CASE REPORT

Adult Pyloric Stenosis Masquerading as Acute Renal Failure

S L Siow, MS, C M Wong, MS, M Sohail, MRCS

Department of Surgery, Sarawak General Hospital, Jalan Hospital, 93586, Kuching, Sarawak, Malaysia

SUMMARY

Gastric outlet obstruction and in particular, pyloric stenosis, is relatively common in developing countries. Acute clinical presentation is often the manifestation of biochemical and electrolyte changes. The presence of metabolic alkalosis in combination with acute renal failure should alarm us to the possibility of adult pyloric stenosis. We report a case of adult pyloric stenosis that presented as acute renal failure and discuss its pathophysiology.

KEY WORDS:
Adult pyloric stenosis, Gastric outlet obstruction, Acute renal failure, Metabolic alkalosis

INTRODUCTION

Understanding of metabolic and electrolyte changes in pyloric stenosis is important, as it has a key role in fluid resuscitation and management.

CASE REPORT

A 26-year-old man was referred from a peripheral hospital to the Medical Unit of Sarawak General Hospital with acute renal failure and anaemia. His renal function had previously been normal. The cause of renal failure was initially unclear. He had no significant past medical history, except for a ruptured appendix 9 months previously. On arrival at the hospital, the patient was dehydrated, lethargic and cachexic but haemodynamically stable. Abdominal examination revealed a lower midline scar. The abdomen was soft, with fullness in the epigastric region. Soon after admission, he became confused, was unable to respond to commands and had a generalized tonic-clonic seizure. The initial laboratory values are shown in Table I.

Ultrasound examination showed normal kidneys with no obstructive uropathy. A computed tomographic scan of the brain showed no abnormality. The results of tests with celebrospinal fluid were normal. An abdominal radiograph showed a dilated stomach, which was mistaken for a dilated bowel.

When attempts to initiate oral feeding resulted in repeated vomiting, the patient was referred to the Surgical Unit for urgent upper endoscopy. Additional history obtained from the patient at that time indicated that he had had epigastric pain on and off, for which he had been prescribed antacids at the district polyclinic. The symptoms had been insignificant until the surgery for the ruptured appendix. He had since been unwell with frequent episodes of vomiting, particularly after meals, on most days, associated with loss of appetite and weight loss of 25kg. He reported that he smoked and drank alcohol daily.

The clinical examination showed a positive succussion splash.

Oesophagogastroduodenoscopy showed pyloric stenosis associated with stagnation of gastric fluid of about 700ml of gastric fluid with some food particles. The rapid urease test showed the presence of *Helicobacter pylori*. The patient was given total parenteral nutrition with continuous nasogastric suction. Gastric lavage was done through a nasogastric tube in preparation for balloon dilatation of the pylorus. Fluoroscopy with gastrograffin showed 2-3cm pyloric stenosis. The duodenum distal to the stenosis was of normal calibre. The dilatation was complicated by duodenal perforation with leakage of contrast medium into the right retroperitoneal space. The patient was managed conservatively for the duodenal perforation, and then underwent elective laparotomy and gastrojejunostomy. He continued to improve and was discharged home once he was eating well and his renal function had normalized.

DISCUSSION

A diagnosis of gastric outlet obstruction is often established from the classical findings of a clinical examination and a plain abdominal radiograph. Late presentation can, however, result in difficult scenarios such as seizures and renal failure. The diagnosis was delayed in this patient, as certain cues were not followed up, such as the unusual presence of metabolic alkalosis and the presence of a succussion splash.

The history obtained after the critical presenting symptoms had been controlled showed that the patient had a long-standing history of duodenal ulcer disease, which had been undiagnosed and inappropriately managed. The ruptured appendix and subsequent laparotomy might have exacerbated the ulcer. The patient's inability to retain food and liquids compounded by prolonged vomiting had led to the complex metabolic and electrolytic changes that gave rise to metabolic alkalosis and acute renal failure.

The pathogenesis of acute renal failure is multifactorial. Both prerenal and intrinsic renal factors are believed to be contributory. Dehydration caused by recurrent vomiting resulted in reduced renal perfusion and subsequent prerenal acute renal failure which if not promptly corrected, could have led to acute tubular necrosis.

