

Colorado Hand Therapy, LLC Case Study Follow Up



Parsonage-Turner Syndrome

The April Clinical Education Meeting at Colorado Hand Therapy consisted of a case study presentation by Tara Haas. Clinically, the case was very complex, and after several months of multiple physician visits, studies, and therapy visits, it was discovered that the patient had a rare condition known as Parsonage-Turner Syndrome.

Parsonage-Turner Syndrome is a rare, neurological condition predominantly seen in males. Those with Parsonage-Turner Syndrome have a characteristic presentation of abrupt and severe onset of shoulder pain. After days to weeks, the acute pain is replaced with a dull ache. Weakness ranging in report from mild to profound, typically occurs after resolution of the initial pain. Sensory disturbances are often reported as well. The cause is largely unknown with varying reports in the literature, though it has been linked to viral illness, has been seen after vaccine administration, and has also occurred in individuals who have had an injury or surgery at a site distal to the shoulder. The literature is very contradictory when reporting outcomes. Some sources report frequent, full recovery, while others report that the majority of their subjects continue with pain and paresthesia at long term follow up.

The patient with Parsonage-Turner Syndrome seen at CHT had sustained a digital nerve laceration of his left small finger. His first surgery for the nerve repair failed and he had a second surgery involving a nerve graft approximately six months later. There was no recent history of vaccination or viral illnesses, however one month after the second surgery, he experienced severe, acute onset of left shoulder pain and left arm numbness. The acute, extreme pain lasted for approximately 2 months, and was gradually replaced with a dull ache. The patient developed a significant decrease in left scapular dynamic stability, with profound weakness of serratus anterior and subscapularis. He proceeded with approximately a total of 3 months of therapy focusing on reestablishing scapular stability, strengthening, and posture. Overall, the patient had a very positive outcome. Though he did not have full strength at discharge, he had demonstrated significant improvement and was reporting very minimal pain, however he continued to have sensory disturbance in his left arm. He had no functional deficits and looks forward to returning to wakeboarding this summer.

Parsonage-Turner Syndrome article reviews follow

Denouement and Discussion

Diagnosis: Parsonage-Turner Syndrome

The image depicts a left-winged scapula resulting from shoulder girdle weakness. After plain radiographs showed no abnormalities, magnetic resonance imaging of the cervical spine and shoulder revealed high T2 signal intensity of the long thoracic, suprascapular, and axillary nerves and fatty atrophy of the muscles, confirming the diagnosis of Parsonage-Turner syndrome (PTS) (otherwise known as brachial neuritis, neuralgic amyotrophy, and idiopathic brachial neuritis).^{1,2}

Parsonage-Turner syndrome is a condition that was first described in 1948 in a case series of 136 patients.^{3,4} Typically, PTS presents with abrupt onset of moderate shoulder pain followed by variable weakness of the shoulder girdle. Patients with PTS usually describe a sharp onset of pain that subsides in days to weeks and is slowly replaced with a dull ache. Weakness develops after the resolution of the initial pain, and there is usually normal sensation.^{1,2} Involved muscles are those innervated by the brachial plexus (C5-C8), most commonly the long thoracic, suprascapular, and axillary nerves.^{1,5} In our patient, a winged scapula was present because of paralysis of the serratus anterior muscle, which is innervated by the long thoracic nerve. Any component of the brachial plexus can be involved, with the lower trunk affected in up to 15% of cases.⁶ Case series have been reported of adult men presenting with phrenic nerve involvement, leading to dyspnea from diaphragmatic paralysis.^{6,7}

The incidence of PTS is estimated at 1.64 per 100 000 in the general population and is highest in the third through seventh decades of life; rare reports have occurred in children as young as 3 months.⁸ There is a male predominance, with reported male to female ratios of 2:1 to 11.5:1.^{3,5,9-11} Although the cause of PTS is undetermined, it has been linked to vaccine administration and viral illnesses in 15% to 25% of cases.^{5,7,12,13} Specific cases after tetanus toxoid immunization and outbreaks in specific clusters have led most to believe that an immune-mediated process is the common pathway in this disease. Most cases are not preceded by trauma.^{2,7,13} Eighty percent of cases spontaneously resolve within 2 years, and patients with severe symptoms at onset may have a more protracted course of weakness.^{1,13} Management is focused on analgesia and physical therapy, with no need for surgery reported in the literature.^{1,5,14}

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Parsonage–Turner Syndrome—Case Report and Literature Review

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Abstract Parsonage–Turner syndrome is the term used to describe a neuritis involving the brachial plexus. It may present with symptoms of an isolated peripheral nerve lesion, although the pathology is thought to lie more proximally. A case describing an isolated anterior interosseus nerve palsy due to an acute brachial neuritis is presented where the electromyographic findings confirmed the diagnosis, but also demonstrated the coexistence of a dual pathology in the form of a cervical radiculopathy. The literature is reviewed regarding etiology, treatment, and prognosis.

