Late compression may be due to callus at the fracture site in the presence of solid union, fibrous tissue at a site of non-union, or a false subclavian artery aneurysm formed as a consequence of traumatic damage to this vessel.^{3,4} In a survey of world literature between 1960 and 1987, Bahnini and Kieffer noted only 76 cases of thoracic outlet syndrome secondary to clavicular trauma. In two thirds, the subclavian vessels were involved in addition to the brachial plexus.⁵ Isolated involvement of the brachial plexus, as in this case, is unusual. Similarly, our patient's presentation of weakness without sensory symptoms is uncommon. In 16 patients with lesions of the brachial plexus after clavicular fractures seen over 20 years at two surgical centres, all had pain and parasthesia in the affected arm.³

Experience in managing brachial plexus damage resulting from callus, is limited by the rarity of this condition. Operative techniques have included resection of the callus, osteotomy and fixation to correct malunion,

and resection of the first rib, or of the clavicle itself, to widen the costoclavicular passage.^{1,3} The results of surgery have been variable, with good arm function returning in only some patients. Outcome is usually better when surgery is performed shortly after the onset of symptoms. In our patient, it was felt that surgery was inappropriate in view of his severe disability resulting from Steele-Richardson-Olszewski syndrome, and the long time from onset of symptoms to the final diagnosis.

Final diagnosis

Diffuse injury to the brachial plexus by callus following healing of a fractured clavicle.

Keywords: clavicular fracture, callus, brachial plexus.

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Hyponatraemia and spontaneous hypoglycaemia

OMP Jolobe, TS Htin

A 76-year-old woman receiving neither diuretic, psychotropic, nor antidiabetic medication, presented with recent-onset mental confusion, in association with a blood pressure of 95/45 mmHg, rectal temperature of 34°C (during the summer), plasma sodium 115 mmol/l, potassium 3.3 mmol/l, urea 2.5 mmol/l, glucose 1.5 mmol/l. Subsequently, she had tests showing a plasma osmolality of 236 mOsm/kg (by osmometry), urine osmolality of 579 mOsm/kg, and a urinary sodium of 118 mmol/l. Chest X-ray showed no abnormality.

Questions

- 1 What is the most probable cause of the association of dilutional hyponatraemia and spontaneous hypoglycaemia in this patient?
- 2 How would you confirm the diagnosis?
- 3 How would you manage this patient?

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Answers

QUESTION 1

Adrenal crisis is the most probable cause of the association of dilutional hyponatraemia and spontaneous hypoglycaemia in a patient with this clinical presentation. In cortisol-deficient states, antidiuretic hormone release is not inhibited by a fall in plasma osmolality,¹ and the resulting failure of excretion of a water load leads to dilutional hyponatraemia. When hypoglycaemia complicates cortisol deficiency, this is a consequence of impairment of gluconeogenesis, along with impairment of hepatic glucose production and glycogen synthesis.² Coexisting growth hormone deficiency can aggravate this phenomenon, sometimes even leading to amelioration of diabetes mellitus after development of hypopituitarism, the so-called Houssay phenomenon.

QUESTION 2

With heightened awareness that 'cortisol deficiency is on the diagnostic list in every case of unexplained hypoglycaemia',² the diagnosis could most speedily be confirmed by opportunistic measurement of plasma cortisol and adrenocorticotrophic hormone (ACTH) levels simultaneously with assay of plasma glucose, since, in a hypoglycaemic patient, a plasma cortisol of <500 nmol/l signifies hypoadrenalism, concomitant elevation of plasma ACTH to >100 ng/l being highly specific for primary hypoadrenalism, although levels below 81 ng/l do not necessarily signify secondary hypoadrenalism.³ More elective dynamic tests involve quantification of the cortisol response to the short synacthen test (SST), which utilises a 250 µg test dose of 1,24-tetracosactrin. The low-dose tetracosactrin test, utilising a 1 µg test dose, has been proposed as a more sensitive alternative, on the basis that relatively physiological test doses might be less likely to mask mild-to-moderate hypoadrenalism.⁴ A useful component of dynamic testing is the quantification of basal levels, not only of plasma cortisol, but also of plasma ACTH, in order to differentiate between primary and secondary hypoadrenalism. Alternatively, the diagnosis of secondary hypoadrenalism can be established by documenting a restoration of adrenal responsiveness to the SST, after stimulating the gland with a three-day course of 1 mg/day depot synacthen. More complicated principles are involved in the use of the metyrapone test, which also incurs the risk of precipitating adrenal crisis. In the present instance, the SST yielded a basal serum cortisol of 196 nmol/l, increasing to 199 nmol/l 30 minutes after the test dose, a normal response being characterised by a peak serum cortisol of >500 nmol/l.

