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Anatomic total shoulder arthroplasty in a patient with Parsonage-Turner syndrome: a case report

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Neuralgic amyotrophy (NA), otherwise known as Parsonage-Turner syndrome (PTS) or brachial neuritis, was described by Parsonage in 1948.[12] It is characterized by a period of intense shoulder pain followed by a period of flaccid paralysis of the shoulder girdle muscles.[4,9,11] The initial pain may be so severe as to awaken a patient from sleep, and typically lasts days to weeks. A period of upper trunk-related weakness typically begins within a month of initial symptom onset. Persistent weakness may occur and most commonly affects the deltoid, supraspinatus, infraspinatus, serratus anterior, biceps, and triceps.[17] In some cases, motor function slowly returns.[2] Maximal recovery typically occurs within 18 months of symptom onset, but may take up to 8 years.[8] Accurate diagnosis is challenging and typically involves a combination of symptomatology, magnetic resonance imaging (MRI), and electromyography findings.[10] Multiple potential etiologies for PTS have been proposed, and the true cause is not always identifiable for individual patients. Potential triggering events include viral or bacterial infection, immunizations, times of increased physiologic stress such as the perioperative and peripartum periods, and strenuous upper body activity.[3,17]

The treatment for PTS is generally conservative, and no specific treatment protocol exists. Physical therapy is utilized to maximize shoulder girdle strength and biomechanical stability of the glenohumeral joint.[1,17] Surgical procedures including nerve exploration, neurolysis, nerve grafting, and nerve or tendon transfers may be performed if there is no evidence of nerve regeneration within 6-9 months of symptom onset.[5,13] Patients with PTS may have coexisting glenohumeral osteoarthritis (OA). The shoulder arthritis may have been present prior to the onset of PTS, or could also develop at any point after the episode of PTS. There is little to no available literature on the management of glenohumeral OA in patients with PTS. We, therefore, report a case of a patient with PTS and concomitant shoulder OA who underwent anatomic total shoulder arthroplasty (TSA).

Case report

A 68-year-old right-hand-dominant man presented with long-standing left shoulder pain, weakness, and decreased range of motion (ROM). Two years prior to presentation, he developed an atraumatic deep, aching pain in the shoulder with use and at rest. After approximately 2 weeks, his pain resolved. However, he developed weakness with elevating his left arm above his head. He reported difficulty with abducting and flexing the shoulder due to weakness. He denied any numbness, tingling, or burning pain in his arm. He remained very active and enjoyed walking and biking several miles per day. He did not pursue specialized medical evaluation at that time. He eventually developed increasing shoulder pain and crepitus with activity, and therefore, presented to our center for evaluation.

Physical examination demonstrated mild fasciculations in the left deltoid and biceps, although there was no deltoid atrophy. Shoulder ROM was 40° active elevation, 90° passive elevation, 0° external rotation, and internal rotation to the iliac crest. Strength was 4/5 forward flexion, 4/5 abduction, 3/5 external rotation, and 4/5 internal rotation. His anterior, lateral, and

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posterior deltoid fired well. He had pain and crepitus with passive ROM.

Plain radiographs and computed tomography demonstrated severe glenohumeral OA with mild superior and central glenoid erosion (Fig. 1). A shoulder MRI demonstrated significant glenohumeral chondromalacia with mild rotator cuff tendinopathy, but no full-thickness rotator cuff tears (Fig. 2, *A*–*C*). He had mild supraspinatus atrophy with preserved deltoid muscle bulk (Fig. 2, *D*). A formal neurology consultation was undertaken. An electromyography showed chronic neurogenic changes in multiple upper trunk-innervated muscles along with fibrillation potentials in the deltoid, infraspinatus, and supraspinatus. Fasciculations were present in the biceps and deltoid, but the cervical paraspinal muscles were normal. Median and ulnar nerve conduction studies were

