CASE REPORTS

Sore throat and hyperferritinaemia

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The conjunction of sore throat and hyperferritinaemia is a diagnostic clue easily missed.

CASE HISTORY

A previously well woman aged 23 had experienced sore throat, rash and polyarthralgia for 3 weeks. Initially, the sore throat had been severe enough to prevent her taking solids. Positive findings on examination were a temperature of 39°C; a widespread, non-pruritic, macular erythematous rash; pharyngeal injection; right wrist swelling; and a sinus tachycardia. The results of preliminary investigations were as follows: haemoglobin 11.4 g/dL (reference range 11.5–16.5), white cell count 40.3×10^9/L (4.0–11.0), neutrophils 38.7×10^9/L (2.0–7.5), platelets 470×10^9/L (150–400). The erythrocyte sedimentation rate (ESR) was 92 mm/h (0–20). Albumin was 21 g/L (35–46), alkaline phosphatase 252 IU/L (31–116), gamma-glutamyl transpeptidase 208 IU/L (0–72), C-reactive protein 411 mg/L (0–7). Urea, electrolytes, creatinine, glucose, calcium, creatine kinase, other liver function tests, thyroid function tests, urinalysis and microscopy were normal.

The profound acute-phase response initially suggested an infective substrate. However, serial blood cultures, antistreptolysin O titres, an atypical-pneumonia screen, throat swab and VDRL were negative, as were titres to Epstein–Barr virus, cytomegalovirus, rubella, parvovirus and hepatitis A and B. Abdominal ultrasound revealed slight hepatosplenomegaly, without lymphadenopathy. Autoantibodies were negative. Serum ferritin was greatly raised at 15 628 μg/L (15–400).

The patient remained unwell over the next few days, with intermittent fevers (Figure 1) often accompanied by transient worsening of the rash. The haemoglobin and serum albumin decreased further. Pleuritic chest pain developed, accompanied by pericardial and bilateral pleural rubs. An electrocardiogram showed new widespread concave-up ST elevation, and transthoracic echocardiography revealed a small pericardial effusion. Adult-onset Still's disease was diagnosed (Box 1) and the pleuropericarditis resolved within 24 hours from the start of prednisolone therapy. Methotrexate was later added, and over the next few weeks the joint pains lessened, her wellbeing improved and acute-phase reactants became normal.

COMMENT

Adult-onset Still's disease is a systemic inflammatory disease of uncertain aetiology with a prevalence of around 1 in 100 000 adults, presenting with a striking constellation of clinical and laboratory features, none of them pathognomonic[1]. Most patients develop fever, rash and arthralgia, but this classic triad is often absent during the first month of illness and diagnostic delays are common[1]. The fever usually exceeds 39°C, with temperature tending to be high in the evening and normal in the morning. The rash is generally non-pruritic, macular or maculopapular and salmon-pink, and is most prominent on the trunk and proximal portions of the limbs. It becomes more apparent during periods of fever and is readily precipitated by thermal or mechanical stimulation. The arthritis is polyarticular in two-thirds of cases, oligoarticular in one-third, and has a predilection for large joints, particularly the wrists. The knees and ankles are also frequently affected[1–3]. Other common features are sore throat, lymphadenopathy, hepatosplenomegaly, pleuropericarditis, raised ESR, hypoalbuminaemia, abnormal liver function tests and anaemia[1]. The classification criteria proposed by Yamaguchi are the most sensitive and specific[2] (Box 1).

Two forms of adult-onset Still's disease are recognized, though they may be difficult to differentiate early on. Two-thirds exhibit a predominantly systemic form, in which articular symptoms may occur but are overshadowed by

Box 1 **Classification criteria for adult-onset Still's disease** (Ref. 2)
(Presence of five or more criteria, including at least two major, has diagnostic sensitivity of 96.2% and specificity of 92.1%)

Major
- Fever (39°C or more for at least 1 week)
- Arthralgia (for at least 2 weeks)
- Typical rash
- Leucocytosis (10×10^9/L or greater, with at least 80% neutrophils)

Minor
- Sore throat
- Lymphadenopathy and/or splenomegaly
- Liver dysfunction
- Negative rheumatoid and antinuclear factor

