CASE REPORT

# Idiopathic Brachial Plexitis After Total Shoulder Replacement with Interscalene Brachial Plexus Block

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We describe a case of idiopathic brachial plexitis (IBP) that occurred after total shoulder replacement with an interscalene brachial plexus block. A unique aspect of the case was full recovery of neurological function before release from the postanesthesia care unit (PACU) with the onset of new neurological symptoms 12–18 h later.

## Case Report

A 65-year old woman was scheduled for right total shoulder replacement secondary to osteoarthritis. Her medical history was positive for hypothyroidism controlled with levothyroxine and for depression treated with fluoxetine.

She was premedicated with 1 mg of midazolam intravenously (IV) and underwent interscalene block with a 25-gauge, blunt bevel needle and 40 mL of 1.4% mepivacaine, 1:200,000 epinephrine freshly added and alkalinized with bicarbonate (1 mL of 1 mEq/mL sodium bicarbonate per 10 mL of mepivacaine). Needle placement within the sheath of the brachial plexus was confirmed with a single, mild paresthesia from the superior trunk of the brachial plexus. After confirmation of complete sensory anesthesia and motor block around the shoulder, a total shoulder replacement was performed routinely without the use of general anesthesia. Estimated blood loss was 300 mL, and there were no intraoperative hemodynamic alterations.

In the PACU, the block began to resolve 3 h after placement, and acute pain was noted. The patient received analgesia with morphine via a patient-controlled analgesia (PCA) device and was released from PACU with good analgesia and full neurological function of the right upper extremity. Morphine, 28 mg, was given over 3 h in the PACU. The patient's admission assessment on the regular nursing floor recorded intact sensation and motor function of the right arm.

Eighteen hours after release from PACU, the patient was awakened by an abrupt increase in shoulder pain. While this pain was being evaluated by the nursing staff, she noted weakness and numbness of her right hand, which had not been present when she fell asleep. When she was evaluated by the orthopedic house staff an hour later, motor function changes were present, and were more dense proximally than distally, with 0–1/5 strength of the biceps, 0–1/5 triceps, 0–1/5 wrist extension, 4/5 wrist flexion, and 4–5/5 for the intrinsic muscles of the hand. There were paresthesiae over the wrist, forearm, and hand. Neurological consultation determined that the lesions were predominantly of the superior trunk of the brachial plexus, but there was patchy involvement of the middle and inferior trunks, manifest by involvement of the median and ulnar nerves. Based on this observation, a lesion at the plexus level was felt to be more likely than a lesion at a more proximal site. A magnetic resonance scan, obtained to rule out a compressive hematoma, showed the brachial plexus to be normal. Conventional radiological findings were unchanged from those obtained in the PACU, and no dislocation of the prosthesis was present.

Her clinical course over the next several days was characterized by extreme pain that was refractory to treatment. Because the pain was poorly localized and disproportionate to the stimulus, an early sympathetic dystrophy was considered possible. Carbamazepine was administered with some success in decreasing the opiate requirement. The patient was also started on prednisone to treat any new edema of the brachial plexus. By the sixth postoperative day, she had recovered some (2/5) wrist and finger extension and had markedly decreased paresthesiae. On the 10th day, an electromyogram (EMG) revealed slowed conduction and positive waves, which were interpreted as a pattern of plexopathy with a primary involvement of the superior trunk. The patient was discharged to home, and physical therapy was prescribed. At 4 wk, a follow-up physical examination and EMG were performed at another institution. The physical examination revealed marked recovery of motor function. The EMG revealed fibrillation and sharp potentials in the right deltoid, biceps, triceps, flexor carpi ulnaris, pronator teres, and interosseous muscles, with some motor unit drop out. An EMG of the contralateral extremity, performed as a control, revealed significant motor-unit drop out in the left infraspinatus, deltoid, biceps, and triceps. In light of the involvement of the entire right brachial plexus and the mild, widespread involvement of the contralateral brachial plexus, the diagnosis of IBP was made. At final follow-up, almost 12 mo postoperatively, neurological recovery was virtually complete. Although repeat EMG was recommended, the patient refused the examination.

