CASE REPORT

Severe Electrolyte Abnormalities and Paralytic Ileus Complicating Delirium Tremens

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(Received for publication on November 7, 1988)

Abstract

Pneumonia and cardiac arrhythmias represent the most common life-threatening complications during delirium tremens. Electrolyte abnormalities are common underlying conditions in chronic alcoholics and they may further complicate the management of patients with alcohol-withdrawal syndrome or with delirium tremens. The authors present two cases in which the clinical picture of severe paralytic ileus complicating delirium tremens was closely associated with electrolyte status and postulate that the two were cause-effect related. A careful electrolyte supplement therapy guided by a strict monitoring of electrolyte balance and renal function proved to be particularly useful in successful management.

Key words: delirium tremens, paralytic ileus, electrolyte imbalance

Introduction

Infectious diseases, especially pneumonia, hyperthermia and cardiac arrhythmias are the most common complications in the course of delirium tremens (DT).1–3
Electrolyte abnormalities, which are almost invariably present, may lead to acute derangement of the neuromuscular activity of the bowel. The purpose of this paper is to draw attention to the latter condition by presenting two cases of generalized ileus secondary to severe electrolyte imbalance during DT.

Case Report

Case 1. A 63-year old alcoholic man developed over a three-day period increasing irritability, diaphoresis and sleeplessness and finally presented with an overt picture of impending DT. On physical examination he appeared restless, was sweating profusely and showed a clouded sensorium. Carpopedal spasms were present and mild pyrexia (37.6°C) was recorded. Multiple spider angiomata were present on his trunk. His chest was clear and heart sounds were normal. Heart rate was 110/min and respiratory rate was 32/min. His blood pressure was 145/85 mmHg. His abdomen was severely distended and tympanic with no bowel sounds. Interrogation of his family members ruled out a concomitant consumption of any drug or any recent trauma. There was, however, a previous history of seizures. During the few months preceding the admission he reportedly had changed his daily drinking habit into heavy week-end alcohol ingestion. A chest X-ray and an EKG were normal. An abdominal X-ray showed small and large bowel dilation without air-fluid levels and no ascites. Blood laboratory data showed that, besides mildly raised AST (GOT; 65 IU/L), ALT (GPT; 72 IU/L) and GGT (γGPT; 95 IU/L), a severe hypomagnesemia (0.4 mmol/L) and hypokalemia (2.4 mmol/L) were present. Serum phosphorus was marginally low (0.9 mmol/L). BUN, creatinine and urinary and serum amylase were normal. Astrup results were in keeping with mild metabolic alkalosis and blood sugar was 2.5 mmol/L (45 mg/dl). He was started on diazepam, nasogastric suction and vigorous i.v. fluids with 6 g magnesium-Na sulphate and 20 mmol phosphate supplements. He was also given dextrose, 3 μg of vitamin B12 and thiamine 100 mg and repeated EKG and blood pressure measurements were performed. Twenty-four hours later the patient was fully conscious and alert, and without fever. No neurological abnormality was present; however, he was complaining of moderate generalized abdominal pain. Abdominal X-ray showed a marginal improvement of the previous finding. Some flatus had passed, but the abdomen remained still distended and only faint bowel sounds were heard. Significant blood laboratory data were as follows: potassium 3.3 mmol/L, magnesium 0.6 mmol/L and phosphorus 0.6 mmol/L. Serum and urinary amylase, and renal function were within normal limits. An 8 hr urine collection showed urinary magnesium and phosphate of 58 mg and 410 mg, respectively. Magnesium i.v. supplement was increased to 8 g/day and i.v. phosphate to 60 mmol/day. On the third day, the abdominal pain subsided and the abdomen was less distended on clinical and radiological examinations. Urinary electrolyte excre-
tion and serum electrolyte values were reverted almost to normal, with the exception of the serum magnesium level which was still 0.9 mmol/L. The total amount of i.v. magnesium and phosphate supplements given during the day was 4 g and 20 mmol, respectively. On the forth day, phosphate supplement was stopped and magnesium was reduced to 2 g. On the fifth day a normal biochemical and clinical picture was restored.

