lesson of the month (2)

An alcoholic presenting with fits

A known alcoholic was admitted for a repeated episode of fits abolished by treating the patient's hypocalcaemia. Several mechanisms of hypocalcaemia in alcoholics have been described but in this patient coeliac disease was also diagnosed. Non-compliance with a gluten-free diet led to poor control of the disease, continued hypocalcaemia and an admission with a repeat seizure leading to death.

Lesson

A 49-year-old known alcoholic was admitted to hospital after being found collapsed with a witnessed minor haematemesis and melaena. The patient had multiple admissions to hospital with seizures that had been attributed to either alcohol intoxication or withdrawal. An emergency endoscopy revealed oesophageal ulcers. He suffered a seizure on admission and while recovering carpopedal spasm was noted and Chvostek's sign was positive. Initial blood investigations included potassium 2.9 mmol/l and calcium of 1.2 mmol/l treated with intravenous (iv) calcium gluconate. Blood tests revealed consistently low calcium levels (Fig 1) and a normal parathyroid hormone, compatible with primary hypoparathyroidism. He was noted to be anaemic and gave a history of diarrhoea. His serological tests for coeliac disease were positive and duodenal biopsy and histology confirmed inflammation and villous atrophy. He was advised to follow a gluten-free diet in addition to standard management for alcohol dependency. The patient re-presented to accident and emergency a few months later with a further seizure and died shortly after admission.

Discussion

Seizures are a frequent reason for admission to emergency departments and can occur on withdrawal as well as during acute intoxication. Hypocalcaemia causes neural hyperexitability leading in the periphery to muscle spasms (Chvostek's sign and Trousseau sign) up to tetany. Changes to the central nervous system include irritability, confusion and in severe cases seizures which require rapid iv calcium gluconate to minimise risk of laryngeal spasm and cardiac arrhythmias. The effects of alcohol ingestion on electrolyte balance are only partly understood but several mechanisms of hypocalcaemia have been implied (Table 1).[1–3]

Coeliac disease is a common condition (prevalence 1:300) with sensitivity to dietary gluten resulting in immune-mediated destruction of small bowel villi, villous atrophy, reduced surface area and malabsorption of calcium and vitamin D.[4] Although detection of tissue transglutimase IgA is a highly sensitive and specific test, the gold standard for diagnosis currently remains duodenal biopsy. Treatment consists of complete avoidance of gluten with restoration of normal mucosa with reversal of malabsorption. Coeliac disease is associated with other autoimmune diseases including idiopathic hypoparathyroidism, which can contribute to hypocalcaemia.

In this case reduced dietary calcium and vitamin intake as well as alcohol-induced metabolic changes might have played a role in the patient's chronic hypocalcaemia, however, it is believed

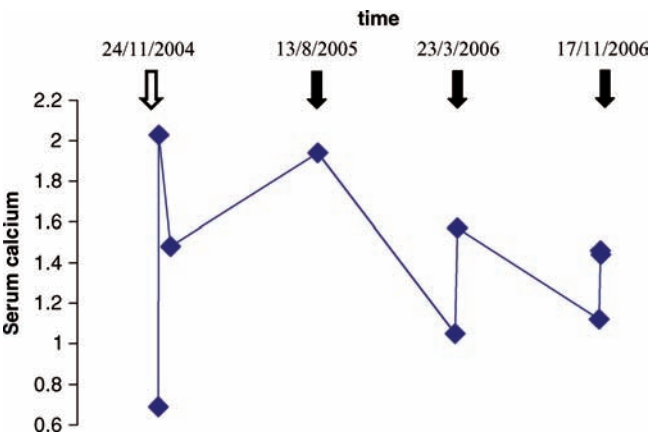

Fig 1. The patient's serum calcium (corrected for albumin). The open arrow indicates a hospital admission for confusion, the closed arrows indicate admissions with presumed 'alcoholic fits'.

Table 1. Possible mechanisms for hypocalcaemia in alcoholics.

