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ELECTRODIAGNOSIS CORNER/REVIEW ARTICLE

Parsonage-Turner Syndrome

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Abstract Parsonage-Turner Syndrome (PTS), also referred to as idiopathic brachial plexopathy or neuralgic amyotrophy, is a rare disorder consisting of a complex constellation of symptoms with abrupt onset of shoulder pain, usually unilaterally, followed by progressive neurologic deficits of motor weakness, dysesthesias, and numbness. Although the etiology of the syndrome is unclear, it is reported in various clinical situations, including postoperatively, postinfectious, posttraumatic, and postvaccination. The identification of the syndrome in the postoperative patient remains a challenge as symptoms may easily be attributed to sequelae of surgical positioning, postoperative recovery, or postanesthetic block pain. The purpose of this review is to bring forth salient, identifiable factors which may assist the surgical clinician in identifying the condition sooner. An early and proper diagnosis affords the opportunity to treat the patient accordingly and to the satisfaction of both surgeon and patient.

Keywords Parsonage-Turner Syndrome · brachial neuritis, brachial amyotrophy

Introduction

Parsonage-Turner Syndrome (PTS) is a rare syndrome that may occur in otherwise normal healthy individuals with sudden, rather abrupt, unilateral shoulder pain that may begin rather insidiously but quickly amplifies in severity and intensity. The acute period of pain is subsequently replaced over a course of a few days to weeks with progressive weakness, reflex changes, and sensory abnormalities in varying presentations that typically involve the shoulder girdle musculature and proximal upper limb muscles. The condition, also known as neuralgic amyotrophy or brachial neuritis, has been reported in numerous clinical situations that involve some sort of antecedent impact on the patient, whether it be surgical, infectious, traumatic, or even therapeutic, such as cases involving vaccinations or antibiotic treatments. The postsurgical patient who develops PTS poses a specific diagnostic dilemma. In these individuals, the patient has undergone a rather traumatic experience to the body considering perioperative administration of antibiotics, intraoperative anesthesia, operative manipulation of tissue, and various positioning techniques used to facilitate surgical techniques. It is no mystery that, when this cohort of patients develops PTS, it is often attributed to intraoperative positioning or even part of the rehabilitative process manifested as shoulder strain with therapy. The purpose, therefore, is to illuminate the subtle clinical signs and symptoms of PTS when they first present to help both clinician and patient in better understanding the implications of the diagnosis and its potential impact in postsurgical care.

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Overview, etiology, and incidence

Idiopathic brachial plexopathy was reported in the Lancet in 1948 by Parsonage and Turner [61]. The condition, subsequently coined Parsonage-Turner Syndrome, had been previously described in the literature as far back as 1897 with many similar clinical presentations of the syndrome reported prior to the extensive study of the syndrome by Parsonage and Turner [2, 19, 23, 27, 79].

The classic description of PTS is a condition in which the patient initially and suddenly develops constant, severe

unilateral shoulder girdle pain [61]. This pain may extend to the trapezius ridge, upper arm, forearm, and hand [14]. The pain is not positional in nature and usually worse at night and may be associated with awakenings from sleep. There are typically no constitutional symptoms associated with the syndrome [47, 61, 89]. The duration of pain is almost always self-limiting, lasting 1 to 2 weeks, but on rare occasion persisting for longer periods of time [70, 75, 78]. Although not present initially, weakness may develop a few days to weeks after the initial onset of symptoms. Sensory deficits are known to occur but vary in prevalence with one study reporting deficits in 66% of patients [15] while another found them in a minority of patients [88]. The sudden loss of strength may not be recognized immediately because of the patient's reluctance to utilize the affected area due to the debilitating nature of the pain. Weakness may also not be recognized if it involves muscles that are difficult to manually isolate, such as the serratus anterior or, to some extent, the supraspinatus muscle. Within a month, the weakness is usually fairly obvious and atrophic changes then become recognized. Scapular winging may be seen in cases where there is involvement of the scapulothoracic nerve innervating the serratus anterior muscle, and this is not always immediately recognized, especially when the patient is not appropriately disrobed and thoroughly examined. Although PTS is a diagnosis partly based on the appearance of specific clinical findings, some of the classical symptoms, including constant incapacitating pain of 1–2-week duration, may not always be present [70]. This can make the diagnosis elusive at times and delay an appropriate targeted treatment regimen for PTS. PTS, therefore, is a diagnosis that should always be entertained in any patient who presents with acute shoulder girdle pain.

