# Thyrotoxicosis and abdominal pain: atypical presentation in a middle-aged man

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Thyrotoxicosis is a common endocrine disorder affecting predominantly young to middle-aged individuals. Young people are known to present with classic features of thyrotoxicosis in contrast to the elderly (Kawabe et al, 1986). We report a middle-aged man with thyrotoxicosis presenting to us with severe abdominal pain.

## DISCUSSION

Thyrotoxicosis is associated with increased excretion of calcium and phosphate in urine and stool. In an elderly woman this can be enough to decrease bone mineral density significantly to reduce the fracture threshold (Blahos et al, 1996). Mild hypercalcaemia occurs in a sizeable proportion in thyrotoxicosis (Lersen and Ingbar, 1992). The change in calcium metabolism in thyrotoxicosis may be the result of thyroxine directly stimulating bone resorption and is reversed when the euthyroid state is restored. However, the effect of thyrotoxicosis on vitamin D metabolism is less well understood. Plasma 25-hydroxy-$D_3$ is decreased in thyrotoxicosis, which could contribute to decreased intestinal absorption of calcium; osteomalacia can supervene in some patients (Eriksen et al, 1985).

In thyrotoxicosis, the magnitude of hypercalcaemia is usually small and patients are rarely symptomatic from hypercalcaemia. Ralston et al (1987) reported an elderly patient with thyrotoxicosis who presented with weight loss and hypercalcaemia leading to an erroneous diagnosis of occult malignant disease. The main presentation of our patient was abdominal pain.

At presentation his main problem was thought to be in the abdomen when investigation from that point took the upper hand. Hypercalcaemia can cause abdominal pain and vomiting as a result of acid peptic disease, nephrolithiasis or pancreatitis, which were unlikely in the index patient. With significant reduction of serum calcium pain did not improve much but it did when thyrotoxicosis was treated.

The temporal relationship of abdominal symptoms with thyrotoxicosis and its response with treatment with antithyroid drug made us think that thyrotoxicosis was responsible for the pain rather than hypercalcaemia, although the exact mechanism remained obscure. We conclude that, although uncommon, thyrotoxicosis can present atypically in younger individuals with severe hypercalcaemia and abdominal pain. HM

## CASE REPORT

A 45-year-old man was referred to the surgical assessment unit by his GP with a 1-week history of vomiting and severe abdominal pain. Marked tenderness was noted in the epigastrium and umbilical region. He was dehydrated with blood pressure 100/70 mmHg and pulse of 110/min. The rest of the initial examination was normal. Abdominal and chest X-ray, electrocardiogram, routine blood tests, including full blood count, liver and kidney functions, cardiac enzymes and serum amylase were normal as were the ultrasound scan of the abdomen and endoscopy of stomach and duodenum. Serum calcium was very high at 3.11 mmol/litre (normal 2.10–2.60) with a normal serum phosphate level.

Treatment with normal saline, frusemide and pamidronate (three daily doses of 30 mg each) reduced the serum calcium to 2.94 mmol/litre with little symptomatic improvement. He also had tremor and a small goitre when thyrotoxicosis was suspected and confirmed (free thyroxine 57.5, normal 9–24 pmol/litre; triiodothyronine 8.9, normal 0.8–2.7 nmol/litre; and undetectable thyroid-stimulating hormone, normal 0.4–4 mu/litre). Serum parathyroid hormone subsequently was reported as low (<5, normal 12–81pg/ml). He was treated with carbimazole 40 mg daily with remarkable improvement. Subsequently alternative causes of hypercalcaemia were excluded by a normal serum and urine electrophoresis, and bone scan. Serum calcium returned to normal in 3 weeks time. He received radioactive iodine, and became hypothyroid 5 months later. He had done well on follow-up with 75 μg thyroxine daily and serum calcium remained normal.

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