent with (although not diagnostic of) continued SIADH or renal concentrating defect. The authors' contention that "the SIADH resolved after appropriate water restriction . . . on the fifth hospital day," is not verifiable by the laboratory results reported. One would need a dilute urine reading, which was not reported, to support this contention.

The authors state that "SIADH is diagnosed by hyponatremia and elevated urine osmolality when compared with serum osmolality in a patient with normal levels of circulating blood plasma." Although technically correct, this definition is open to misinterpretation. Specifically, it can be interpreted to mean that the urine osmolality must be numerically greater than the serum osmolality ("elevated . . . when compared with"). In fact, such is actually the case in the patient reported both initially and at discharge, although this is not always the case. A better statement is that, in a patient who is euvolic, a urine osmolality that is too high for the clinical

picture is diagnostic of SIADH. Rephrased, in the presence of hypotonic serum and in the absence of hypovolemia (confirmed by urinary sodium concentration), a urine osmolality that is anything other than maximally dilute is suggestive of SIADH. For example, in the reported patient, with a serum osmolality of 248 mOsm/kg, any urine osmolality of greater than 120–150 mOsm/kg would be suggestive of SIADH. As quoted from *Williams Textbook of Endocrinology*, "Nonosmotic water conservation in SIADH and in volume contraction is recognized by the presence of a urine osmolality value higher than 120 to 150 mmol/L in association with reduced serum osmolality."² In SIADH, the elevation in urinary osmolality is an elevation relative to the clinical situation, not the serum osmolality per se, and anything higher than 100 mOsm/kg in someone with hyponatremia who just drank 6 L of fluid would be highly suggestive of SIADH.

A better title of the article would have been "Conversion of asymptomatic (or inapparent) to symptomatic (or apparent) SIADH by fluid loading for pelvic ultrasonography." The numbers presented do not make a convincing case that the patient was able to appropriately excrete a free water load.

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References

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2. Reeves WB, Bichet DG, Andreoli TE. Posterior pituitary and water metabolism. In: Wilson JD, Foster DW, Kronenberg HM, Larsen PR, editors. *Williams textbook of endocrinology*. 9th ed. Philadelphia: W B Saunders, 1998: 376–7.

The above letter was referred to the authors of the article in question, who offer the following reply.

To the Editor: We would like to thank Dr. Fox for his spirited critique of our case study. After receiving his letter, we checked our original manuscript and noted that the patient's serum sodium level at the time of discharge from the hospital was omitted from the published version. The patient went home 5 days after admission with a serum sodium of 139 mEq/L.

We contacted the patient's family physician and verified that the patient is still asymptomatic nearly 30 months after the incident. Although the physician has had no need to check the urine chemistries of this patient, he has assured us that the patient has not had any similar signs or symptoms of SIADH since her hospitalization (J Rogers, personal communication, 19 December 2000).

Table 1. Positive Cultures for Methicillin-Resistant *Staphylococcus aureus* (MRSA).

Symptom	Grain Stain Leukocytes	Culture Result
Ear discharge	Absent	MRSA coagulase-negative staphylococci, diphtheroids
Surgical wound	Few*	MRSA, diphtheroids, <i>Streptococcus viridans</i>
Abdomen wound	Absent	MRSA, streptococcus group B, coagulase-negative <i>Staphylococcus</i>
Eye discharge	Absent	MRSA, (few, absent on Gram stain)
Eye discharge	Absent	MRSA, <i>Streptococcus viridans</i> , diphtheroids
Ear discharge	Absent	MRSA, <i>Pseudomonas aeruginosa</i> , <i>Proteus mirabilis</i>

*Presence of a few leukocytes without pus in a healing wound is not important.

Overall, we feel that Dr. Fox might have made several errors in his otherwise insightful analysis. We do not believe that the patient had asymptomatic SIADH prior to admission. The patient did not meet the criteria for SIADH, either before admission or at discharge, because she was not hyponatremic (serum sodium 135 mEq/L).

