Shoulder Reanimation with Nerve-Related Procedures in Chronic Parsonage-Turner Syndrome: A Pilot Study

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Abstract

Background Parsonage–Turner syndrome (PTS), also known as brachial neuritis or neuralgic amyotrophy, is an uncommon disorder affecting peripheral nerves of upper extremity, more predominantly those innervating the shoulder. In the majority of patients, paralysis improves with conservative treatment within a few weeks to a few months. Patients with persistent paralysis improve with nerve-related procedures.

Methods We report our experience with nerve-related procedures in chronic PTS. A total of 21 cases of PTS with wasting of the shoulder muscles and restricted movements were evaluated at a tertiary care center between March 2014 and May 2022. According to the involvement of peripheral nerve, patients were divided in three groups; Group A consisted of 15 patients with involvement of both the suprascapular nerve (SSN) and axillary nerve (AXN), Group B had four patients with SSN involvement, and Group C included two patients with isolated AXN involvement. Four patients in Group A also experienced weakness of elbow flexion. Thirteen cases resolved over a period of 3 to 7 months, with minor residual deficits. Five cases with paralysis lasting more than 7 months were treated with nerve decompression, with or without external neurolysis, epineurolysis, and nerve transfer procedures.

Results A total of 21 male patients with an average age of 35 years were assessed. Sixteen patients recovered almost completely over a period of 3 to 7 months. Five patients with persistent paralysis underwent neurosurgical procedures. At the 36-month postoperative review, three patients in Group A had regained active shoulder abduction (range: 140–180 °, average 153°) and external rotation (30–60°, average 43°). A single patient in Group B regained 160° of active shoulder abduction and 50° of external rotation. Nerve transfer failed in the single patient in Group C with isolated involvement of AXN.

Conclusion Nerve-related procedures are viable options in patients with PTS who fail to recover with conservative management. We recommend an observation period of 7 months before considering surgical intervention.

Keywords
- nerve decompression
- nerve transfer
- neurolysis
- Parsonage–Turner syndrome
- shoulder reanimation

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Introduction

PTS, also known as neuralgic amyotrophy, is an idiopathic brachial plexopathy of upper extremity peripheral nerves,[1] affecting most commonly the suprascapular nerve (SSN), axillary nerve (AXN), and anterior interosseous nerves.[2] The musculocutaneous, radial, posterior interosseous, and median nerves are affected less commonly.[3] Rarely the spinal accessory nerve, long thoracic nerve, ulnar nerve, recurrent laryngeal nerve, and phrenic nerve are affected.[2,4] The incidence of PTS has been reported from 2 to 3 per 100,000 per year[5] to a very high estimate of 100 per 100,000 per year.[6] This disorder typically manifests with an abrupt onset of shoulder pain, which may extend to the upper arm, forearm, and hand. The pain usually lasts for 1 to 2 weeks, followed by motor weakness, dysesthesias, and numbness. The etiology of PTS remains unclear. The condition has been reported following trauma, surgery, viral infections, vaccination, antibiotic treatment, and autoimmune conditions.[7,8] Recently it has been reported following COVID-19 infection[9] and COVID-19 vaccination.[10]

Most cases of PTS resolve with conservative management. Nonsteroidal anti-inflammatory drugs (NSAIDs) are commonly used to treat the pain syndrome, which is present invariably in the first few days to weeks of the illness. The role of oral or parenteral corticosteroids in the treatment of brachial neuritis is debatable.[11] A few studies suggest that very early treatment with corticosteroids in some cases may result in prompt pain relief, with no or minimal motor dysfunction in selected patients. Steroids therefore do not affect the progression of the disease. It has been observed that a sizable number of patients with PTS do not improve with conservative regime and develop significant muscle weakness and dysfunction. Neurosurgical procedures can help restore form and function in this subgroup of patients.

Methods

Patients

This study comprised 21 male patients with PTS who presented to a tertiary care referral center between March 2014 and May 2022. All patients offered a history of an abrupt onset of pain in the shoulder with infrequent radiation to the upper arm, forearm, and hand, followed 2 to 3 weeks later by motor weakness.

According to the involvement of peripheral nerves, patients were divided into three groups. Group A consisted of 15 patients with involvement of both the SSN and AXN. Three of them had slight scapular winging. Group B had four patients with SSN involvement, and Group C included two patients with isolated AXN involvement. Four patients in Group A also experienced weakness in elbow flexion.

