

Causes of neck pain

Neck pain most commonly arises from the cervical spine:

Collagen disease
 Congenital abnormality
 Degenerative changes
 Osteoporosis
 Post-surgical pain
 Pus/sepsis (eg, osteomyelitis, discitis)
 Torticollis
 Trauma (eg, fracture, dislocation, whiplash)
 Tuberculosis
 Tumour

Less frequently, pain results from lesions of the brain, spinal cord, or meninges. It is the meninges which are pain sensitive. The pain may mimic musculoskeletal neck pain:

Haematomyelia
 Meningitis/sepsis
 Neuralgic pain (eg, post-herpetic neuralgia)
 Subarachnoid haemorrhage
 Tonsillar herniation (secondary to any cause of raised intracranial pressure)
 Trauma
 Tumour (benign, eg, neurofibroma, or malignant, eg, deposit from medulloblastoma)

Other structures within the neck may give rise to pain (and any advanced tumour may give rise to pain):

Carotid artery
 Aneurysm (eg, in Marfan disease, syphilis)
 False aneurysm (secondary to trauma)
 Lymphatics
 Sepsis
 Thyroid
 Acute bacterial thyroiditis
 Viral thyroiditis (eg, mumps)
 Skin
 Cellulitis
 Trauma
 Larynx/trachea
 Trauma (eg, transection)
 Oesophagus (there is associated dysphagia)
 Foreign body
 Oesophagitis
 Stricture
 Trauma
 Tumour

Importantly, pain may be referred to the neck from the heart, diaphragm, or teeth:

Heart
 Ischaemia
 Myocardial infarction

Rarely neck pain may be psychiatric:

Atypical facial pain
 Globus hystericus

neck pain in our patient, which was worse on waking, to this cause.

Other serious causes for neck pain, which may mimic musculoskeletal neck pain, include subarachnoid haemorrhage, which is characterised by sudden, severe pain. This arises either because the haemorrhage is spinal in origin or because blood from a cerebral bleed tracks into the spinal subarachnoid space. Blood may even gravitate into the lumbar cule-sac, producing back pain and sciatica at presentation.³ It is therefore important to consider the various causes of neck pain when assessing patients so that serious, uncommon emergencies are not missed (table). The history may be acute, as in subarachnoid haemorrhage or myocardial infarction. How-

ever, in the present case the patient had a 10 day history and had already seen her GP. We therefore recommend that history taking includes a brief inquiry about the timing and onset of symptoms, associated headache, and general wellbeing.

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Necrotising fasciitis as a complication of steroid injection

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Abstract

Necrotising fasciitis is described as a complication of steroid injection of a

painful shoulder in a previously well female. This case highlights a very rare life threatening emergency after steroid

injection. Early recognition, resuscitation, and aggressive surgical management are essential to prevent mortality in this condition.

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Keywords: necrotising fasciitis; non-steroidal anti-inflammatory drugs; steroids

Case history

A 41 year old woman, who was previously well, presented to her general practitioner (GP) with a five day history of a painful arc on moving her left shoulder. He prescribed oral diclofenac and gave an intra-articular injection of Depo-Medrone 40 mg with lignocaine. This procedure was carried out observing a strict aseptic technique. Two days later she complained of malaise, diarrhoea, and vomiting. She was advised to take fluids and rest. Over the next 12 hours her condition deteriorated. Her diarrhoea worsened, she became sweaty, confused, and eventually collapsed. She was seen by the GP and referred as an emergency to the accident and emergency (A&E) department.

On arrival to the resuscitation room she was confused, agitated, cyanosed, and required immediate resuscitation. Her temperature was 38°C and her blood pressure was 90/40 mm Hg with a pulse of 150 beats/min. She had a fine petechial rash over her trunk, particularly across her shoulders and groin. Over her left upper arm there was a well demarcated area of purplish discolouration with associated skin peeling and the arm was grossly oedematous (figure) The examination of cardiovascular, respiratory, gastrointestinal and neurological systems was otherwise unremarkable.

Results of initial blood tests showed a normal full blood count including platelets and normal coagulation studies. Abnormal laboratory findings included hyponatraemia (plasma sodium 124 mmol/litre), hypoglycaemia (plasma glucose 2.0 mmol/litre), metabolic acidosis (pH 7.25, bicarbonate 7.3 mmol/litre) with raised urea (15.9 mmol/litre) and creatinine (282 µmol/litre). A diagnosis of septic shock secondary to necrotising fasciitis of the upper arm was made. High flow oxygen and rapid intravenous fluid were given, and penicillin G (4 mega-units) and flucloxacillin (2 g) were injected intravenously. In view of her low blood sugar she was also given 50 ml of 50% dextrose and arrangements were made for transfer to intensive care. Six litres of intravenous fluids were given but her blood pressure failed to improve. She then had a ventricular fibrillation cardiac arrest during transfer to intensive care. Defibrillation, intubation, and cardiopulmonary resuscitation were carried out. The cardiac rhythm converted to electromechanical dissociation and despite full advanced cardiac life support she failed to respond and was pronounced dead 60 minutes later.

