Severe Metabolic Alkalosis due to Choledochoduodenal Fistula

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sediment. Serum electrolyte concentrations: sodium 141 mEq/l, potassium 3.87, chloride 77.3, and bicarbonate 54.7 mEq/l. Blood urea nitrogen and serum creatinine concentrations were 41 and 5.6 mg/dl, respectively. The blood pH was 7.731. Serum calcium and phosphate concentrations were 10.1 and 8.8 mg/dl, respectively. Isotonic sodium chloride and adding KCl at a concentration of 10 mEq/l reversed the metabolic alkalosis and the impaired renal functions. Barium

Dear Sir,

An increase in the incidence of spontaneous biliary enteric fistulas has been reported during the last decades. The major cause of biliary enteric fistulas is cholelithiasis. A choledochoduodenal fistula due to duodenal ulcer is a rare phenomenon [1]. No significant clinical symptoms are seen in the majority of the patients. Clinical symptoms are usually epigastric pain and vomiting. Signs and symptoms of extracellular volume contraction and metabolic alkalosis may be seen due to severe vomiting. We present a case with severe metabolic alkalosis due to choledochoduodenal fistula secondary to duodenal ulcer.

A 37-year-old man was admitted to our hospital because of severe vomiting and numbness in his hands. He had been suffering from epigastric pain and vomiting during the last 6 years, and an antacid preparation was initiated. During the last months frequency and severity of the complaints increased despite continuing the treatment, and during the last week before his admission he had severe vomiting attacks. On his admission, the blood pressure was 100/ 60 mm Hg, the heart rate 104 beats/min the body temperature 36 °C, and the respiratory rate was 28/min. The skin turgor was decreased, and Chvostek and Trousseau signs were negative. Laboratory studies revealed hemoglobin 18.2 g/l, hematocrit 55.4%, and white blood cell count 17,000 mm³. Urinalysis revealed the following: pH 9.0, osmolality 300 mosm/kg, protein 15 mg/dl, no glucose, sodium 6.6 mEq/l, potassium 7, chloride 15 mEq/l, and microscopically normal urine

Fig. 1. Passage of contrast material into the ductus choledochus via the choledochoduodenal fistula.

examination of the upper gastrointestinal tract was performed to detect the cause of the severe vomiting. A choledochoduodenal fistula secondary to duodenal ulcer was found (fig. 1). 1 week later, when he was discharged after prescription of a H₂ receptor antagonist, serum creatinine and blood urea nitrogen levels were 1.1 and 16 mg/dl, respectively.

Among the spontaneous biliary enteric fistulas the choledochoduodenal fistula has

an incidence of 3.5-20% [2]. As a duodenal ulcer complication, a choledochoduodenal fistula is rare. The reported incidence of biliary enteric fistulas in patients who underwent gastrectomy is between 0.3 and 4.1% [2]. The low incidence may partly be due to the position of the ulcer which is usually 4 cm distal from the pylorus, while the retro-pancreatic and duodenal portions of the common bile duct are situated at least 7 cm distally from the pylorus. The symptoms of the patients are usually epigastric pain and vomiting. Rarely ascending cholangitis may develop due to inadequate drainage of the bile. In the management of the choledochoduodenal fistula secondary to duodenal ul-

cer, the major therapeutic objective is the healing of the ulcer which is usually followed by healing of the fistula. Our patient was admitted to the hospital because of severe vomiting and numbness in his hands. A severe metabolic alkalosis and increased serum creatinine and blood urea nitrogen levels were found during laboratory investigations. Severe vomiting due to choledochoduodenal fistula secondary to duodenal ulcer was the cause of marked metabolic alkalosis and impaired renal functions [3]. Although most of the patients are asymptomatic, a choledochoduodenal fistula should be kept in mind as an unusual cause of metabolic alkalosis due to vomiting.

References

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