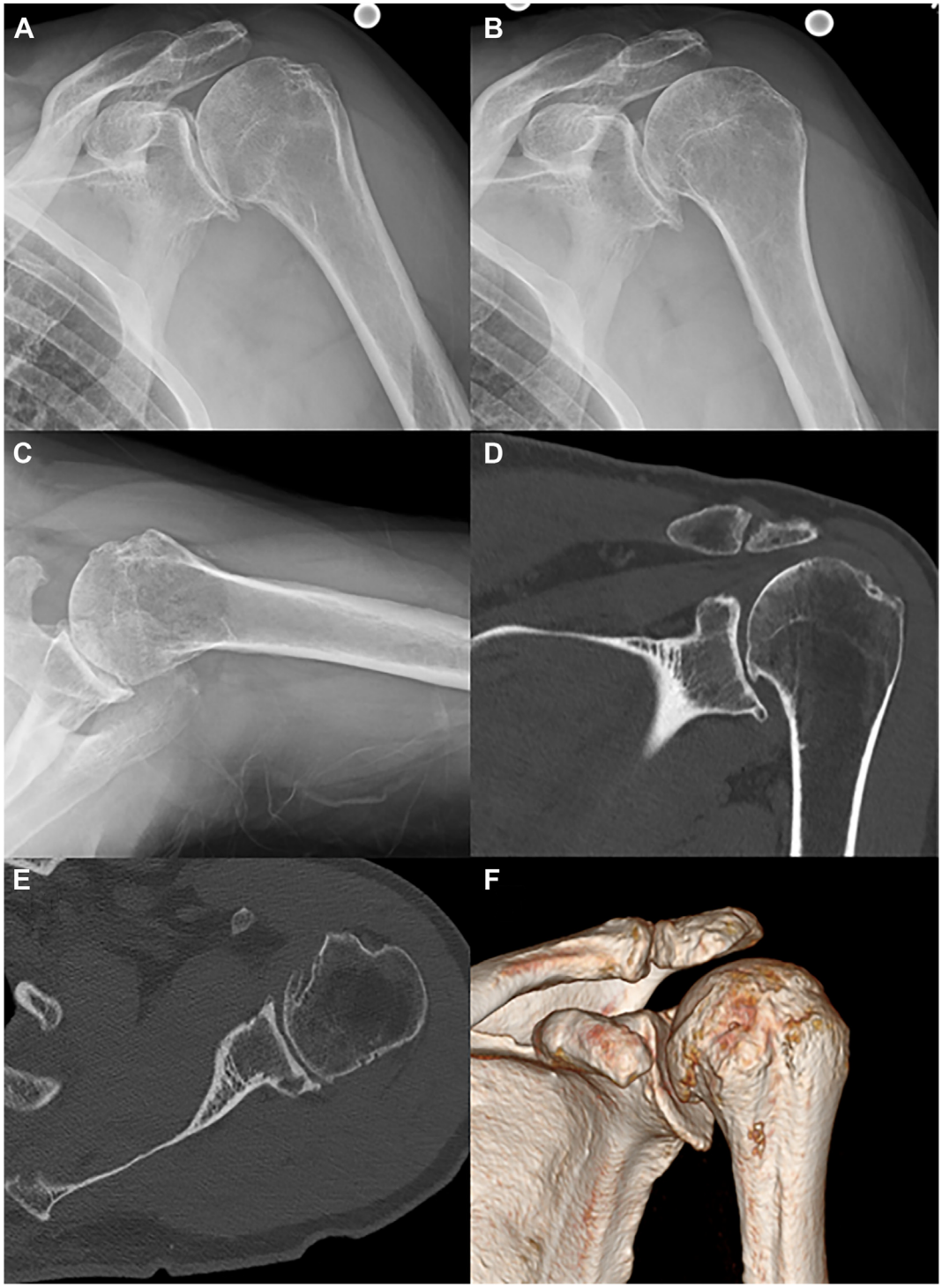

Figure 1 Preoperative anterior-posterior internal rotation, external rotation, and axillary lateral radiographs (**A-C**) as well as preoperative coronal, axial, and 3D reconstruction computed tomography images (**D-F**) demonstrating end-stage osteoarthritic changes with joint space narrowing, osteophyte formation, and minimal glenoid erosion. There is no significant humeral head subluxation.