In a recent review article, Kugler and Husted¹ listed the diagnostic criteria for SIADH: (1) hypotonic hyponatremia, (2) urine osmolality 100 mOsm/kg, (3) absence of extracellular volume depletion, (4) normal thyroid and adrenal function, and (5) normal cardiac, hepatic, and renal function. Because this patient had a normal serum sodium level both before and after admission (142 mEq/L and 139 mEq/L), she cannot be classified as having SIADH, as Dr. Fox contends.

Dr. Fox also errs in stating that a hypotonic fluid load, in and of itself, cannot impair the concentrating ability of the kidney. After submission of our case report, a review of hyponatremia by Adroque and Madias² was published that would appear to refute this contention. The authors note that “if water intake exceeds the capacity of the kidneys to excrete water, dilution of body solutes results, causing hypo-osmolality and hypotonicity. . . . Hypotonicity, in turn, can lead to cerebral edema, a potentially life-threatening complication.”

We make no presumptions as to the cause of this patient’s acute onset of SIADH but believe that it was due to no other reason than hypotonic fluid load overwhelming the ability of the kidney to excrete free water. We doubt that an acute fluid load can trigger central secretion of ADH but instead believe that the ability of the kidney to excrete a hypotonic bolus of fluid reaches a limit at some point—clearly less than the 6 L that this patient ingested within 1 hour. Further research is needed to elucidate the complex endocrine and metabolic pathways at work in this process. The main purpose of our case report was to warn of this phenomenon and possibly prevent it from occurring again.

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References

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Pseudo Outbreak of MRSA

To the Editor: An outbreak refers to the occurrence in a given population of cases of an illness clearly in excess of the usual expectancy. This report illustrates how misinterpretation of laboratory reports can create a pseudo outbreak!

In one unit of 36 residents in a large developmental center (850 residents), a culture sample was taken from 6 (16%) patients with various symptoms (Table 1). Methicillin-resistant *Staphylococcus aureus* (MRSA) grew in all. Upon learning of this “exotic bug,” the residents’ parents became alarmed and demanded isolation of the “infected” residents.

Further investigation showed that no resident was actually “infected” by MRSA. No patient had classic local (pus) or systemic (fever, malaise, leukocytosis) signs of *S aureus* infection.¹ Gram stain showed no leukocytes, meaning that the pathogen had not invaded tissues to activate host defense. Multiple organisms grew in 5 (83%) cultures; the sixth culture had scant MRSA, which suggested contamination. All samples were from unsterile exterior body surfaces, rich in resident flora.² The residents have mental retardation and might be heavily colonized because of suboptimal personal hygiene, despite the caretakers’ best efforts. Generally, MRSA cannot be even diagnosed in such cases, as laboratories perform no antibiotic sensitivities for mixed growths of uncertain clinical importance.³ Nevertheless, sensitivities were performed in our cases, because the physician specifically wanted to rule out MRSA as a result of overzealous concern about the antibiotic resistance in this institutionalized population.

All residents improved with topical treatment. Their symptoms were probably related to a mechanical (scratching, rubbing by self-mutilating residents), anatomic (poor drainage of natural secretions because of a deformed external ear canal or nasolacrimal duct), or viral cause. We educated parents and staff about MRSA and emphasized standard infection control procedures, including frequent handwashing for residents and employees. Isolation was considered to be futile and unnecessarily restrictive to the residents’ developmental needs.⁴

S aureus is more frequently a colonizer than an invader. It colonizes the nose or skin in 20% to 40% of healthy adults.⁴ With frequent antibiotic exposure, it might become resistant to methicillin. Our residents are more likely to receive antibiotics because of a predisposition to infections (severe anatomic deformities, aspiration, pica, unhygienic habits) and easy physician access. It is likely that many of our residents are colonized by MRSA.

Proper interpretation of Gram stain and culture results is crucial for sound practice. Isolation of an organism on culture should not be taken at face value. Clinicians must differentiate between colonization (positive culture without infection) and infection (clinical disease, leukocytes in the Gram stain, and isolation of the pathogen in pure culture.)¹

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