Bilateral femoral neck fractures as a result of coeliac disease

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Summary

An elderly patient presenting with severe tetany secondary to hypocalcaemia causing bilateral fractures of the femoral necks, was proved to have coeliac disease. Severe muscle pains, an organic mental syndrome and personality changes are rare complications of coeliac disease and are reversible with treatment.

Introduction

The typical presentation of coeliac disease is a malabsorption syndrome with abdominal distention, diarrhoea, and steatorrhoea.

However, some patients present with other abnormalities not immediately suggestive of coeliac disease.

Some patients show metabolic bone disease with fractures, osteomalacia and bone pains. Others have obscure neurological and neuromuscular complaints, tetany fits or convulsions (Cores, Telerman-Toppet and Cremer, 1972), and yet others present with psychiatric and personality disorders (Bosak, Wang and Adlersberg, 1957).

The authors describe a case of coeliac disease which presented with severe tetany leading to fractures of both femoral necks, severe muscle and bone pains and an organic mental syndrome which was reversible after treatment.

Case report

A 79-year-old male presented with vigorous seizures which occurred during sleep, lasted 30 min, and were followed by a semi-stuporous state, hypersalivation and severe biting of the tongue. Physical examination revealed a pale confused unco-operative patient. Blood pressure 90/60 mmHg. All muscles were extremely tender. The legs were in external rotation. Neurological examination was unremarkable.

Past history revealed 2 episodes of renal colic due to stones, and an exploratory laparotomy for suspected stomach cancer 8 years previously. Six years before the present admission he had broken his left femoral neck and right ischium on falling from a height of 10 metres.

Five years before, coeliac disease had been diagnosed in another hospital on the basis of the clinical picture, small bowel biopsy and a good response to gluten-free diet. The patient did not keep to the gluten-free diet.

Over the past year he had been admitted to hospital twice for bone and muscle pains, especially in the legs, but had left before investigation.

Investigations

X-rays revealed recent bilateral fractures of the femoral necks. Hb, 5·8 g/dl; urea, 17-43 mmol/l; creatinine, 212-2 μmol/l; calcium, 1·4 mmol/l; magnesium, 0·78 mmol/l; phosphorus, 1·45 mmol/l. Alkaline phosphatase was high (190 i.u./l), and electrophoresis showed it to be of bony origin. Vitamin B12, 350 μg/ml. Creatine kinase was 193 i.u./l, mostly of muscle origin (MM) and there was a creatinuria of 194 mg/24 hr. Calcium urinary excretion was 0·5 mmol/24 hr and phosphorus 29·07 mmol/24 hr.

Xylose test, oral glucose tolerance test and faecal fat indicated malabsorption.

The serum 25-hydroxy-vitamin D (25(OH)D3) was 4·8 ng/ml (normal 10–35 ng/ml) and the serum 24,25 dihydroxy-vitamin D was undetectable (<0·39 ng/ml) (normal values 0·7–2·5 ng/ml).

The intestinal absorption of standard single oral dose of 25(OH)D3 in arachidic oil 10 ng/kg body weight was studied after an 8-hr fast and blood samples obtained at 0, 4, and 6 hr were 5·8 ng/ml, 48 ng/ml and 51 ng/ml respectively (controls, 0 hr = 15±2·2 ng/ml; 4 hr = 82·4±5·6 ng/ml; 6 hr = 82·1±5·8 ng/ml).
Parathyroid hormone was 9.8 i.u./l (normal, 2–5 i.u./l).

Electrocardiogram revealed prolonged ST segment. Electroencephalogram, brain scan and computerized tomography of the head were unremarkable.

**Progress**

The patient was treated with a gluten-free diet, calcium, and vitamin D preparations.

After 2 years on treatment, all the blood tests and absorption tests were normal, and he could walk with the help of 2 sticks. His personality had changed completely to being friendly and cooperative.

**Discussion**

The systemic manifestations of gluten enteropathy are variable (Ross, Gibb and Hoffman, 1966). The present case is of interest in its musculo-skeletal and neurological presentation. The patient had been admitted twice previously for undiagnosed bone pains and myalgia and presented again with severe convulsions causing simultaneous fractures of both femoral necks.

Osteomalacia in coeliac disease is well documented (Melvine, Hepner and Bordier, 1970) and is the result of long-standing hypovitaminosis D and hypocalcaemia. Tetany due to hypocalcaemia may cause bone fractures in these patients, as happened in the present patient.

It is assumed that the patient’s severe muscle pains accompanied by creatininuria and elevation of serum muscular creatine kinase were secondary to the tetany caused by the hypocalcaemia. Weakness and muscle pains may also accompany osteomalacia (Schott and Wills, 1976). Pleasure et al. (1979) demonstrated objective weakness as a result of myopathy in vitamin D-deficient chicks and provided evidence that vitamin D-deficiency has effects on skeletal muscle calcium metabolism, not secondary to altered plasma concentration of calcium and phosphate.

Another point of interest was the change of negative behaviour and lack of co-operation which could be caused by long-standing hypocalcaemia or malabsorption (Bossak et al., 1957). On treatment the patient’s personality and behaviour changed and he became co-operative and friendly. Other investigators have not described personality or behaviour abnormalities (Haas and Haas, 1950).

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**References**