The pathophysiologic events which result in PTS have, as yet, not been fully described but various risk factors and antecedent events can be identified in 28–83% of presenting patients [9, 18, 30, 45, 47, 52, 53, 61, 71, 78, 79, 85, 86, 88]. Table 1 summarizes several of the etiological associations found with Parsonage-Turner Syndrome.

Several risk factors associated with the development of PTS have been suggested. The most common associated risk factor is a recent viral illness. One study reported 25% of patients had a recent viral illness before the appearance of PTS symptoms [30]. Recent immunization accounts for the second most common risk factor with cases reported in 15% of individuals who developed PTS [30]. Therefore, two competing theories in the development of PTS in the most common cases of PTS are attributed to either a viral illness directly involving the brachial plexus or an autoimmune response to the viral infection or to the viral antigen in the immunization. There appears to be an association between certain diseases and the pathophysiologic development of PTS. Since systemic lupus erythematous, temporal arteritis, and polyarteritis nodosa all have a vasculitic component to their disorders, a vascular etiology is proposed as a cause of PTS in these conditions. There are also theories regarding the symptoms of pain of PTS being related to ischemia. Ischemia, whether it is inflammatory or mechanical [25, 37, 38, 58, 59], might be one explanation for the sudden onset of severe pain. Nerve biopsies in postsurgical patients show evidence that ischemic changes demonstrated by perineural thickening, neovascularization, and focal fiber loss in PTS may have an immune pathogenesis [73].

Table 1 Conditions associated with the development of Parsonage-Turner Syndrome

Infection
Bacterial
Parasitic
Viral
Surgery
Anesthesia
Hereditary [40, 63, 77]
Rheumatic disease
Connective tissue disorders (i.e., Ehlers-Danlos Syndrome) [17, 43]
Systemic lupus erythematosus [6]
Temporal arteritis [67]
Polyarteritis nodosa [1]
Trauma
Remote from the shoulder girdle
Stressful exercise
Immunizations [90]
Tetanus toxoid and antitoxin
Diphtheria, pertussis, tetanus (DPT) vaccine
Smallpox
Swine flu
Other
Pregnancy and childbirth
Miscellaneous conditions [62]
Radiation therapy
Lumbar puncture
Pneumoencephalogram
Radiologic contrast dye administration
Allergy desensitization [92]

In rare cases without an antecedent event, patients who have developed PTS often struggle to try to identify a specific event or physical trauma which may serve as a trigger of their symptoms. These reported events, usually mild in nature, often seem inconsequential to the treating physician. Nevertheless, potential antecedent biomechanical trauma should always be considered as part of a thorough history and physical to exclude other areas or causes of injury.

PTS occurs with an overall reported incidence of 1.64 cases per 100,000 people [4]. The true incidence may be higher as a result of underreporting due to the difficult nature of making the appropriate diagnosis in challenging clinical presentations. Sexual dimorphism may also apply to PTS syndrome. Overall, there appears to be a higher incidence in men than in women. Reports vary from a slightly increased ratio of 11 to 9 [51] up to reports of 11.5 to 1 [47]. There does not appear to be a prevalence for hand dominance nor is there a general trend towards development of the condition more on the left versus right [29, 47]. Patients affected range from 3 months of age [78] to 75 years [41]. However, the highest incidence of PTS occurs in individuals between the third and seventh decades [78].

