Thirst and polyuria

An interesting case of thirst and polyuria

P K Moulik, C Nethaji, A A Khaleeli

Answers on p 251.

A 24 year old woman presented with a three week history of weight loss, thirst, polyuria, sweating, and nausea. For three days before admission she had abdominal pains and vomiting after meals. Past medical history included acromegaly treated by trans-sphenoidal resection of a pituitary macroadenoma at the age of 19 years. Postoperatively, she did not have diabetes insipidus; however, she developed anterior hypopituitarism replaced with hydrocortisone 10 mg twice daily, thyroxine 100 µg daily, and ethinyl-oestradiol and desogestrel for three weeks every month. She also had persistent postoperative acromegaly that was well controlled with bromocriptine 7.5 mg daily. She had not received postoperative radiotherapy.

Clinical examination revealed postural hypotension with supine blood pressure 130/70 mm Hg and standing blood pressure 85/45 mm Hg. She was in sinus rhythm with a heart rate of 94 beats/min. Visual acuity was 6/24 in the right eye and 6/5 in the left eye. Visual field examination showed temporal hemianopia in the right eye and full field in the left eye. Fundoscopy showed bilateral optic atrophy, right more than left, likely the result of her pituitary macroadenoma. She had mild periumbilical tenderness to deep palpation without any guarding or rigidity. Bowel sounds were present. The rest of the clinical examination was normal.

Investigations showed normal electrolytes, urea, creatinine, glucose 3.2 mmol/l, serum osmolality 297 mmol/kg water (reference range 288–298) and urine osmolality 178 mmol/kg water. Corrected serum calcium was raised at 3.01 mmol/l (reference range 2.15–2.55 mmol/l) with parathyroid hormone (PTH) <0.08 pmol/l indicating non-parathyroid cause of hypercalcaemia. Insulin like growth factor 1 was normal at 17 nmol/l (reference range 15–64). She was rehydrated with intravenous isotonic saline and her dose of hydrocortisone temporarily increased but her hypercalcaemia persisted.

A water deprivation test was normal, with urine osmolality 1032 mmol/kg water and serum osmolality 287 mmol/kg water after eight hours of water deprivation. Results of thyroid function tests were available at this stage showing free thyroxine 111 pmol/l with thyroid stimulating hormone <0.03 mU/l. Her thyroxine replacement was stopped and a technetium (99mTc) pertechnetate thyroid uptake scan was done after seven days (fig 1). Thyroid stimulating hormone receptor autoantibodies (TSHRAb) were positive at 75 U/l (reference range <10 U/l). Magnetic resonance imaging revealed an enlarged empty pituitary fossa with a thin rim of pituitary tissue in the periphery.

QUESTIONS

(1) What are the possible causes of thirst and polyuria in this woman?
(2) What are the possible causes of hypercalcaemia?
(3) What is the cause of her thyrotoxicosis?
(4) Is there an association between acromegaly and thyrotoxicosis?

Figure 1 Pertechnate thyroid uptake scan.

Skin lesion

A proliferating pimple

S Puppala, V P Muralikrishnan, D M Wayte, L R Jenkinson

Answers on p 251.

A 63 year old man was referred by the general practitioner with a skin lesion on the right buttock which he had for three months. The lesion started as a small “pimple” and gradually increased in size. It was associated with occasional minor bleeding and pain when touched. There were no other systemic symptoms and no associated past medical history. Physical examination revealed a 5 cm firm erythematous skin nodule with involvement of the subcutaneous tissue as shown in the fig 1A. There was an indurated erythematous “halo” around its base. A biopsy of the lesion was performed and the histology was as shown in the fig 1B.
A 72 year old woman presented with a fleeting rash, lasting several days at a time, on both thighs and lower trunk, over a four month period (fig 1). The eruption was non-pruritic and non-palpable, and occurred on different parts of the legs at different times. She had a 30 year history of seropositive, erosive rheumatoid arthritis and a five year history of secondary Sjogren’s syndrome. The patient also had two sinuses on the sole of her right foot, which had been present for a year, and which were being treated conservatively with cleansing and dressing. There were no other new symptoms on admission. Her medication had not changed in over a year, and consisted of methotrexate 15 mg/week (plus folinic acid), prednisolone 15 mg/day, diclofenac, thioridazine, and doxepin.

She had a mild normocytic anaemia (haemoglobin concentration 117 g/l) with a raised ferritin level (531 µg/l), while leucocyte and platelet counts were normal. Renal and liver function blood tests, as well as plasma calcium and phosphate concentrations were all within the normal range. The erythrocyte sedimentation rate (Westergren) was 42 mm in the first hour, and C-reactive protein level was 62 mg/l. Rheumatoid factor titre was 320; tests for antinuclear antibodies, antibodies for extractable nuclear antigen, and cryoglobulins were negative.

**QUESTIONS**

(1) What is the name given to this rash, and how is the appearance described?