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Corresponding Author: Siow Sze Li, Department of Surgery, Sarawak General Hospital, Jalan Hospital, 93586, Kuching, Sarawak, Malaysia
Email: szeli18@yahoo.com

Table I: Initial laboratory values

Parameter		Value
Haemoglobin		6.7 g/dl
Serum urea		19.5 mmol/l
Serum sodium		141 mmol/l
Serum potassium		3.1 mmol/l
Serum chloride		58 mmol/l
Serum creatinine		685 mmol/l
Corrected calcium		1.68 mmol/l
Arterial blood gas at room air	pH	7.65
	Pco_2	78 mmHg
	Po_2	94.9 mmHg
	Calculated HCO_3-	60 mmol/l

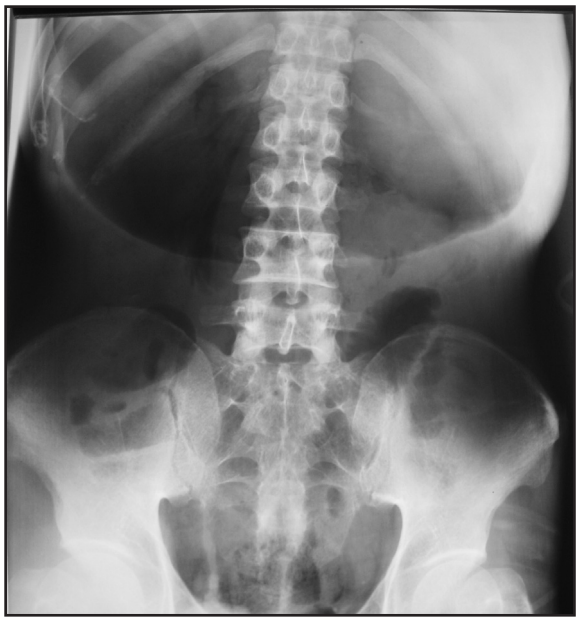

Fig. 1: AXR showing a dilated stomach containing a large amount of air

Sodium plays a key role in volume homeostasis of extracellular fluids, as it accounts for over 90% of the osmotically active solute in the plasma and interstitial fluid[1,2]. The dehydration that follows vomiting causes a reduction in extracellular fluids volume that results in a fall in blood pressure, the glomerular filtration rate and the amount of sodium filtered. As a result, aldosterone secretion is stimulated, which causes sodium and water retention. Aldosterone acts on the renal tubules to reabsorb sodium in exchange for potassium and hydrogen, producing a potassium diuresis and increased urine acidity. The loss of potassium and hydrogen due to vomiting further leads to severe metabolic alkalosis and potassium deficiency. As chloride is lost in the vomitus, sodium reabsoption in the proximal tubule is less effective than in the distal tubule. Sodium is reabsorbed with bicarbonate in the proximal tubule[3].

If the vomiting persists, relative intracellular acidosis will occur due to movement of potassium out of the cells to correct the hypokalaemia, and hydrogen will move into the cells to maintain electroneutrality[4].

Extracellular (metabolic) alkalosis is due to an increase in the plasma bicarbonate concentration[5]. As hydrogen is derived from the intracellular dissociation of carbonic acid into hydrogen and bicarbonate, 1 mEq bicarbonate is generated with every mEq loss of hydrogen[3]. In addition, the intracellular acidosis stimulates sodium-hydrogen exchange, which further enhances renal bicarbonate reabsorption[4]. In the presence of potassium deficiency, the kidneys preferentially excrete hydrogen, which further worsens the alkalosis[3,5].

Severe metabolic alkalosis is potentially life-threatening and requires urgent treatment. It affects the major organ systems, including cardiac dysrhythmias and vascular collapse, and neurological effects, which are primarily seizures[4]. This patient had manifestations of metabolic alkalosis both clinically, in the form of confusion and generalized tonic-clonic seizure, and biochemically, in the form of reduced ionized calcium, which were not recognized.

CONCLUSION

Acute renal failure due to adult pyloric stenosis is rare. However, it is reversible if treated promptly and appropriately. The case highlights the importance of complete history and clinical examination in identifying unusual causes of acute renal failure.

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