Case Report: A Patient with Hypercalcaemia

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A 55 year old man was admitted to the Accident & Emergency Department in May 1998 with a 3-day history of tiredness malaise and nausea. He had developed a urinary and upper respiratory infections a few days earlier and his general practitioner treated with a course of Ciprofloxin. Over the next 3 days, his condition deteriorated with gastrointestinal symptoms and unsteadiness. He was taken to the hospital following a fall and on admission his blood urea and creatinine were noted to be 49mmol/L and 800μmol/L respectively. A CPK 419mmol, calcium 3.47mmol and bicarbonate 31mmol were also noted, GT 28, WBC count of 14.4 and an ESR 105mmol were also noted. Dipstick urine showed +3 blood and +3 proteins and +2 WBC with no casts or crystals. He was normoglycaemic. A radiograph of the chest was unremarkable and a brain CT scan excluded a focal neurological lesion. His urine output remained stable at around 1500 mis a day.

In view of marked hypercalcaemia a working diagnosis of acute renal failure secondary to a possible immunoproliferative malignancy e.g. multiple myeloma was made. Hawemodialysis was started on a low calcium bath and further investigations requested.

His immunological markers concluded normal autoantibody screen p and c ANCA complements and plasma electrophoresis. He was hepatitis B & C negative. A skeletal survey was normal. Abdominal ultrasound revealed two normal sized unobstructed kidneys. Renal biopsy showed normal glomeruli but there was dens deposition of calcium in the tubules and the interstitium giving a characteristic appearance.

A repeat history was taken with special emphasis to drug intake as hypercalcaemia was unexplained. He admitted to taking an over the counter tablet for indigestion for the last several months average of upto 36 tablets a week. This tablet was identified as Rennie.

All self medicating tablets were stopped. The patient made a swift recovery requiring no more than two dialysis sessions. His hypercalcaemia and renal function improved with conservative treatment. Two months post discharge his serum calcium was down to 200μmol/L with a calcium level between 2.0-2.5mmol/L. The patient was instructed not to self medicate any over the counter medications for indigestion in future.

This gentleman presented with a classic picture of milk alkali syndrome, which consists of hypercalcaemia renal impairment and metabolic alkalosis[1,2]. Once a well known cause of hypercalcaemia following ingestion of "Sippy" powder for the treatment of peptic ulcer, the incidence has risen again and it is now considered to be the third commonest cause of hypercalcaemia following primary hyperparathyroidism and malignancy.

The "Sippy regimen"[3] consisted of hourly administration of milk or cream with Sippy powder (600mg of magnesium carbonate and 600mg of sodium bicarbonate alternating with a powder containing 600mg-bismuth subcarbonate and 1200-1800mgofsodium bicarbonate). Toxic reactions were noted shortly[4]. The clinical spectrum depends on the amount and duration of alkali and calcium intake. Three clinical syndromes have been recognized. Acute, with hypercalcaemia, GI symptoms, elevated to normal plasma phosphage concentration and acute renal insufficiency, presenting after approximately one week of treatment[5].

Chronic form (Burnett's syndrome)with long history of high milk/alkali intake with evidence of metastatic calcification with band keratopathy and nephrocalcinosismyalgias and pruritis[6]. Laboratory abnormalities are similar to the acute form. Myalgias and pruritis improve slowly to normalization of calcium but there is often minimal orno improvement in renal function. The subacute or intermediate form (also called Cope's syndrome) is usually seen following intermittent intake of alkali/milk for several years[7]. Symptoms are those of acute and chronic types and although patients improve on withdrawal of the offending agents, mild renal impairment persists.

Our patient was taking approximately 234mg of calcium per week for the past many months and although left with mild renal impairment, his symptoms and biochemistry improved on stopping his antacids. It is therefore important to consider milk/alkali syndrome in a patient with unexplained hypercalcaemia and renal failure especially when several over the counter preparations are available for the symptomatic relief from indigestion.

References

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