Suprascapular nerve injury secondary to cement extravasation in an anatomic total shoulder arthroplasty: a case report

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Shoulder arthroplasty is a common method of treatment for degenerative shoulder disorders with high rates of successful outcomes. There are various reasons that may affect the outcomes of the replacement, including failure of the glenoid component. Cementless glenoid components in conventional total shoulder arthroplasty have a significantly higher revision rate than cemented glenoid components. The Food and Drug Administration states that all polyethylene glenoid components are intended for cemented use only when used in a total shoulder application. (Code of Federal Regulations Title 21, Volume 8, 2018; Section 888.3650 Shoulder joint metal/polymer non-constrained cemented prosthesis).

It is uncommon for patients to have suprascapular nerve (SSN) impairment, which is typically a diagnosis of exclusion for anatomic total shoulder arthroplasty. Potential causes of SSN injury include compression from adjacent ganglia, abnormal morphology of the suprascapular notch, neuritis, direct trauma or traction injury, massive rotator cuff tear, and iatrogenic injury. Iatrogenic injury to the SSN has been reported in reverse total shoulder arthroplasty (RTSA) upon drilling or placement of baseplate screws. Iatrogenic cement extravasation injuries have been reported in other orthopedic surgical procedures including spine procedures along with hip and knee arthroplasty where the cement was intended to provide structural support. There have been no reported cases to date regarding SSN damage secondary to cement extravasation in an anatomic total shoulder replacement. We present a case of cement extravasation through a drill hole in the glenoid during total shoulder arthroplasty procedure causing neurological deficit to the SSN.

Case report

A 68-year-old man presented for right shoulder glenohumeral degenerative joint disease. He had a 4-year history of progressive loss of motion with diminished function in the prior 6 months. Treatment to date had included corticosteroid injections and physical therapy. The patient reported that injections worked except within the last year and that physical therapy did not improve his symptoms. Preoperative imaging included X-rays showed mild flattening of his glenoid consistent with progressive degenerative changes. Following anatomic shoulder arthroplasty, the patient progressed normally in the early postoperative period. Three months after surgery, his physical therapist expressed concern with his strength and function. His range of motion on the affected shoulder was 90/140 forward flexion, 90/140 abduction, and 0° of external rotation with shoulder in 0° of abduction. His strength was 3/5 for external rotation with 0° of shoulder abduction, 3+ for subscapularis.

Investigation performed at the Department of Orthopaedic Surgery, The University of Alabama at Birmingham, Birmingham, AL, USA. Institutional review board approval was not required for this case report.

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C5-6. A computed tomography of the shoulder demonstrated cement extravasation within the suprascapular notch (Fig. 1, a and b), as well as the expansive nature of the cement extravasation (Fig. 1, c).

Upon recognition of the cement mass, the SSN was decompressed through an open trapezius splitting approach. Fibrous tissue and a 4-cm maximum length cement fragment was noted in the area surrounding the area of the suprascapular notch surrounding the nerve (Fig. 2). It was cleared in a careful fashion and gently elevated with a Freer Elevator along its lateral margin near the glenoid surface. The fragment measured 4 cm × 1.5 cm × 1.0 cm (Fig. 3, a) and contained a clear course of the nerve within the inferior portion of the cement (Fig. 3, b). The nerve remained intact though there was some stenosis of the nerve through the area of the suprascapular notch. General neurolysis was performed and the suprascapular ligament was released. He was seen approximately 2 months after his cement removal surgical date and progressed well. His right shoulder range of motion improved to 135°/0°/90° abduction, 4+/-5 with forward flexion, and 4+/5 for the subscapularis testing. At his last office visit, approximately 8 months from the date of surgery, his range of motion was 130° forward flexion, 120° abduction, 45° of external rotation with shoulder in 0° of abduction, 80° of external rotation with the shoulder abducted to 90°, and internal rotation to his back pocket. Strength of the right shoulder was 4+/-5 for external rotation with 0° of shoulder abduction, 4+/-5 with external rotation at 90° of shoulder abduction, 5/-5 with forward flexion, and 5/-5 for the subscapularis testing. Overall, the patient recovered well with decreased pain and improved functionality compared to preoperative measures. After the patient’s 8-month follow-up, he did not have subsequent follow-up for long period due to geographical remoteness. The patient then followed up with outside institution at 14-year status post-right shoulder arthroplasty with complaints of progressive pain after pushing up from a seated position. Computed tomography scan at that time indicated superior anterior migration of humeral head consistent with rotator cuff tear arthropathy and a poorly visualized glenoid component with suggested loosening. Of greater concern at that time, the patient is in diminishing health and has been diagnosed with dementia. These factors ultimately lead to the family pursuing nonoperative measures for this patient.