Exclusions
- Infections
- Malignancies
  Other rheumatological disorders

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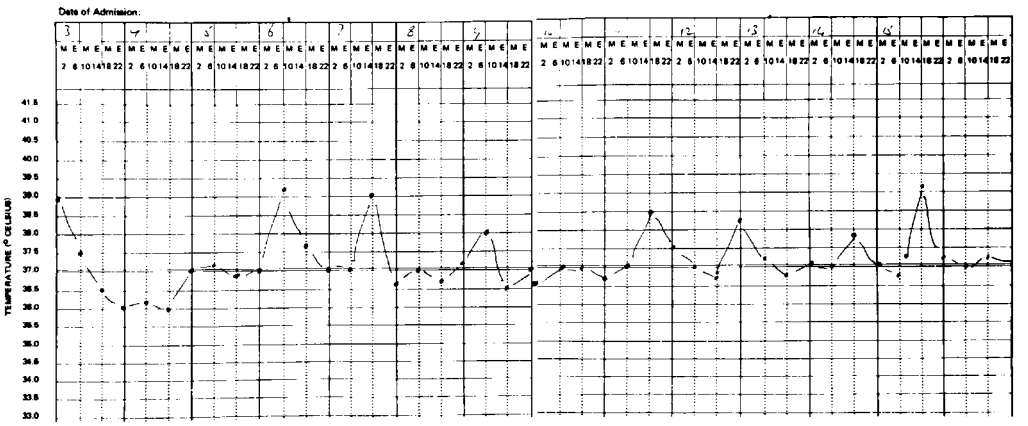

Figure 1 **Temperature chart**

systemic features. The other third have a chronic articular form, in which joint involvement predominates over systemic features. This group have a worse prognosis and are characterized by chronic progressive polyarthritis with joint ankylosis and destruction[1,3]. The clinical course in adult-onset Still's disease is therefore highly variable, just under a quarter of patients presenting with an episode of predominantly systemic illness followed by prolonged remission at one end of the spectrum, another third with a chronic persistent disorder who develop progressive joint damage at the other, and a group characterized by intermittent exacerbations in between[1,3]. Overall, half the patients still require medication 10 years after diagnosis but pain and disability are less than in other rheumatic diseases and functional outcomes tend not to be compromised[4].

Treatments for adult-onset Still's disease have not been evaluated in controlled studies and remain empirical. Non-steroidal anti-inflammatory drugs are occasionally effective as monotherapy but most patients require steroids, which may be needed in high doses for 6 months, followed by long-term low-dose maintenance in severe disease. Disease modifying drugs and immunosuppressives are used, and there is some evidence that methotrexate is effective[5].

This case illustrates two features which are important early diagnostic clues—sore throat and extreme elevation of serum ferritin. In a review of 341 cases, sore throat was reported in 69%[6]. This symptom is therefore a cardinal feature of adult-onset Still's disease and is particularly prevalent in the first month of illness, often predating other symptoms. Typically, it persists for a few days and may be associated with odynophagia, resolving shortly after the full range of systemic symptoms are manifest. Examination reveals mild pharyngeal injection only, and throat cultures are negative. An awareness of this association is important since it is uncommon in other rheumatic diseases and considerably diminishes the differential diagnosis in a patient with fever, rash and arthralgia[1,6].

Serum ferritin is usually high in active adult-onset Still's disease. The concentration is five or more times the upper limit of normal in almost three-quarters of patients and values above 10 000 μg/L have been recorded[7] (as here). As an acute-phase reactant, ferritin is also raised in infectious and inflammatory conditions, hepatocellular disease, haemochromatosis and secondary iron overload, and malignancy. However, the increment in adult-onset Still's disease is disproportionate to the inflammatory state alone and, with the exception of acute hepatic necrosis, concentrations exceeding 5000 μg/L are seldom encountered in other disorders[7,8].

Hyperferritinaemia in an appropriate clinical context, such as an undefined rheumatological disorder or pyrexia of unknown origin, is therefore suggestive of adult-onset Still's disease[7,8]. Furthermore, serum ferritin becomes normal with treatment and can be used to monitor disease activity.

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Jet-ski injury: severe blunt neck trauma with survival

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Few people survive blunt trauma in which the trachea and oesophagus are transected.

CASE HISTORY

A 10-year-old girl sustained a severe neck injury following blunt trauma to the neck in a high-speed jet-skiing accident in Egypt, in which she hit a line outstretched between two boats. After rescue from the sea she was unconscious with an obstructed airway. An emergency cricothyroidotomy was performed by a passing anaesthetist, followed by immediate intubation via the cricothyroidotomy site to relieve the airway obstruction. She was then transferred to a local hospital.

