Parsonage-Turner syndrome

- Neuralgic amyotrophy
- Cubital Tunnel Release in a Patient With Parsonage-Turner Syndrome: A Case Report
- Parsonage-Turner Syndrome After Bacterial Pharyngitis in Childhood With Resulting Glenohumeral Joint Subluxation in Adulthood
- Neurological Manifestations of HEV Infection: A Rare Phenomenon or an Underrecognized Reality?
- Comment on the article: Hourglass-like constriction is a hallmark of neuralgic amyotrophy (Parsonage-Turner syndrome). Hand Surg Rehabil. 2025;44(2):102090"
- Post-Transplant Pain and Paralysis: Neurologic Amyotrophy as an Atypical Cause of Shoulder Dysfunction Following Hematopoietic Stem Cell Transplant
- Isolated Pectoralis Major and Tricep Atrophy Secondary to Neuralgic Amyotrophy
- Endoscopy for neurogenic thoracic outlet syndrome: effectiveness and safety assessments

It is named after Maurice Parsonage and John Turner and published in The Lancet in 1948 by Parsonage and Turner [1]. 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.

Parsonage Turner syndrome is also known as acute brachial neuropathy and acute brachial radiculitis.

Other names used are Parsonage–Aldren–Turner syndrome, neuralgic amyotrophy, brachial neuritis, brachial plexus neuropathy, or brachial plexitis.

Parsonage-Turner syndrome and hereditary brachial plexus neuropathy (HBPN) present with indistinguishable attacks of rapid-onset severe shoulder and arm pain, disabling weakness, and early muscle atrophy. Their combined incidence ranges from 3 to 100 in 100,000 persons per year. Dominant mutations of SEPT9 are the only known mutations responsible for HBPN. Parsonage and Turner termed the disorder “brachial neuralgic amyotrophy,” highlighting neuropathic pain and muscle atrophy. Modern electrodiagnostic and imaging testing assists the diagnosis in distinction from mimicking disorders. Shoulder and upper limb nerves outside the brachial plexus are commonly affected including the phrenic nerve where diaphragm ultrasound improves diagnosis. Magnetic resonance imaging can show multifocal T2 nerve and muscle hyperintensities with nerve hourglass swellings and constrictions identifiable also by ultrasound. An inflammatory immune component is suggested by nerve biopsies and associated infectious, immunization, trauma, surgery, and childbirth triggers. High-dose pulsed steroids assist initial pain control; however, weakness and subsequent pain are not clearly responsive to steroids and instead benefit from time, physical therapy, and non-narcotic pain medications. Recurrent attacks in HBPN are common and prophylactic steroids or intravenous immunoglobulin may reduce surgical- or childbirth-induced attacks. Rehabilitation focusing on restoring functional scapular mechanics, energy conservation, contracture prevention, and pain management are critical. Lifetime residual pain and weakness are rare with most making dramatic functional recovery. Tendon transfers can be used when recovery does not occur after 18 months. Early neurolysis and nerve grafts are controversial [2]

Epidemiology

Parsonage-Turner Syndrome (PTS) is a rare syndrome with an incidence of 1.64 cases in 100,000 people.

Etiology

The etiology is still unknown, although an immune mediated mechanism is thought to be involved. Hematopoietic stem cell transplantation is a well-established treatment for hematological malignancies, but with a growing implication in the treatment of autoimmune diseases. The neurological side effects are probably underdiagnosed. The association of the Parsonage-Turner syndrome and the hematopoietic stem cell transplantation is scarce. We describe two clinical cases of idiopathic brachial plexopathy after hematopoietic stem cell transplantation. The reconstruction of the immune system after a transplant may be the trigger of a brachial plexopathy, but more studies are necessary for the etiology of this disease to be understood and to establish a cause-effect relation with the transplant [3)]

HEV can cause diverse neurological manifestations, especially Parsonage-Turner syndrome. Here, we used a large-scale human genomic approach to search for genetic determinants of severe clinical presentations of HEV infection.

Approach and results: We performed whole genome sequencing in 3 groups of study participants with PCR-proven acute HEV infection: (1) 24 patients with symptomatic acute hepatitis E; (2) 12 patients with HEV-associated Parsonage-Turner syndrome; and (3) 16 asymptomatic blood donors (controls). For variant calling and annotation, we used GATK4 best practices followed by Variant Effect Predictor (VEP) and Annovar. For variant classification, we implemented the ACMG/AMP Bayesian classification framework in R. Variants with a probability of pathogenicity >0.9 were considered damaging. We used all genes with at least 1 damaging variant as input for pathway enrichment analyses.We observed a significant enrichment of type I interferon response pathways in the symptomatic hepatitis group: 10 out of 24 patients carried a damaging variant in one of 9 genes encoding either intracellular viral sensors (IFIH1, DDX58, TLR3, POLR3B, POLR3C) or other molecules involved in type I interferon response [interferon regulatory factor 7 (IRF7), MYD88, OAS3, GAPDH]. We did not find any enriched pathway in the Parsonage-Turner syndrome group or in the controls.

The results highlight the essential role of type I interferon in preventing symptomatic acute hepatitis E [4)]

A total of 258 NMD cases following COVID-19 have been reported globally, of which 171 cases were Guillain-Barré syndrome (GBS), 40 Parsonage-Turner syndrome (PTS), 22 Myasthenia Gravis (MG), 19 facial nerve palsy (FNP), 5 single fiber neuropathy, and 1 Tolosa-Hunt syndrome. All (100%) SFN patients and 58% of FNP patients were female; in the remaining NMDs, patients were predominantly male, including MG (82%), GBS (63%), and PTS (62.5%). The median time from vaccine to symptom was less than 2 weeks in all groups. Symptoms mainly appeared following the first dose of vector vaccine, but there was no specific pattern for mRNA-based.

