presence of epithelial trauma and also helped to localise any remaining pieces of solidified glue, which were removed by fine forceps at the slit lamp. Corneal abrasions were treated with topical antibiotic and mydriatic drops and these patients were followed up until their injuries had healed.

The eye lashes were stuck together in five patients, four adults and one child. The lashes were carefully cut in three of the adults, as the lids were almost completely closed. The lids were then gently prised apart and a full ocular examination was completed. The two patients who were treated conservatively had only a few lashes bonded together which did not obscure vision or prevent a thorough ophthalmic examination.

All patients were followed until their symptoms had resolved. No patients suffered any long term ocular morbidity.

Discussion

There are various published reports of ocular,[5–8] aural,[4] and oral[9] injuries associated with superglue. The cause of ocular injuries has been attributed to patient carelessness, poor eyesight, and childhood curiosity. A new category of deliberate ocular superglue injury during assault has been added by this study.

The list of ocular injuries that may be caused by superglue includes: dermatitis, loss of eye lashes, ankyloblepharon (an abnormal fusion of the upper and lower eyelid margins), severe eye pain, conjunctival epithelial abrasion, corneal abrasion, and punctate epithelial keratopathy. On contact with the cornea and conjunctiva, superglue causes a chemical keratitis. However, cyanoacrylate superglue will only bond with dry surfaces and so tends to collect in the lower conjunctival fornix as an irregular cast, causing a traumatic keratopathy. For the same reason, superglue causes ankyloblepharon mainly by bonding the eyelashes, as the dry anterior margin of the eyelid provides only a small surface area for bonding. A small degree of lid closure may be observed, as the lids usually separate spontaneously within a week. Superglue ankyloblepharon can be treated by trimming the eye lashes and gently separating the lids. This technique was used in three adult patients in this study, who had a significant degree of eyelid closure. In children under the age of eight years, obstruction of the visual axis by superglue ankyloblepharon may lead to amblyopia if left untreated. In this age group, treatment may necessitate a general anaesthetic.

In this study 13 of the 14 patients suffered their injury as the result of an accident. Half of these patients suffered their injury while opening the glue container. A printed message on the container warning of the possibility of ocular injury may reduce the incidence of such accidents. Four patients in this study were children less than six years old, which illustrates the need to store superglue containers away from young children. However, in the event of an infant finding a superglue bottle, a childproof cap would prevent injury. Many medicine bottles are now made childproof by employing tops which only open when pushed down and twisted or when arrows on the top and on the bottle are correctly aligned. Such childproofing would also reduce the likelihood of adults inadvertently pouring superglue into their eyes rather than their prescribed eye drops, as they would have to scrutinise the bottle much more carefully in order to remove the top and would thus realise they had picked up the wrong bottle.

1 Weiss JL, Williams P, Lindstrom RL. The use of tissue adhesive in corneal perforations. Ophthalmology 1983;90:61.
2 Carton SA, Kessler LA, Seidenberg B, Hurwitt ES. A plastic adhesive method of small blood vessel surgery. World Neurol 1960;1:356.
3 Refojo MF, Dohlman CH, Koliopoulos J. Adhesives in ophthalmology: a review. Surv Ophthalmol 1971;15:217–36.
4 Wight RG, Bull PD. Superglue ear. J Laryngol Otol 1987;101:706–7.
5 Kimbrough RL, Okereke PC, Stewart RH. Conservative management of cyanoacrylate ankyloblepharon: a case report. Ophthal Surg 1986;17:176–7.
6 Silverman CM. Corneal abrasion from accidental instillation of cyanoacrylate into the eye. Arch Ophthalmol 1988; 106:1029–30.
7 Lyons C, Stevens J, Bloom J. Superglue inadvertently used as eyedrops. BMJ 1990;300:328.
8 Derespinis PA. Cyanoacrylate nail glue mistaken for eye drops. JAMA 1990;263:2301.
9 Cousin GCS. Accidental application of cyanoacrylate to the mouth. Br Dent J 1990;169:293.

Neuralgic amyotrophy presenting to an accident and emergency department

M J Darby, A R Wass, D I Fodden

Accident and Emergency Department, Pinderfields General Hospital, Aberford Road, Wakefield, Yorkshire WF1 4DG, United Kingdom
M J Darby
A R Wass
D I Fodden

Correspondence to:
A R Wass FRCS, Senior Registrar in Accident and Emergency Medicine.