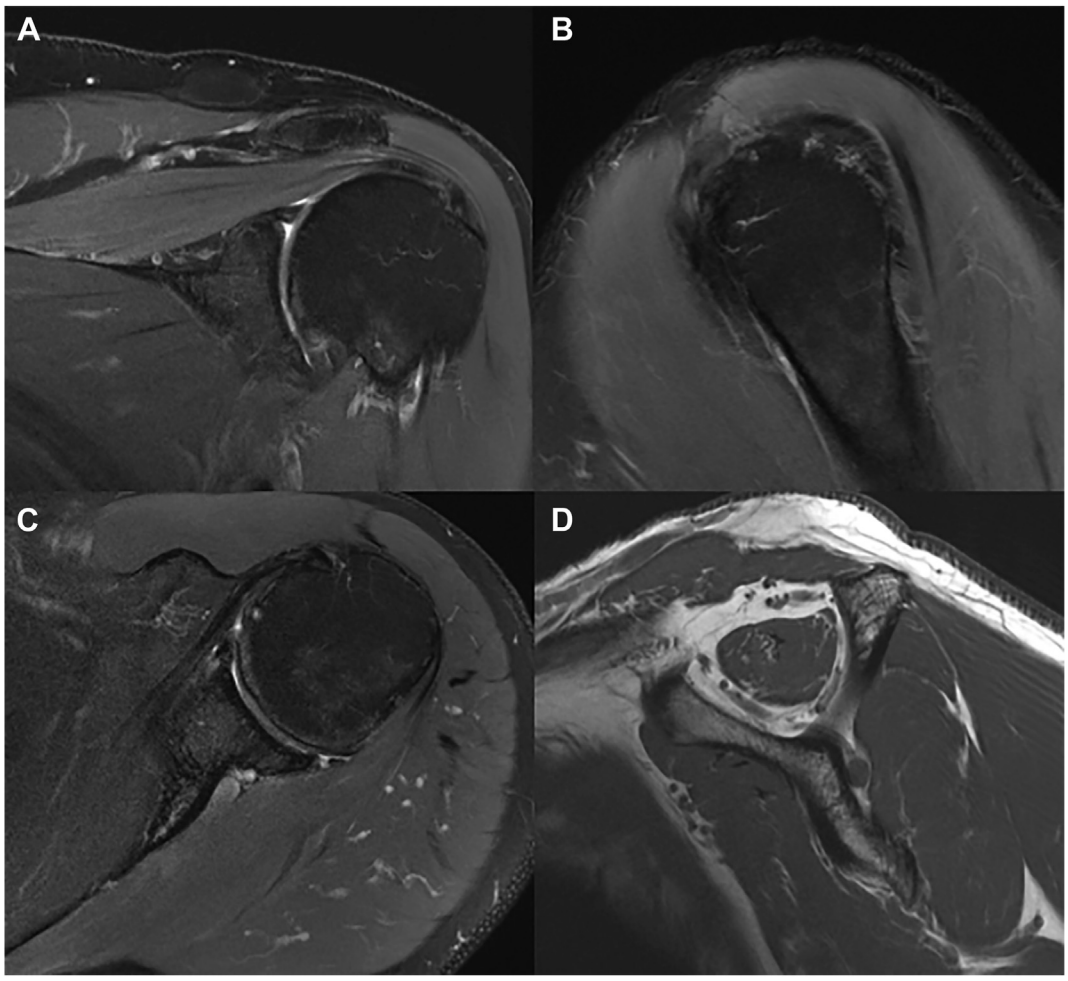

Figure 2 (**A**) Preoperative magnetic resonance imaging demonstrating a T2 coronal view of the partial supraspinatus undersurface fraying, (**B**) a T2 sagittal view of mild partial-thickness tearing of the supraspinatus and infraspinatus at the insertion, (**C**) a T2 axial view demonstrating an intact subscapularis tendon, and (**D**) a T1 sagittal view at the base of the scapular spine showing Goutallier stage 1 supraspinatus atrophy.

normal, while lateral antebrachial cutaneous responses were absent bilaterally. An MRI of the cervical spine noted moderate left C5-C6 foraminal narrowing. An MRI of the brachial plexus showed subtle fascicular swelling and T2 hyperintensity with minimal superficial enhancement of the C5 and C6 nerve roots extending into the upper trunk of the brachial plexus (Fig. 3), along with similar changes in the posterior cord. There were mild chronic denervation changes in the supraspinatus and infraspinatus. This constellation of symptoms, physical exam findings, and specialized testing was most suggestive of a chronic, active left upper trunk brachial plexopathy such as PTS.

We had a long discussion with the patient regarding his pain and weakness. Our neurology colleagues suspected a most likely diagnosis of PTS in the setting of radiographic and clinical evidence of end-stage glenohumeral OA. Given his severe OA, the patient made the informed decision to proceed with a left shoulder arthroplasty. An anatomic TSA was planned given his lack of full-thickness rotator cuff tears and concern for stretching and further injuring his brachial plexus with a reverse shoulder arthroplasty (RSA). Additionally, he was a relatively young and active patient making TSA more attractive than RSA. This research received an institutional review board exemption, and the patient provided written authorization for the usage of his medical information for research purposes.