Keywords Parsonage–Turner syndrome · Cervical radiculopathy

Introduction

The diagnosis of Parsonage–Turner syndrome can be a cause of angst for both the patient and the attending physician. Not only is it debilitating for the patient, the clinical presentation may mirror other pathologies making accurate diagnosis

difficult. It was first described in 1897 by Feinburg [7] who reported a case of unilateral brachial plexus neuritis associated with influenza. It was not until 1943, however, that Spillane described it as a distinct clinical entity [22]. In 1948, Parsonage and Turner described 136 cases of the condition as a neuralgic amyotrophy and gave it the eponymous term “shoulder–girdle syndrome” [20]. They described a typical presentation of sudden onset of shoulder pain, followed by a flaccid paralysis of the shoulder girdle and upper arm, at which time the pain subsided. They noted that power gradually was restored spontaneously to the affected side over a matter of months. A much later review of this condition revealed a longer recovery period, with almost 90% of patient attaining complete recovery only by the end of the third year [23]. Other smaller series have shown that almost half of patients had some residual deficit at long-term evaluation [17].

Although the original descriptions of Parsonage–Turner Syndrome or acute brachial neuritis describe a global palsy affecting muscles about the shoulder region, there have been many subsequent reports of patients presenting with seemingly isolated peripheral nerve lesions whose pathophysiology lies in diffuse proximal inflammation of the upper limb [5, 14, 17, 24]. The presentation of acute brachial plexus neuritis as a palsy of the anterior interosseus nerve (AIN) has already been published [9, 10, 12, 18, 26]. We describe another such case of an isolated AIN palsy where electromyographic (EMG) findings pointed to a proximal neuritis as the etiology of the lesion and where the conservative measures employed had a successful outcome. In this report, however, the EMG studies revealed a concomitant pathology, which was explained by careful patient history, but otherwise may have added to the diagnostic dilemma. We also review the literature regarding the etiology, treatment, and outcome of this condition.

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Case Report

A 43-year-old right-hand-dominant man was referred to the principal author for evaluation of a swelling on the radiovolar aspect of his left non-dominant thumb. He underwent excision of the same under general anesthesia without complication. Histology confirmed the lesions to be benign inclusion cysts.

Five days post-operatively, the patient developed a sudden onset of severe pain affecting his left shoulder and upper arm. There was some sensory disturbance along his lateral arm. A further 10 days later, this discomfort had subsided, but the patient had become aware of his inability to flex both the distal interphalangeal joint of his left index finger and the interphalangeal joint of the left thumb. The patient denied any recent trauma or recent viral or systemic illness. Of note, in his past medical history, he had played rugby as a teenager, but had not played competitively in the previous 20 years. He denied any history of neck pain. Clinical examination confirmed paralysis of flexor pollicis longus (FPL) and flexor digitorum profundus (FDP) of his index finger (4/5 weakness, Medical Research Council grade). There was no sensory loss in his left upper limb, and shoulder and elbow strength were graded as normal. No tenderness or positive Tinels sign could be elicited over the course of the median nerve. Blood investigations showed a normal full blood count and erythrocyte sedimentation rate, with immunoglobulin levels within normal limits.

EMG carried out 3 weeks after onset of symptoms revealed denervation in the left pronator quadratus and left flexor pollicis longus muscles with positive sharp waves and a reduced polyphasic recruitment pattern. The left biceps, extensor carpi radialis, triceps, and pronator teres also showed signs of denervation consistent with a brachial plexus neuritis. What is also noteworthy is that the paraspinal segments on the left side showed evidence of membrane instability, with small positive sharp waves (less than 50 μ V). This latter finding does not fit the classical pattern of Parsonage–Turner syndrome and may represent a concomitant cervical radiculopathy.

Repeat assessment carried out 2 months later revealed a continued reduction in function of the affected hand. EMG studies at this time showed normal insertion activity and evidence of recovery reinnervation potentials in the biceps, extensor carpi radialis, triceps, and pronator teres, indicating ongoing recovery. The anterior interosseus innervated pronator quadratus and FPL demonstrated poor recovery. After 4 months, electromyographically, the extensor carpi radialis, flexor carpi radialis, and pronator quadratus were all normal. Flexor pollicis longus, however, showed a recruitment pattern of approximately 70% of normal. This was confirmed clinically by the inability to form the “O” sign between thumb and forefinger. By 7 months, the patient had

recovered both flexion of the distal joint of the index finger and the interphalangeal joint of the thumb.

Discussion

Parsonage–Turner syndrome is the term given to describe an entity that is also known as acute brachial neuritis, neuralgic amyotrophy, brachial neuropathy, or neuritis of the shoulder girdle to name but a few. This plethora of descriptive terminology does not lead to a ready diagnosis, with its being easily confused with other upper extremity abnormalities, including anomalies of the rotator cuff, acute calcific tendinitis, adhesive capsulitis, cervical spondylosis, peripheral nerve compression, tumor, acute poliomyelitis, and amyotrophic lateral sclerosis [17]. It is relatively rare, with an incidence of 1.64 cases per 100,000 population being reported [3]. It appears to affect males more than females, with a peak in incidence in patients in their third and seventh decades [15, 16]. There does not seem to be any relationship to hand dominance, although the condition occurs bilaterally in one third of cases [5, 15].

