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MORNING REPORT

Is it Really Just a Number?

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A 77-year-old woman presents to the emergency department with altered mental status. There is no immediate family member available for further history. The patient does not know what medical conditions she has, what medications she is taking, or the details of her social and family history.

The differential diagnosis for altered mental status is broad. It includes infectious causes, such as urinary tract infection, pneumonia, meningitis, and endocarditis; metabolic conditions, such as hypoglycemia, hyper/hyponatremia, hypercalcemia, hypoxia, hypercapnia, hepatic encephalopathy, uremic encephalopathy, drug intoxication or withdrawal, and Wernicke encephalopathy; intracranial events, such as cerebrovascular accident, intracranial hemorrhage, primary or metastatic tumors, and encephalitis; cardiac events, such as arrhythmias, heart failure, and hypertensive emergencies; and vasculitis. The differential diagnosis of altered mental status is based on historical and physical findings. It is especially important to distinguish delirium from dementia.

Unfortunately, in this case, no collaborative information is available, and the patient is not able to provide any additional history. Therefore, physical exam and laboratory and radiology data will have to play a more critical role in this patient assessment.

On physical exam, the patient is drowsy and oriented to self only. The patient is afebrile with a pulse of 80, blood pressure of 130/70, respirations of 25, and oxygen saturation of 97% on room air. She has moist mucus membranes, clear lungs to auscultation, a regular heart rate and rhythm without murmurs, a soft nontender abdomen without hepatosplenomegaly, and no edema of the extremities. She has bruises on her forehead and knees. Her neurological exam is nonfocal.

There is clear evidence of altered alertness in this patient. Her exam is nonfocal, making stroke or tumors less likely. She does have tachypnea, which especially in the elderly can be a sign of sepsis syndrome even without fever. Tachypnea can also be present in pulmonary embolism, hyperthyroidism, anxiety, and respiratory acidosis. Electrolyte and metabolic abnormalities, especially with the tachypnea, will need to be ruled out. I would recommend a full basic metabolic panel, complete blood count, and arterial blood gas if the basic metabolic panel is abnormal. The bruises on the forehead and knees could indicate a problem with platelets or abnormal coagulation (e.g. from warfarin, liver disease), so a head CT to rule out bleed and INR, PT, and PTT should be obtained.

The patient's labs are as follows: sodium 138, potassium 3.7, chloride 116 (H), bicarbonate 16 (L), creatitine 0.8, bun 26; CBC, AST, ALT, alkaline phosphatase, albumin, and PT/INR/PTT are normal. Arterial blood gas reveals a pH 7.45, pCO_2 26.1 (L), calculated HCO_3 16.0 (L), and pO_2 97.

The patient has a mixed-acid base picture with a pH in the normal range but with a low serum bicarbonate and low serum CO_2. With the PCO_2 less than 40, this patient has a respiratory alkalosis. However, in order to determine if there is a secondary disorder, we need to calculate the corrected bicarbonate, which is equal to the actual HCO_3 plus the anion gap minus 12. If the corrected bicarb is more than 30, then there is a primary metabolic alkalosis. If the correct bicarb is 23 to 30, then there is no additional disorder. If the corrected bicarb is less than 23, then a hidden metabolic acidosis exists. In our case, the bicarb is 16 and the anion gap (Na-(Cl+HCO_3)) is 6, so the corrected bicarb is 10. Therefore, our patient has a respiratory alkalosis with a non-anion gap metabolic acidosis.

The differential diagnosis for a normal anion gap metabolic acidosis is divided into two groups: GI losses, such as diarrhea, fistulae, or ileal loop, and renal causes, such as renal tubular acidosis, carbonic anhydrase inhibitor use, or post hypocapnia. The differential diagnosis of respiratory alkalosis includes hypoxemia, stimulation of pulmonary or pleural receptors (i.e. pneumonia, pulmonary embolism, pulmonary edema, asthma), psychogenic hyperventilation, medications (e.g. theophylline, catecholamines, salicylates, and progesterone), CNS disorders, fever, early sepsis, and increased minute ventilation secondary to ventilator management. The differential diagnosis can be narrowed if both respiratory alkalosis and metabolic acidosis are present, and it includes gram-negative sepsis, acute cardiopulmonary arrest, severe pulmonary edema, and salicylate intoxication.

This patient has a negative head CT. Urine toxicology is negative. Salicyclate level is high at 88 mg/dL. (Therapeutic level is 10 to 30 mg/dL.) When the patient's family arrives, they explain that the patient was consuming 20 baby aspirins a day for three months for osteoarthritis.

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The patient is transferred to ICU. A bicarbonate drip is started to alkalinize the urine; however, the salicylate level continues to increase to a peak of 98 mg/dL. Emergent hemodialysis is initiated. After two sessions of hemodialysis, the salicylate levels normalize.

Management of salicyclate toxicity consists of: 1) detoxification with activated charcoal; 2) alkalinization of urine, although hypokalemia must be corrected in order for alkalinization to be effective; and 3) hemodialysis when the patient has altered mental status, pulmonary or cerebral edema, renal insufficiency, fluid overload, a serum salicylate level greater than 100 mmol/L in acute overdose, or clinical deterioration despite aggressive supportive care.

Aspirin is a commonly used medication and is frequently associated with intentional and suicidal ingestion. The classic presentation of salicylate toxicity is anion gap metabolic acidosis secondary to salicyclic acid, lactic acid, and ketones in addition to respiratory alkalosis and respiratory acidosis. Our patient had an anion gap of 6 with hyperchloremia (116). Renal, central, and other causes of hyperchloremia were ruled out with history, physical, and labs. On review of prior case reports, similar elevation of serum chloride has been reported and is a lab error due to high concentration of salicylate.[1] Our lab uses the Roche Cobas Integra analyzer, which has been known to cause falsely elevated chloride levels in the presence of elevated salicylate levels.[2]

Take Home Points

1. Aspirin toxicity can present as non-anion gap metabolic acidosis.
2. Lab results can "over-measure" serum chloride, explaining "pseudo-normal" anion gap in the setting of salicylate toxicity.

References

1. Jacob, J, Lavonas, EJ. Falsely normal anion gap in severe salicylate poisoning caused by laboratory interference. J Ann Emerg Med 2011; 58(3):280-1.
2. Mori L, Waldhuber S. Salicylate interference with the Roche Cobas Integra chloride electrode. Clin Chem 1997; 43:1249-50. SGIM