Diagnosis and Follow-up

The Medical Research Council (MRC) scale was utilized to assess the motor power of the affected muscle(s). All of the patients were subjected to magnetic resonance imaging (MRI) of brachial plexus which invariably revealed thickening and edema of the upper roots, trunk, and suprascapular nerve. Electromyographic (EMG) examinations were performed on the target muscle(s) at the time of the first examination and at 4-week intervals thereafter. Measuring the abduction and external rotation with a goniometer helped in clinically assessing the shoulder function.

All patients were followed for a period of 7 months from the onset of the disease. Patients showing improvement on clinical examination and EMG tests were managed with strengthening exercises. Patients with absent shoulder abduction, progressive muscle wasting, and Electromyography evidence of denervation were subjected to surgical procedures, viz. nerve decompression, neurolysis, and nerve transfers.

Twelve patients in Group A, three in Group B, and one in Group C recovered spontaneously during an observation period of 7 months. Scapular winging improved in all three patients in Group A. Three patients in Group A and one patient each in Groups B and C failed to recover with conservative management and were subjected to neurosurgical procedures.

Nerve-Related Procedures

Decompression of the suprascapular nerve at the suprascapular notch was performed in all patients with SSN involvement. Nerve decompression, neurolysis, and nerve transfer were performed in three patients in Group A (►**Table 1**). A single patient in Group B with hourglass constriction of SSN underwent decompression and external neurolysis under an operating microscope (►**Table 2**). Another single patient in Group C was treated with distal nerve transfer between the long head triceps branch and the AXN (Group C).

Nerve Decompression

SSN decompression in the tight osseo-ligamentous tunnel was achieved by division of the superior transverse scapular ligament (STSL). We preferred an open approach, considering it safe and reproducible. With the patient in semi-lateral position, a transverse incision was made parallel to and just above the scapular spine and continued toward the tip of the shoulder. The trapezius muscle was elevated from the scapular spine, and the space between the trapezius and the supraspinatus muscle was defined. The supraspinatus muscle was retracted downward from the upper border of scapula, revealing the glistening white STSL bridging the suprascapular notch. Suprascapular vessels coursing superficial to the ligament were safeguarded. The SSN, encased in a sleeve of fibrous connective tissue, was isolated within the fatty tissue just proximal to the suprascapular notch. The STSL was divided using a No. 15 scalpel blade on a long-handled knife. A current of 2 mA was delivered with a nerve stimulator to stimulate supraspinatus muscle. Occurrence of convincing contractions confirmed the completeness of decompression and the viability of the supraspinatus muscle.

Table 1 Surgical procedures and functional outcomes in Group A ($N = 6$)

Case no	Age (y)	Interval between pain and palsy (d)	Duration of palsy (mo)	Surgical procedure	Shoulder abduction (°)	External rotation (°)
1	35	19	7 mo	Decompression of SSN LHT branch to anterior branch of AXN	180	60
2	36	17	9 mo	Decompression and microneurolysis of SSN LHT branch to anterior branch of AXN	140	30
3	40	19	9 mo	Decompression of SSN SAN to SSN transfer LHT branch to anterior branch of AXN	140	40

Abbreviations: AXN, axillary nerve; LHT, long head triceps; SAN, spinal accessory nerve; SSN, suprascapular nerve.

The trapezius muscle was sutured back to the spine of scapula using a 3–0 polyglactin suture. Finally, the skin incision was closed with a drain placed in the subcutaneous plane.

Neurolysis

Decompression followed by neurolysis of the SSN was indicated when the nerve anatomy was distorted by constrictions or fibrous bands. Two cases required epineurolysis and perineurolysis in the distal part of the SSN. One of the them (case no 2, ►**Table 1**) had kinking of SSN by a fibrous band proximal to the notch. Division of the fibrous band coupled with an epineurolysis, restored the normal anatomy of the nerve.

Nerve Transfers

Patients with progressive muscle wasting, lack of movement, and fibrillation potentials in EMG, were treated with distal nerve transfers, viz. distal spinal accessory nerve (SAN) to SSN and/or long head triceps (LHT) branch of the radial nerve to the anterior branch of the AXN.

Distal SAN to SSN Transfer

The distal part of the SAN was reached through an incision over the scapular spine, as described previously. SAN, identified on the undersurface of the trapezius, was cautiously isolated from the thin vessels. The nerve was then traced distally along the medial border of scapula and sectioned with sharp scissor. The SSN was isolated within the fatty tissue just proximal to the suprascapular notch, and divided. The SAN was sutured to the distal segment of the SSN using 10–0 nylon suture under an operating microscope.