(2) What underlying diseases does it suggest?

**Answers on p 252.**

**Figure 1** Rash on medial aspect of both legs (reproduced with patient’s permission).

**Figure 1 (A)** Erythematous skin nodule and (B) histology of lesion.
A 15 year old girl with fever, jaundice, haemolysis, and sudden clinical deterioration

M Pirisi, B Branca, C Avellini, A Solinas

Answers on p 253.

A 15 year old girl presented to an internal medicine ward with a two day history of jaundice and intermittent, high grade fever. Low grade fever and constitutional symptoms had been present for three weeks, during which the only medication taken was ibuprofen. Her mother recalled having found rat excrement in her knapsack, after the girl had returned from a rural resort, a few days before the onset of her illness. All family members had eaten seafood on a single occasion, two weeks before admission. She was not sexually active, had never been transfused, had no known exposure to hepatotoxic chemicals, and did not drink alcohol or use recreational drugs. There was no family history of liver disease or of recent travel to foreign countries. On physical examination, she was alert and cooperative. There was hepatomegaly and diffuse abdominal tenderness; ascites and stigmata of chronic liver disease were absent.

Laboratory data are shown in table 1. There were no schistocytes on the blood smear, nor were corneal opacities found at slit lamp examination. Abdominal ultrasound and duplex Doppler sonography confirmed the presence of marked liver enlargement (longitudinal diameter 20 cm); spleen longitudinal diameter was 14 cm. Bile ducts were not dilated, the hepatic artery, portal vein and hepatic veins were patent, and there was no free peritoneal fluid.

In the next six hours confusion, anuria, and respiratory distress ensued. The patient was transferred to an intensive care unit and a liver biopsy was performed (fig 1).

**QUESTIONS**

(1) What does the liver biopsy show?

(2) What is the most likely diagnosis?

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**Table 1** Haematological and blood chemistry values

<table>
<thead>
<tr>
<th>Variable*</th>
<th>Value</th>
<th>Normal range</th>
</tr>
</thead>
<tbody>
<tr>
<td>White blood cells ($\times 10^9/l$)</td>
<td>26.2</td>
<td>4.5–9.8</td>
</tr>
<tr>
<td>Red blood cells ($\times 10^{12}/l$)</td>
<td>1.91</td>
<td>4.2–5.6</td>
</tr>
<tr>
<td>Haemoglobin (g/l)</td>
<td>65</td>
<td>120–160</td>
</tr>
<tr>
<td>Packed cell volume</td>
<td>0.19</td>
<td>0.37–0.47</td>
</tr>
<tr>
<td>Platelets ($\times 10^9/l$)</td>
<td>364</td>
<td>140–400</td>
</tr>
<tr>
<td>MCV (fl)</td>
<td>99</td>
<td>82–98</td>
</tr>
<tr>
<td>Absolute reticulocyte count ($\times 10^9/l$)</td>
<td>191</td>
<td></td>
</tr>
<tr>
<td>Prothrombin time (sec)</td>
<td>26.3</td>
<td></td>
</tr>
<tr>
<td>INR</td>
<td>2.64</td>
<td></td>
</tr>
<tr>
<td>Bilirubin ($\mu mol/l$)</td>
<td>347</td>
<td>3–17</td>
</tr>
<tr>
<td>Alkaline phosphatase (U/l)</td>
<td>8</td>
<td>32–104</td>
</tr>
<tr>
<td>Alanine aminotransferase (U/l)</td>
<td>20</td>
<td>10–36</td>
</tr>
<tr>
<td>Lactate dehydrogenase (U/l)</td>
<td>817</td>
<td>211–423</td>
</tr>
<tr>
<td>Haptoglobin</td>
<td>Undetectable</td>
<td></td>
</tr>
<tr>
<td>Coombs test (direct and indirect)</td>
<td>Negative</td>
<td></td>
</tr>
<tr>
<td>Glucose-6-phosphate dehydrogenase</td>
<td>Normal</td>
<td></td>
</tr>
<tr>
<td>Pyruvate kinase</td>
<td>Normal</td>
<td></td>
</tr>
<tr>
<td>Autoantibodies (SMA, ANA, and MIT)</td>
<td>Negative</td>
<td></td>
</tr>
<tr>
<td>Serology for hepatitis viruses A, B, and C</td>
<td>Negative</td>
<td></td>
</tr>
<tr>
<td>Ceruloplasmin (mg/dl)</td>
<td>20.8</td>
<td>20–60</td>
</tr>
</tbody>
</table>

*MCV, mean corpuscular volume; INR, international normalised ratio; SMA, antismooth muscle antibodies; ANA, antinuclear antibodies; MIT, antimitochondrial antibodies.

**Figure 1** Liver biopsy specimen: (A) haematoxylin and eosin × 100 and (B) haematoxylin and eosin × 400.
An episodic eruption

P Bentley and A Keat

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