Association with surgical procedures

The association of Parsonage-Turner Syndrome with surgical procedures has been reported extensively [30, 48, 53]. In these cases, PTS typically occurs in the perioperative timeframe during acute postoperative recovery and, in some cases, rehabilitation. The surgeries reported include a variety of orthopedic procedures, coronary artery bypass surgery, hysteroscopy, and oral surgery [3, 12, 16, 24, 30, 39, 48, 69]. Several mechanisms have been postulated for the onset of this condition in such situations. The two leading theories regarding the etiology of postsurgical PTS include traction injury to the brachial plexus resulting from improper positioning and immune-mediated inflammation of the brachial plexus. It seems reasonable that PTS can stem from inappropriate position, traction, or pressure injuries during operative procedures. In coronary artery bypass surgery, sternal traction has been postulated to result in brachial plexus compression. Injury to the brachial plexus can also occur in surgical situations when the arms are overabducted in a prone or supine position. However, this theory of injury seems unlikely in the face of mounting evidence that PTS also occurs in surgical procedures that involve little to no traction or compression injury to the brachial plexus [24, 39, 48].

The development of PTS symptoms following surgery can be quite variable occurring within 24 h of the procedure or up to a week or more following surgery. Although postsurgical neurological changes can be iatrogenic, there are many cases where there appears to be no causal relationship to the surgical technique or anesthesia used. This is especially obvious in cases where there is isolated and complete involvement of one of the following nerves or a combination of them: the suprascapular nerve, the long thoracic nerve, the anterior interosseous, the axillary nerve, or the phrenic nerve. The unique feature here is that the nerves involved, when there is more than one, do not have a common innervation pattern. That is, the nerves vary in the originating root level or brachial plexus trunk or cord distribution. Furthermore, there may be other nerves, which do have the same root level or plexus innervation pattern as the nerve involved but are completely normal. This distinction is key in examining the electrodiagnostic characteristics (i.e., degree of denervation and denervation pattern) following injury which can help determine whether there has been a true neurological postoperative complication or development of a postsurgical Parsonage-Turner Syndrome. A greater awareness of postsurgical Parsonage-Turner Syndrome can help alleviate the fears and concerns of the patient of a surgical complication and also help avoid unnecessary intervention by surgeons. Early recognition of PTS, taking steps for an accurate and early diagnosis, and close follow-up care can best insure a satisfactory outcome for both patient and surgeon.

The differential diagnosis in patients presenting with severe, unilateral shoulder pain should also include several similar presenting conditions. Cervical disk herniations or foraminal stenosis causing a cervical radiculopathy or mass lesions compressing the brachial plexus or individual nerves are a few examples. In some of these cases, PTS becomes a diagnosis of exclusion. This is especially true in cases where a magnetic resonance imaging (MRI) of the cervical spine reveals foraminal stenosis or a small cervical intervertebral disk protrusion that does not appear to be clinically significant but still corresponds to the level of involvement [7]. In these cases, it can be difficult to clearly determine whether the pathology identified on imaging studies is a major contributing factor and whether further treatment options such as epidural injections and/or surgical decompression should be considered. While cervical radiculopathy is probably the most commonly considered clinical diagnosis, conditions such as postherpetic neuralgia, calcific tendonitis, acute subacromial bursitis, and adhesive capsulitis can all present initially and acutely with similar symptoms. Other causes of brachial plexopathies such as thoracic outlet syndrome may present with less acute and severe pain and should also be entertained [33, 36, 54, 55, 82]. The classic skin lesion associated with shingles will usually be identified in cases of postherpetic neuralgia, making this diagnosis fairly obvious; however, cases of shingles can present without rash, making the diagnosis in these cases more elusive [28]. Patients with cervical radiculopathies will commonly have a positive Spurling maneuver [72]. However, this maneuver is typically negative in patients with PTS. Symptoms that are secondary to calcific tendonitis or an acute subacromial bursitis will be aggravated with shoulder motion, particularly impingement-like maneuvers, and these patients can usually find positions of greater comfort. Cortisone injections (or even a diagnostic lidocaine injection) may help establish a definitive diagnosis and also provide immediate relief.