LHT Branch into AXN Transfer

With the patient in the supine position, an incision was made along the posterolateral aspect of the arm, with the upper part of incision curving along the posterior border of the deltoid, and the lower part centered over the triangular space between the long and lateral heads of the triceps muscle. LHT branch of radial nerve was identified along the lower border of teres major muscle. This branch was sectioned close to the triceps muscle. AXN with its anterior and posterior branches was exposed in the quadrangular space. The anterior branch was transected proximally and sutured to the LHT branch with 10–0 nylon suture under an operating microscope.

Postoperative Care and Assessment

Postoperatively, the operated arm was strapped to the chest for a period of 3 weeks, following which patients were offered stretching exercises. Beginning from the 20th postoperative week, monthly EMG examinations were performed on the target muscles. Abduction and external rotation were measured with a goniometer to clinically assess shoulder function. Abduction was recorded by measuring the angle formed between the arm axis and parallel to

Table 2 Surgical procedures and functional outcomes in Group B ($N = 1$)

Case no	Age (y)	Interval between pain and palsy (d)	Duration of palsy (mo)	Surgical procedure	Shoulder abduction (°)	External rotation (°)
1	39	21	7 mo	Decompression and external neurolysis of SSN	160	50

Abbreviation: SSN, suprascapular nerve.

Table 3 Surgical procedures and functional outcomes in Group C ($N = 1$)

Case no	Age (y)	Interval between pain and palsy (d)	Duration of palsy (mo)	Surgical procedure	Shoulder abduction (°)	External rotation (°)
1	36	18	11 mo	LHT branch to anterior branch of AXN transfer	80	60

Abbreviations: AXN, axillary nerve; LHT, long head triceps.

the spinal cord axis. External rotation was measured with the patient standing, the shoulder fully internally rotated, the elbow flexed, and the forearm positioned transversely over the abdomen. Any rotation from this position was measured and recorded as the range of external rotation.

Results

All patients were males, with an average age of 35 years (range 25–41 years). Sixteen patients recovered over a period of 3 to 7 months, with some residual deficits. Five of the patients with progressive or persistent weakness required surgical intervention. When reviewed 36 months after surgery, three patients in Group A had restored active shoulder abduction (range 140–180°, average 153°) and external rotation (30–60°, average 43°) (►**Table 1**). A single patient in Group B restored 160° of active shoulder abduction and 50° of external rotation (►**Table 2**). Nerve transfer failed in a single patient in Group C with isolated involvement of the AXN (►**Table 3**). Preoperative shoulder abduction, operative procedures, and clinical recovery following nerve-related procedures in case no. 1 (►**Table 1**) are depicted in ►**Figs. 1** to **6**.

Discussion

A majority of patients with Parsonage–Turner syndrome (PTS) improves with conservative treatment using non-steroid anti-inflammatory drugs, physical therapy, and rehabilitative exercises. In selected cases, steroids might alleviate pain in the acute stage, but do not affect disease progression. Functional recovery, partial to near complete, is usually apparent within a few weeks to a few months of time. However, in a small number of patients, paralysis worsens with the passage of time and most typically involves the shoulder girdle and upper arm musculature. Muscle weakness may go unnoticed at first, until atrophy of the affected muscles progresses to subluxation of the shoulder joint. Development of adhesive capsulitis and muscle contractures can severely restrict joint functions. Though

Fig. 1 Severely restricted shoulder abduction.

