PNEUMATIC TOURNIQUET PARALYSIS

CASE REPORT

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We describe a 31-year-old man in whom a paresis and sensory defect of the left arm developed after amputation of the index finger. The operation was performed in a bloodless field, using a pneumatic tourniquet. The sensory defect resolved in two months and the paresis in five and a half months. We consider that direct pressure produced by the tourniquet caused the nerve lesion. It is probable that the tourniquet was inflated to a pressure of 500 millimetres of mercury instead of the intended 250 millimetres of mercury because of a faulty gauge. In order to avoid this rare complication, it is advisable to check the tourniquet gauge each time before use.

Paralysis of an upper limb after the use of a tourniquet for establishing a bloodless field was first described 100 years ago by Montes and Putnam (Speigel and Lewin 1945). When rubber tubing or an elastic bandage were used the complication was more common, but even now, when pneumatic tourniquets are in common use, this complication is estimated to occur once in 5000 to 8000 operations performed in a bloodless field (Love 1978; Yates, Hurst and Brown 1981). However, only a few case reports have been published. We now describe one such case.

CASE REPORT

A 31-year-old man presented on January 1, 1982, after a firework he had been holding in his left hand exploded. Because of an extensive laceration the index finger had to be amputated. The operation was performed under general anaesthesia and a bloodless field was used. His arm was emptied of blood using an Eschar ms bandage, after which a pneumatic cuff was applied to the upper arm and the pressure adjusted to 250 millimetres of mercury. The tourniquet time was 1 hour 15 minutes. After the operation, the patient noticed weakness and a sensory defect in his arm.

On January 11, examination revealed that the left wrist and fingers were totally paralysed, and flexion of the elbow was weaker than normal; the remaining muscles functioned normally. There was wasting of the upper arm and forearm amounting to two centimetres.

The biceps and radial reflexes were nearly absent, the triceps reflex normal. The patient stated that his sensory response to pin prick in the forearm and hand was more “superficial” than normal.

By January 18 some active flexion of the wrist had returned but no other active movements were detected. At the end of February, the fingers began to move and sensation to return. The patient returned to work on March 1. On March 23, the wasting and reflexes were found to be unchanged; the muscles of the wrist and fingers were a little weaker than normal, as were the flexors of the elbow. Sensation to pin prick was normal.

Examination on September 10 revealed only 0.5 to 0.8 centimetres of wasting in the upper arm and forearm. The biceps and radial reflexes were still clearly diminished. Extension of the thumb was weak, but other muscles were normal and no sensory defects were found. Electromyography. An electromyogram on February 19 showed a severe lesion in the muscles of the forearm and hand. There was abundant pathological spontaneous activity and a clear loss of motor units. No muscle was, however, totally denervated. In the triceps muscle (supplied by the radial nerve) and in the biceps (supplied by the musculocutaneous nerve) the lesion was mild. The deltoid muscle was intact. The motor nerve conduction velocities of the median and ulnar nerves in the upper arm were somewhat less than in the forearm. The muscle action potentials elicited by stimulation of both nerves had a lower amplitude when stimulated in the proximal upper arm than when stimulated in the distal upper arm. Two months later, the lesion was clearly milder: the loss of motor units and the pathological spontaneous activity had lessened, and muscle action potentials were equal when stimulated in the proximal or distal upper arm. By September 11, 1982, the lesion had healed and the only abnormal finding was altered polyphasic and long-lasting motor unit potentials.

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DISCUSSION

We found reports of 55 cases of tourniquet paralysis in the literature (Eckhoff 1931; Speigel and Lewin 1945; Bruner 1951; Middleton and Varian 1973; Rudge 1974; Trojaborg 1977; Bolton and McFarlane 1978). However, most were caused by rubber tubing or an elastic bandage; only 15 were caused by a pneumatic tourniquet (Bruner 1951; Moldauer 1954; Middleton and Varian 1973; Rudge 1974; Mukherjee 1977; Bolton and McFarlane 1978); and only two cases of tourniquet paralysis have been described in detail (Rudge 1974; Bolton and McFarlane 1978).

The radial nerve is the most vulnerable, but the ulnar and median nerves also are often damaged (Sunderland 1968). Damage to the musculocutaneous nerve, as in our patient, is rare and only one case has been reported previously (Bolton and McFarlane 1978). Sensory defects are usually minor and heal more rapidly than motor defects (Eckhoff 1931), as was found in our patient. Disturbance of sweating, and causalgia, as a sequel to damaged autonomic nerve fibres has been described (Bolton and McFarlane 1978), but were not present in our patient.

Nerve lesions heal spontaneously in three to six months (Sunderland 1968) and are only exceptionally permanent (Speigel and Lewin 1945). In our patient, the sensory loss resolved in two months and the paresis in five and a half months; by eight months from the onset of symptoms abnormal findings were insignificant.

Direct pressure caused by the pneumatic cuff is usually regarded as the cause of the nerve lesion as shown by clinical studies (Eckhoff 1931; Moldauer 1954; Bolton and McFarlane 1978; Love 1978) and experimental findings (Allen 1938; Paleta, Willman and Ship 1960; Fowler, Danta and Gilliatt 1972); only a few authors regard ischaemia as the most important cause of the lesion (Denny-Brown and Brenner 1944).

Speigel and Lewin (1945) surgically explored three cases of tourniquet paralysis. They noted that at the site of compression the diameter of the nerve was reduced to one-half or one-quarter of the normal. In addition, scarring and neuromata were found. Ochoa, Fowler and Gilliatt (1972) noted that in baboons a tourniquet caused both a displacement of the nodes of Ranvier and an invagination of myelin.

In cases of paralysis described in the literature the tourniquet time varies from 28 minutes to 2 hours 40 minutes. The significance of the duration of exsanguination in the causation of the neural lesion remains unclear. Ischaemia of more than two hours is not usually recommended, because in experimental studies even muscle tissue begins to suffer from acidosis and other metabolic changes after that time (Solonen and Hjelt 1968; Wilgis 1971).

The pressure recommended for a pneumatic cuff applied to the upper arm is 250 millimetres of mercury in adults and 200 millimetres in children (Flatt 1972). In our case, this recommendation was followed, but when damage was detected our tourniquets were checked; of the two which may have been used for the operation one had a gauge which showed a pressure only half the actual pressure. (In retrospect, it was impossible to determine which tourniquet had been used.) In four of the 15 cases described in the literature, the gauges had shown, or were suspected to have shown, less than the actual pressure (Bruner 1951; Flatt 1972; Bolton and McFarlane 1978). We believe, therefore, that a faulty gauge had probably been used in our case.

A corresponding lesion in the lower extremity occurs more rarely; it has been described only in connection with the use of an elastic bandage and not with a pneumatic tourniquet (Love 1978). However, after meniscectomy using a pneumatic tourniquet, 18 out of 25 patients had transient electromyographic changes (Weingarden, Louis and Waylonis 1979).

Tourniquet paralysis nearly always resolves spontaneously. However, the patient is unable to work for a considerable length of time, and his recovery from operation is clearly impeded. Moreover, the causalgic syndrome, with its distressing features, may appear during convalescence (Bolton and McFarlane 1978).

In order to avoid tourniquet paralysis, the gauge should be checked each time before use (Flatt 1972). A safety valve which prevents excessive pressure can also be installed in the device (Wheeler and Lipscomb 1964).

REFERENCES


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