The exact cause of Parsonage–Turner syndrome is unknown. Many factors have been proposed to cause the neuritis including trauma, infection, viral disease, heavy exercise, recent surgery, immunization, and autoimmune conditions [5, 15, 16, 20, 23, 25]. A rare hereditary form has also been described [11]. Infection seems to precede the onset of symptoms in up to one quarter of patients, with Tsairis et al. [23] reporting 25 out of 99 patients with an antecedent flu-like illness. Heavy exercise was described in 8 out of 12 patients by Weikers et al. [25] as being a causative factor. Surgery in areas away from the shoulder region, as in this case, is also common in the literature [8, 20, 25]. A viral etiology has also been postulated, with the Coxsackie B virus been implicated in an epidemic reported [2].

The characteristic pattern of pain followed by profound weakness are generally the clues to the diagnosis of Parsonage–Turner syndrome, with confirmation being sought by electromyography. This pain is often described as a severe ache or throbbing radiating from the shoulder distally down the arm or proximally into the neck [16]. These symptoms may last for a few hours or may persist for up to three or more weeks. Usually, the patient will describe weakness after the pain has subsided, with an estimated 70% of sufferers experiencing weakness 2 weeks after the onset of symptoms [23]. It is this particular clinical course in this case that helps to distinguish between a brachial neuritis and a true compression of the anterior interosseus nerve. Although the patient ultimately presented with an inability to pinch between thumb and index finger, it is important to note that these motor signs were preceded by severe pain in the shoulder

girdle area which resolved on commencement of the paralysis of the flexor pollicis longus and the flexor digitorum profundus to the index. These are important distinctions to make, as treatment of Parsonage–Turner syndrome is conservative, whereas anterior interosseus nerve palsy, as classically described, represents a compression at an isolated known anatomical site and warrants surgical exploration [6]. Another point to note from the history of this case is the sensory disturbance described by the patient. This is often present with Parsonage–Turner syndrome and commonly affects the lateral arm [5]. These sensory defects are for the most part generally incomplete, unlike the associated motor defects.

In general, the results of basic laboratory investigations (full blood count, blood biochemistry, immunoglobulin analysis, and urinalysis), as in this case, are usually normal [16]. Electromyography is often crucial in confirmation of the diagnosis. In this report, while denervation (in the form of abnormal latency and prolonged duration of the action potential) of FPL, FDP to the index finger and pronator quadratus would be seen with all anterior interosseus nerve palsies, it is crucial that the neurophysiologist sample the musculature more proximally (deltoid, biceps) to demonstrate the pathology affecting the brachial plexus more diffusely [17]. In general, these electromyographic findings are generally found usually 3 weeks after the onset of symptoms [25]. Sampling of the proximal paraspinous nerves in this case also revealed abnormalities not seen with Parsonage–Turner syndrome, but more suggestive of a cervical radiculopathy. As these changes were small (less than 50 μ V), they probably represented an old concomitant pathology as may be explained by the patient's previous history of contact sports in the form rugby football [13, 19]. This form of cervical degeneration is common and often asymptomatic in the rugby playing population [19]. An early polyneuropathy could also show a similar EMG pattern [13]. The history and presentation, however, is not usually as time-specific as in this case. This abnormality seen on electrodiagnostic testing of the paraspinous musculature may represent dual pathology or indeed may represent a further manifestation of the syndrome, as it is unlikely that this muscle group would be tested in all cases. However, it must be stated that the cervical paraspinous musculature is usually spared from any abnormalities on electromyography [16, 25]. Magnetic resonance imaging has, in recent times, been shown to show some promise in the diagnosis of brachial neuritis, and this will improve with higher resolution and imaging quality [21].

Overall, Parsonage–Turner syndrome is a self-limiting condition, with the mainstay of treatment being supportive. In their review of 99 patients, Tsairis et al. found no significant benefit for patients who had had corticosteroids administered. They also found that physical therapy did not appear to im-

prove time to functional recovery. For the most part, provision of adequate analgesia during the initial phase of this condition is required. There does not appear to be any substantial supportive evidence for the use of other modalities such as massage, ultrasound or electrical stimulation therapy [1]. Surgery should be reserved for patients who have plateaued. DePalma [4] describes two cases where surgery was indicated in patients who remained without improvement for more than 2 years—one patient with persistent weakness of serratus anterior and rhomboids who benefited from surgery to stabilize his scapula to the thorax, the other in a patient who had no recovery to a radial nerve lesion who underwent tendon transfers. The prognosis from this condition for the most part is good, with an estimated three-quarters of all patients making a complete recovery within 2 years [23]. A few patients may experience a relapse of symptoms, but these tend to be less intense and last a much shorter duration [15, 21].

This case report illustrates the importance of a careful clinical history, along with examination, and electromyography not only of affected muscle group is mandatory in this type of case. The conservative treatment of anterior interosseus nerve palsy secondary to Parsonage–Turner syndrome has a favorable outcome.

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