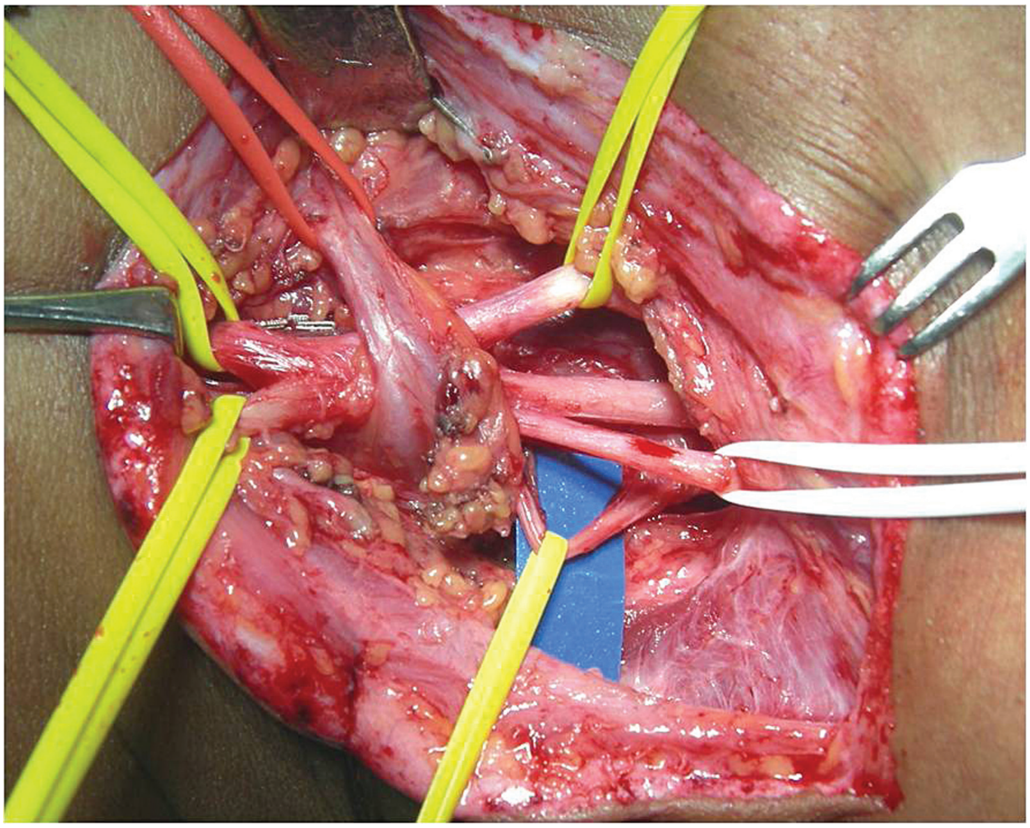

Fig. 2 Enlarged and swollen C5, C6 nerve roots, upper trunk, anterior and posterior divisions and suprascapular nerve.

shoulder involvement is commonest, a few studies indicate a predomination of palsies involving more peripheral nerves like anterior and posterior interosseous nerves.[5]

Despite extensive documentation of this entity as early as 1948 by Parsonage and Turner, there has not been much change in the management protocol. With conservative treatment, the majority of patients do recover, but a few continue to suffer from pain and paralysis due to a long standing inflammation and its consequences, such as post-inflammatory adhesions and constrictions affecting peripheral nerves. van Alfen et al, in a series of 246 cases, reported that the majority of patients exhibited persisting functional deficits with an average follow-up of more than 6 years. In another study, Misamore et al noticed some residual deficit in three of seven patients at long-term follow-up.[12] Based on our experience and in consideration of these studies, we also recommend an early surgical exploration in cases of severe nerve involvement. We are of the opinion that nerve constrictions and torsions from adhesions and fibrous bands are unlikely to resolve with conservative management.

We also hypothesize that an inflamed nerve is more likely to get compressed where it courses through a narrow tunnel, e.g., suprascapular nerve passing through the notch beneath the STSL. In these circumstances, decompression of the tunnel by division of the ligament is most likely to reverse the paralysis, as noted with the release of median nerve in carpal tunnel syndrome. In the present study, suprascapular nerve decompression was performed as a primary procedure in four of five cases.

The presence of hourglass constrictions of peripheral nerves in PTS has recently been well documented.[13] Pan et al,[14] in a small series of five cases, proposed these constrictions as an explanation for failure to recover from this syndrome. A recent report by Krishnan et al[3] highlights the utility of microneurolysis in hourglass constrictions of peripheral nerves in chronic neuralgic amyotrophy. Patients who underwent microsurgical epineurolysis and perineurolysis of hourglass constrictions performed better compared with nonsurgical management. In our study, neurolysis of SSN in two patients yielded good functional outcomes in shoulder functions. In dealing with more severe constrictions of peripheral nerves, Pan et al[14] suggested resection of the diseased segment followed by direct nerve repair or nerve grafting. Of five nerves treated with resection and direct repair or nerve graft, two obtained full recovery, and three had partial recovery.

