

Objectives

Pseudohyperchloremia refers to a spuriously elevated blood chloride level. A rare finding, it can be associated with marked hyperlipidemia, bromide ingestion, and, in this case, salicylate toxicity.¹⁻⁵ Although of no intrinsic clinical significance, as a clinical sign and a confounder of gap acidosis calculations, it can have important clinical implications.

Case Description

A 32 year old woman with a complex psychiatric history and prior suicidal attempts presented after ingesting 4 bottles of aspirin. Aside from methamphetamine and opiates, she reported no other ingestions.

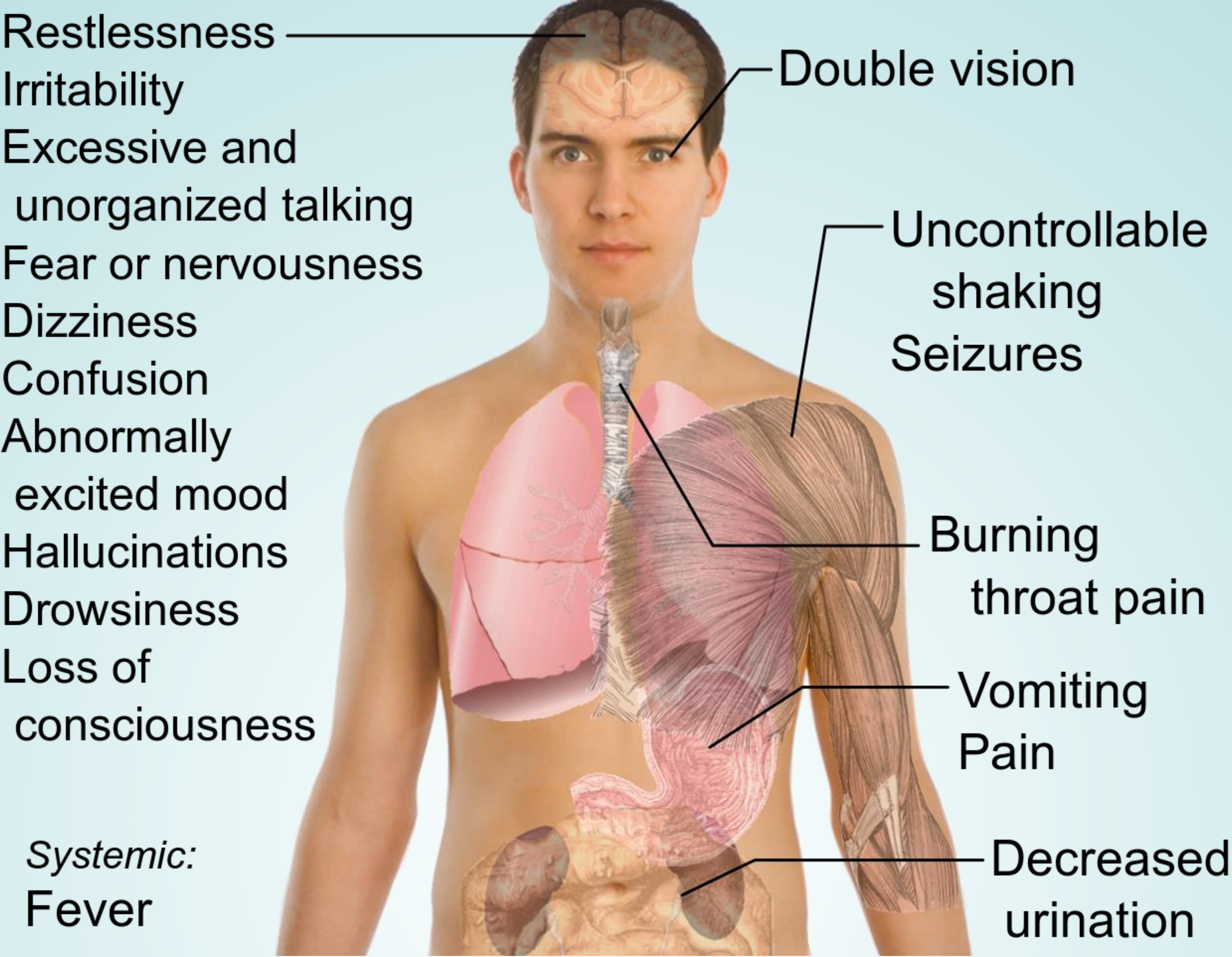
- At presentation to outside hospital
- Vitals: T: 36.1 HR: 110 BP: 140/100 RR: 26 SP02: 96%
 - Physical Exam: anxious, communicative, flushed and diaphoretic, nauseated, diffuse TTP abdomen
 - VBG: 7.46 / 23 / 112 / 16.7
 - Salicylate Level: 71.5
 - Chemistries: Na 147, Cl 109, Bicarb 17, Anion Gap 21

She later developed altered mental status and persistent acidosis despite three ampules of bicarbonate and 5% dextrose maintenance fluids; she received no chloride-containing products. Transferred to OHSU for urgent HD.