Surgical procedure

With the patient in a beach chair position, a longitudinal incision was carried out starting 1 cm lateral to the coracoid and extending distally approximately 10 cm. Dissection was carried out through subcutaneous fat until the deltoid was encountered. The deltoid musculature was preserved without evidence of significant atrophy (Fig. 4, *A*). A full-thickness medial skin flap was developed until the coracoid became palpable. The deltopectoral interval was opened starting proximally just medial to the coracoid and working distally, retracting the cephalic vein medially and cauterizing any branches encountered on the lateral aspect of the vein. The clavipectoral fascia was then opened, allowing access to the subacromial space. A combination of blunt and sharp dissection was used to free up the subacromial and subdeltoid space. The fascia along the lateral border of the conjoint tendon was incised allowing medial retraction of this structure. The anterior humeral circumflex artery and its 2 venous communicantes were identified and ligated using electrocautery. The subscapularis was identified and the overlying bursal tissue was cleared off. The subscapularis tendon appeared healthy without tears and there was no obvious subscapularis muscle atrophy. A standard subscapularis tenotomy was performed and the cut edge of the tendon was tagged for later repair (Fig. 4, *B*). A biceps tenodesis was performed by suturing the

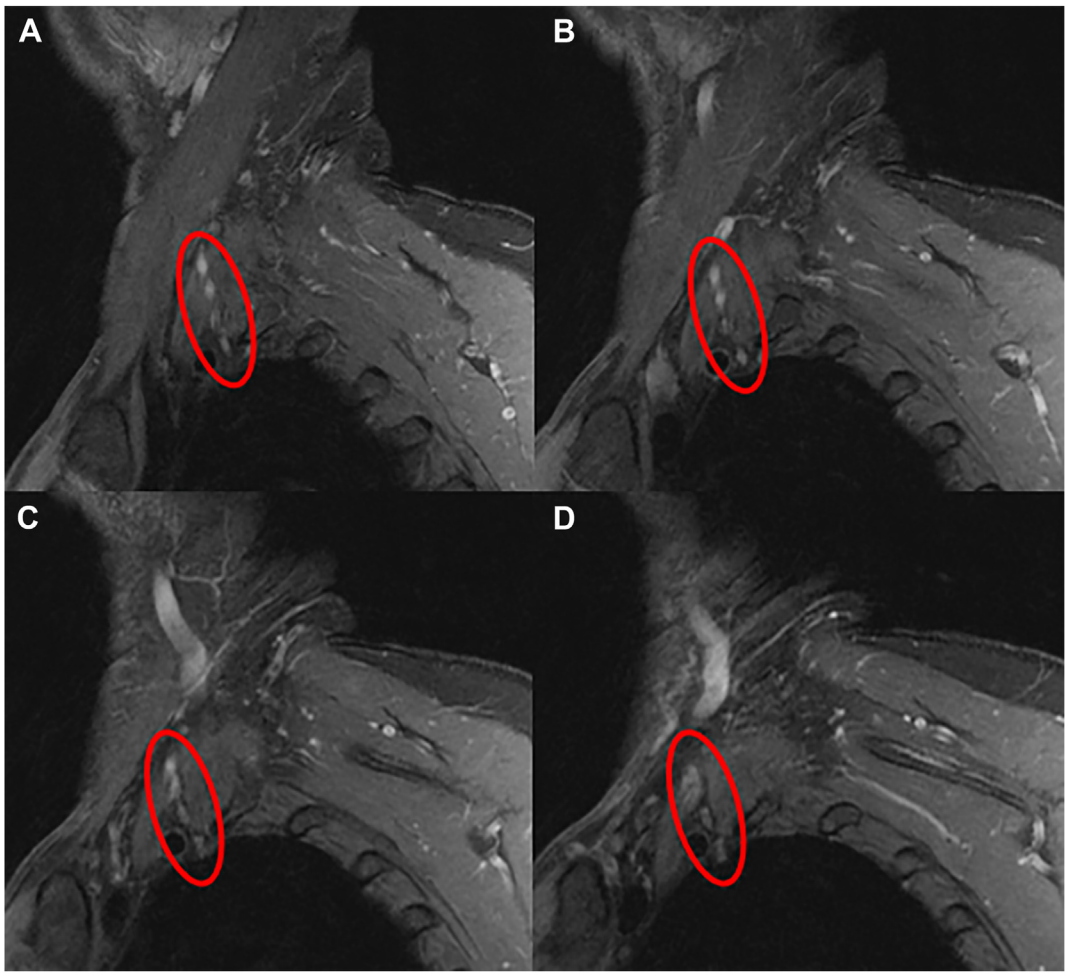

Figure 3 (**A**-**D**) Preoperative brachial plexus magnetic resonance imaging with sagittal views progressing from medial (**A**) to lateral (**D**). Mild swelling and edema are present in the C5 and C6 nerve roots extending into the *Upper* trunk of the brachial plexus.

