Reminder of important clinical lesson

A reminder of the classical biochemical sequelae of adult gastric outlet obstruction

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Summary

The commonest cause of gastric outlet obstruction (GOO) is pyloric stenosis secondary to peptic ulcer disease or gastric carcinoma. Patients with GOO have unique metabolic sequelae, namely hypochloraemic, hypokalaemic metabolic alkalosis with paradoxical aciduria and hypocalcaemia. A case of a patient presenting as GOO is discussed. The aim of this report is to highlight the metabolic abnormalities and management in patients with GOO.

BACKGROUND

The classical biochemical sequelae of adult gastric outlet obstruction (GOO), namely hypochloraemic, hypokalaemic metabolic alkalosis with paradoxical aciduria and hypocalcaemia, is rarely seen in the modern era of medicine as these patients are identified early and a prompt management of their chronic dehydration prevents the development of full-blown metabolic derangements.

Once the metabolic sequelae develops, it is important to recognise the classical combination and these patients should be managed urgently with intravenous resuscitation.

CASE PRESENTATION

Introduction

The commonest cause of GOO is pyloric stenosis secondary to peptic ulcer disease or gastric carcinoma. Patients with GOO have unique metabolic sequelae named hypochloraemic, hypokalaemic metabolic alkalosis with paradoxical aciduria and hypocalcaemia.

A case of a patient presenting as GOO is discussed. The aim of this report is to highlight the metabolic abnormalities and management in patients with GOO.

Case

A 72-year-old gentleman with a history of duodenal ulceration, gastro-oesophageal reflux disease and prostate carcinoma was admitted with increasing confusion and vomiting. He had been feeling unwell for over a month and was unable to tolerate oral fluids at presentation. Biochemical analysis revealed low sodium and potassium levels with mild derangement of renal function. This initial diagnosis was gastroenteritis but the patient failed to improve. He had developed hypocalcaemia and his urine had a very low pH.

A surgical consult was requested. By now his abdomen was distended and a prominent succussion splash present. A wide bore nasogastric tube was inserted and 11 litres of altered food and fluid were aspirated.

On review of the initial plain abdominal radiograph, a markedly distended stomach was noted (figure 1).

Gastroscopy and a barium meal examination subsequently confirmed oedema and narrowing in the first part of the duodenum secondary to benign acid peptic disease. The patient received intravenous proton-pump inhibitor, nasogastric suctioning and conservative measures and he remains well 6 months after discharge.

INVESTIGATIONS

An oesophago-gastro-duodenoscopy and barium meal examination subsequently confirmed oedema and narrowing in the first part of the duodenum secondary to benign acid peptic disease.

DISCUSSION

The biochemical syndrome of hypochloraemic, hypokalaemic metabolic alkalosis with paradoxical aciduria and hypocalcaemia is rarely seen in the modern medicine. This is primarily because complicated peptic ulcer disease has become uncommon as the medical treatment of the early disease has revolutionised its management. Majority of the adult patients seen with GOO are secondary to gastric carcinoma; however, these patients do not present with classical biochemical syndrome of GOO as many of them have relative hypochlorhydria. Primary adult hypertrophic pyloric stenosis is another condition that can present with the typical biochemical features of GOO.¹

The pathophysiology of the biochemical aberrations in GOO is as a result of persistent vomiting of gastric hydrochloric acid resulting in hypochloraemia and

Figure 1 °Plain abdominal x-ray showing significantly distended stomach.

Learning points

- ▶ Benign adult GOO is uncommon and a malignant cause must be ruled out.
- ▶ The diagnosis of GOO is usually straightforward on a good history and clinical examination.
- ▶ Plain abdominal x-ray may show a hugely distended stomach in these cases.
- ▶ These patients need urgent fluid resuscitation as they are chronically dehydrated and may have compromised renal functions.

metabolic acidosis. As dehydration progresses, metabolic abnormalities become more profound and renal function is overwhelmed. Initially, the urine has low chloride and high bicarbonate content to compensate for gastric losses of hydrochloric acid and is appropriately

alkaline. As a result of continuous dehydration, sodium gets reabsorbed preferentially over potassium and hydrogen ions that are excreted by kidneys. This results in urine becoming paradoxically acidic and hypokalaemia ensues.² Alkalosis leads to a reduction of the circulating ionised calcium thereby completing the biochemical syndrome.³

Upper gastrointestinal endoscopy with or without barium studies is invaluable in establishing a diagnosis and excluding gastric cancer. The primary treatment of metabolic syndrome associated with GOO is prevention. The treatment of established GOO with biochemical syndrome is conservative with a wide bore nasogastric tube and rehydration along with replacement of sodium and potassium.⁴ Acid–base imbalance is soon corrected once kidneys are better perfused.⁵ An indication of successful resuscitation is urine becoming alkaline. Management includes nasogastric aspiration, intravenous proton-pump inhibitors and fluid replacement.

Competing interests None.

Patient consent Obtained.

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