QUESTION 3

Emergency treatment should include the administration of intravenous dextrose and hydrocortisone, followed by intravenous saline, and the identification and correction of the

Learning points

- the association of dilutional hyponatraemia and spontaneous hypoglycaemia is highly characteristic of adrenal failure
- the management of adrenal crisis should include a search for intercurrent infection, which may be of relevance, even when apparently trivial
- even in old age, women with obstetric histories remain potential candidates for the late presentation of adrenal complications of Sheehan's syndrome

precipitating cause (in this instance, urinary tract infection). None of these measures should await the performance of elective dynamic tests outlined above, because diagnostic accuracy is not impaired by replacement therapy, provided care is taken to substitute dexamethasone 0.5–1.0 mg/day for hydrocortisone (or prednisolone) prior to dynamic testing, so as not to interfere with the cortisol assay.² For definitive management of the underlying cause, a distinction needs to be made between primary and secondary adrenal failure, so that underlying pathogenetic mechanisms can be identified with appropriate imaging modalities. In the present case, evidence supporting the presence of pituitary disease, and hence, secondary hypoadrenalism, came from laboratory tests showing stigmata of hypogonadotropic hypogonadism, namely, serum luteinising hormone 0.8 IU/l (13–70), serum follicle-stimulating hormone 3.6 IU/l (35–151), and serum oestradiol 62 pmol/l (<130). Serum prolactin was also low (<10 mU/l), but serum thyroxine and thyroid-stimulating hormone levels were normal. On computed tomography of the pituitary and adrenals, there was no focal abnormality to rule out the diagnosis based on her history of menstrual and lactatory failure following postpartum haemorrhage at 40, namely, Sheehan's syndrome. In the modern era this is a diagnosis for which the alternatives include pituitary necrosis complicating diabetic pregnancy, and autoimmune lymphoid hypophysitis.⁵

Outcome

The patient responded well to the measures outlined above, and she was discharged with arrangements for obtaining a steroid 'bracelet', indicating her dependence on replacement therapy. Such patients should also keep a supply of parenteral hydrocortisone (or equivalent corticosteroid) in the refrigerator for emergency use in an adrenal crisis.

Final diagnosis

Adrenal crisis.

Keywords: adrenal crisis, hyponatraemia, hypoglycaemia

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Skin rash in a jaundiced patient

M Akasheh, K Omari, F Madanat

A 62-year-old woman presented with a violaceous, pruritic skin rash affecting her face, central chest, forearms and thighs (figure 1). She had lost 12.7 kg over the last two months. She also complained of difficulty in combing her hair, and had mild dysphagia to solid food.

On examination, in addition to the rash, she was found to be jaundiced, with proximal muscle tenderness and weakness. There were no palpable organs or lymph node enlargement.

Laboratory findings were as follows: haemoglobin 11.0 g/dl, packed-cell volume 0.31, white blood cell count $13.8 \times 10^9/l$, platelets $149 \times 10^9/l$, erythrocyte sedimentation rate 72 mm/h, total bilirubin 107 $\mu\text{mol/l}$, alanine transaminase 70 IU/l, aspartate transaminase 67 IU/l, lactate dehydrogenase 420 IU/l. Abdominal computed tomography (CT) scan is shown in figure 2.

Figure 1

Figure 2

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Questions

- 1 What is the skin rash due to?
- 2 What other investigations should be performed?
- 3 What abnormality is shown on the abdominal CT scan?
- 4 What is the probable cause of her dysphagia?
- 5 What is the underlying diagnosis?