Several similarities exist between Parsonage-Turner Syndrome and adhesive capsulitis. Both conditions present with severe pain, worse at night, and, initially, are unremitting regardless of position. Both conditions are idiopathic, have a nonspecific inflammatory component, and will resolve spontaneously with a relatively good long-term prognosis for recovery of function [60, 63]. However, one major difference exists. Patients with PTS, unlike patients with adhesive capsulitis, do not experience the significant loss of glenohumeral range of motion. In fact, glenohumeral motion is usually completely preserved in PTS. This may largely be the result of the different structures involved (capsule vs. nerve) and not a reflection of the etiologic factors that cause the conditions. The unique relationship of the shoulder girdle region to both disorders may raise the question of whether anatomic proximity of the thorax/chest wall/lung plays a role. There are no current reports of a patient presenting with simultaneous PTS and adhesive capsulitis.

PTS generally involves one upper limb, but bilateral involvement has been reported and, in some cases, abnormal electromyograph (EMG) changes can be identified in the contralateral asymptomatic limb. The upper trunk of the brachial plexus, the suprascapular nerve, the long thoracic nerve, and the axillary nerves are the most commonly involved [32, 47, 56, 64, 72, 81]. Frequently reported, but less common, are the anterior interosseous,

musculocutaneous, and spinal accessory nerves [5, 15, 32, 42, 50, 57, 64, 83, 84, 93]. Least commonly involved are the ulnar, radial, and median nerves [61, 74, 76, 78]. There is also rare involvement of the middle and lower trunks [78]. Because the paraspinal muscles are usually spared, root-level involvement is felt to be rare [13, 50]. Phrenic nerve involvement has also been reported [10, 35, 46, 85]. These patients present with shortness of breath and will have an elevated hemidiaphragm on plain chest X-rays. Reports of lumbosacral plexus involvement exist, but most of these patients have underlying medical illnesses, suggesting that there may be other contributing factors [11, 26, 50, 68]. The lateral antebrachial cutaneous (LAC) sensory nerve is affected in 32% of cases [21]. One unique and often diagnostic feature of PTS is that involvement may not follow a classic nerve or plexus pattern. One can see involvement of the suprascapular and long thoracic nerves while sparing other nerves and muscles (i.e., deltoid, biceps) that may share the same root or plexus level.

Electromyographic study of PTS

Parsonage-Turner Syndrome is believed to be an axonal process, and the diagnosis is therefore very dependent on the EMG (needle) portion of the electrodiagnostic exam [51, 88]. Widespread denervation is usually seen in involved muscles, and complete denervation is often the case. Because of the often atypical distribution, electromyographers need to be extremely detail-oriented in the systematic approach to testing specific muscles of the upper limb. Testing of muscles that are not routinely examined during EMG studies should be interrogated by needle EMG even when they appear to be clinically asymptomatic. A thorough physical exam with extensive manual muscle testing is essential before performing a complete EMG exam. Since PTS is an axonal disorder commonly affecting proximal muscles of the upper limb, the motor and sensory nerve conduction velocities and distal latencies routinely tested on the distal upper limb are usually normal. Because of the common involvement of the LAC, one should consider testing of the LAC sensory nerve bilaterally for comparison [21]. When there is a significant asymmetry (greater than 50% drop) in the involved LAC sensory amplitude, one should consider testing of the superficial radial sensory amplitudes to help rule out a brachial plexopathy from other causes, such as mass lesions in the supraclavicular fossa or other possible pathologies [31, 91].

Prognosis for functional recovery is usually good in most cases [80]. One of the largest natural history studies by Tsairis found that 89% of patients had fully functional recovery at 3 years [78]. However, this study based recovery strictly on functional strength and did not look at EMG findings to determine whether there was complete muscle reinnervation. It is therefore possible that functional recovery was secondary to compensatory changes and not necessarily due to full EMG recovery.

While no large natural history studies on recovery of PTS based on EMG findings exist, reinnervation usually begins to occur somewhere between 6 months and 1 year based on this author's preliminary findings of a current study. This time, variable is based on the fact that axonal regeneration and eventual muscle reinnervation are length-dependent processes. The rate of axonal regeneration is classically described as occurring at a rate of 1–4 mm/day. So reinnervation of the supraspinatus following a suprascapular neuropathy will occur before innervation of the infraspinatus. It will also more commonly occur earlier than reinnervation, following a long thoracic neuropathy or anterior interosseous neuropathy due to the length-dependent recovery of those conditions.