Her injuries included a fracture/dislocation of the second on third cervical vertebrae (C2-C3) without evidence of spinal cord or spinal nerve root injury. Exploration of the neck revealed a transected trachea and oesophagus. She was electively paralysed and mechanically ventilated via a formal tracheostomy and her cervical spine was stabilized by halotraction. A feeding gastrostomy was inserted.

Three weeks following the injury she was transferred, paralysed and mechanically ventilated, to Great Ormond Street Hospital, London. The neck wound now was grossly infected and she was extremely malnourished: free gastro-oesophageal reflux had caused difficulties in establishing gastrostomy feeds. The proximal oesophagus had closed off, requiring her to spit out her saliva. The distal oesophagus remained open as a fistula in the neck wound. Plain X-ray (Figure 1), computerized tomography (CT) and magnetic resonance imaging (MRI) of the cervical spine showed a well opposed but clear subluxation of C2 on C3. Four days after admission she underwent an occiput to C4 posterior spinal fusion with split calvarial bone, and a feeding jejunostomy was inserted to provide full enteral nutrition. Immobilization of the neck was maintained for three months after this operation, with a halo-jacket, until CT scanning suggested the fusion was complete. She was mobilizing with assistance within one month.

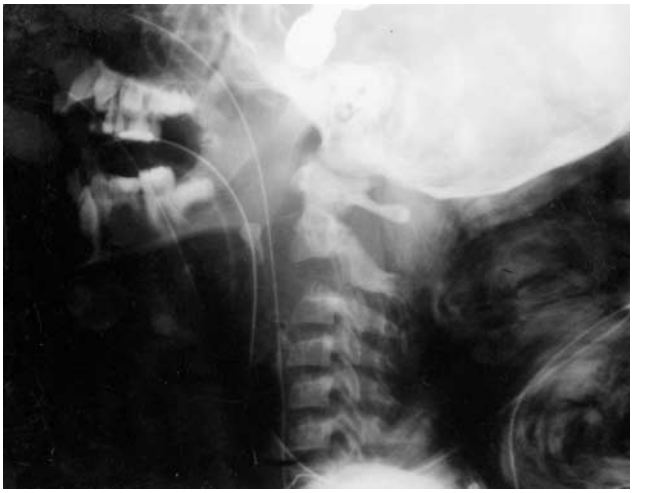

Figure 1 **X-ray of cervical spine showing subluxation of C2 on C3**

Reconstruction of her oesophagus was undertaken four months after admission. Flexible bronchoscopy and oesophagoscopy showed paralysed and abducted vocal cords, indicating bilateral recurrent laryngeal nerve injury. The fistulous opening in the neck communicated with the lower oesophagus. The distal part of the cervical oesophagus was mobilized into the superior mediastinum and anastomosed end-to-end with the proximally transected oesophagus. The trachea was reconstructed three weeks later. Laryngeal release and mobilization of the thoracic trachea was necessary to allow closure without tension. A new tracheostomy was constructed distal to the repair, during which a laryngocutaneous fistula and an anterior oesophageal fistula were identified and closed. The oesophageal reconstruction healed with a minor stricture which responded to bouginage. Continuing problems with aspiration and recurrent respiratory infections developed because of laryngeal incompetence and necessitated an epiglottopexy to protect the airway. As a result she was aphonic but could communicate with an electrolarynx.

At one-year follow-up, swallowing was unimpeded with no evidence of aspiration and weight gain was satisfactory. She was coping well with her permanent tracheostomy and was again enjoying jet-skiing.

COMMENT

Few cases of combined oesophageal and tracheal transection in association with a cervical spine injury have been reported previously in blunt neck trauma. Hermon *et al.*[1], Ayabe *et al.*[2], and Aseoka *et al.*[3] reported combined tracheal and oesophageal rupture, but in these cases transection was

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not complete. Chen *et al.*[4] reported complete cricotracheal separation in association with cervical spine injury, but their patient did not sustain an oesophageal injury.

Complete disruption of the trachea is usually fatal. Severe neck trauma is often associated with cervical spine injury, recurrent laryngeal nerve damage and damage to other vital structures. Pneumomediastinum and pneumothorax requires emergency tube thoracostomy and tracheostomy[5]. Rupprecht *et al.*[6] reported acute haemorrhage from rupture of the thyroid gland following blunt neck trauma. Other complications include acquired tracheo-oesophageal fistula[7,3] and retropharyngeal abscesses[8].

