

Parsonage–turner syndrome after interscalene block for diagnostic arthroscopic surgery of shoulder joint

Sir,

A 45-year-old male patient, ASA physical status I, was scheduled for diagnostic arthroscopy of right shoulder joint. Pre-anesthetic examination was unremarkable for any pathological antecedent. On the day of surgery, he was shifted to the preoperative room and monitoring of ECG, pulse oximetry, and blood pressure was started. Patient was administered oxygen via ventimask. Inj. Ranitidine 50 mg IV and inj. Metoclopramide 10 mg IV were administered to the patient as pre-medication. General anesthesia was induced with inj. Propofol 180 mg IV and inj. Vecuronium 7 mg IV. An ultrasound-guided interscalene block was performed and 20 ml of 0.25% Bupivacaine was administered without any incident. Anesthesia was maintained as per the standard protocol of our institute. After 2 h of uneventful surgery, he was transferred to the post anesthesia care unit. Twelve hours after the surgery, the patient complained of right arm weakness and continuous severe shooting pain in the right shoulder, radiating to the arm. Patient was immediately administered inj. Diclofenac 75 mg IM, but there was no relief in symptoms of pain. X-ray of shoulder joint was unremarkable. The following morning, the pain subsided on rest, but was precipitated on movement of right arm, along with motor weakness. Magnetic resonance imaging (MRI) of cervical spine and brachial plexus, done the following day, did not show any acute lesion. A neurology consultation on the postoperative day 2 found decreased sensation to temperature throughout dermatomes C4–T1, with absent vibration, joint position perception, and light touch sensation in C6–T1. Subsequently, the electromyography showed sensory and motor involvement being compatible with brachial polyneuritis. Over a course of next few days, the patient complained of a tingling sensation on the skin of right arm which was painful on palpation. After 7 days of motor weakness and sensory paresthesias, an immunologic study reported positive antibody titers for IgM and IgG anti-ganglioside antibodies. Immunoglobulin treatment along with oral prednisolone was started, and clinical improvement was observed during the following months.

Idiopathic amyotrophic neuralgia or Parsonage–Turner syndrome (PTS) refers to a rare set of symptoms resulting from inflammation of unknown etiology and an immunological pathology of the brachial plexus

leading to hypersensitivity of the damaged nerves to stretching or pressure.^[1] This syndrome can present with sudden-onset pain radiating from the shoulder to the upper arm, followed by weakness and numbness. Subsequently, there is generally a phase in which there is no pain while resting but, with specific movements or positions, sudden sharp shooting pain can occur, which subsides within a couple of hours. Affected muscles become weak and atrophied, and in advanced cases, paralyzed.^[2] The most common presentation is involvement of nerves of brachial plexus, but the nerves of upper and lower legs and feet, diaphragm or the vocal cords, skin and muscles of the abdomen, muscles of the face and ear and organ of Corti can also be affected.^[3] Surgery, trauma, or exhausting exercise, infection, vaccinations, and treatments with blood products or immunotherapy could be predisposing factors. Although it is a well-documented entity with autosomal dominant variant, to our knowledge, there are few reported cases of PTS precipitated by interscalene block in a healthy adult in anesthesia literature. Tetzlaff *et al.*^[4] have reported a case of idiopathic brachial plexitis after total shoulder replacement for osteoarthritis with interscalene brachial plexus block in a 65-year-old hypothyroid patient. Provisional diagnosis is based on thorough history and examination and appearance of a sudden and severe shoulder and upper arm pain, paralysis, and amyotrophy. Accurate diagnosis can be challenging, but nerve conduction velocity and imaging studies assist in the evaluation.^[5] Although the mechanisms of this injury are unclear, the potential preexisting occult pathology of the peripheral nervous system may have predisposed him to development of a peripheral autoimmune injury leading to a brachial neuritis. Treatment is symptomatic and requires combination of a long-acting nonsteroidal anti-inflammatory drug (NSAID) with long-acting opioids for acute phase. A short therapy of oral prednisone along with immunoglobulin therapy has a favorable effect in some cases.^[6] Later on, during the chronic phase of the disease, patient may require psychotherapy to cope with psychological trauma of chronic pain and chiropractic care or physiotherapy to regain function in the limb and to prevent contractures. Patients and physicians may misunderstand the persistence of pain and paresthesias in the early postoperative period to be a reflection of poor surgical sequelae or anesthetic complication.

In conclusion, it is interesting that anesthesiologist include the PTS in the differential diagnoses of any kind of extended sensory and motor block because it supposes a different diagnostic and clinical approach. Thorough clinical examination is important to exclude any neurological complications due to regional anesthesia or the surgery performed, like compressive neuropathies, cervical radiculopathy, brachial stretch injuries, or ulnar nerve compressive injury. All of these latter conditions can place the anesthesiologist at legal risk. Patient should be adequately counseled, reassured, and explained about this phenomenon.

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