biceps tendon to the tendon sheath and surrounding soft tissue with Vicryl sutures (Johnson & Johnson, New Brunswick, NJ, USA). The intra-articular biceps tendon was then retrieved using a curved clamp and tenotomized. The shoulder was placed in external rotation, allowing an inferior capsular release to be performed. The shoulder was then dislocated in a position of extension and external rotation. The rotator cuff was readily visible and its integrity was confirmed (Fig. 4, *C*). An extramedullary cutting guide set to 30° of retroversion was placed on the lateral border of the humerus with the version rod parallel to the arm, and the depth of resection was set just superior to the rotator cuff insertion (Fig. 4, *D*). The humeral cut was performed and the resected head was saved for potential bone grafting as well as assistance with implant sizing. The humerus was then sized, reamed, and broached to receive the Comprehensive Nano Stemless Shoulder humeral implant (Zimmer Biomet, Warsaw, IN, USA). The broach trial protector was placed on the broach. The shoulder was then placed back into a neutral position, and the glenoid was exposed. The biceps tendon stump and labrum were excised. The glenoid was then prepared to receive a hybrid anatomic glenoid component (Fig. 4, *E*). Cement was placed in the 3 peripheral holes and the real glenoid component was impacted in place (Fig. 4, *F*). The shoulder was placed back into extension and external rotation, and the humeral broach protector was removed. The Versa-Dial humeral head trial (Versadial Solutions, Irivine, CA, USA) was then placed, and the shoulder was reduced. Once satisfied with the arc of motion and soft tissue tensioning, the trial humeral head and broach were removed. The real stemless component was placed (Fig. 5, *A*) and the correct head was impacted onto the humeral component (Fig. 5, *B*). Prior to relocation, several suture anchors were placed in the subscapularis footprint (Fig. 5, *C*). The shoulder was then reduced. The subscapularis tendon was tied down to the medial row anchors, and additional fixation with several lateral row anchors was performed (Fig. 5, *D* and *E*). The tendon edge was oversewn with several Vicryl sutures. The deltopectoral interval was reapproximated (Fig. 5, *F*) and skin closure was performed in a standard fashion. A simple shoulder sling was placed, and the patient was awoken from anesthesia and transferred to the postanesthesia care unit. Radiographs obtained 4 weeks postoperatively demonstrated satisfactory implant placement without increased acromiohumeral interval or humeral head subluxation (Fig. 6). By 6 weeks postoperatively, the patient was neurovascularly unchanged from preoperatively and reported resolution of his nighttime pain and crepitus. He was allowed to begin working with physical therapy on shoulder strengthening and ROM.

Discussion

PTS is a relatively uncommon brachial plexopathy which may result in long-term weakness and dysfunction of the shoulder

