Final diagnosis

Adenocarcinoma of the cervix with lymphangitis carcinomatosis.

Keywords: adenocarcinoma, cervical carcinoma, lymphangitis carcinomatosis

A flaccid arm

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A 58-year-old man presented in April 1996 with painless weakness of his left arm. Steele-Richardson-Olszewski syndrome had been diagnosed three years previously, causing increasing difficulty with mobility. He also had non-insulin-dependent diabetes, which was well controlled on metformin, with no diabetic complications. In February 1996 he fell, and sustained a left clavicular fracture, which was managed conservatively. Eight weeks later, he developed weakness of the left arm which gradually worsened over two weeks. He was otherwise well, with no weight loss, respiratory symptoms, or recent immunisations, and was a non-smoker.

On examination, there was a swelling over the mid third of the left clavicle. The left arm was flaccid and areflexic with grade 3/5 power in all muscle groups. Vibration and joint position sensation were impaired. There was no discolouration or swelling, and temperature and pulses were normal. The only other findings were of increased tone and bradykinesia in the right arm and lower limbs, which together with restriction in gaze and reduction in facial movements were consistent with a diagnosis of Steele-Richardson-Olszewski syndrome. The plantar responses were flexor.

Questions

1 What is the most probable site of the lesion?
2 What is the most probable pathology?
3 How would you investigate this patient?
Answers

QUESTION 1
Flaccid weakness implies a lower motor neuron lesion. Involvement of all muscle groups in the arm, from deltoid to the small muscles of the hand, suggests damage to motor fibres arising from anterior horn cells from C5 to T1. Impairment of vibration and joint position sense means the lesion is not restricted to motor components. Therefore, the most likely site of the pathology is the left brachial plexus.

Other possibilities include an intraspinal lesion, involving anterior horn cells and dorsal columns, or an involvement of multiple nerve roots, but these are unlikely in the absence of other focal neurological signs.

QUESTION 2
With the history of clavicular fracture eight weeks before the onset of weakness, and the finding of a swelling over the mid third of the clavicle, the most probable diagnosis is compression of the brachial plexus by callus or fibrous tissue at the site of the fracture.

Box 1 lists other causes of brachial plexus damage. A traction injury would be expected to cause symptoms immediately, and is therefore unlikely in this case. Thoracic outlet syndromes are due to angulation of the lower roots of the brachial plexus over an abnormal rib, or a fibrous band between the seventh cervical vertebra and the first rib. This predominantly involves C8 and T1. The involvement of all roots from C5 to T1 makes this diagnosis improbable. Neuralgic amyotrophy may occur after trauma or immunisation, and is characterised by pain and weakness around the shoulder girdle. Although involvement of distal arm muscles, and sensory loss, can occur, the absence of pain makes this a less likely diagnosis. There was no clinical evidence to suggest neoplastic infiltration or history of irradiation in this case.

QUESTION 3
Electrophysiological investigations helped localise the lesion to the brachial plexus. Electromyography of left arm muscles revealed florid spontaneous activity, indicating widespread denervation. Somatosensory evoked potentials were abnormal in the sensory cortex, cervical region and at Erb’s point suggesting a post-ganglionic lesion. As the sensory action potential amplitude in the distal segment of the median nerve was normal, the post-ganglionic lesion was likely to be in the brachial plexus. A chest X-ray showed callus around a displaced fracture of the mid clavicle (figure 1). An MRI of the upper thorax revealed exuberant callus around the site of the left clavicular fracture with involvement of the brachial plexus. The subclavian vessels were normal (figure 2).

Discussion
Damage to the brachial plexus following fracture of the clavicle is rare. In a series of 193 such fractures, only two early transient lesions of the brachial plexus were recorded. Brachial plexus damage following fracture of the clavicle may occur early, if the plexus is compressed by displaced fragments of bone.

Mechanisms of brachial plexus injury
- traction injuries affecting the shoulder girdle
- compression (by eg, cervical rib, displaced bone fragments, callus)
- neuralgic amyotrophy
- neoplastic infiltration (by eg, apical lung carcinoma, lymphoma, neurofibroma)
- post-irradiation

Box 1

Learning points
- a brachial plexus lesion may develop several weeks after a fracture of the clavicle.
- the lesion may result from callus or fibrous tissue at the site of fracture.
- early recognition and surgical intervention can result in improvement or resolution of neurological symptoms.

Box 2
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. In a survey of world literature between 1960 and 1987, Bahnni 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. 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. 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. 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

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A 76-year-old woman receiving neither diuretic, psychotropic, nor anti-diabetic 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?