Case 2. A 46-year old chronic alcohol abuser received from his general practitioner two injections of a long acting steroid over a ten-day period because of severe allergic rhinitis. Subsequently, he developed incoherent verbal expression alternating with momentary confusion that culminated in a generalized seizure. He presented with manifest DT. Chvostek's sign and Trousseau's sign were present. His abdomen was severely distended and mildly tender. A three-finger hepatomegaly could be palpated but ascites was not detected. Only faint bowel sounds were heard. A chest X-ray showed bilateral features of mild COPD and an EKG showed supraventricular tachycardia with 3 premature ventricular beats/min. Blood pressure was 160/90 mmHg and temperature was 36.8°C. Heart rate was 104/min and respiratory rate was 34/minute. An abdominal X-ray demonstrated small and large bowel dilation without signs of localized obstruction. Blood laboratory data showed a raised MCV (102 fl), AST (90 IU/L), GGT (169 IU/L) and low albumin (26 g/L). The electrolyte balance was abnormal with the following results: magnesium 0.4 mmol/L, potassium 3.1 mmol/L, calcium 1.88 mmol/L, sodium 138 mmol/L and phosphorus 0.5 mmol/L. Metabolic alkalosis was shown with Astrup. BUN, creatinine and serum and urinary amylase values were within normal limits. A 12 hr urine collection showed grossly elevated magnesium and phosphorus levels, 110 mg and 530 mg, respectively. A bolus, i.v. dose of 8 mmol magnesium-Na sulphate was given with a resulting normalization of the EKG. The patient was then started on chlordiazepoxide and i.v. fluids with 10 g magnesium-Na and 30 mmol phosphate supplements and blood pressure was recorded every four hours. After twenty-four hours his mental condition was very much improved and a repeated EKG was within normal limits; however, he complained of severe generalized abdominal pain. The abdomen was still distended with some rebound tenderness. No persitalsis had been resumed. Later during the day his general condition deteriorated and his temperature went up to 38.9°C. A full blood counts showed a white cell counts of 18,000/mm³ with a left shift. A blood culture showed no organism growth and a microscopic urinalysis was negative. Blood laboratory data showed an improvement in potassium level (3.7 mmol/L) and a normal calcium level (2.10 mmol/L); however, magnesium was 0.6 mmol/L and phosphorus was 0.7 mmol/L. Serum iso-amylase and urine amylase were normal, renal function was unchanged. A repeated chest X-ray and EKG were unchanged. On an abdominal X-ray, the most striking feature was a very dilated transverse colon with a maximum diameter of 13 cm (picture 1). The wall of the colon seemed slightly edematous but the mucosal pattern and
Fig. 1  Case 2. Close-up of the left part of the transverse colon and the splenic flexure. Both are extremely dilated with a maximum diameter of 13 cm. Slight edema of the wall can be seen, however there is no obvious mucosal abnormality and haustration is preserved.

hastration looked otherwise preserved. In particular, no sign of “thumbprinting” was seen. These findings were felt to be in keeping with a megacolon supervening a paralytic ileus. Nonetheless, a careful colonoscopy was performed in order to rule out a large bowel obstruction or to detect ischemic colitis signs. An attempt to endoscopically decompress the colon was partially successful. No obvious colonic pathology was found on colonoscopy. An abdominal ultrasound was not very contributory because of overshadowing intestinal gas. No obvious abnormality, however, was noted in the biliary tree or in the pancreas. The patient was started on i.v. metronidazole, ampicillin and tetracycline therapy and it was decided to pursue a closely monitored conservative management. A nasogastric tube was inserted and i.v. magnesium and phosphate supplementation was continued at the previous loading dose. Twenty-four hours later the general condition improved and his temperature was 37.6°C. His abdomen was soft,
no longer tender and less distended on clinical and radiological examinations. Some peristalsis was heard and flatus passed. Marked improvement in serum electrolyte values was achieved (magnesium 1.3 mmol/L, phosphorus 0.9 mmol/L). Renal and hemodynamic parameters were unchanged. Intravenous phosphate supplement was stopped and i.v. magnesium was reduced to 6 g over 24 hr. The following day the temperature settled down and the patient was feeling better. By the sixth day, normal serum and urinary data were obtained. Repeated abdominal films confirmed a continuous improvement from the previous picture and, on the tenth day, a complete restoration of normal bowel gas pattern was demonstrated. Small and large bowel enemas, done before discharge, were normal.