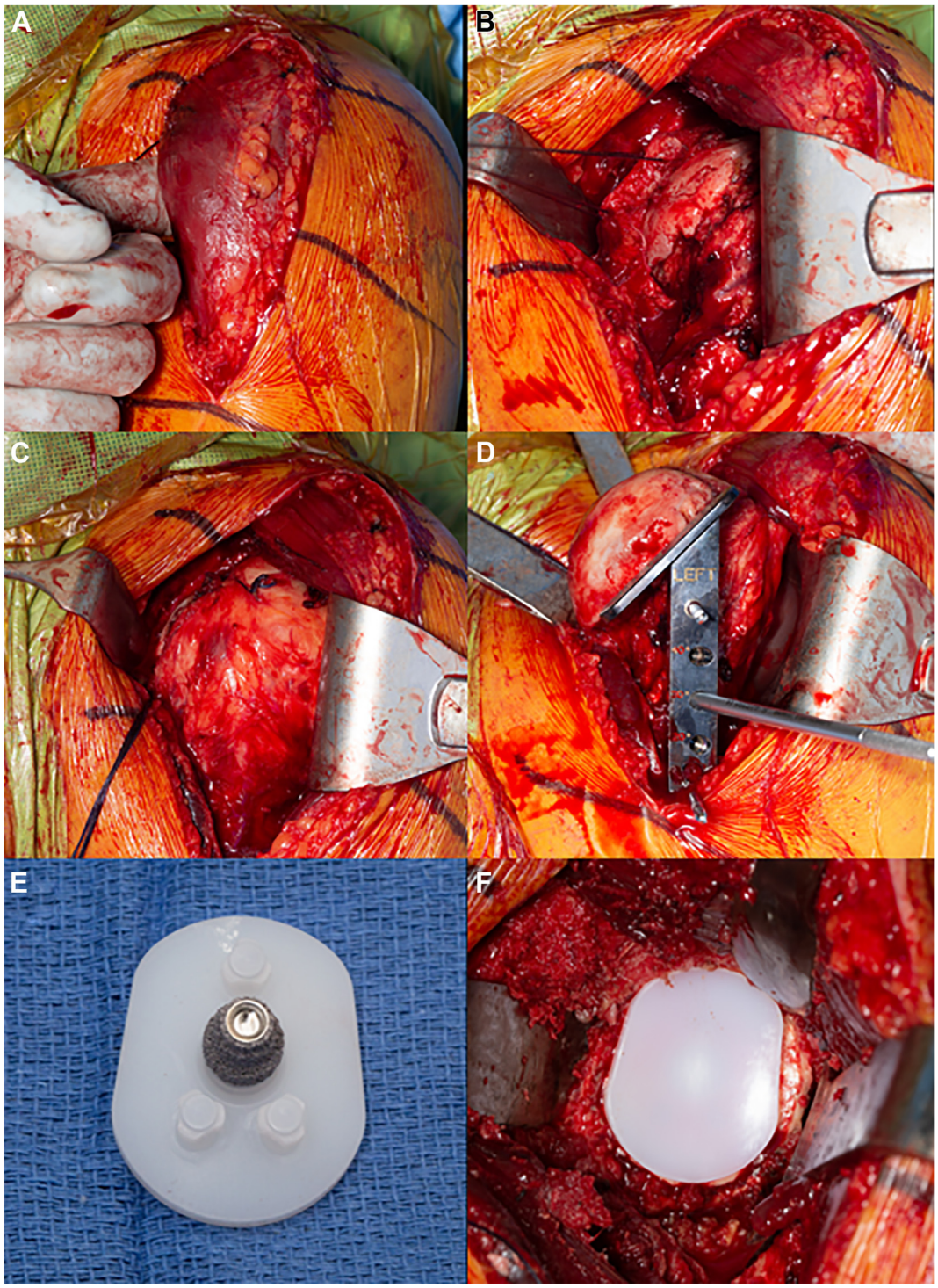

Figure 4 Intraoperative photography at the time of anatomic total shoulder arthroplasty. (**A**) Healthy-appearing deltoid. (**B**) Thick, intact subscapularis tendon. (**C**) Intact rotator cuff. (**D**) Extramedullary humeral cutting guide set to 30° of retroversion. (**E**) Biomet Comprehensive hybrid glenoid component. (**F**) Glenoid exposure with final implant in place.

girdle.[13,15] The condition generally does not result in long-term pain. The initial painful episode typically lasts a few weeks and is followed by chronic weakness.[16] We present a case of a patient with a remote history of PTS who presented with increasing shoulder pain and OA. He subsequently underwent successful anatomic TSA.

The treatment for glenohumeral OA in the setting of PTS is not well-studied. We hypothesize that outcomes following shoulder arthroplasty in patient with PTS may be less favorable than in patients without PTS due to persistent rotator cuff and deltoid weakness as well as overall shoulder dysfunction preoperatively. However, the patient in this report retained relatively good function of his shoulder and was even able to bike several miles per day preoperatively. His shoulder pain increased over time and was beginning to limit his ability to ride his bike and perform other more strenuous activities. His activity level, relatively young age of 68 years, and concern for further irritation of the brachial plexus