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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 neuromuscular 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,[1] 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.[2] Most patients present between the ages of 30 and 60 years, though it has been reported from 3 months to 75 years.[3] The upper branches of the brachial plexus are affected more often.[2] 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.[2] Rarely cases of neuralgic amyotrophy of the lumbosacral plexus have been reported.[4] 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,[5] and Mulvey *et al* described 16 cases of diaphragmatic involvement presenting with dyspnoea.[6] 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.[1 7] Other cases have been reported after infection (for example, Weil's disease),[8] vaccination (for example, hepatitis B),[9] radiotherapy for Hodgkins's disease,[10] and occasionally in small epidemics.[11 12] These antecedents raise the possibility that neuralgic amyotrophy is an immunologically related disorder. This hypothesis was investigated by Sierre *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.[13]

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.[14]

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.[1 3 14] Strong analgesia is recommended during the early painful stages of the disease. The benefit of oral, parenteral, or intra-articular steroids remains uncertain.[2] 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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1 Parsonage NJ, Turner JWA. Neuralgic amyotrophy—the shoulder girdle syndrome. Lancet 1948;i:973–8.
2 Aymond JK, Goldner JL, Hardaker WT. Neuralgic amyotrophy. Orthop Rev 1989;12:1275–9.
3 Tonali P, Uncini A, Di Pasqua DG. So called neuralgic amyotrophy: clinical features and long-term follow-up. Ital J Neurol Sci 1983;4:431–7.
4 Rifisch A, van Laack W. Neuralgic amyotrophy of the lumbar area. Arch Orthop Trauma Surg 1989;108:329–32.
5 Pierre PA, Laterre CE, van den Bergh PY. Neuralgic amyotrophy with involvement of cranial nerves IX, X, XI and XII. Muscle Nerve 1990;13:704–7.
6 Mulvey DA, Aquilina RJ, Elliot MW, Moxham J, Green M. Diaphragmatic dysfunction in neuralgic amyotrophy: an electrophysiological evaluation of 16 patients presenting with dyspnoea. Am Rev Respir Dis 1993;147:66–71.
7 Ferrini L, Della Torre P, Preticoni G, Cantisani TA. Neuralgic amyotrophy of the shoulder girdle; the Parsonage-Turner syndrome. Ital J Orthop Traumatol 1986;12:223–31.
8 Cumming WJK, Thrush DC, Kenwood DH. Bilateral neuralgic amyotrophy complicating Weil's disease. Postgrad Med J 1978;54:680–1.
9 Reutens DC, Dunne JW, Leather H. Neuralgic amyotrophy following recombinant DNA hepatitis B vaccination. Muscle Nerve 1990;13;461.
10 Malow MA, Dawson DM. Neuralgic amyotrophy in association with radiotherapy for Hodgkin's disease. Neurology 1991;41:440–1.
11 Wyburn-Mason R. Brachial neuritis occurring in epidemic form. Lancet 1941;ii:662–3.
12 Bardos V, Somodska V. Epidemoilogic study of a brachial plexus neuritis outbreak in north east Czechoslovakia. World Neurol 1961;2:973–9.
13 Sierra A, Prat J, Bas J, Romeu A, Montero J, Matos JAM, et al. Blood lymphocytes are sensitised to brachial plexus nerves in patients with neuralgic amyotrophy. Acta Neurol Scand 1991;82:183–6.
14 Devathasan G, Tong HI. Neuralgic amyotrophy: criteria for diagnosis and a clinical with electromyographical study of 21 cases. Aust NZ J Med 1980;10:188–91.

CASE REPORTS

Potassium permanganate poisoning—a rare cause of fatal self poisoning

K L Ong, T H Tan, W L Cheung

Department of Accident and Emergency, Prince of Wales Hospital, Shatin, Hong Kong
K L Ong
W L Cheung

Department of Diagnostic Radiology, Caritas Medical Centre, Kowloon, Hong Kong
T H Tan

Correspondence to:
Dr Ong Kim Lian, Department of Accident and Emergency, Prince of Wales Hospital, Shatin, New Territories, Hong Kong.

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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

Potassium permanganate is a readily available over the counter agent that is widely used as an antiseptic for baths in patients with eczema—particularly the exudative types—due to its astringent properties. Historically its uses have been interesting: it has been used as an abortifacient,[1] as a urethral irrigation fluid for treatment of gonorrhea, as a fluid for stomach washout in cases of alkaloid poisoning, and in the solid form as a local remedy for snake bite.[2] 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,