

LETTER TO THE EDITOR**Toxicology**

No evidence for propylene glycol toxicity due to amylophagia

Dear Editor,

We read with interest the case report by Downey et al¹. However, the authors fail to corroborate their speculation that propylene glycol (1,2-propanediol; PG) was the cause of her elevated lactate at the second hospital. We believe that the history of chronic cornstarch ingestion (amylophagia) is a “red herring.”

PG toxicity includes high anion gap metabolic acidosis, hyperosmolality, acute kidney injury, and elevated lactate *without other explanation*.^{2–4} However, other conditions—such as diabetic ketoacidosis and shock—have similar features. The diagnosis requires a verified PG exposure and, ideally, laboratory detection of PG. This case has neither of these.

The authors assert without any reference that “cornstarch contains PG.” The authors cite another case report mentioning a call to an unidentified cornstarch manufacturing company stating they do “not have specific data regarding PG content in our corn starch.”⁵ The two leading brands of cornstarch in the US are Argo® (ACH Food Companies, Inc., Oakbrook Terrace, IL) and Clabber Girl® (Clabber Girl Corp., Terre Haute, IN). Both display “100% pure” and list cornstarch as the only ingredient. These do not contain PG.

Her apparent rise in lactate concentration has an alternate explanation. Between the first lactate of 63.96 mg/dL (7.1 mmol/L) and the second lactate concentration of 173.69 mg/dL (19.3 mmol/L), she received 1 L of intravenous lactated Ringer’s solution (LR). Lactated Ringer’s solution contains 28 mmol/L of lactate.⁶ If the second blood draw came from the line used to infuse the LR, this would artificially increase the apparent lactate concentration.⁷

The reported serum osmolality from the referring hospital laboratory might be unreliable. Pires et al⁸ found that some hospitals could not easily measure osmolality on site. At least three hospital laboratories (among 38 respondents) acknowledged calculating osmolality but reporting it as “measured” osmolality. If the referring hospital used this practice, it would explain the reportedly normal osmolal gap at the referring emergency department (ED) followed by an increased osmolal gap at the children’s hospital without speculating about PG exposure. Shock, which became manifest in this case, can also elevate osmolality.⁹

The patient reported daily ingestion of “one box” of cornstarch for 6 months. There is no clear mechanism to explain how amylophagia abruptly caused her acute illness. The patient had acute chest pain,

shock requiring norepinephrine infusion, and a sharp rise in troponin concentrations. The authors offer no explanation for the troponin concentration peaking at 4832.90 ng/L—nearly 350 times the upper limit of normal (<14 ng/L). An acute viral illness with viral myocarditis would explain all the clinical and laboratory features of this case.

The present case has no measurement of PG and no evidence of PG in the cornstarch. Neither PG ingestion nor amylophagia caused her acute illness.

In the words of the fictional sleuth Sherlock Holmes, “It is a capital mistake to theorize before one has data. Insensibly one begins to twist facts to suit theories, instead of theories to suit facts.”¹⁰

CONFLICT OF INTEREST STATEMENT

The authors declare no conflict of interest.

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PRIOR PRESENTATIONS

There was no prior presentations by the authors on this topic.

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