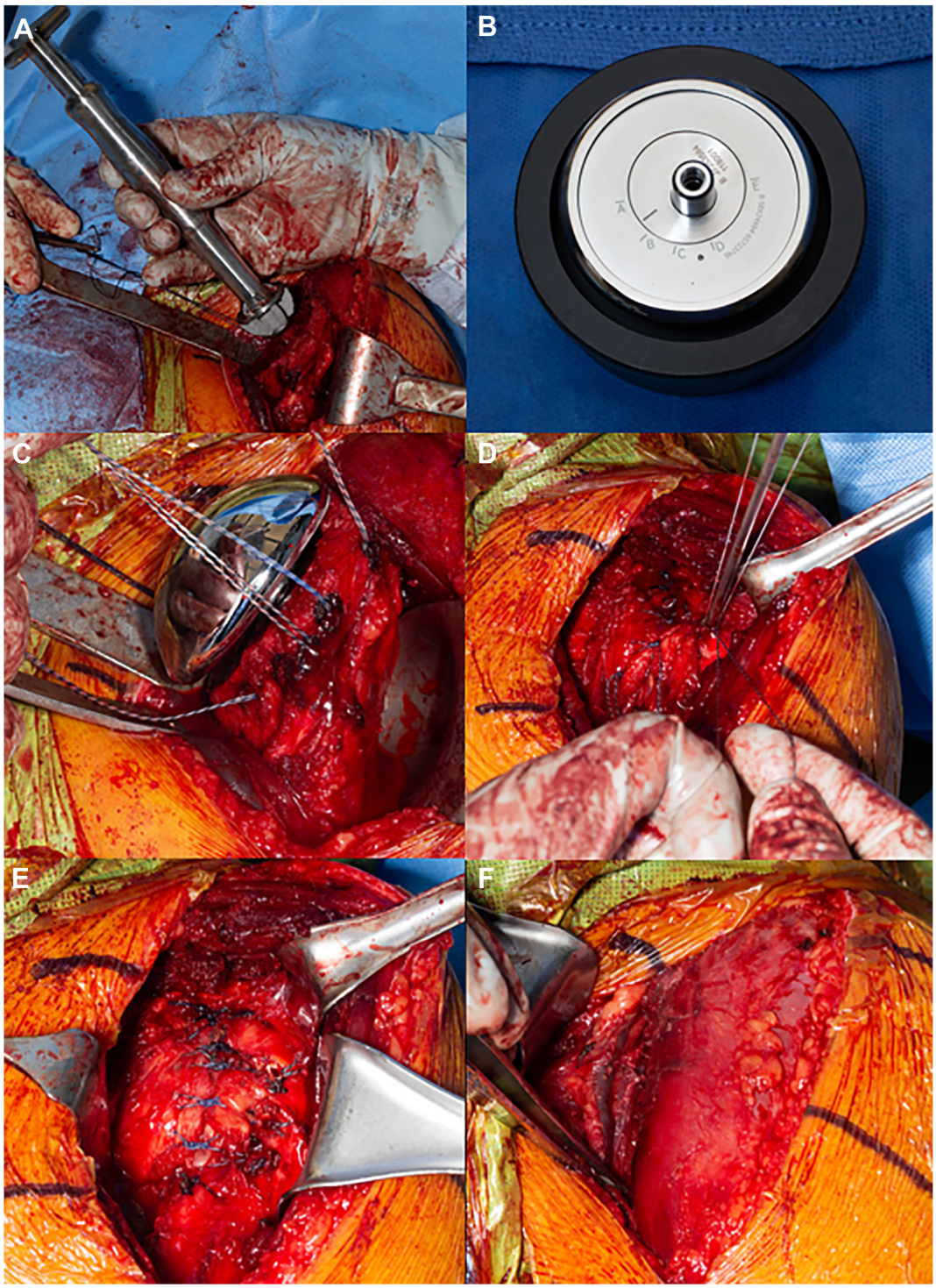

Figure 5 Intraoperative photography at the time of anatomic total shoulder arthroplasty. (**A**) Implantation of the Biomet Comprehensive Nano Stemless Shoulder humeral implant. (**B**) Versa-Dial humeral head. (**C**) Medial row suture anchors in the subscapularis footprint placed prior to relocation. (**D**) Placement of lateral row anchors. (**E**) Double row subscapularis repair. (**F**) Reapproximation of the deltopectoral interval.

due to lengthening after a RSA led to the shared decision of proceeding with an anatomic TSA.

A recent case report described a failed anatomic TSA in a patient with a history of PTS.[18] This patient had preoperative anterior deltoid weakness and had previously undergone a failed nerve transfer. Prior to his TSA, he had significant weakness and atrophy of the anterior deltoid with anterior humeral head subluxation. He subsequently developed pseudoparalysis after TSA with an irreparable supraspinatus and subscapularis tear. He, therefore, was revised to RSA with a pectoralis major transfer. Elhassan et al reported the outcomes of RSA with pectoralis transfer in patient with deltoid paralysis.[5] In 29 patients with a