A specialist multidisciplinary approach to management of these rare cases is important and is best achieved in a tertiary referral centre. The extent of the injury must be delineated accurately by CT, MRI and contrast studies. The timing of the various stages of the operative reconstruction requires careful planning.

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An epitrochlear tumour

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Surgical exploration of a painful epitrochlear tumour gave a surprising result.

CASE HISTORY

A woman of 46 noticed a painful mass 4 cm proximal to the medial epicondyle of her right arm; there was no history of trauma or infection. The mass was $3 \times 4 \times 2$ cm when measured clinically and did not involve the skin or underlying musculature. There were no signs of acute infection, lymphangitis or axillary lymphadenopathy and the white blood cell count was normal. Magnetic resonance imaging showed a 6×3 cm encapsulated tumour superficial to the fascia (Figure 1) and seeming to involve the epifascial veins. On surgical exploration it proved to be an encapsulated abscess, histologically a vascular inflammatory granuloma. Special staining excluded typical and atypical

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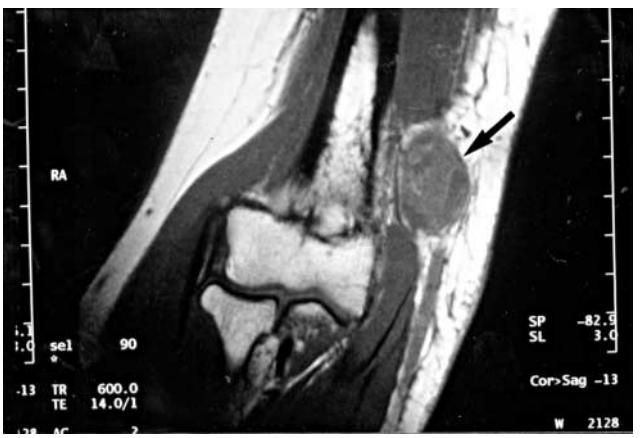

Figure 1 **T1 weighted magnetic resonance image of right elbow demonstrating epifascial mass (arrow) consistent with neurovascular tumour, abscess or haematoma**

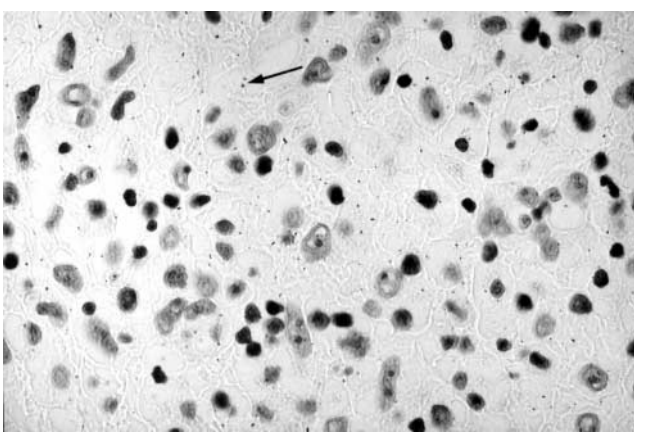

Figure 2 **Warthin–Starry silver stain demonstrating argyrophilic aggregates of bacteria (arrow) (× 63)**

Figure 3 **Peripigmented papule between fourth and fifth metacarpal heads (arrows)**

mycobacteria. Warthin–Starry silver staining revealed aggregates of Gram-negative bacteria characteristic of *Bartonella henselae* infection (Figure 2). IgG antibodies to *Bartonella*, measured by an indirect immunofluorescence assay, rose postoperatively from normal ($<$1:64) to $>$1:512, confirming the diagnosis. The patient's history was re-evaluated. She had recently acquired a one-year-old cat and had noticed a red papule on the skin of her right hand 3½ weeks before the onset of the painful swelling in her elbow which was still present as hyperpigmentation 6 weeks later (Figure 3). She also mentioned an episode with headache and general fatigue without fever, after the appearance of the papule. Her postoperative course was uneventful.

