Neuralgic amyotrophy in A&E

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Abstract

Two patients with neuralgic amyotrophy (Parsonage-Turner syndrome) are described. Problems arising from the shoulder girdle commonly present to accident and emergency (A&E) departments. Neuralgic amyotrophy is an infrequent neurological disorder which predominantly affects the shoulder girdle. Characterised by severe pain followed by muscle weakness, atrophy, and variable sensory deficits, the diagnosis is based on history and physical findings and is confirmed by electromyography. The prognosis is excel-
lent and treatment is supportive using analgesia and physiotherapy.

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Keywords: neuralgic amyotrophy; accident and emergency; differential diagnosis; muscle wasting

Case 1
A 16 year old male presented with a short history of pain in the right shoulder. His work involved heavy lifting but there was no history of trauma to the shoulder. Initial examination revealed diffuse joint tenderness and all active movements, especially abduction, were severely restricted by pain. Painful arc syndrome was diagnosed and the patient referred for physiotherapy.

Two weeks later the patient returned with inability to lift his right arm. Repeat examination revealed wasting of the supraspinatus, infraspinatus, and deltoid. Shoulder abduction was graded 1/5. Sensation was normal and all reflexes were preserved. Subsequent EMG revealed severe denervation of supraspinatus on the affected side compatible with the diagnosis of neuralgic amyotrophy.

Case 2
A 20 year old male presented complaining of two months of neck pain. He had been involved in a road traffic accident seven months before presentation but had been asymptomatic for five months. There was no other history of trauma. Initial examination was normal, the patient was reassured and given analgesia.

The patient returned four months later complaining of pain in the left shoulder and weakness of the left arm. Examination now revealed tenderness over trapezius, wasting of deltoid, and reduction in power of all shoulder movements, especially abduction. He had developed "trick" scapulothoracic movements to compensate for this weakness. He had reduced sensation over the shoulder with absent biceps and supinator jerks on the affected side.

A cervical CT myelogram excluded a compressive lesion at C5/6. Subsequent EMG showed a reduction in functioning motor units, most pronounced in the left deltoid, consistent with a diagnosis of neuralgic amyotrophy.

Discussion
Neuralgic amyotrophy is a rare but well described condition of unknown aetiology. Described by Parsonage and Turner in 1948, the condition is characterised by severe pain, classically located around the shoulder girdle, occasionally radiating into the neck or arm. Patients often complain of associated tiredness and irritability as they have difficulty sleeping at night. The pain may last from a few hours to several months before the onset of paralysis and muscle wasting. Classically as paralysis develops, pain decreases. The muscle wasting is often dramatic. There may be sensory loss but this is not a constant feature and although the pathology involves lower motor neurones, fasciculation is rarely seen.

Neuralgic amyotrophy affects men more often than women. Most patients present between the ages of 30 and 60 years, though it has been reported from 3 months to 75 years. The upper branches of the brachial plexus are affected more often. Muscular involvement is most often seen in the deltoid, followed by supraspinatus, infraspinatus, biceps, triceps, and also serratus anterior which results in winging of the scapula. Involvement of the wrist flexors and extensors is also seen. Rarely cases of neuralgic amyotrophy of the lumbar-sacral plexus have been reported. Involvement of nerves other than of the brachial and lumbosacral plexi is well described. The accessory nerve is most commonly involved but cases are reported of multiple cranial nerve involvement, and Mulvey et al described 16 cases of diaphragmatic involvement presenting with dyspnoea. Although in the majority of cases no precipitating factors are apparent, in many there is an antecedent illness. Several series have reported a preponderance of cases in postoperative patients. Other cases have been reported after infection (for example, Weil's disease), vaccination (for example, hepatitis B), radiotherapy for Hodgkins disease, and occasionally in small epidemics. These antecedents raise the possibility that neuralgic amyotrophy is an immunologically related disorder. This hypothesis was investigated by Sierie et al who showed that lymphocytes were sensitised to brachial plexus nerve in six patients with neuralgic amyotrophy, suggesting that this is an immune mediated disease.

The differential diagnoses of neuralgic amyotrophy includes adhesive capsulitis and rotator cuff tendonitis, neither of which is associated with muscle wasting or sensory dysfunction. Acute poliomyelitis may produce a similar clinical picture but is only of clinical relevance in patients coming from the developing world who have not been immunised. Cervical disc prolapse may produce pain in the shoulder as well as both motor and sensory signs, and a CT myelogram or magnetic resonance imaging may be necessary to exclude this condition. The most useful diagnostic investigation for neuralgic amyotrophy is EMG, which will show evidence of denervation and a reduction in functioning motor units in the majority of cases.

The prognosis of neuralgic amyotrophy is excellent, with most studies showing full recovery of function within two years in over 80% of patients treated purely symptomatically. Strong analgesia is recommended during the early painful stages of the disease. The benefit of oral, parenteral, or intra-articular steroids remains uncertain. Physiotherapy helps to maintain a full range of joint movement and assists return of muscle function.

Conclusion
Neuralgic amyotrophy is a rare cause of shoulder girdle symptoms. The diagnosis should be considered in patients presenting with severe shoulder girdle pain in the absence of trauma. It must be remembered that pain precedes
motor signs. For this reason we believe patients presenting with severe, unexplained shoulder girdle pain should be offered follow up, preferably by their general practitioner. If motor signs develop, the diagnosis can be confirmed with EMG and the patient reassured that the prognosis is excellent.

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CASE REPORTS

Potassium permanganate poisoning—a rare cause of fatal self poisoning

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Abstract

Attempted suicide by self poisoning is common because of the ready availability of drugs, whether prescribed or bought over the counter. In some cases, the ingestion of seemingly innocuous household products or chemicals can result in death.

Potassium permanganate is an example. Poisoning with potassium permanganate can be fatal when a significant amount is ingested, as shown by a patient who suffered both the corrosive and systemic toxic effects of this chemical.

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Keywords: potassium permanganate poisoning; liver damage; kidney damage; laryngeal oedema

Although the chemical is readily available, potassium permanganate poisoning is not common.

Case report

Our patient was a 24 year Chinese female with no previous psychiatric history. She ingested an unknown quantity of potassium permanganate crystals after a domestic quarrel. She was immediately stopped by her relative and then drank a large amount of water. No other drug was ingested. She was first seen at a district hospital where gastric lavage was performed before her transfer to our department.

On arrival, she was alert and oriented. Her airway was patent and she had no stridor. There were brownish black stains on her hands and lips. There were similar staining of the entire oropharynx. The tongue and lips were swollen and bled on contact. Direct laryngoscopy under local anaesthesia showed a stained and oedematous pharynx, with gross swelling of the laryngeal structures. She was anaesthetised, intubated, and ventilated and was admitted to the intensive care unit.

Initial chest x ray was normal. Oesophagoscopy soon after admission showed staining of the upper oesophagus. The rest of the oesophagus, stomach, and duodenum were normal.