Letters to the Editor

Heavy metal intoxication from homeopathic and herbal remedies

Sir,
We read with interest the paper by Keen and colleagues.1 The case bears striking similarity to that of a 55 year old Indian man recently treated in our hospital.

For 9 months he had taken 2–4 tablets daily of a resin extract from the Balsamodedron mukul plant. Each tablet contained 6.5–7.5 mg of lead as well as recordable levels of mercury and arsenic. This was associated with anaemia, abdominal colic, constipation and a lead line of the gums; toxicology screen revealed grossly elevated serum lead levels on two occasions at 1,300 μg/l and 990 μg/l respectively (normal < 50 μg/l) but no trace of other heavy metals. Intravenous and subsequently oral chelation therapy with dimercaptosuccinic acid was instituted with prompt response.

Statistics show that poisoning from homeopathic remedies is an increasing problem with Guy’s Hospital Poisons Unit recording 5–15 cases per annum (personal communication). This is probably an underestimate as there is considerable evidence that subclinical cases exist with short-term exposure. The majority of people affected are from the ethnic minorities and the tablets are taken for a wide range of medical conditions. Investigations by the first and third authors confirm that the tablets, though largely manufactured abroad, are freely purchasable in the UK. There continues to be an influx of reports1–4 concerning heavy metal intoxication from herbal and homeopathic remedies despite pleas for standardized practice.5

A. Olujohungbe
P.A. Fields
A.F. Sandford1
A.V. Hoffbrand
Department of Haematology,
Royal Free Hospital and School of Medicine,
Pond Street,
London NW3
1Information Services,
Cheltenham, UK.

References

Cerebellar dysfunction following dextropropoxyphene-induced carbamazepine toxicity

Sir,
The interaction between dextropropoxyphene and carbamazepine was reported in the Postgraduate Medical Journal by Yu et al.1 There are few case reports of this interaction despite the widespread use of both drugs, in clinical practice. The following case report is interesting not only in the dramatic nature of the interaction but also in the presentation of the toxic state.

A 24 year old man had been on long-term carbamazepine therapy for epilepsy and had been prescribed dextropropoxyphene for an ear infection. He presented with an acute onset of slurred speech, incoordination, marked intention tremor, multidirectional nystagmus and ataxia such that the patient could hardly stand. He had taken a total of eight tablets of coproxamol (dextropropoxyphene 32.5 mg/paracetamol 325 mg) over the preceding 24 hours.

An urgent carbamazepine level showed a dramatic four-fold increase in the serum carbamazepine concentration, which was previously within the normal range.

The patient’s signs and symptoms rapidly resolved following 48 hours cessation of the carbamazepine until normal serum levels were achieved.

S. Allen,
Department of Cardiology,
Northwick Park Hospital,
Watford Road,
Harrow,
Middlesex HA1 3UJ, UK.

Reference

Selenium deficiency, reversible cardiomyopathy and short-term intravenous feeding

Sir,
Levy et al.1 reported a patient who developed a cardiomyopathy due to selenium deficiency whilst receiving total parenteral nutrition. A further metabolic derangement that should enter the differential diagnosis of such a patient is hypophosphataemia. Symptomatic hypophosphataemia is a well-recognized complication of parenteral nutrition,2 and its neuromuscular consequences include dysfunction of both skeletal2 and cardiac muscle.3 Phosphate levels are said to fall from the fourth day onwards in patients receiving intravenous alimentation without adequate phosphate supplementation,4 perhaps related to chronic respiratory alkalosis.5 Hence, hypo-

A. Olujohungbe
P.A. Fields
A.F. Sandford1
A.V. Hoffbrand
Department of Haematology,
Royal Free Hospital and School of Medicine,
Pond Street,
London NW3
1Information Services,
Cheltenham, UK.

References
Phosphatemia may have contributed to the relatively acute onset of cardiomyopathy in the patient reported.1

A.J. Larner
Department of Anatomy,
University of Cambridge,
Downing Street,
Cambridge CB2 3DY, UK.

References

Dr Levy replies as follows:

‘Dr Larner is entirely correct to point out the importance of hypophosphatemia as a cause of significant muscle dysfunction, and not simply in patients receiving parenteral nutrition. Our patient repeatedly had an entirely normal serum phosphate concentration.

