

Axillary brachial plexus block complicated by cervical disc protrusion and radial nerve injury

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Sir,

The reported incidence of nerve injury after peripheral nerve block ranges from 0.2% to 19%.^{1,2} There are several possible mechanisms including direct transection, intraneural injection, pressure or vasoconstrictor-induced ischaemic injury, direct drug toxicity and neurovascular compression.³

A 50-year-old healthy female scheduled for fusion of the right index metacarpal joint received an ultrasound-guided axillary brachial plexus block with 35 ml of ropivacaine, 5 mg/ml with adrenaline 2.5 µg/ml. There were no signs/symptoms indicating nerve injury. Good spread of the local anaesthetic (LA) was seen under real-time ultrasound. An upper arm tourniquet inflated at 250 mmHg was used for the uneventful procedure, which lasted 110 min. She developed numbness on the dorsum of the right hand after 48 h. On examination, tingling on the dorsum of the right thumb worsening with elbow extension was present. Sensations were normal proximally but decreased in the right radial forearm with normal motor power and deep tendon reflexes. There was pain on extension of the right arm and passive abduction of the shoulder. Nerve conduction velocity (NCV) demonstrated absent right and normal left radial sensory potential and conduction velocity. Median and ulnar motor as well as sensory NCV were normal, bilaterally. The electromyographic (EMG) examination of right arm muscles revealed no abnormal spontaneous activity, indicating denervation.

A magnetic resonance imaging (MRI) of the cervical spine and the entire brachial plexus carried out at 8 weeks showed multilevel C3–6 disc bulging, ventral cord flattening with osteophyte formation at C4–5 and paracentral cord flattening at C5–6, with marked right foraminal compromise involving the C6 nerve root (see figure 1). No distal brachial plexus lesions were seen. A diagnosis of postganglionic C6 nerve root injury was made and she was referred to neurosurgery for



further management. She is being managed conservatively with analgesics, physical therapy to maintain range of motion and strengthening exercises. Her condition continues to improve but the sensory deficit remains.

Factors initially implicated included LA toxicity, adrenaline use, intraneural injection, tourniquet compression and pre-existing nerve injury. The use of adrenaline-containing LA may contribute to the neurological dysfunction by reducing endoneurial blood flow and producing peripheral nerve ischaemia. However, adrenalin at a concentration of 2.5 µg/ml does not appear to affect nerve blood flow.⁴ A pre-existing neurological condition may cause the 'double crush' phenomenon, producing a clinically significant injury⁵ and yet the EMG showed no signs of denervation. Tourniquet palsies are due to direct extrinsic pressure or axonal hypoxia on the nerves beneath the tourniquet and are related to the cuff pressure and duration of application.⁶

The radial nerve (C5-T1) travels through the posterior cord, dividing into a superficial and a deep branch at the elbow. The sensory fibres of the superficial branch originate from the C6 nerve root, providing sensory supply to the dorsum of the wrist, hand, thumb and lateral 1.5 (or 2) digits. Absent distal sensory NCV in this patient most likely represented a component of post-ganglionic C6 nerve root injury.⁷

This report should serve as a reminder to evaluate patients for other reasons of nerve injury, especially if the neurological compromise does not correlate with the block.

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