## Discussion

This case identifies a diagnosis of which anesthesiologists who practice regional anesthesia and surgeons

Accepted for publication May 23, 1997.

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0003-2999/97/$5.00

who perform shoulder surgery should be aware in the postoperative period. IBP should be considered in the context of postoperative neurological changes after shoulder surgery or brachial plexus anesthesia, because brachial plexus stretch, direct nerve trauma, and compressive neuropathy are well known serious sequelae with potential medicolegal implications.

Shoulder surgery, particularly total shoulder replacement, is associated with a risk of injury to the brachial plexus. Interscalene block, as in all brachial plexus anesthesia, can be associated with injury to the brachial plexus (1). The association of intentional elicitation of paresthesiae with adverse outcome after brachial plexus anesthesia has been suggested (2), although the issue remains controversial.

The diagnosis of IBP in the postoperative period has been reported (3–5), although not in conjunction with regional anesthesia. This condition is thought to be related to a diverse family of neurological syndromes with names such as "brachial plexus neuropathy" and "acute brachial radiculitis." A wide variety of events has been associated with IBP, including vaccination (6), infection (7), trauma (8), radiation (9), and pregnancy (10). There are numerous reports of cases without apparent cause (11–13) and of some with a congenital origin (14). The diverse etiologies of IBP suggest a possible immune-mediated mechanism as a common denominator (15).

The relationship of surgery to IBP is unclear, but some of the tissue events of surgery are similar to other associated potential causes. One of the proposed mechanisms of immune-mediated IBP is that some event activates a dormant virus that resides in the tissues of the brachial plexus, a situation analogous to the reactivation of herpes zoster that occurs with shingles (16). It could be that some of the physical events associated with surgery or brachial plexus anesthesia are involved in activating dormant viruses in these cases. Suppression of the immune system is known to occur with general anesthesia but was not a factor in this case, because general anesthesia was not used.

The events in the clinical course of this patient are characteristic of IBP. Severe pain often precedes the onset of paresthesiae and motor deficits with IBP (17,18). This patient had severe pain 12 h postoperatively that awakened her from sleep and preceded the onset of her motor deficit. The severe pain that occurred later in the course of her PACU stay could also have been the onset of IBP. The asymmetric involvement of the brachial plexus is another characteristic of IBP that was present in this case (19–21), as is the evolving nature of the motor lesions. Abnormal anatomy, analogous to the postoperative conditions after shoulder surgery, has been associated with IBP (22). The natural history of IBP also matches the clinical course of this patient, with recovery usually being rapid and nearly complete by 12 mo (5,15,23,24). Other causes of brachial plexus injury do not match the clinical pattern of this case. The widespread involvement of the brachial plexus makes injury from block placement unlikely. The full recovery of function, with severe pain and sensory changes preceding motor deficit at an interval after normal function, precludes stretch or sharp injury to the brachial plexus during surgery, because the neurological consequences are invariably present immediately after surgery. Because chemicals were added to the local anesthetic (epinephrine, sodium bicarbonate), there is the possibility of chemical contamination, although this explanation also fails to explain the evolution of this case, because fresh ampules were used for each additive, and the myelitis from contamination should have been immediately apparent.

In summary, we report a postoperative neurological event consistent with IBP. Full recovery of neurological function after total shoulder replacement under interscalene block was followed by the onset of intense pain, paresthesiae, and mixed motor and sensory defect. Partial recovery within 2 wk, EMG findings, including the contralateral shoulder, and the near-complete resolution of the symptoms within 12 mo are consistent with the diagnosis of IBP. It is important to consider this diagnosis after shoulder surgery and brachial plexus anesthesia, because stretch injury from the surgery and nerve trauma from conduction block are often the first diagnoses considered, and both have potential medicolegal implications.

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