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Re-feeding syndrome

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Abstract
The effect of a therapeutically administered high calorie diet in a severely malnourished patient is discussed in this case report. In patients with advanced head and neck cancer prolonged periods of malnutrition prior to admission are frequently encountered. This case report highlights the need to constantly monitor the electrolyte and vitamin levels during the early stages of instituting enteral or parenteral nutrition. By vigilant monitoring and a high index of suspicion re-feeding syndrome or severe hypophosphataemia and its associated complications can be avoided.

Key words: Neoplasms, Squamous Cell; Feeding Methods, Complications

Introduction

Re-feeding syndrome is a term used to describe severe fluid and electrolyte shifts with its related metabolic implications in malnourished patients undergoing caloric replacement. These are patients being replenished by total parenteral nutrition utilizing concentrated calories. The consequences of such high caloric input following prolonged starvation include hypophosphataemia, hypokalaemia, hypomagnesaemia as well as altered glucose, fluid and vitamin balance. We report such a case following total enteral replacement of calories.

Case report

A 56-year-old man was admitted with a three-week history of dysphagia, melaena and weight loss. He was a heavy smoker and chronic alcoholic. He was suffering from depression and symptoms of alcohol withdrawal secondary to his dysphagia. On examination he was cachexic with tender smooth hepatosplenomegaly. ENT examination revealed marked trismus and drooling of saliva. Cervical lymphadenopathy was not palpable. On flexible endoscopy a mass was seen to extend from the nasopharynx to the lower lateral pharyngeal wall. On admission his haemoglobin was 7.8 g/dl and his potassium was 3.2 mmol/l (normal range 2.8–5.5 mmol/l). The rest of his blood indices were normal. His CT scan showed the mass in the lateral pharyngeal wall to be extending into the thoracic inlet and causing tracheal compression.

Initial management was by blood transfusion and oesophago-gastroduodenoscopy. The same revealed a duodenal ulcer for which appropriate management was started. Arrangements for a percutaneous endoscopic gastrostromy were made and he was fed via a nasogastric tube in the interim. However, he was confused and withdrawn throughout this period. Despite all our attempts we were unable to communicate our management plans to him. He persistently refused all forms of treatment and also kept removing the nasogastric tubes. By day five his dysphagia was absolute. Attempts to place a gastrostomy via endoscopic or ultrasound guidance also failed. These factors led to a prolonged period of intravenous replacement. During this time his corrected calcium slowly rose but he remained hypokalaemic and hypernatraemic. A bone scan did not show any metastatic disease.

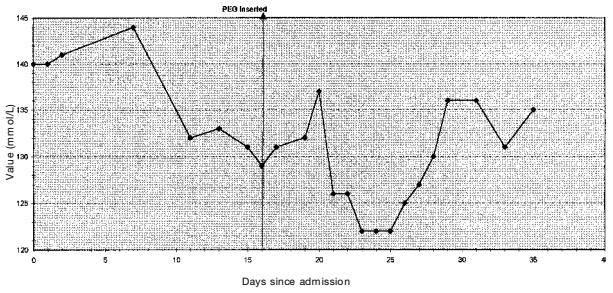

Fig. 1
Serum sodium values during admission.

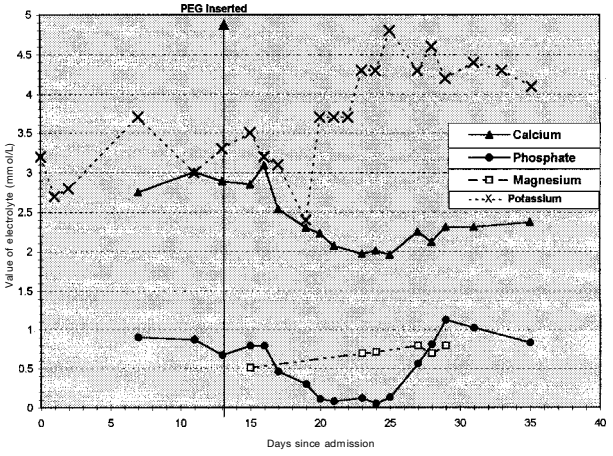

Fig. 2
Electrolyte values during admission.