Discussion

In alcoholics, severe electrolyte imbalance as well as blood sugar and gas abnormalities may occur unpredictably. It has been shown that seven to ten hours after alcohol withdrawal, respiratory alkalosis induces a rise in arterial pH and a drop in serum magnesium.4 The incidence of hypomagnesemia among alcoholics is over 20% and is even more frequent in patients in DT or in alcohol withdrawal.5,6 The major contributing mechanism of this deficiency is an alcohol-induced magnesium diuresis which may lead to urinary losses two to three times normal value even in the presence of total body magnesium deficit.7,8 This is in contrast to what happens in other situations of magnesium deficiency of extrarenal origin in which tubular reabsorption is enhanced so that magnesium virtually disappears from urine.

We have presented two cases in which severe electrolyte abnormalities played a major role in the management of paralytic ileus complicating DT. In the first case alcohol intoxication was the cause of DT, whereas in the second, administration of steroids probably triggered the onset. The latter case showed more profound and prolonged electrolyte derangements affecting the cardiac function and the recovery of normal gut motility. Both our patients presented with frank phosphaturia and magnesuria at a time when blood levels of these divalent ions were abnormally low. Since we cannot ascribe this finding to massive volume expansion, it suggests defective proximal tubular reabsorption of these minerals. Unlike chronic ethanol intake, acute ingestion may give bouts of urinary magnesium losses. It was of interest that in case 1 there had been a recent change in drinking habits with heavy week-end alcohol binges. Hypomagnesemia, in turn, may cause a large phosphouresis9 which can worsen the neurological and cardiac disturbances. An underlying phosphate deficiency is usually observed in alcoholics as a result of malnutrition, repeated episodes of acidosis and vomiting.10 Further, a marked phosphorus cell influx takes place due to hyperventilation, often present in these patients. An excessive urinary phosphate excretion was a common
feature of both cases and in patient 2 persisted for a few days. In patients 1, the serum phosphorus level was slightly low at the admission and, despite cautious phosphate supplementation, dropped lower the following day. This could have been the result of two factors: a) intravenous magnesium administration has been shown to induce a transient rise of urinary phosphorus excretion perhaps via an enhanced parathyroid hormone production; b) alcoholics with underlying phosphate deficiency sometimes show a refeeding-induced hypophosphatemia. In alcoholics, fever can be caused by alcohol intoxication itself as it seems to be the case in the first patient in which no alternative cause could be found. However, in the second case, a prolonged paralytic ileus and persisting electrolyte abnormalities sustained each other in a vicious circle and caused a megacolon with a septic picture. Because spontaneous bacterial peritonitis carries a very high mortality, it should always be carefully considered whenever a patient with chronic liver disease presents with fever, abdominal pain and decreased bowel sounds. The presence of ascites is a commonly associated finding but in this case no evidence of it was found. Acute pancreatitis sometimes presents with mental disturbances indistinguishable from DT and may be also associated to hypomagnesemia and hypophosphatemia. We performed repeated serum and urinary amylase determinations in view of this possible diagnosis. A concern was as to whether the case 2 had ischemic bowel precipitated by accumulation of calcium in smooth muscle of the bowel or its vessels as a result of magnesium deficiency. Further, an excessive urine phosphate excretion has been described during this condition. However, the clinical, radiological and endoscopic findings made this diagnosis unlikely.

Although all the above mentioned electrolyte derangements are well recognized, it is not wise to give electrolyte supplements empirically in such patients. A careful monitoring of electrolyte balance, renal function and hemodynamic status is mandatory. Raised serum potassium, magnesium or phosphorus levels, inadvertently caused, may significantly increase morbidity. On the other hand, rehydration and dextrose infusions may adversely affect phosphorus depletion. In severe hypomagnesemia, total body deficit is about 2 mEq/kg/body wt and replacement of magnesium storage by i.v. administration must be closely monitored since a considerable amount of parenterally administered magnesium will be lost in the urine. Further, a normal magnesium level does not exclude persistent total body deficit; therefore, after serum magnesium normalizes, it is better to continue supplementation for a while. Hypokalemia is a well-recognized manifestation of magnesium deficiency, in both cases reported here it was normalized by magnesium administration and balanced i.v. fluids without any extra potassium supplements.

In summary, we have presented two cases of severe paralytic ileus associated with marked electrolyte abnormalities complicating DT. The improvement of the clinical picture followed the improvement of the biochemical one. The awareness of this con-
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dition may lead to a prompt diagnosis and to a rational treatment strategy, avoiding unnecessary surgery which carries a high risk in this group of patients.

Acknowledgements: We are very grateful to Mrs. Elizabeth K. Harley for reviewing the English manuscript.

References