Instead of nerve grafts, we favor distal nerve transfers when dealing with diffuse segmental fibrosis or severe hourglass constrictions of the suprascapular and AXNs. In the present report of four patients, all patients had satisfactory recovery following distal nerve transfer, except a single patient in Group C having isolated AXN involvement. A long denervation period could be the reason for failure of nerve transfer. Our recommendation for nerve transfers, therefore, is at 7 months after the onset of the disease.

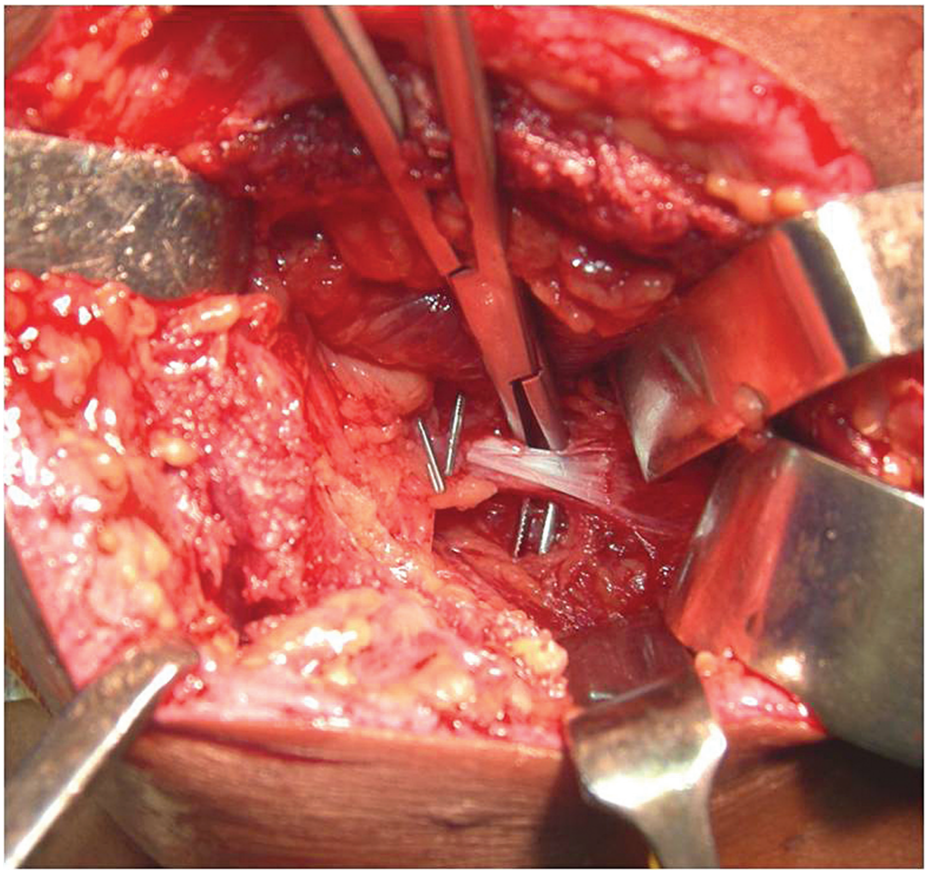

Fig. 3 Thickened and hypertrophic suprascapular ligament.