COMMENT

In 1889, Parinaud reported an oculoglandular syndrome of lymphadenopathy and conjunctivitis that is now known as cat-scratch disease (CSD). In 1950, Debré *et al.*[1] described the relationship of adenopathy and house-cat contact. Several genetically related organisms have been implicated in the aetiology. Initially, *Afipia felis* was believed to be the causal agent. Subsequently, *Rochalimaea* species were associated with an immune response in patients with CSD. These were renamed as *Bartonella* in view of extensive 16S rRNA sequence homology with organisms in this genus[2]. Recently *B. henselae* has been identified as a major cause for CSD[1,2]. Cats may infect humans either directly through scratches, bites or licks, or indirectly via an arthropod vector. The cutaneous lesion is typically a round, red-brown, nontender papule that develops one week after contact with a cat, most often a newly acquired kitten. This minor injury often goes unrecognized. In the next 1–2 weeks regional lymph nodes that drain the area gradually enlarge to several centimetres over 2–3 weeks and may stay for another 3 weeks[3]. Some cases are more severe and last several months; many others go undiagnosed. In about 10% of patients the nodes become infected and suppurate. The lymph nodes most often involved are in the axilla, then the neck and jaw region and the groin[3].

Epitrochlear swellings, as seen in our patient, are infrequent. Early in the course of infection lymph nodes show hyperplasia with vascular proliferation. As the infection progresses granulomas appear, and later multiple microabscesses form, fusing to larger abscesses in those nodes that undergo suppuration. Gram-negative, argyrophilic, non-acid-fast, pleomorphic bacilli may be seen in lymph node preparations or may be noted on biopsy of the primary papules. Usually there is a local inflammation of the involved lymph node and not an encapsulated abscess as in our patient. The course of CSD, however, is usually benign and often self-resolving. Malaise, headache, and fever occur in fewer than half the patients[3].

In the past, histopathological examination of the involved lymph node specimens was thought to be the most reliable diagnostic test for CSD. Typical findings include stellate caseating granulomas, microabscesses, and lymphoid follicular hyperplasia. Histological examination with Warthin–Starry silver stain or Brown–Hopps tissue Gram stain, with electron microscopy or immunofluorescence, reveals argyrophilic aggregates of bacteria[4]. However, the tissue stains do not distinguish between species of *Bartonella*. Although *B. henselae* organisms can technically be cultured from specimens of tissue or blood, incubation for up to 6 weeks is required[1–3].

Serological testing for the presence of antibodies to *B. henselae* is the most widely used test for confirmation of diagnosis. An indirect fluorescent antibody technique is at present the most effective test, with up to 93% sensitivity and 98% specificity in selected populations[5]; the most sensitive test is a polymerase chain reaction to detect the presence of *B. henselae*-specific DNA sequences[6], but it is not widely available and the quality may vary between laboratories.

Cat-scratch disease is most often seen in children or in association with human immunodeficiency virus infection. It is usually unresponsive to antibiotics, even when the organism is sensitive *in vitro*. Whereas healthy individuals tend to recover spontaneously, immunocompromised patients are at risk of progressive and fatal infections. The most effective antibiotics seem to be erythromycin, rifampicin, doxycycline and gentamicin[2–4].

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Endocrine evaluation for muscle pain

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In occasional patients with low adrenal cortical or thyroid function, the main symptoms are musculoskeletal[1,2]. Indeed, when the features are those of a myopathy, the patient is likely to be seen first by a neurologist rather than an endocrinologist, as happened in the following two cases.

CASE HISTORIES

Case 1

A boy of 13 was admitted with a 2-year history of muscle cramps. Serum creatine kinase was 270 U/L (reference range 25–200). Electromyographic findings were normal but muscle biopsy (vastus lateralis) showed atrophic fibres and intermediate type 2c fibres. Because of recent episodes suggestive of postural hypotension the endocrine evaluation focused on possible adrenal cortical hypofunction. Serum cortisol was very low at 95 nmol/L (reference range 160–580), plasma aldosterone was slightly depressed at 48 pmol/L (56–360) and serum corticotropin was very high at 35.6 pmol/L (2.2–13.3). Adrenal cortex antibody was present in a titre of 1:16. Addison's disease was diagnosed and he was started on cortisone acetate 37.5 mg/day and fludrocortisone 50–100 μg/day. After several months' treatment the muscle cramps were no better and the patient was re-evaluated. He proved to have primary hypothyroidism in addition to the adrenal failure—free thyroxine (FT4) 12 pmol/L (reference range 11.7–24.6), basal thyrotropin (TSH) 6 mU/L (0.23–4.0), TRH-stimulated TSH 40 mU/L (5–25). Thyroid microsomal antibodies were present at 1:400; thyroglobulin antibody was not detected. On sonography the thyroid gland was atrophic. When L-thyroxine 100 μg/day was added to the steroid replacement therapy, the cramps disappeared.

