CASE REPORT

Acquired pyloric stenosis resulting in hypokalaemic, hyperchloraemic normal anion gap metabolic acidosis. Persistent vomiting in an adult: cause and effect

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SUMMARY

A 24-year-old woman presented with a history of persistent vomiting for at least 3 months. This resulted in severe dehydration with risk of acute kidney injury. In addition to volume depletion, loss of gastric fluid resulted in a specific metabolic derangement—hypokalaemic, hypochloraemic normal anion gap metabolic alkalosis with a reduced ionised calcium concentration and paradoxical aciduria. These metabolic changes were reflected in her ECG. Investigation demonstrated acquired gastric outflow tract obstruction secondary to a pyloric peptic ulcer. The patient was resuscitated with intravenous crystalloid and electrolyte supplements. The acquired pyloric stenosis was treated medically with a proton pump inhibitor and *Helicobacter pylori* eradication therapy with excellent recovery.

early treatment with fluid and electrolyte replacement and rapid resolution of these risks.

CASE PRESENTATION

A 24-year-old woman presented with a history of persistent vomiting for at least 3 months. She was constantly nauseated and described an intermittent dull ache in the epigastrium. She had worsening malaise and generalised weakness associated with weight loss of approximately 20 kg. She stopped smoking diamorphine at about the time the symptoms first manifested. HIV and hepatitis screens were negative. There was no other history of note. On examination she was dehydrated and cachexic. Her heart rate was 110 with a blood pressure of 90/60. Her abdomen was distended and tender with normal bowel sounds. Examination was otherwise normal.

BACKGROUND

Persistent vomiting is common. If severe it can cause dehydration with risk of acute kidney injury or hypovolaemic shock. In addition, it causes a unique and complex metabolic disturbance which can affect myocardial conduction and induce a malignant arrhythmia. Prompt recognition ensures

INVESTIGATIONS

The patient's estimated glomerular filtration rate was normal but she had significant electrolyte and acid-base disturbance consistent with persistent vomiting: pH 7.57, bicarbonate 53, base excess 25.9, lactate 2.7, potassium 1.7, magnesium 0.2,

Figure 1 ECG.

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Figure 2 CT abdomen.

chloride 75 and ionised calcium 1.0. Urinalysis, beta-human chorionic gonadotropin and thyroid stimulating hormone were normal. An ECG (figure 1) showed a sinus tachycardia with widespread ST depression and a prolonged QTc. CT abdomen (figure 2) revealed gastric dilation. An upper gastrointestinal (GI) endoscopy demonstrated acquired gastric outflow tract obstruction secondary to *Helicobacter pylori* positive pyloric peptic ulcer.

TREATMENT

The patient was resuscitated with intravenous crystalloid and electrolyte supplements. The acquired pyloric stenosis was treated medically with a proton pump inhibitor and *H. pylori* eradication therapy with excellent recovery.

OUTCOME AND FOLLOW-UP

As above.

DISCUSSION

Persistent vomiting in adults has a wide differential diagnosis. This includes hyperemesis gravidarum (which occurs in 1% of pregnant women), gastric outflow tract or intestinal obstruction, gastroparesis secondary to autonomic neuropathy or drugs, intestinal or urinary infection and raised intracranial pressure.

Acquired pyloric stenosis in adults is increasingly rare. Peptic ulcer-related disease has steadily reduced in incidence on a worldwide basis. Interestingly, this trend started prior to the introduction of acid-suppressing medication suggesting a more fundamental change in the natural course of the disease process.¹ Rarer causes are adult-onset idiopathic hypertrophy, gastric neoplasms, gastric diverticulosis and iatrogenic following endoscopic submucosal dissection.

Learning points

- ▶ Persistent vomiting results in dehydration and ultimately prerenal renal failure with risk of acute tubular necrosis.
- ▶ In addition to volume depletion, loss of gastric fluid also results in a specific metabolic derangement—hypokalaemic, hypochloaemic normal anion gap metabolic alkalosis.
- ▶ Severe hypokalaemia results in muscular weakness and increased myocardial excitability with risk of arrhythmia.
- ▶ Opioid dependence may mask painful medical conditions.

Persistent vomiting results in dehydration and ultimately prerenal renal failure with risk of acute tubular necrosis. In addition to volume depletion, loss of gastric fluid also results in a specific metabolic derangement—hypokalaemic, hypochloaemic normal anion gap metabolic alkalosis. This is associated with a reduced ionised calcium concentration and paradoxical aciduria. Pathophysiology:

- a. Direct loss of hydrogen ions from the GI tract results in metabolic alkalosis. This induces respiratory compensation by CO₂ retention but this is limited by hypoxia. Metabolic compensation for elevated bicarbonate is usually rapidly effective. However, in persistent vomiting, chloride loss and volume depletion (with activation of the renin-angiotensin-aldosterone (RAA) system reducing the filtered load of bicarbonate) both stimulate bicarbonate reabsorption in the renal tubules exacerbating the alkalosis. Activation of the RAA system also increases sodium absorption throughout the nephron in exchange for hydrogen ions further exacerbating the alkalosis and resulting in aciduria.²
- b. Hypokalaemia is induced by potassium exchange for hydrogen and sodium ions in the renal tubules and hydrogen ions between the intracellular and extracellular space. Severe hypokalaemia (<2.5 mmol/L) results in muscular weakness and increased myocardial excitability with risk of arrhythmia. ECG changes include increased P wave amplitude and duration, prolonged PR interval, ST depression, T wave flattening, prolonged QTc and prominent U waves. There is an increased risk of supraventricular and ventricular tachycardia (VT) including polymorphic torsade de pointes VT.^{3–5}
- c. Reduced serum ionised calcium results from increased binding of calcium to albumin which is ionised in an alkaline environment. Hypocalcaemia (absolute or due to a reduction in ionised calcium levels) also affects the myocardium resulting in a prolonged QTc with risk of polymorphic VT as well as a direct effect on myocardial contractility.^{4,5}

Contributors PK is the sole author of this manuscript.

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