not mention thiamine deficiency as an important cause of lactic acidosis.

In 2004, Klein et al. described thiamine deficiency as a cause of fatal metabolic acidosis.1 Their patients were Thai workers who lived in Israel and ate polished rice. Since then, we have also treated several patients with thiamine deficiency that has caused life-threatening lactic acidosis. These patients were either workers from the Far East or malnourished persons with alcoholism.

Thiamine deficiency can be responsible for a number of serious medical problems, none more so than lactic acidosis. Thiamine is a cofactor in the normal functioning of pyruvate dehydrogenase, and with its deficiency, lactate accumulates, causing lactic acidosis.2 Fortunately, early therapy with intravenous thiamine is lifesaving.3

Thiamine deficiency should also be considered in infants who are receiving parenteral nutrition without adequate thiamine supplements.4

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To the Editor:


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To the Editor: We would like to emphasize three peculiarities of metformin-induced lactic acidosis. First, hypoglycemia is not the rule, possibly because metformin primarily inhibits the endogenous overproduction of glucose, with a minor effect on peripheral consumption.1 Second, venous hyperoxia is common, since mitochondrial respiration is globally inhibited.2,4 As a consequence, oxygen extraction decreases and venous oxygen content increases. Third, the outcome is usually favorable.2,4

We have reviewed the data sheets of 17 patients (13 women and 4 men; mean ±SD age, 68±8 years) with metformin intoxication (mean serum drug level, 52±26 μg per milliliter) and lactic acidosis (mean arterial pH, 7.03±0.18, and mean lactate level, 18±9 mmol per liter) in whom central or mixed venous blood oxygen-saturation levels were monitored.2,4 The initial mean blood glucose level was 117±84 mg per deciliter (6.5±4.7 mmol per liter) (only 6 patients had a
glucose level of <80 mg per deciliter (<4.4 mmol per liter]) and the mean venous oxygen saturation was 80±10% (normal value, 65 to 70%). Three patients (18%) died in the hospital.

In line with these findings, the patient presented in the case had an initial blood glucose level of 116 mg per deciliter (6.4 mmol per liter), venous partial pressure of oxygen of 73 mm Hg, and a good clinical course.

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THE DISCUSSANT REPLIES: Consistent with the comments by Goldman and Shtaynberg, methanol or ethylene glycol toxicity cannot be ruled out exclusively on the basis of a narrow osmolar gap. After initial exposure to these alcohols, the osmolar gap may decrease over time as parent alcohol is converted into other metabolites and hydrogen ions, leading to changes in the anion gap. This patient’s multiple-organ involvement, lactic acidosis, and negative toxicologic screening make such alcohol toxicities less likely. Whereas fomepizole is usually well tolerated, data to support its use are inconclusive.

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