- Upon arrival to OHSU Medical ICU
- Vitals: T: 36.6 HR: 118 BP: 100/69 RR: 23 SP02: 100%
 - Physical Exam: agitated, disoriented, increasingly altered, diaphoretic, MMM, pupils minimally reactive, otherwise normal
 - VBG: 7.54 / 22 / 71 / 18.5
 - Salicylate Level: 117
 - Chemistries: Na 142, Cl 127, Bicarb 17, Anion Gap -2
 - Lactate: 0.6

For worsening mental status, she was intubated, given activated charcoal and underwent emergent hemodialysis. After dialysis her mental status returned to baseline; salicylate level was 24 mg/dL, chloride 109meq/L, anion gap 5 and normal blood gas. (The trend of her salicylate and serum chloride levels are graphed to the right.) On HD2, she was discharged to outpatient rehabilitation facility.

Symptoms of Aspirin overdose⁶

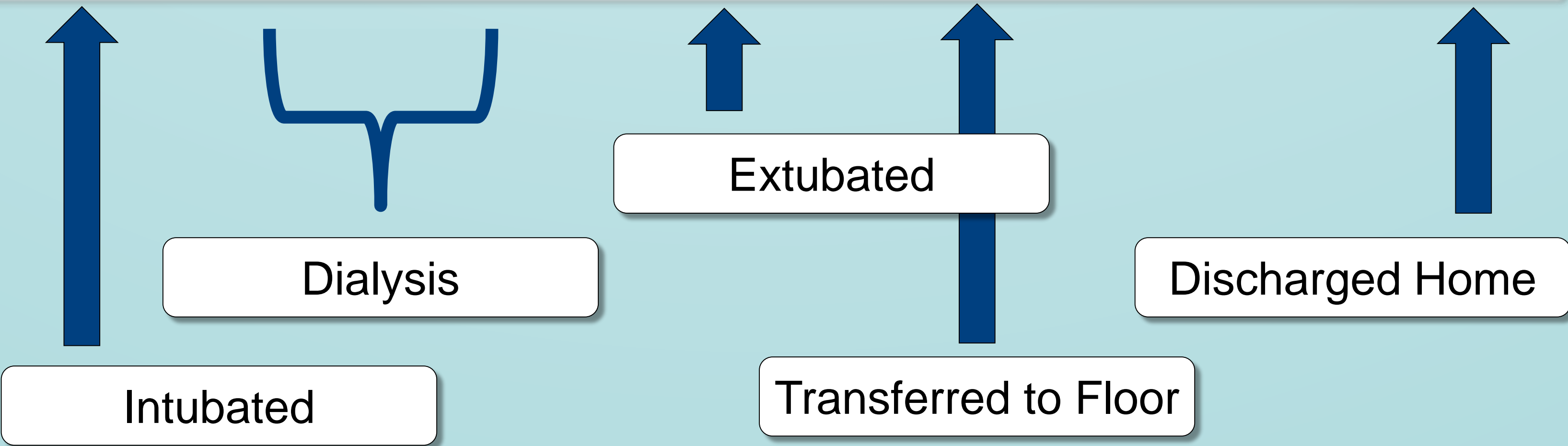
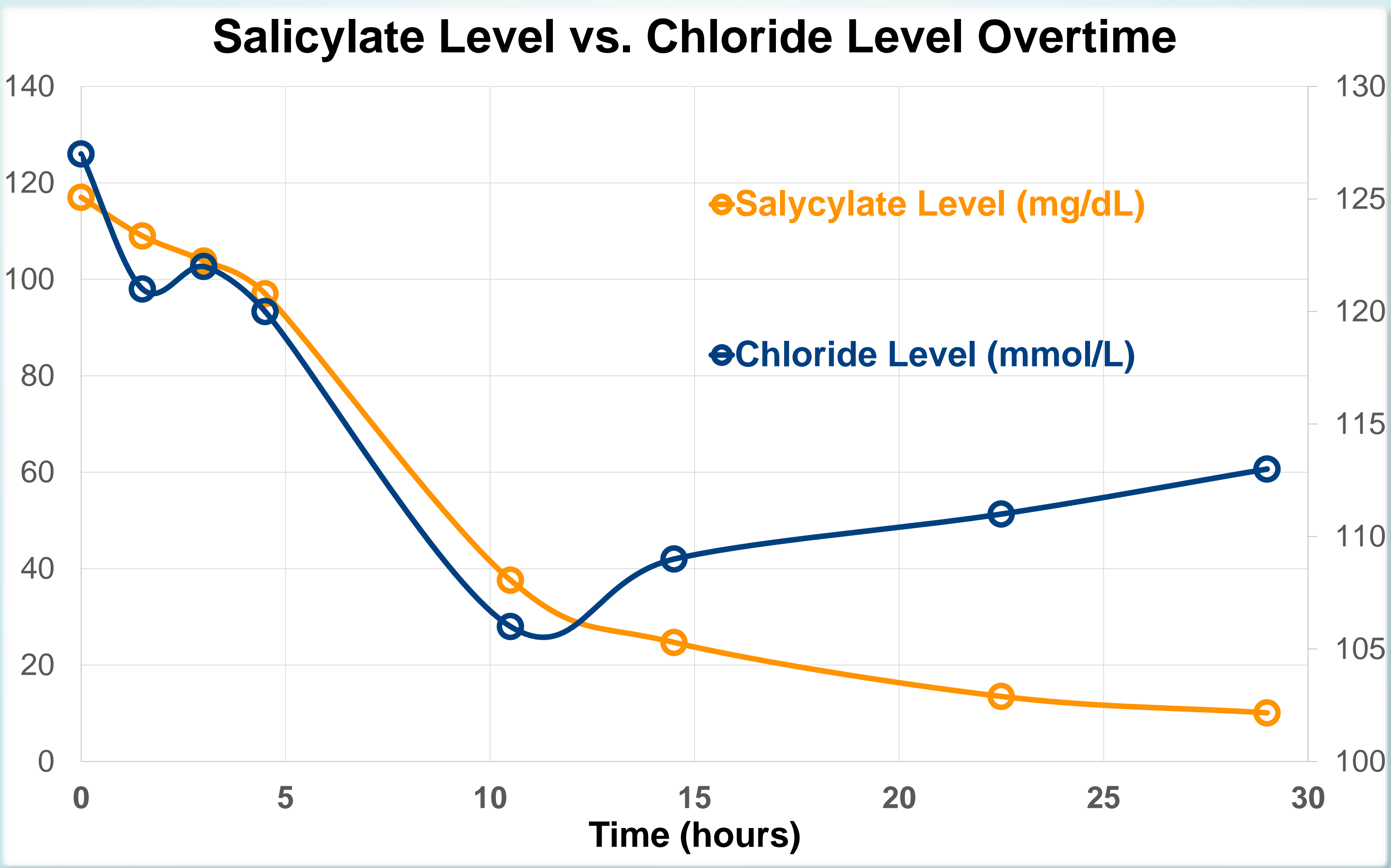