Although PTS is a clinical diagnosis, EMG is a form of diagnostic testing that can better identify, isolate, and grade severity of denervation and, after a period of time, reinnervation of the involved muscles. Imaging studies (usually an MRI) are often indicated to rule out a mass lesion (i.e., tumor, ganglion cyst, disk herniation) and may show a nonspecific inflammatory response in the brachial plexus in conditions of PTS. Once other causes have been ruled out, a treatment plan can be outlined.

Treatment

In the earliest stages of this condition (the first few weeks), pain management with opiates, NSAIDS, and neuroleptics is the mainstay of treatment [49, 56, 87]. Acupuncture and transcutaneous electrical nerve stimulation (TENS) can also be adjuncts to medications. Oral steroids have been recommended by some, but there is poor literature evidence to support its efficacy [51]. Oral prednisone given the first month after onset may shorten duration of symptoms based on one study, but further studies need to be performed to establish efficacy of treatment with corticosteroids or other immune-modulating therapies [83]. A recent study of postsurgical patients who developed peripheral neuropathies was performed at the Mayo Clinic. Immunotherapy was used as a form of treatment. All eight patients who were treated had meaningful improvement of neurological symptoms and impairments [73].

In cases where imaging studies demonstrate cervical spine pathology that is suggestive of nerve root-level compression, a cervical epidural may be helpful to distinguish between pain due to a cervical radiculopathy and PTS. Antiviral medications should be considered in cases presenting with a classic shingles rash or when postherpetic neuralgia is otherwise suspected [8, 14, 20, 22, 28, 34, 44, 65, 66, 81].

Physical therapy plays an important role in the treatment of this condition. Modalities such as TENS can help in pain management. Acupuncture may be helpful, but positive results here are mostly anecdotal. A more aggressive therapeutic treatment plan can be instituted once the painful stage has abated. The timing and the role of strengthening exercises are dependent on the degree of muscle denervation, the degree of weakness, and the degree of altered biomechanics and to the premorbid functional level of activity for that patient. Range of motion exercises, which

are clearly important and dependent on the muscles involved, may require the assistance of a physical or occupational therapist. Strengthening exercises are not indicated in completely denervated muscles for obvious reasons, and the role of electrical stimulation is controversial but should be considered especially when the denervated state is prolonged, i.e., greater than 4 months. Any degree of exercise can overload injured muscles in their early reinnervated state. In these cases, axonal regeneration and muscle reinnervation may be retarded. Limited EMG follow-up testing of the involved muscles can be used to demonstrate the extent of reinnervation. This information can be useful in helping determine when muscles can tolerate more aggressive strengthening. Observing functional motion and carefully scrutinizing the biomechanics and the flow of motion can help one determine what type of exercises are appropriate and what levels of aggressive training should be implemented. Sound biomechanics should be established before high-level athletes begin to resume more rigorous and higher-level activities. This is particularly true for the overhead athlete. Initiating strengthening of the shoulder girdle when muscle denervation exists can exacerbate the varying muscle strength imbalances that already exist. This not only alters the biomechanics of the shoulder but also establishes a dysfunctional pattern of motion that may lead to further injury of the denervated muscle from overstretching and eccentric overload.

Conclusion

PTS is a condition that usually presents suddenly with disabling pain and is often difficult to diagnose in its acute state. The pain is generally self-limiting, and the natural history of PTS leads to a generally favorable functional recovery. Early diagnosis with a good clinical evaluation, electrodiagnostics, and relevant imaging can allow for aggressive short-term pain management, help outline proper therapy, and provide comfort to both patient and physician in establishing a diagnosis. Proper diagnosis can also avoid unnecessary additional testing or surgical exploration. Limited follow-up EMG testing can be useful in demonstrating early electrodiagnostic signs of recovery which can precede clinical evidence of recovery. This finding can help alleviate the patient's psychological concerns about their disability and functional recovery and also provide the treating medical team with information regarding the patient's prognosis and, ultimately, recovery.

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