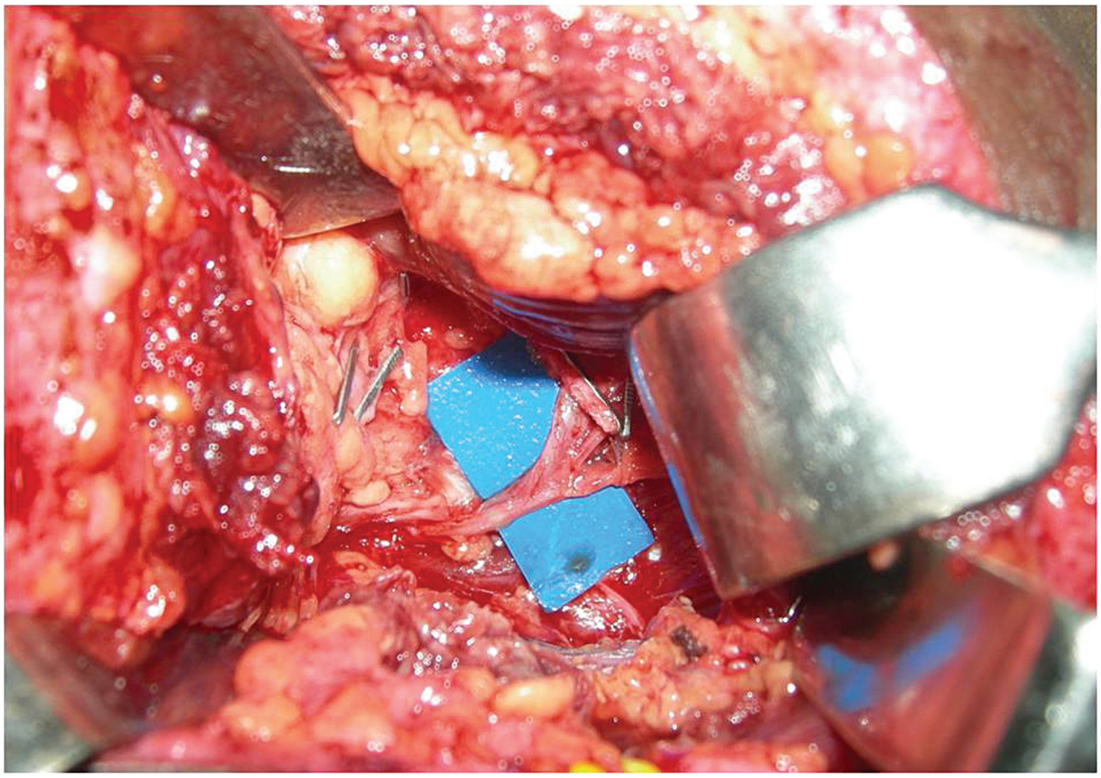

Fig. 4 Suprascapular nerve following decompression.

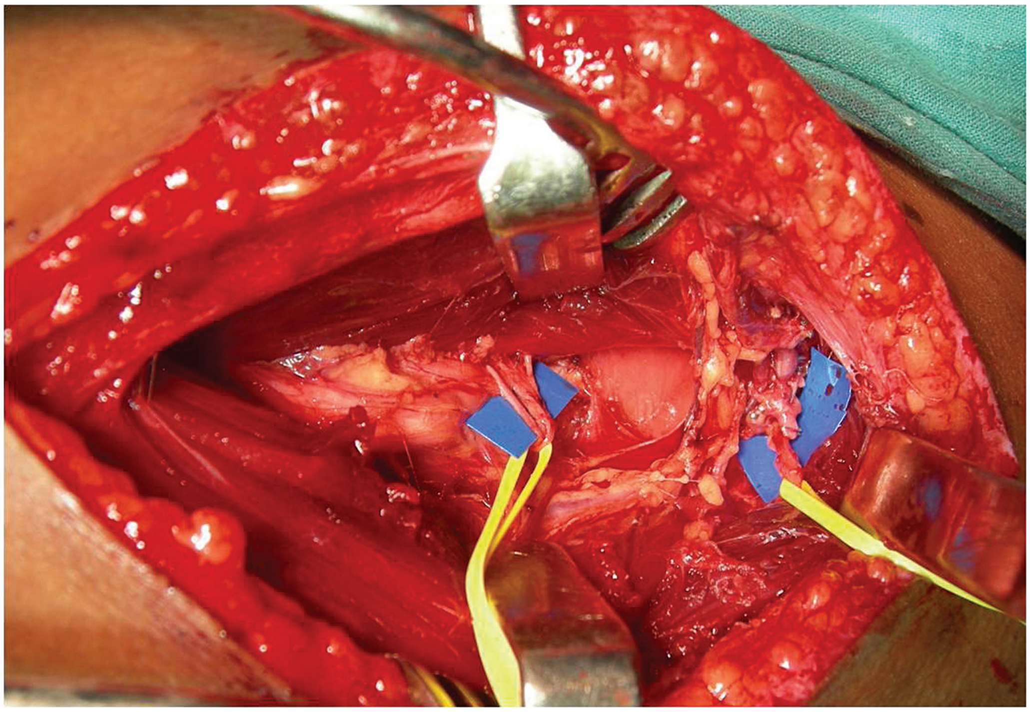

Fig. 5 Donor and recipient nerves for deltoid reinnervation.

Fig. 6 Improved shoulder abduction 36 months after nerve-related procedures.

Conclusion

We recommend nerve procedures in patients with PTS who fail to recover with conservative treatment. However, it is difficult to draw any firm conclusions on the basis of small sample size and the nonrandomized, retrospective design.

Conflict of Interest
None declared.

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