The take-home message is that patients receiving parenteral nutrition, even for short periods, which contain apparently adequate levels of vitamins, inorganic salts and trace elements, may still become biochemically deficient and manifest clinical signs and symptoms thereof.’

Oesophageal carcinoma presenting as isolated malignant hypercalcaemia

Sir,

Occult malignancy may present as hypercalcaemia and increased levels of parathyroid hormone-related protein (PTHrp), as recently reported in the Journal by Hutcheson et al.1 Only six patients with oesophageal carcinoma presenting as hypercalcaemia have been reported. Only one patient had no local obstructing symptoms or bone metastasis, and serum parathyroid hormone was found to be mildly elevated.2 We report a patient with squamous cell carcinoma of the oesophagus (SCCE) and no obstructing symptoms, presenting as malignant hypercalcaemia, with intact parathyroid hormone (iPTH) suppressed, and increased PTHrp.

A 56 year old man was admitted in June 1991 for acute hypercalcaemia. He had had surgery for duodenal ulcer and cholelithiasis in 1988 and 1989, when serum calcium was 2.1 mmol/l (normal range (NR) 2.1–2.6), and albumin 40 g/l. In the 15 days prior to admission asthenia, anorexia, weight loss, polyuria, polydypsia and altered level of consciousness progressively developed. Investigations disclosed metabolic alkalosis, total serum calcium, 3.69 mmol/l, total serum proteins 75 g/l, phosphorus 0.8 mmol/l. The serum iPTH was <0.3 pmol/l (NR 37–80). Serum PTHrp was determined by competitive radioimmunoanalysis using specific antibody against fragment 1–40 of human PTHrp, INCSTAR, USA and was 11.2 (range in normocalcaemic controls 0.99–7.39). Skeletal X-ray films were normal. Despite rehydration, total serum calcium reached 4.65 mmol/l. Calcitonin, steroids and intravenous biphosphonates were initiated, but no response was observed.

As neck ultrasound disclosed a round-shaped lesion, exploratory surgery was performed. A normal thyroid and scattered lymph nodes were found along the internal jugular chain. Biopsy of three parathyroids showed no abnormalities. Histological examination of one lymph node revealed metastasis of undifferentiated carcinoma. After surgery, hypercalcaemia persisted refractory to treatment and the patient finally died. Necropsy revealed a SCCE of the distal portion of the oesophagus, and metastasis to bone, liver and lungs.

PTHrp plays an important role in the humoral hypercalcaemia of malignancy of SCCE. Tachimori et al.3 have studied 11 SCCE tumour extracts obtained from patients with hypercalcaemia and all had detectable immunoreactive PTHrp. Northern blot hybridization for PTHrp mRNA revealed the expression of two bands which undergo hybridization. The incidence of hypercalcaemia in SCCE ranges between 16 and 56%,4 but in almost all cases local obstructing symptoms are present in our patient. Rapidly progressive dissemination involved the skeleton. However, hypercalcaemia in SCCE is rarely caused by bone metastasis5 and the presence of bone metastasis does not rule out the possibility of humoral hypercalcaemia of malignancy.6 PTHrp causes hypercalcaemia, activating preformed osteoclasts and inhibiting renal phosphate reabsorption.3 Patients with SCCE and hypercalcaemia have lower overall survival rates even in those cases without clinical evidence of bone metastases (survival rate, 9.1% at the 24th month in comparison with 37.8% in those SCCE without hypercalcaemia).6 This poor survival could be related to dedifferentiation of carcinoma cells.

J.M. Fernández-Real
C. Villabona
J. Soler
Department of Endocrinology,
Ciutat Sanitària de Bellvitge,
Universitat de Barcelona,
L’Hospitalet de Llobregat,
Barcelona, Spain.

References
Selenium deficiency, reversible cardiomyopathy and short-term intravenous feeding.

A. J. Larner

doi: 10.1136/pgmj.70.828.764-b

Updated information and services can be found at:
http://pmj.bmj.com/content/70/828/764.3.citation

These include:

Email alerting service
Receive free email alerts when new articles cite this article. Sign up in the box at the top right corner of the online article.

Notes

To request permissions go to:
http://group.bmj.com/group/rights-licensing/permissions

To order reprints go to:
http://journals.bmj.com/cgi/reprintform

To subscribe to BMJ go to:
http://group.bmj.com/subscribe/