Discussion

The anatomical course of the SSN has been well defined. The SSN ascends from the upper trunk of the brachial plexus involving roots from C5 and C6. The nerve passes downward to the superior border of the scapula and through the suprascapular notch, inferior to the superior transverse scapular ligament, where motor nerves to the supraspinatus muscle branch. From the suprascapular notch the nerve tracks obliquely and laterally along the supraspinatus muscle in the supraspinatus fossa to the base of the scapular spine. The nerve begins to curve medially once it reaches the base of the scapular spine and passes under the infraspinatus scapular ligament through the spinoglenoid notch, terminating its course by giving off glenohumeral posterior capsule articular branches before innervating the infraspinatus muscle (Fig. 4). Given that the SSN is essentially in a fixed position throughout its course along the fossa, it is susceptible to intrinsic and extrinsic damage which could result in neural pathology.

There are numerous potential etiologies for SSN neuropathy, but to our knowledge, this is the first case of SSN injury from poly-methylmethacrylate cement extravasation during ATSA. Extruded cement can cause nerve damage via mass effect or via heat generated during cement polymerization. Nerves can undergo permanent damage when heated to 45°C-47°C for 1-2 minutes. Cement polymerization temperatures are directly correlated with cement thickness and have been recorded at over 55°C (131°F). We assessed the thermal increase from 2 separate cylinders of Simplex (Stryker, Kalamazoo, MI, USA) bone cement measuring 4 × 1.5 × 1.0 cm with a Raytek Raynger ST infrared thermometer (Raytek Corp., Santa Cruz, CA, USA) and identified a maximum temperature of >95°C (>203°F). Thus, prevention or immediate removal of leaked bone cement is imperative in the prevention of possible nerve injury from both thermal damage and mass effect. Thermal nerve injury requiring cement removal appears to not be frequently reported in revision shoulder arthroplasty surgery, but is noted in other arthroplasty literature. The heat generated by ultrasound devices used during cement removal may injure surrounding nerves. Measured tissue temperatures during ultrasound humeral cement removal have raised the adjacent bone to 62.8°C (145°F) and radial nerve to 51.7°C (125°F). Because of this concern, it has been recommended that surgeons employ safe strategies during cement removal, such as intermittent use of the ultrasound device with the application of cold irrigation during intervals.
Potential nerve injury related to shoulder surgery has been previously reported. Iatrogenic SSN injuries have been reported during SLAP repairs secondary to an errant drill hole or suture anchor. The available bone stock at the posterior superior glenoid rim is much smaller than that of the anterior superior rim, thus putting the patient at risk of injury if the posterior superior bone is drilled from an anterior approach or anywhere in the upper glenoid if the drill angle does not keep the anchor within the glenoid vault. Regarding shoulder arthroplasty, RTSA has been associated with a higher incidence of nerve injury compared to ATSA. Lädermann et al. in 2011 identified that the frequency of acute postoperative nerve injury was 10.9 times higher in RTSA compared to ATSA. This was likely be secondary to the arm lengthening sustained during RTSA and/or external rotation during humeral and glenoid preparation. Avoidance of prolonged periods in these at-risk arm positions, along with intermittent recovery phases in the neutral position, may prove beneficial to decrease the rate of nerve injury. Wang et al. in 2010 described an incidence of a malpositioned superior screw entering the suprascapular fossa following RTSA causing SSN entrapment leading to diffuse shoulder pain. Intraoperatively, 2 cm of the screw tip was observed to be encased with scar tissue and entrapping the SSN from beneath. Six months following the removal of the insulting screw tip and nerve decompression, the patient was reported to have improved pain and regained range of motion. Additionally, a recent cadaveric study established drilling safe zones and concluded that superior/posterior drilling and extraosseous screw employment during baseplate implantation exposes the SSN to possible injury.

Conclusion

We report an unusual SSN injury from extravagated cement through a drilled polyethylene glenoid hole in ATSA which required removal and resulted in residual functional deficits. SSN neuropathy secondary to shoulder surgery is infrequent and likely often overlooked as a potential source of lower than anticipated functional outcome. Clinical features of SSN neuropathy may include supraspinatus/infraspinatus atrophy (depending on location of lesion), weakness, and posterolateral shoulder pain. SSN neuropathy is typically a diagnosis of exclusion and is usually diagnosed based on clinical features, EMG, and imaging. Treatment outcomes vary based on etiology. This case highlights the importance of surgeons to appreciate vault perforation when drilling into the glenoid and inserting polymethylmethacrylate cement in anatomic total shoulder arthroplasty.

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Figure 3 (a) Extruded cement viewed from superior viewpoint. (b) Inferior view revealing course of suprascapular nerve (white dotted line) through the cement.

Figure 4 Illustration demonstrating course of suprascapular nerve. (Illustration used with permission of David Fisher.)