Case 2

A bricklayer aged 45 was admitted with a 5-year history of asthenia, myalgias and cramps in the legs; lately his symptoms had included poor memory and apathy, and vitiligo had appeared. At the age of 35 he had been investigated for similar symptoms and Hashimoto's thyroiditis had been diagnosed, on the basis of a high TSH and positive thyroglobulin and thyroid microsomal antibodies. Taking L-thyroxine 100 μg/day he had then been well for 5 years. Thereafter the symptoms had returned, and in subsequent years evaluations by internists, orthopaedic surgeons, neurologists and psychologists had been unrewarding. Seemingly the possibility of adrenal failure had been considered, because serum cortisol was measured on one occasion, but the result had been normal (233 mmol/L, reference range 193–666).

When referred to us the patient had no abnormal physical signs; blood pressure was 95/60 mmHg. Findings on electromyography and muscle biopsy (vastus lateralis) were normal. Serum creatine kinase was slightly raised at 209 U/L and serum potassium was 5.0–5.3. Morning serum cortisol fluctuated from low-normal 152 mmol/L to normal and responded normally to 250 μg intravenous tetracosactrin with a peak of 588 mmol/L. Adrenal cortex antibodies were present at a titre of 1:64. As well as thyroid microsomal antibody, thyroglobulin and thyroid peroxidase antibodies were positive. We diagnosed polyglandular autoimmune syndrome type II (PGA–II) and added oral cortisone acetate (37.5 mg/day) to the thyroxine therapy. However, the patient did not improve. Therefore we asked him to stop all treatment for 8 weeks and re-evaluated him. We confirmed the primary hypothyroidism (basal TSH 4.3 mU/L; TRH-stimulated TSH 35 mU/L) and borderline hypocortisolism (baseline 150–179 nmol/L; peak after tetracosactrin 560). We also found frank hyperreninaemic

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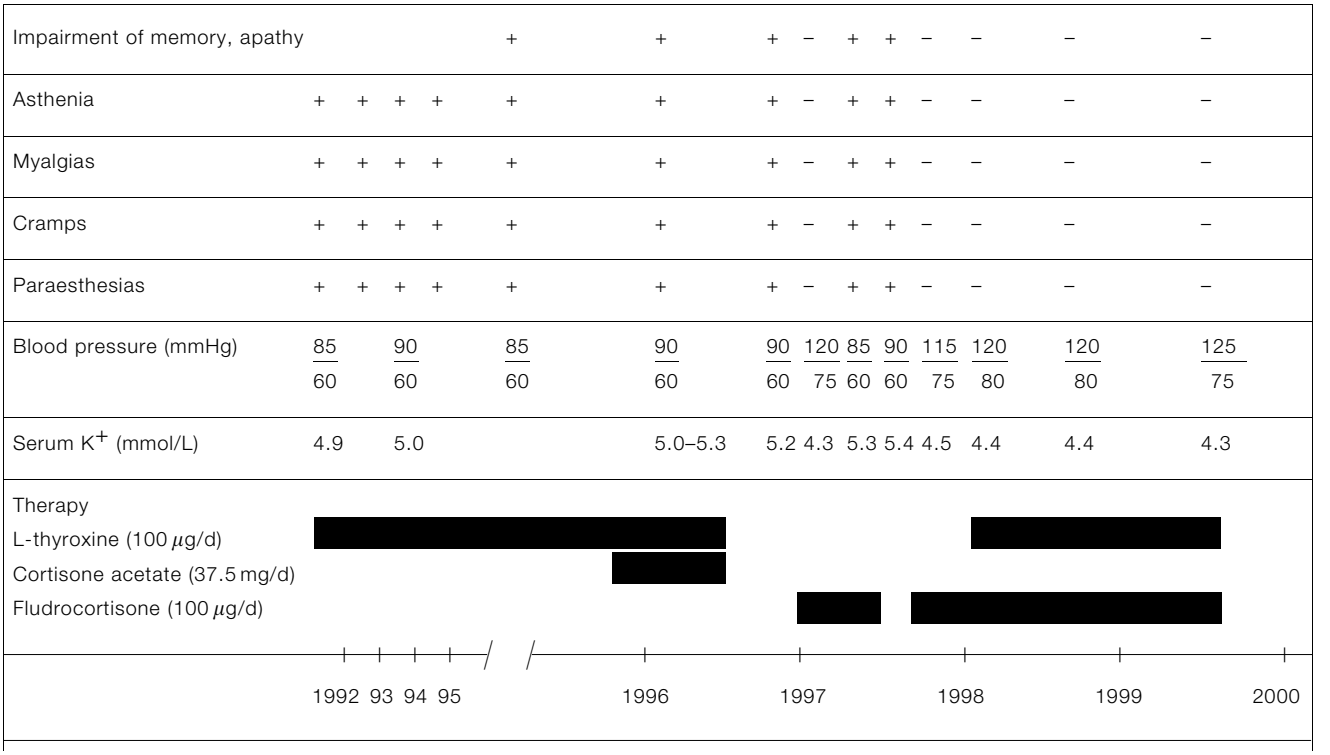

