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Neuropathy following Axillary Brachial Plexus Block: Is It the Tourniquet?

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AXILLARY brachial plexus block is an accepted and effective means of providing anesthesia for outpatient upper extremity procedures.[1] The incidence of nerve injury after axillary blockade is between 0.2% and 19%, however.[2] The mechanism of injury is unknown, but case series imply an association with identification of the cords of the brachial plexus by needle-seeking paresthesiae.[3,4] We describe a case of postoperative neuropathy affecting the upper limb, which was initially attributed to axillary blockade but, in fact, was caused by ischemic monomelic neuropathy (IMN) secondary to the use of a pneumatic tourniquet.

Case Report

A 63-yr-old woman, American Society of Anesthesiology (ASA) physical class II, presented for open reduction and internal fixation of comminuted and displaced spiral fractures of her left third and fourth metacarpals. Relevant medical history included essential hypertension, which was controlled on hydrochlorothiazide. Bilateral carpal tunnel syndrome had been diagnosed clinically and confirmed by nerve conduction studies 17 yr before presentation. Her symptoms had resolved without surgery. The physical examination was, apart from the fractures, unremarkable. Her blood pressure was 130/60 mmHg, and her heart rate was 74 beats/min in sinus rhythm.

After intravenous administration of 1 mg midazolam and 50 μg fentanyl, an axillary brachial plexus block was performed *via* the elicitation of paresthesia technique using a 25-gauge, 2-in needle. Ulnar nerve paresthesia was obtained on the first pass, and 35 ml 1.25% mepivacaine and 1:200,000 epinephrine mixture was deposited. On the second pass, radial paresthesia was obtained and 15 ml was deposited. An additional 10 ml local anesthetic was deposited anterior to the axillary artery. A total of 60 ml 1.25% mepivacaine with 1:200,000 epinephrine was deposited into the brachial plexus sheath. There was no pain during injection of the local anesthetic. The intercostobrachial and musculocutaneous nerves were blocked *via* subcutaneous injection of 3 ml and 7 ml 2% lidocaine at the axilla and antecubital fossa, respectively.

The patient's left arm was abducted at no more than 90° to the torso and positioned level on an arm table. A tourniquet (Zimmer ATS 2000; Zimmer, Dover, OH) was applied to the midpoint of the left upper arm and inflated to 300 mmHg. The block was satisfactory for the procedure in that neither supplemental nerve block, excessive sedation, nor general anesthesia was required. The patient underwent open reduction and internal fixation of the fractures. After 59 min, the tourniquet was deflated and a loose bulky dressing was applied. With the block still intact, the patient was dismissed to the outpatient area and, subsequently, home.

On the following day, surgical evaluation noted that there was "persistence of total ulnar paresthesias. . .now almost 24 h after the block." These symptoms persisted after the dressing was loosened. Three weeks later, the patient had "ulnar aching and dysesthesias up the forearm, and occasional shocks and discomfort on abduction of the arm." Surgical opinion was that the neurologic deficit was not caused by operative trauma but by injury resulting from the axillary block.

Three weeks after surgery, a neurologic consultation was obtained; examination revealed decreased hypothenar and interossei strength, decreased ulnar digit flexion, and decreased light touch and pinprick sensation on the left. At the time of the consultation, nerve conduction and electromyographic studies were obtained (Tables 1 and 2). Nerves were stimulated at the elbow and wrist, and responses were recorded in the hand. The amplitudes of the left ulnar and radial sensory and median motor responses were reduced. Motor and sensory distal latencies, conduction velocities, and F-wave latency were normal. Needle electrode examination of the muscles on the left demonstrated fibrillation potentials in all muscles sampled below the elbow and in the biceps above the elbow, consistent with a polyneuropathy. Voluntary motor unit potentials were normal throughout. The results indicated partial axonal degeneration of the ulnar, median, radial, and musculocutaneous nerves below the level of the tourniquet in the midupper arm. (There was also evidence of a mild bilateral median neuropathy at the wrists, consistent with her previous nerve conduction studies.) By 6 months, her symptoms had completely resolved.

Discussion

The tourniquet is used in peripheral limb surgery to produce a bloodless surgical field. Post-tourniquet nerve palsy or "tourniquet paralysis" is well documented, and the incidence of tourniquet paralysis has been reported as 1 in 8,000 operations.[5] Recommendations for appropriate use of the tourniquet include the maintenance of a pressure no more than 150 mmHg greater than the systolic blood pressure and deflation of the tourniquet every 90–120 min.[6] Even the modern-day tourniquet used according to these recommendations may result in tourniquet paralysis, however.[7] A recent prospective observational study of upper and lower limb blockade using a nerve stimulator and the multiple injection technique demonstrated that higher tourniquet inflation pressure (> 400 mmHg) was associated with an increased risk of transient nerve injury.[8] Prolonged tourniquet time is also associated with an increased incidence

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Table 1. Nerve Conduction Studies

	Amplitude (mV and μV)			Velocity (m/s)			Distal Latency (ms)			F-Wave Latency (ms)		
Nerve	Right	Left	Normal	Right	Left	Normal	Right	Left	Normal	Right	Left	Normal
Median, motor	7.8	5.9	>4.0	51	55	>48	5.4	4.0	<4.5	27.5	25.2	<32
Median, sensory	24	28	>15		58	>56	4.5	3.3	<3.6			
Radial, sensory	51	36	>20			>49	2.3	2.2	<2.9			
Ulnar, motor	8.3	5.1	>6.0	61	58	>51	2.7	2.6	<3.6		23.9	<32
Ulnar, sensory	53	24	>10	59	57	>54	2.7	2.5	<3.1			

The findings of the nerve conduction studies should be compared with the reference values and the results from the contralateral side.

of neuropathy.[2] Although underreported in the anesthesia literature, there have been case reports of upper extremity tourniquet paralysis. In 1992, Hidou *et al.*[9] described an ASA physical class I patient undergoing axillary brachial plexus block for hand surgery. The tourniquet remained at 300 mmHg for 45 min, and, after surgery, electrophysiologic testing demonstrated a severe conduction block of sensory and motor fibers localized to the lower margin of the tourniquet.

