Case Report

Extreme Metabolic Alkalosis and Acute Kidney Injury in a 38-Year-Old Male Patient

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Abstract

Repeated vomiting may lead to profound loss of fluid and electrolytes. We describe a case with life-threatening acid-base disturbances due to vomiting. A 38-year-old man presented to an emergency department with weakness and decreased urine output after having vomited up to 20 times per day over a period of 7 days. Arterial blood gas analysis revealed a metabolic alkalosis with partial respiratory compensation. Initial management consisted of oxygen therapy and intravenous fluid therapy with normal saline and potassium chloride. To prevent further gastric losses of HCl, proton-pump inhibitors and metoclopramide were administered. The vomiting was caused most likely by a temporary duodenal stenosis due to portal hypertension of unknown etiology. In our opinion, this case demonstrates the successful management of a life-threatening condition by simple measures. Despite extensive diagnostic procedures, the effective treatment of the underlying condition consisted of watchful waiting.

Keywords: Duodenal stenosis, metabolic alkalosis, vomiting

Introduction

Repeated vomiting may lead to loss of fluid and electrolytes. We describe a case of a 38-year-old patient with repeated vomiting over a period of 7 days, leading to life-threatening acid-base disturbances.

Case Report

A 38-year-old man presented to an emergency department with progressive weakness and decreased urine output after having vomited up to 20 times per day over a period of 7 days. The patient was referred to our intensive care unit. Before medical consultation, he was healthy, and no other symptoms were reported. Diarrhea, fever, chills, or abdominal pain was absent.

Clinical examination revealed a patient of normal weight (height: 180 cm, weight: 70 kg). Blood pressure and heart rate were elevated (150/80 mmHg, 105/min); the respiratory rate was normal (13/min), but oxygen saturation was low (89%). During blood pressure measurement, a positive Trousseau sign was noted. The mucous membranes were dry, and the physical examination was otherwise unremarkable.

Arterial blood gas analysis revealed a metabolic alkalosis with partial respiratory compensation (pH: 7.71 [7.35–7.45], base excess: 40.5 mmol/l [-2-3 mmol/l], pCO_2: 56.1 mmHg [32–45 mmHg], pO_2: 43.5 mmHg [83–108 mmHg], bicarbonate: 72.2 mmol/l [21–26 mmol/l], lactate: 2.8 mmol/l [0.5–1.6 mmol/l], chloride: 52 mmol/l [98–106 mmol/l], sodium: 130 mmol/l [136–146 mmol/l], potassium: 2.4 mmol/l [3.5–5.1 mmol/l], ionized calcium: 0.77 mmol/l [1.15–1.29 mmol/l] and glucose: 165 mg/dl [70–105 mg/dl]).

Ultrasound revealed a distended and full stomach; the inferior vena cava was collapsing on inspiration. Otherwise, the ultrasound was unremarkable. The electrocardiography [Figure 1] showed a prolonged QT interval (QTc 547 ms).

Laboratory tests were consistent with acute kidney injury (creatinine: 5.2 mg/dl, urea: 176 mg/dl); liver function tests, coagulation tests, albumin, lipase, procalcitonin, total calcium, hemoglobin, and thyroid-stimulating hormone were normal. Leukocytes were minimally elevated (11500/µl).

We suspected a prerenal acute kidney injury and a hypochloremic metabolic alkalosis due to gastric losses of HCl and fluids.

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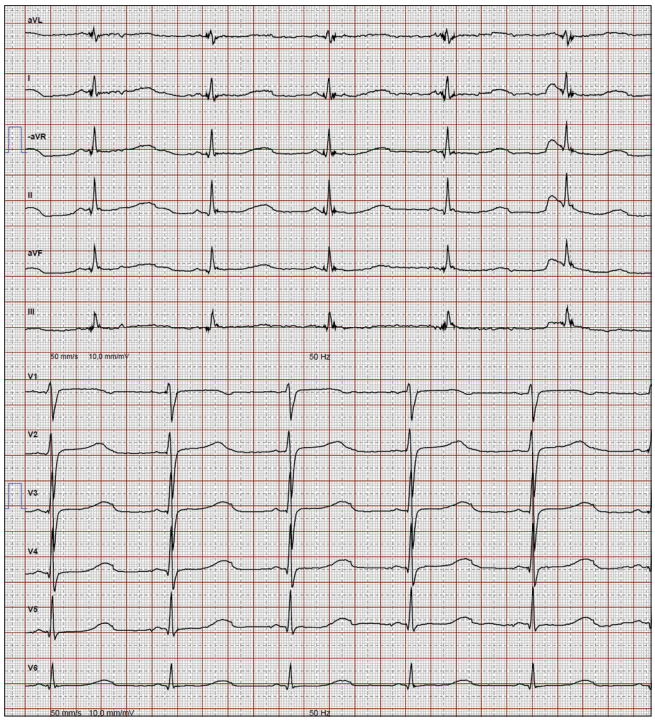

Figure 1: Electrocardiography on admission. The electrocardiography on admission showing an increased QT interval consistent with the electrolyte abnormalities

Initial management consisted of oxygen therapy to correct hypoxemia and intravenous fluid therapy with normal saline and potassium chloride. Initially, a balanced crystalloid solution was infused, and later normal saline with KCl (40 mmol/l) was used because of the higher chloride content. Metoclopramide was administered to promote gastric emptying. To prevent further gastric losses of HCl, a proton-pump inhibitor was given. The patient was put on nihil per os for 24 h.