Figure 1 **Clinical and biochemical course, and response to therapy in case 2.** Once he had experienced the benefits of fludrocortisone, he refused to continue with cortisone in addition

hypoaldosteronism (renin 0.57–0.65 nmol/L [reference range 0.16–0.44]; aldosterone 20–35 pmol/L [56–360]. A cause-and-effect relation between hypoaldosteronism and the symptoms is indicated by the disappearance of symptoms with fludrocortisone therapy (Figure 1).

COMMENT

In primary adrenal insufficiency, weakness and fatigue are universal while muscle or joint pains occur in 6–13% of patients[1]. Investigations of muscle usually show nothing abnormal. In hypothyroidism the features can be similar[2], but serum creatine kinase is often raised and electromyographic and muscle biopsy findings are more likely to be abnormal. The two patients here had Schmidt's syndrome—coexistent adrenal and thyroid failure—in the context of PGA–II. PGA–II can include specific neuromuscular disorders such as myasthenia gravis, which we excluded in these cases.

Our patients show the variable spectrum of symptoms in the non-classic presentation of PGA–II. In patient 1, the sole symptom was muscle cramps; in patient 2, cramps were associated with myalgias, weakness and latterly neuropsychological disturbances. In patient 2, myalgias occurred twice—the first time when he was hypothyroid, the second time when he was hypoaldosteronaemic and euthyroid. This fact leads us to another consideration. We show that, even though the same two endocrine glands were functionally impaired, muscle symptoms seem to have had different origins. Patient 1, despite the long duration of symptoms, had normal muscle histology, whereas patient 2 had the histological appearances previously reported in hypothyroid patients, who present with myopathy[4].

Adrenal insufficiency is the most frequent cause of hyperreninaemic hypoaldosteronism[5], of which hyperkalaemia is a hallmark. The relapses of neuromuscular symptoms in patient 2 coincided with moderate increases in serum potassium. While we do not know the reason for his hypersensitivity to small changes in kalaemia, it does seem that hypoaldosteronism tends to prevail over hypocortisolism. In a few cases of Addison's disease the autoimmune damage centres on the aldosterone-producing layer (zona glomerulosa) rather than all three layers of the adrenal cortex[6].

In conclusion, we underscore the necessity of a thorough endocrine investigation for otherwise unexplained neuromuscular disturbances. Early recognition will be helped by awareness that the muscle symptoms can arise with only marginally raised serum potassium, that such complaints can for a long time be the only feature of adrenal insufficiency and that aldosterone deficiency may outweigh the importance of cortisol deficiency.

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A knife in the back: anaesthetic management

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In a patient urgently requiring anaesthesia for surgical investigation of thoracic injury, rapid-sequence induction with one-lung ventilation may be the best option.

CASE HISTORY

A man aged 18 was brought to the accident and emergency department after being stabbed in the back. The knife had been left *in situ*. On arrival his pulse rate was 130/min blood pressure 114/45 mmHg and respiration rate 16/min. Air entry to the lungs was equal and oxygen saturation was 99% with the patient on oxygen 15 L/min. A kitchen-knife handle was protruding from the right posterior thoracic wall in the paravertebral region at the level of T6. There was said to have been little blood loss at the scene of the injury. Primary and secondary surveys, performed with the patient sitting, identified no further injuries. During his initial assessment he was given 500 mL crystalloid, 1500 mg cefuroxime, 3 mg morphine and 10 mg metaclopramide intravenously. The heart rate fell to 95/min and all other indices remained stable. Chest X-rays were taken (Figure 1). It was decided that the patient should undergo emergency thoracoscopy to exclude vascular injury, ascertain the position of the knife and remove it under direct vision. The patient had normal dentition, a non-protruding mandible, full range of neck and jaw movements and a Mallampati[1] score of 1. He had eaten immediately before the assault. After transfer to the operating theatre the right radial artery was cannulated and invasive blood pressure monitoring was started. He was preoxygenated in the left lateral position for 5 min (Figure 2). With the patient supported by theatre personnel in the left lateral position, a modified rapid sequence induction was performed with 20 mg etomidate and 70 mg rocuronium. Direct laryngoscopy revealed a Cormack and Lehane[2] grade 1 view, and a size 37 mm left double-lumen endobronchial tube (Bronchocath) was inserted. Two-handed cricoid pressure, as originally described by Sellick[3], was maintained until tracheal cuff inflation and satisfactory tube placement had been confirmed by auscultation. Correct endobronchial placement was further verified by the use of a flexible fibreoptic bronchoscope. Anaesthesia was maintained with isoflurane, air and oxygen, plus 2 μg/kg fentanyl. Ventilation of the left lung continued whilst the right lung was deflated. On video-assisted thoracoscopy the knife was found not to have penetrated any major vascular structures or the oesophagus. It was removed without incident. Afterwards a chest drain was inserted and the patient was extubated. He was discharged home the following evening.