Discussion

This case illustrates an interesting phenomenon in salicylate intoxication—a falsely elevated serum chloride caused by salicylate interfering with ion-sensitive electrodes in many lab assays.³ While these spurious values have been previously reported,²⁻³ the clinical implications of this spurious value has not been fully explored. Though she had a negative anion gap, in itself physiologically impossible, the effect on artificially lowering the anion gap may provide false reassurance in patients presenting with normal anion gaps. Appropriate treatment with urgent dialysis may have been delayed because of an inaccurately low anion gap and normal pH, despite a clear mixed metabolic acidosis and respiratory alkalosis.

Conclusion

This lab error may also provide valuable diagnostic information in the undifferentiated patient with altered mental status. Severe hyperchloremia is an uncommon presentation, and most etiologies are either chronic (renal tubular acidosis, genetic diseases, kidney disease), iatrogenic (normal saline, medications) or ascertainable from clinical history (GI losses). There are few other syndromes that present with hyperchloremia and altered mental status—toluene ingestion, bromide ingestion and hypovolemic shock from diarrhea. While not all patients with salicylate intoxication present with pseudohyperchloremia, given the rarity of non-diarrheal causes of altered mental status changes and severe hyperchloremia, it may be a reasonably specific sign. This warrants further study as a case when information can be simultaneously valuable and untrue. In undifferentiated patients with altered mental status, the presence of severe hyperchloremia should place salicylate intoxication on the differential, and warrants obtaining a serum level.



References

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- 3) Mori L, Waldburger S. Salicylate interference with the Roche Cobas Integra chloride electrode. Clinical chemistry. July 1997;44:1111-1112.
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- 6) Salicylate poisoning. Wikipedia. https://en.wikipedia.org/wiki/Salicylate_poisoning. Published October 21, 2017