After 24 h, a total of 4.5 l of crystalloid fluid was infused. There was no further vomiting. A repeated bedside ultrasound revealed an empty stomach. The pH was corrected to 7.49, bicarbonate was reduced to 48.6 mmol/l, base excess was 23.4, chloride was 69 mmol/l, and potassium was still low at 2.8 mmol/l. There was hypoventilation with a pCO_2 of 68.4 mmHg. We noted onset of diuresis. After 72 h of fluid therapy, the acid-base and electrolyte status was normal. Creatinine had decreased to 3.5 mg/dl. Small meals were tolerated.

Since there was no evidence of an infectious etiology for the excessive vomiting and the ultrasound had shown a distended stomach, an upper gastrointestinal endoscopy was performed [Figure 2]. A significant stenosis of the duodenum was diagnosed. Although there was no ulcer visible, an occult ulcer was deemed to be possible. A portal hypertensive gastropathy was diagnosed while esophageal varices were absent. *Helicobacter pylori* testing was positive, so an eradication therapy was initiated. A repeated endoscopy confirmed the stenosis, but again, no ulcer was seen. Since a

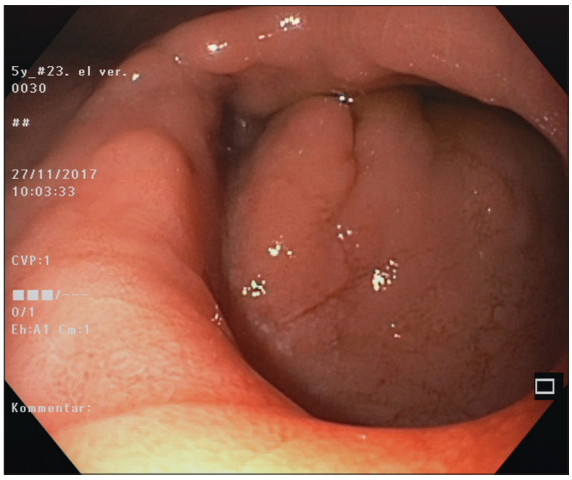

Figure 2: Gastrointestinal endoscopy. Endoscopy revealing an obstruction of the duodenum. Further, imaging with magnetic resonance imaging and endosonography excluded a compression by a neoplastic process

focused abdominal ultrasound did not reveal the reason for the stenosis, a magnetic resonance imaging scan was performed to rule out malignancy such as pancreatic or gallbladder cancer or a thrombosis of the mesenteric vein. There was no evidence of malignancy or cirrhosis. An endosonography was done without evidence of malignancy or mesenteric vein thrombosis. At that point, the stenosis had partly resolved, and endoscopic passage was possible. We concluded that the temporary duodenal stenosis was caused most likely by portal hypertension of unknown etiology, and our differential diagnosis included idiopathic noncirrhotic portal hypertension after exclusion of cirrhosis and portal vein obstruction.

Discussion and Conclusion

Cases of metabolic alkalosis have been reported previously but due to the high mortality cases with a pH >7.65 are extremely rare. Often, patients present with seizures or decreased state of consciousness. We found a similar derangement of acid-base and electrolyte status reported in a case of a malpositioned gastrostomy tube but no similar case that was caused by vomiting alone.[1]

Cases of metabolic alkalosis may occur due to inherited ion channel diseases such as Gitelman syndrome or iatrogenic due to gastric suction or ingestion of alkaline substances. Metabolic alkalosis may also occur in hyperaldosteronism.[2]

There are reports of aggressive or invasive regimens such as intravenous HCl or hemodialysis.[3] The use of high cation-gap amino acid solution has also been reported.[4] Since our patient was alert and there was no acute hemodynamic compromise, we took a conservative approach and started with oxygen therapy to correct the hypoxemia and started normal saline with potassium chloride as intravenous fluid therapy. After initiation of oxygen therapy, we saw a more adequate respiratory compensation with pCO_2 rising to values of 69.8 mmHg leading to an improvement in pH. Since we

saw a steady improvement with infusion of normal saline and at the same time gastric losses were minimized by application of proton-pump inhibitors, there was no indication for dialysis or application of hydrochloric acid.

Since history, laboratory tests, and ultrasound did not reveal the reason for the vomiting, an endoscopy was planned. The endoscopy was done after acid base, and electrolytes were stabilized due to possible complications during anesthesia.[5] Endoscopy revealed an obstruction of the duodenum. Duodenal obstruction as a cause of vomiting with symptomatic alkalosis has been reported recently in the veterinary literature.[6] The diagnosis of a duodenal obstruction due to a portal hypertension without cirrhosis was not confirmed by invasive testing since the patient was stable and the stenosis had resolved.

In our opinion, this case demonstrates the successful management of a life-threatening condition by simple measures. Despite extensive diagnostic procedures, the effective treatment of the underlying condition consisted of watchful waiting until the stenosis resolved spontaneously.

Consent for publication

Written consent was given by the patient.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given his consent for his images and other clinical information to be reported in the journal. The patient understands that name and initials will not be published and due efforts will be made to conceal identity, but anonymity cannot be guaranteed.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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