Necropsy showed evidence of widespread cellulitis involving the left upper arm and extending onto the anterior chest and breast, and cerebral oedema consistent with septicaemia. No other foci of infection were identified. A microbiological examination of fatty tissue



Necrotising fasciitis of the left upper arm.

from the upper arm taken at necropsy and blood cultures sent on presentation to the A&E department showed group A streptococcal infection.

Discussion

The term necrotising fasciitis was first used by Wilson in 1952¹ when he described a rapidly progressive inflammation and necrosis of subcutaneous tissue and adjacent fascia with secondary necrosis of the overlying skin. In the earlier stages there is usually diffuse swelling of a limb, followed by the appearance of bullae filled with clear fluid, which rapidly take on a maroon or violaceous colour. Unless appropriate intervention is undertaken, there is often a rapid progression to frank cutaneous gangrene, muscle necrosis, and moderate or severe systemic toxicity which carries a high rate of morbidity and mortality.²

The diagnosis is clinical but microscopy of aspirate from subcutaneous tissue may show organisms.³ Aspirate should be taken from the advancing edge of the lesion where the organisms are most plentiful. The most common causative organisms are a mixture of aerobic and anaerobic bacteria, particularly group A β haemolytic streptococci (*Streptococcus pyogenes*), but no specific combination has been shown to be the cause of the disease, and many other organisms have been isolated from patients with necrotising fasciitis. These include aerobes such as staphylococci, *Escherichia coli*, pseudomonas, klebsiella, *Proteus mirabilis*, *Haemophilus parainfluenzae*, and anaerobes such as bacteroides, *Clostridium perfringens*, and peptococcus.⁵

Initiating factors may be minor traumata including abrasions, lacerations, burns, and substance injection, or the condition may occur as a complication of chronic disorders

such as a chronic skin ulcer. There have been reports where no evidence of an initiating injury was found. There is a higher incidence of necrotising fasciitis associated with diabetes mellitus, obesity, advancing age, and atherosclerosis.^{6,7} The primary treatment for necrotising fasciitis is early aggressive surgical debridement after initial resuscitation.

Early intravenous broad spectrum antibiotics should be given until culture results dictate specific antimicrobials. Some clinicians strongly advocate the use of hyperbaric oxygen,⁸ though evidence for the value of this is lacking.⁹ If hyperbaric oxygen is to be employed it should not delay surgical intervention. Mortality varies with the interval from the onset of the disease and its treatment, but has been reported to be as high as 70-80%.^{10,11}

Necrotising fasciitis specifically following steroid injection has been reported previously.^{12,13} Steroids are well known to have an inhibitory effect on leucocyte function, both locally and systemically, and may have been a predisposing factor in our patient. Steroids inhibit monocyte chemotaxis as well as the production of interleukin 1 and other monokines.¹⁴ They reduce phagocytosis and the production of prostaglandins, thromboxanes, and leukotrienes.^{15,16}

Our patient had also recently been started on non-steroidal anti-inflammatory drugs which, through their effect in lymphopenia and depression of lymphocyte function, have been specifically implicated in the pathogenesis of necrotising fasciitis.^{17,18} No other predisposing condition was present.

There is a small possibility that the Depo-Medrone with lignocaine could have been contaminated, although the GP reported he had

not had any problems from any other drugs in the same batch. The Committee on Safety of Medicines and the drug company were contacted. The drug company had not had any other cases reported to them.

Where a diagnosis of necrotising fasciitis is suspected, early aggressive surgical treatment after the initial resuscitation is essential if mortality and morbidity is to be prevented.

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Oesophageal “cross” — a sinister foreign body

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Abstract

A young jail inmate purposely ingested a foreign body formed of sewing needles, specially designed to be arrested in the gut and cause perforation. Immediate surgical removal of such ingested foreign objects is recommended because the chances of distal passage are nil.

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Keywords: oesophagus; self inflicted perforation; foreign body

Case report

A 23 year old prisoner presented with dysphagia of 10 hours' duration. He gave a history of having purposely ingested a metallic foreign body 16 hours previously in an attempt to escape jail temporarily. Chest x ray showed a

rather unusual foreign object which appeared to be situated within the cervical oesophagus (figure).

Following uncomplicated surgical removal, the patient explained how he made the device. Two sewing needles, each measuring approximately 5 cm, are tied crosswise with a rubber band, thus forming a “cross”. The construction of the cross is elastic—its two branches can be pulled together, but when released they return to their original position. With the branches lying parallel, the cross is wrapped in a small strip of paper and then ingested with some water.

Discussion

The foreign body created and ingested by our patient warrants special consideration because it has two potential puncturing points, cranial

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