Pancreatitis
Malabsorption
Malnutrition with hypoalbuniaemia
Parathyroid hormone (PTH) deficiency
Reduced vitamin D production
Increased magnesium excretion with altered PTH sensitivity

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that the main cause of the fits was his addiction to alcoholic beverages containing gluten, maintaining mucosal inflammation and villous atrophy.

In summary:

- metabolic causes of fits, such as hypocalcaemia, have to be considered and treated even when more common causes are apparent triggers
- alcohol influences calcium metabolism leading to hypocalcaemia
- coeliac disease is a frequent and well-established cause of hypocalcaemia
- in alcoholics with coeliac disease, poor compliance, consumption of beer and other gluten containing beverages as well as generally poor nutrition may all contribute to hypocalcaemia.

References

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book reviews

Axel Munthe. The road to San Michele

By Bengt Jangfeldt. IB Tauris, London 2008. 381 pp. £25.00

A mixture of facts and conscious fiction – Axel Munthe

I read *The story of San Michele* as a boy in the 1930s and was bowled over. Its author, Axel Munthe, was a swashbuckling, Swedish doctor who treated rich European patients, had all sorts of hair-raising adventures, and wrote about his dream house on the isle of Capri. Sixty years on I contributed a profile of Munthe to a medical journal. Few of my colleagues had heard of him, let alone the one-time bestseller, and there was no copy of the book in the city library; the author remained something of a mystery. All is now revealed by the distinguished Swedish professor of literature, Bengt Jangfeldt, in *Axel Munthe. The road to San Michele*, translated by Harry Watson. What must have been a daunting task ordering a mass of correspondence, diaries, personal testimonies, and interviews with relatives has produced a story as gripping as the original, though both authors share a fondness for fantasy.

A recluse who moved in the *grand monde*

The bare bones of Jangfeldt's blow-by-blow account (a chronological table would have been helpful) may be summarised thus. Axel Munthe was born in Sweden in 1857. He qualified as a doctor in Paris and rapidly made a name for himself as a 'neuropsychiatrist'. After six years, he was 'forced into exile' in Capri – an island he had fallen in love with as a young man – by ill health, a failed marriage and, according to him, a quarrel with Charcot over hypnosis. Around 1889 he returned to Rome where he practised from Keats's former house near the Spanish Steps, and soon had an extensive clientele among wealthy expatriates, as well as the city's poor who he treated for nothing. In 1892 he began to look after the Crown Princess Victoria of Sweden and was appointed her personal physician when she became Queen in 1903.

In 1895 he bought the ruins of what was said to be the villa of the Roman Emperor Tiberius at Anacapri and spent five years restoring it to what became San Michele. He left Rome in 1902 to live there in solitude, but still travelled extensively with the Queen and saw patients in Sweden and increasingly in London. In 1907 he married a young Englishwoman, Hilda Pennington-Mellor, an only child of wealthy parents, with whom he had two sons, but they soon agreed to live apart. By this time his eyesight was failing and he moved to Torre Materita, an old castle on the other side of the island to avoid the bright light. There he began laboriously to write *The story of San Michele*; it was published by his friend John Murray in 1929 and became an instant bestseller. He was forced to leave Capri in 1945, due to the second world war, and went to live with the King of Sweden in Stockholm. He died there in 1949 at the age of 91, having failed to return to his beloved Capri.

Bacillus niger

This brief portrait does scant justice to a complex character, full of contradictions and prone to odd behaviour. All his life Munthe complained of hypochondria, melancholy, insomnia, fear of death, and he wrote a piece about being infected with *bacillus niger* (remember Churchill – black dog and Hemingway – black ass?). Surely he suffered from clinical depression? At other times he 'rushed to help' the victims of a cholera epidemic in Naples and an earthquake in Messina; climbed in the Alps in dangerous conditions that cost him three toes from frostbite; and dashed to France in 1914, at the age of 57 and partially blind, to help the Red Cross. He himself called one of these episodes 'demonic'; it would not be too fanciful to label it