“Pseudobulimia”: A remarkable case of metabolic alkalosis and hypokalemia

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CASE PRESENTATION

Ms. S is a 29-year-old female with a past medical history of unspecified eating disorder with rapid intentional weight loss resulting in superior mesenteric artery (SMA) syndrome. This led to proximal bowel obstruction, for which she underwent open gastrostomy and jejunostomy approximately 2 months prior to presentation. She now receives feedings and medications through a jejunostomy tube and has an additional gastrostomy tube which functions to drain gastric secretions and oral pleasure feedings. Although clinically underweight prior to her surgery, her weight loss had progressed. She remained dependent on jejunostomy tube feedings with additional electrolyte supplementation including potassium bicarbonate.

After numerous hospital readmissions, our patient presented to the ED again with chief complaint of weakness and lethargy.

Pertinent physical examination:
- Cachexia
- BMI of 14.2.

LABS

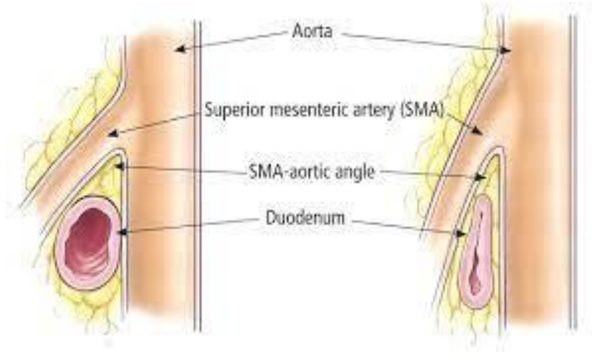
132	82	16	132
2.7	43	0.4	

(WNL prior to SMA surgery)

Urine Analysis: pH: 8.0

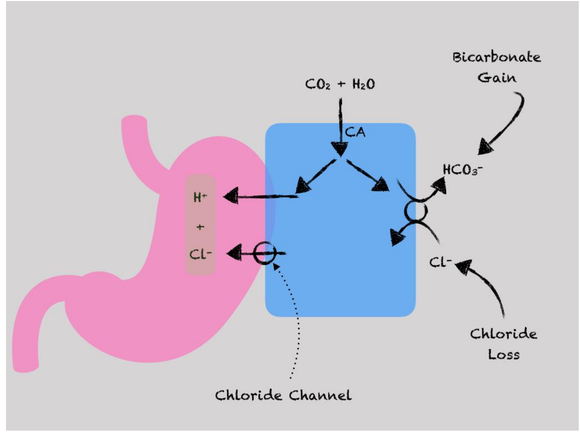
SMA SYNDROME

SMA syndrome can be seen with loss of the mesenteric fat pad surrounding the proximal duodenum. As an effect the SMA and aorta impinge on the duodenum, causing bowel obstruction. First line treatment is weight gain to regain mesenteric fat, although surgical intervention may also be necessary.



CHLORIDE SENSITIVE METABOLIC SLKALOSIS

This case displays chloride-sensitive metabolic alkalosis akin to what is seen in bulimia nervosa and cyclical vomiting syndromes. Loss of hydrochloric acid from vomiting, or in this case gastrostomy tube drainage, promotes generation of alkalosis due to volume contraction. Moreover, hypochloremia and hypokalemia are responsible for the maintenance phase of metabolic alkalosis due to secondary hyperreninemic hyperaldosteronism. This mechanism is inherently distinct from chloride-resistant metabolic alkalosis seen in primary hyperaldosteronism, Bartter Syndrome, Gitelman Syndrome, and Liddle Syndrome.



MANAGEMENT

In our patient, we used normal saline and potassium chloride to counteract her alkalosis with chloride supplementation. She responded well with resolution of alkalosis, hypokalemia and lethargy. For ease of outpatient management and keeping in perspective her nutritional needs, she was transitioned to short term TPN with potassium chloride and close electrolyte monitoring.

CONCLUSION

Recognition of stimuli for both the generation and maintenance phase of metabolic alkalosis will help in effective and prompt treatment with resolution as in our case.

REFERENCES

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