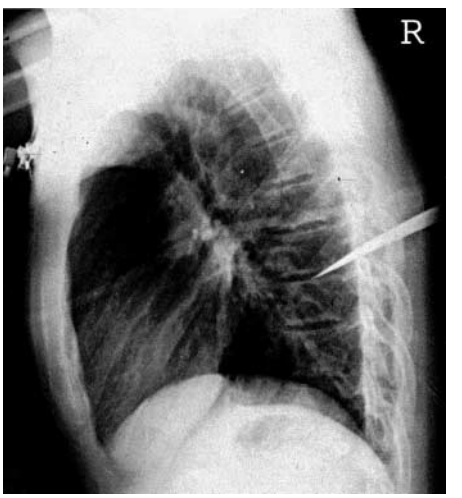

Figure 1 **Lateral chest radiograph**

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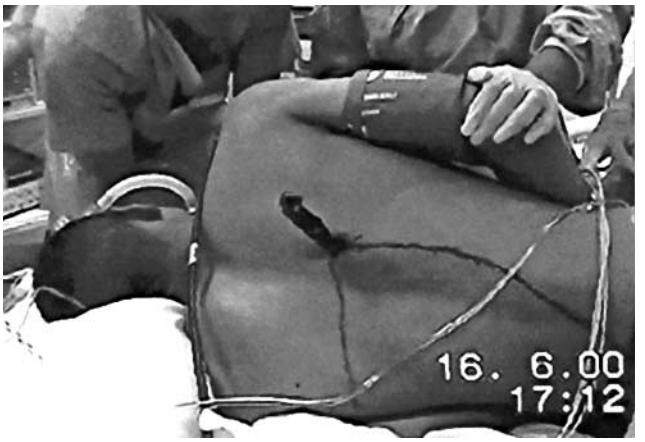

Figure 2 **Position during preoxygenation**

COMMENT

For a patient suspected of having a full stomach, rapid-sequence induction of anaesthesia, with preoxygenation, application of cricoid pressure and avoidance of manual inflation of the lungs, is the recommended way to limit the risk of aspiration of stomach contents[4]. This patient presented a particular challenge because, as well as having a full stomach, he was unable to lie supine, had a possibility of a mediastinal or thoracic injury, and one-lung ventilation was preferable for the planned procedure. Because of the nature of the injury and the lack of a pneumothorax on chest radiography a bronchopleural fistula was thought unlikely.

Rapid-sequence induction is possible in the lateral position, although intubation may take longer, mainly because of operator unfamiliarity[5]. Placement of a double-lumen tube has been described with a bougie[6]. One-lung ventilation can be provided with a single-lumen endotracheal tube, either by the use of an endobronchial blocker or by endobronchial placement of a single-lumen tube, although intubation of the left main bronchus can be difficult. The use of an endobronchial blocker has been described during rapid-sequence induction[7].

One-lung ventilation is not an absolute requirement for thoracic surgery but improves surgical access for video-assisted thoracoscopy. The left lateral position was perceived to aid the positioning of a left-sided double-lumen tube.

Before proceeding, we discussed several other options for this patient. A gaseous induction was considered, to maintain negative-pressure ventilation in case of a bronchopulmonary fistula, but the risk of aspiration was deemed greater. Other suggestions were the use of a Montreal mattress or a Toronto frame to support the patient in the supine position with the knife remaining in his back. We used rocuronium instead of suxamethonium, to allow more time for manipulation of the airway.

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