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

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References

Dr Levy replies as follows:
‘Dr Larner is entirely correct to point out the importance of hypophosphataemia 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 Hutchesson 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 normalcalcaemic 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 parathyroid glands 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.5 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.

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References
Community-acquired Acinetobacter pneumonia

Sir, Achar and colleagues report a case of community-acquired Acinetobacter calcoaceticus var. anitrus (now known as Acinetobacter baumannii) pneumonia from the Middle East. They summarize cases from the literature but do not include our series of 11 blood culture-positive cases from Darwin, Northern Territory, Australia, with an accompanying review of 34 previously reported cases.

In our ongoing study of adult bacteraemic community-acquired pneumonia at Royal Darwin Hospital since 1986, A. baumannii accounts for 16 of 148 cases (11%). A. baumannii is the third commonest cause of death (nine deaths, case fatality rate 56%), behind Burkholderia (formerly Pseudomonas) pseudomallei (16 deaths) and Streptococcus pneumoniae (12 deaths). Our cases of A. baumannii are characterized by male predominance (14/16), presence of underlying risk factors such as alcoholism or chronic lung disease (16/16), and severe presentation with unilateral chest X-ray consolidation (15/16) and death within 24 hours in fatal cases (6/9). All our community-acquired isolates have been gentamicin sensitive and cefotaxime/ceftriaxone resistant. Pleural effusions are unusual.

Because B. pseudomallei and A. baumannii account for 48% of adult mortality from community-acquired pneumonia in our region of tropical Australia, our protocol for empirical therapy of severe pneumonia is to add gentamicin to ceftriaxone in patients with underlying risk factors. If B. pseudomallei is isolated or strongly suspected (for example, in diabetics), ceftazidime is substituted. If A. baumannii is isolated, piperacillin is added to the gentamicin and ceftriaxone ceased, or imipenem monotherapy is substituted. Despite a high level of suspicion, intensive-care facilities and appropriate antibiotics, mortality from these two organisms remains high. This is consistent with studies from Thailand demonstrating that ceftazidime therapy, while halving mortality from septicaemic melioidosis, does not substantially decrease mortality in the first 48 hours.

References

Watermelon stomach in the CREST syndrome

Sir, El Omar and colleagues describe the interesting association between severe bleeding from gastric telangiectasia and the CREST syndrome, and the authors conclude that this association is surprisingly rare with only four previous reports. They also comment that gastric vascular abnormalities can be very extensive giving rise to the watermelon stomach. I have recently dealt with such a problem in a patient with the CREST syndrome and this case illustrates several important points in the management of such patients.

A 46 year old female with CREST syndrome was referred for assessment of chronic iron deficiency. Two years previously gastritis and oesophagitis had been treated with omeprazole, but because of continued bleeding, with negative investigations including angiography, a laparotomy and on-table enteroscopy had been performed. Small bowel telangiectasias were found and the most severely affected segment of ileum was excised.

At the time it was known that some small bowel vascular lesions had been left in situ. This procedure was initially successful but after 18 months iron deficiency anaemia resistant to oral iron was again apparent; at this stage she was receiving approximately 3 units of blood every 3 weeks.

At upper gastrointestinal endoscopy red streaks radiating out from the pylorus were seen, characteristic of watermelon stomach. Histological examination of endoscopic biopsies confirmed the diagnosis. Selective visceral angiography showed subtle abnormalities with hypervascularity of the antrum but the small bowel vasculature appeared normal. A technetium-99 labelled red cell scan suggested that the blood loss was from the gastroduodenal region rather than the distal small bowel.

Her bleeding has been controlled by endoscopic treatment with heater probing and injection of alcohol subsequently because of a technical problem with the heater probe. Following ablation of the endoscopically visible lesions she has remained stable for 13 months, she has required no blood transfusions and there is no further evidence of bleeding.

This report illustrates one further case of symptomatically bleeding gastric telangiectasias with the CREST syndrome in addition to those described by El Omar et al. and is unusual amongst these in demonstrating the extensive lesion called watermelon stomach.

It shows that such patients may have multifocal gastrointestinal vascular lesions and recurrence of anaemia following surgery may occur. Careful assessment using endoscopy, biopsy, angiography and radio-