Bolton *et al.*[10] published the first well-documented report of multiple limb neuropathies as a result of ischemia, a complication of arteriovenous shunt placement in uremic patients. Similar reports appeared subsequently.[11,12] Wilbourn *et al.*[13] described IMN after intra-aortic balloon insertion, after cannulation for cardiopulmonary bypass, and in thromboembolism in patients with an atheromatous aorta as well as a result of the placement of arteriovenous shunts. They attributed the ischemia associated with IMN to arterial occlusion or low flow states. Bolton *et al.*[10] and Wilbourn *et al.*[13] noted a predominance of sensory symptoms and pain over physical signs. Our patient's presentation was consistent with this.

Electrophysiologic findings of IMN include axon-loss lesions of motor and sensory nerves supplying the distal portion of the limbs. In addition, there is evidence of denervation, distal greater than proximal. Nerve conduction studies reveal motor and sensory responses that are low in amplitude or unobtainable. Sensory fibers are more affected than motor fibers, and the decrease in amplitude is more pronounced distally. Latencies and conduction velocities are normal.[14] Our patient's electrophysiologic study was consistent with these findings. In tourniquet paralysis, besides the axonal neuropathy changes seen in IMN, there are electrophysiologic changes of conduction block, which may predominate. Thus, electrophysiologic studies will determine the degree of demyelination *versus* axonal degeneration. This has clinical significance, because lesions that are predominantly demyelinating in nature result in early recovery, whereas those caused by axonal degeneration are associated with delayed recovery. Autonomic changes consistent with causalgia may also occur.[15] A component of our patient's symptoms may have been attributable to autonomic dysfunction.

Table 2. Electromyographic Studies

Muscle	Insertional Activity	Fibrillation Potentials	Voluntary Motor Unit Potentials
R, first dorsal interosseous	Normal	0	Normal
R, flexor carpi radialis	Normal	0	Normal
L, abductor digiti minimi	Increased	+++	
L, biceps brachii	Increased	++	
L, brachioradialis	Increased	++	
L, deltoid	Normal	0	Normal
L, extensor indicis proprius	Increased	++	
L, first dorsal interosseous	Increased	++	
L, flexor carpi radialis	Increased	+++	
L, flexor pollicis longus	Increased	+++	
L, infraspinatus	Normal	0	Normal
L, latissimus dorsi	Normal	0	Normal
L, pronator teres	Increased	+++	
L, triceps	Normal	0	Normal

Note the normal voluntary motor unit potentials and the distal left fibrillation potentials and increased insertional activity.

L = left; R = right.

The connection or overlap between the entity of tourniquet paralysis and the syndrome of IMN is not well documented. Generally, tourniquet paralysis is thought to be the result of direct compressive injury of the nerve. Experimentally, local nerve compression from a pneumatic tourniquet displaced the nodes of Ranvier 200 μm away from the site of compression, caused an intussusception of paranodal myelin and axon into the myelin sheath, and resulted in demyelination in the area of intussusception.[16] The investigators concluded that these changes were caused by direct mechanical compression. Ischemia, however, may play a role in tourniquet paralysis. Wilbourn *et al.*[13] hypothesized that the peripheral nerve was relatively more sensitive to ischemia than muscle or skin. Even low tourniquet inflation pressures produce nerve injury and abnormal microvascular permeability.[17] Neural microvascular permeability has also been demonstrated after tourniquet inflation pressures of 50–200 mmHg, especially at the edge of the compression.[18] Thus, there is experimental evidence to suggest both ischemic and compressive mechanisms in post-tourniquet paralysis.

Nerve injury is a significant issue for anesthesiologists. It accounts for 16% of the claims filed in the ASA Closed Claims Study database.[19] Of these, ulnar and brachial

plexus injuries account for 28% and 20%, respectively. Caplan[20] notes that only 9% of medicolegal cases of perioperative neuropathy have a recognizable cause despite extensive investigation. In a retrospective study of patients at high risk for neuropathy undergoing upper extremity surgery under axillary block, Horlocker *et al.*[2] determined that 11.3% of nerve injuries were anesthesia related and 88.7% were surgery related. Only clinical criteria (*i.e.*, level and distribution of the neuropathy) were used to differentiate anesthesia-related nerve injury from surgery-related nerve injury, however.

Mechanical pressure on nerve tissue has long been recognized in the pathogenesis of tourniquet paralysis. Our report illustrates that interruption of the blood supply, as occurs in IMN, may also play a role. When presented with neuropathy after upper limb surgery under axillary brachial plexus block, the anesthesiologist needs to consider all risk factors, including the tourniquet, in the differential diagnosis of the neuropathy. Importantly, electrophysiology studies, as illustrated in our case report, will determine the site and mechanism of the injury as well as the prognosis for recovery.

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