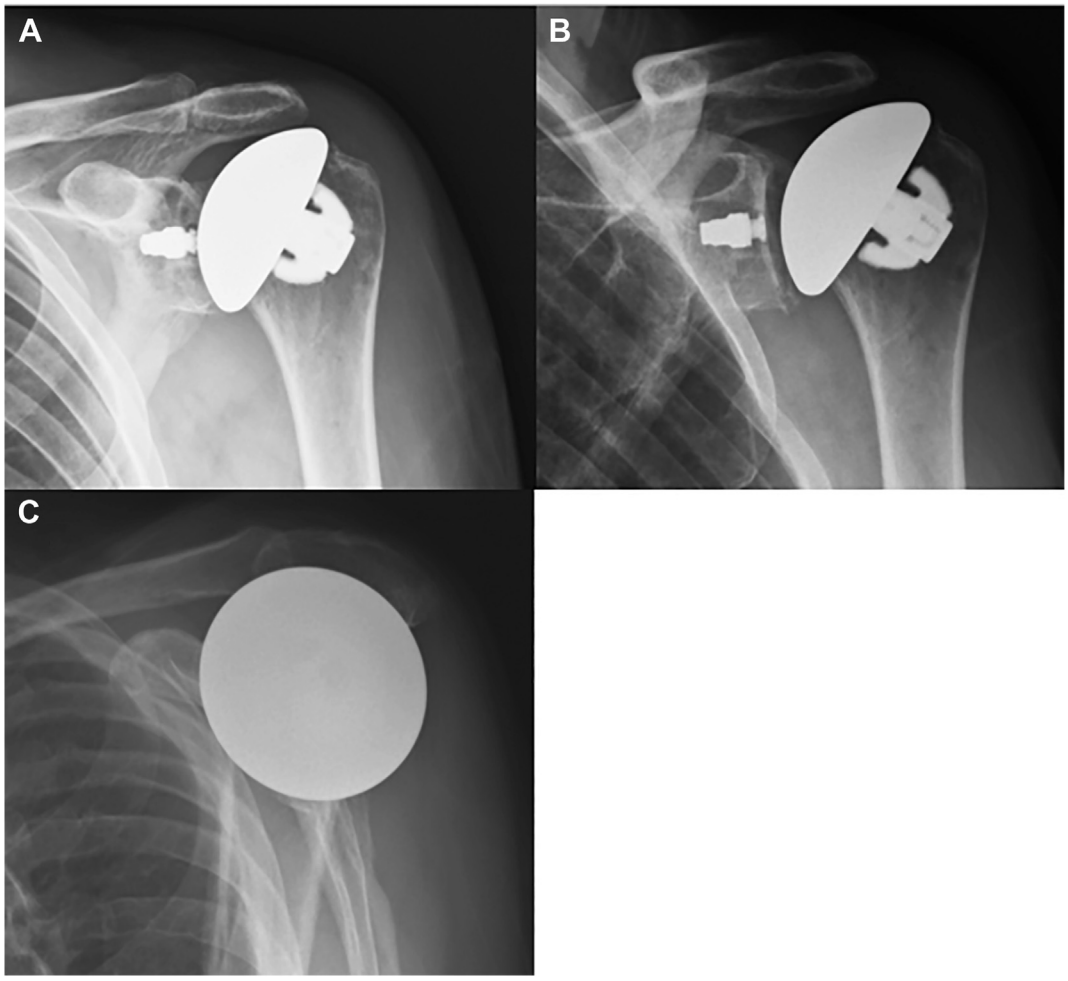

Figure 6 (**A-C**) Radiographs taken 4 weeks postoperatively demonstrating satisfactory implant placement without increased acromiohumeral interval or humeral head subluxation.

mean follow-up of 37 months, these patients had significant improvements in pain, subjective shoulder value, and ROM. Nine patients in this cohort had a prior brachial plexus injury, but it was not specified if any cases were due to PTS. The patient in our case report had relatively preserved deltoid strength without significant deltoid atrophy. He did have moderate rotator cuff weakness on examination, but no significant fatty atrophy or tearing on MRI. Imaging also did not demonstrate significant preoperative humeral head subluxation.

Several studies have analyzed the effect of RSA on the brachial plexus. Ladermann et al reported a mean lengthening of 2.7 cm after RSA compared to the contralateral shoulder.[6] In their study, the risk of postoperative nerve injury was over 10 times higher in the RSA group compared to the TSA group and mostly affected the axillary nerve. Serrano Mateo et al performed RSAs on 20 cadavers and demonstrated significant elongation of the median, musculocutaneous, ulnar, radial, and axillary nerves postoperatively.[14] By contrast, Lowe et al demonstrated a potentially decreased risk of nerve injury in RSA by utilizing a lower neck-shaft angle in conjunction with lateralization of the glenosphere. These modifications decreased the amount of postoperative arm-lengthening in their cohort. This modification could negate the potentially detrimental effects of RSA compared to TSA on the brachial plexus.[7] These studies were performed in patients without known PTS, so the effect of lengthening the brachial plexus in a patient with PTS is not known.

Conclusions

Anatomic TSA remains a good arthroplasty option for younger patients with an intact rotator cuff. The outcomes of TSA in patients with PTS are not known. However, RSA is known to lengthen the brachial plexus and may result in a serious neurologic insult to an already diseased brachial plexus in patients with PTS. We report a case of a successful TSA in a relatively young patient with PTS, glenohumeral arthritis, and an intact rotator cuff. Large cohort studies in this population will be challenging given the low prevalence of PTS, so the choice between TSA and RSA in these patients should be made on a patient-to-patient basis.

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