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TABLE I
NORMAL SERUM ELECTROLYTE VALUES

Sodium	136 – 146 mmol/L
Potassium	3.5 – 5.1 mmol/L
Calcium	2.15 – 2.65 mmol/L (corrected)
Phosphate	0.8 – 1.5 mmol/L
Magnesium	0.72 – 1.00 mmol/L
Zinc	11 – 24 µmol/L

It was on the 16th day after admission that an open gastrostomy tube was placed under general anaesthetic. The dieticians then placed him on a feeding regime suitable for a 40.4 kg individual. Despite continued appropriate enteral feeding he became lethargic, drowsy and finally confused. During the same seven days his electrolytes also deteriorated, most notably his phosphate level. This was accompanied by hyponatraemia (Figure 1), hypocalcaemia (serum corrected level) and hypomagnesaemia (Figure 2). His electrolytes were constantly monitored and adjusted, as were his vitamin B1 levels. His total proteins ranged throughout from 50–61 gm/l (normal range 62–77 gm/l). His albumin levels during this time were 28–32 gm/l (normal range 35–55 gm/l). For one week despite regular assessment and replacement it was not possible to correct his electrolyte balance. This was reflected in his persistently confused and lethargic state. He developed arrhythmia/tachycardia, polyuria and tremors. A diagnosis of re-feeding syndrome was made and he was treated according to the guidelines provided by the Oxford Radcliffe Hospital.[1] Eventually, eight days after commencement of this regime the electrolytes started to correct themselves. Within a week with continued supplements and vigilant monitoring the electrolytes returned to their normal levels. The improvement in the electrolytes was clearly reflected in the concomitant recovery in the patient's clinical and conscious level.

Discussion

Severe hypophosphataemia and its associated complications in patients being re-fed by total parenteral nutrition after severe weight loss is termed re-feeding syndrome.[2] The altered metabolism leads to cardiac, respiratory, neuromuscular, renal, haematological, hepatic and gastrointestinal problems. Our patient who was chronically malnourished when placed on enteral therapy quickly developed this syndrome. The pathogenesis of the re-feeding syndrome is secondary to the shift in metabolism from fat to carbohydrates during re-feeding itself.[3] The consequent high glucose levels leads to insulin release. The increased insulin production and carbohydrate depletion causes an intracellular shift of potassium, phosphorus, glucose and water. The preceeding prolonged starvation in these patients and the intracellular shift following re-feeding leads to severe extracellular hypophosphataemia and hypokalaemia and hypomagnesaemia. It is therefore in the early stages of re-feeding that biochemical shifts occur and electrolytes should be more rigorously monitored. Red and white blood cell dysfunction secondary to the phosphate level and nitrogen levels is also noted along with renal impairment.[4] This sequence of events was clearly demonstrated by our patient and as our index of suspicion was low it slightly delayed our diagnosis.

The patients at risk of developing this condition are those suffering from anorexia nervosa.[5] Other susceptible patients include chronic alcoholics, the chronically malnutritioned/underfed, patients undergoing prolonged intravenous hydration, classic marasmus and the obese with massive weight loss.

Re-feeding syndrome is best avoided by raised awareness of the condition as well as testing and correcting electrolyte disturbances before replacement therapy.[6] Careful and slow restoration of circulatory volume, calories and vitamins is mandatory. If potassium is below 2.8 mmol/l, phosphorus below 0.3 mmol/l and magnesium below 0.5 mmol/l then replacement needs to be instituted. We would recommend that a dose of thiamine (50 mg) be regularly administered 30 minutes before each feed. Feeding should be started at 20 kcals/kg for the first 24 hours, to achieve full feeding capacity of any patient within a week. Full feeding capacity varies between individuals and can be up to 100 kcals/kg. Following this particular protocol we were able to successfully manage our patient.

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