K4 False Low Bicarbonate Level With Propofol Infusion and Hypertriglyceridemia

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The goals of this presentation are to: 1) explore the association of Propofol with fatal metabolic acidosis, 2) use the Henderson-Hasselbach equation to resolve discrepancies in acid-base analysis, and 3) recognize interfering substances that may give rise to false lab results.

A 72-year-old male who was admitted with respiratory distress and confusion was found to have a right upper lobe lung mass and hypercalcemia. His measured CO_2 level on the 1st and 2nd days of hospitalization were 25 and 23 mmol/L, respectively. On the 2nd day, he was intubated for worsening respiratory status, administered levofloxacin for presumptive pneumonia, and administered Propofol for sedation. His arterial blood gases after intubation showed a pH 7.38, PCO_2 38, PO_2 143 and his measured CO_2 26 was mmol/L. Over the following 4 days, his measured CO_2 progressively decreased to 8 mmol/L with an anion gap of 419, negative ketones, and normal serum lactate with no corresponding significant changes in his arterial blood gases. On the 7th day of hospitalization, he received lipid infusion with total parentral nutrition. A grossly lipemic serum specimen showed a CO_2 level of 3 mmol/L. Propofol was discontinued. Four hours later, a 2nd lipemic specimen showed, after ultracentrifugation to remove the chylous material, a CO_2 level of 21 mmol/L. A lipid panel showed a triglyceride level of 4426 mgldL. The patient's condition continued to deteriorate and he died later on the 7th day. At autopsy, the cause of death was poorly differentiated small cell carcinoma in the right upper and middle lung lobes with liver and lymph node metastasis.

Propofol® is a short-acting anesthetic agent. It is a hydrophobic compound, which is formulated in a lipid emulsion (Intralipid) to facilitate intravenous use. Several cases have been reported in which an association between the use of Propofol and a clinical presentation of metabolic acidosis, cardiac dysrhythmias, and lipemia has been suggested. Some of these cases were complicated by fatality. Most of these fatal cases involved children who were ventilated for laryngotracheobronchitis. The cause of metabolic acidosis in these cases was not determined. It was also suggested that the Intralipid in the Propofol preparation might interfere with lactate metabolism in the liver causing accumulation of lactate and acidosis.

In this case, there was a consistent, progressive decrease in the measured serum bicarbonate level during Propofol infusion. However, the patient acid-base status, as simultaneously measured by arterial blood gases did not show a corresponding change that would match the very low level of serum bicarbonate. There was also a marked hypertriglyceridemia that may be related to both Propofol infusion and lipid infusion for nutritional support. After ultracentrifugation of the serum, the measured bicarbonate level in the supernatant returned to the patient's baseline value prior to Propofol and lipid administration. That bicarbonate value was consistent with the patient's acid-base status measured by arterial blood gases. Ultracentrifugation removes the chylous material from serum. It may also remove Propofol from serum since it's a hydrophobic compound. Neither Propofol nor hypertriglyceridemia have been reported as a potential interfering substance with serum bicarbonate assays. In most of the previously reported cases, metabolic acidosis, dysrhythmias, cardiac failure, and death have been related to Propofol by exclusion of all other causes. No conclusive evidence has been reported to prove or disprove this association. This is the first report suggesting that low bicarbonate level associated with Propofol infusion may be largely due to an interfering substance or substances with the bicarbonate assay. Further studies are needed to determine the role of Propofol and hypertriglyceridemia in serum bicarbonate measurement.

Propofol, Fetal Metabolic Acidosis, Bicarbonate

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