COVID-19 vaccines might induce some NMDs, mainly in adults. The age distribution and gender characteristics of affected patients may differ based on the NMD type. About two-thirds of the cases probably occur less than 2 weeks after vaccination [5].

The syndrome is idiopathic; although many specific risk factors have been identified (such as; post-operatively, post-infectious, post-traumatic or post-vaccination) the aetiology is still unknown. The condition manifests as a rare set of symptoms most likely resulting from autoimmune inflammation of unknown etiology of the brachial plexus (The brachial plexus is a complex network of nerves through which impulses reach the arms, shoulders and chest.)

This syndrome can begin with severe shoulder or arm pain followed by weakness and numbness.

Those who suffer from Parsonage–Turner experience acute, sudden-onset pain radiating from the shoulder to the upper arm. Affected muscles become weak and atrophied, and in advanced cases, paralyzed. Occasionally, there will be no pain and just paralysis, and sometimes just pain, not ending in paralysis.

Clinical Features

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.

Diagnosis

Parsonage-Turner syndrome diagnosis

Differential diagnosis

Because these symptoms may mimic other conditions, such as brachial plexus injury, PTS may be difficult to recognize. Other diagnoses that present similarly include cervical radiculopathy (could be caused by degenerative disc disease, disc bulge, etc.), compression of the brachial plexus by mass lesion, postherpetic neuralgia, calcific tendonitis, acute subacromial bursitis, and adhesive capsulitis [6]

The differential diagnosis in patients presenting with severe, unilateral shoulder pain should also include several similar presenting conditions. Cervical disc 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.

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.

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. Patients with cervical radiculopathies will commonly have a positive Spurling's test. 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.

Prognosis

Despite its wasting and at times long-lasting effects, most cases resolve themselves and recovery is usually good in 18–24 months, depending on how old the person in question is. For instance, a six-year-old could have brachial neuritis for only around 6 months, but a person in their early fifties could have it for over 3 years.

Mixed martial artist Todd Duffee was diagnosed with the condition in early 2013. He returned to professional fighting in December 2014.

Case reports

A rare case in which a 79-year-old female, with no significant past medical history, was diagnosed with PTS two months after a biopsy of the right levator scapulae muscle. Forty-eight hours after the procedure, she developed sudden-onset pain and weakness in the right scapulae and neck, followed by worsened weakness. This case report highlights the importance of considering PTS before proceeding with treatment. Patients with suspected PTS should undergo electromyography (EMG) to confirm diagnosis and monitor disease progression and resolution [7)]

1)
PARSONAGE MJ, TURNER JW. Neuralgic amyotrophy; the shoulder-girdle syndrome. Lancet. 1948 Jun 26;1(6513):973-8. PubMed PMID: 18866299
2)
Meiling JB, Boon AJ, Niu Z, Howe BM, Hoskote SS, Spinner RJ, Klein CJ. Parsonage-Turner Syndrome and Hereditary Brachial Plexus Neuropathy. Mayo Clin Proc. 2024 Jan;99(1):124-140. doi: 10.1016/j.mayocp.2023.06.011. PMID: 38176820.
3)

Taranu MA, Ezcurra Díaz G, Olivé Marqués A, Coll-Fernández R, Montané E, Ferra Coll C, Lucente G. Asociación de trasplante de progenitores hematopoyéticos y síndrome de Parsonage-Turner: ¿coincidencia o relación causa-efecto? [Association between hematopoietic stem cell transplantation and Parsonage Turner syndrome: coincidence or cause-effect relationship?]. Rehabilitacion (Madr). 2023 Dec 22;58(2):100835. Spanish. doi: 10.1016/j.rh.2023.100835. Epub ahead of print. PMID: 38141426.
4)

Saadat A, Gouttenoire J, Ripellino P, Semela D, Amar S, Frey BM, Fontana S, Mdawar-Bailly E, Moradpour D, Fellay J, Fraga M; HEV Human Genetics Collaborators. Inborn errors of type I interferon immunity in patients with symptomatic acute hepatitis E. Hepatology. 2023 Dec 8. doi: 10.1097/HEP.0000000000000701. Epub ahead of print. PMID: 38079352.
5)

Tayebi A, Samimisedeh P, Jafari Afshar E, Mahmoudnia S, Milan N, Ayati A, Madady A, Rastad H. Neuromuscular diseases associated with COVID-19 vaccines: a systematic review and pooled analysis of 258 patients. BMC Neurol. 2023 Dec 11;23(1):437. doi: 10.1186/s12883-023-03486-y. PMID: 38082244; PMCID: PMC10712145.
6)

Feinberg JH, Radecki J. Parsonage-turner syndrome. HSS J. 2010 Sep;6(2):199-205. doi: 10.1007/s11420-010-9176-x. Epub 2010 Jul 30. PMID: 21886536; PMCID: PMC2926354.
7)

Dalati Y, Ashraf U. Parsonage-Turner Syndrome With an Uncommon Trigger: A Case Study and Literature Review. Cureus. 2023 Nov 28;15(11):e49589. doi: 10.7759/cureus.49589. PMID: 38156184; PMCID: PMC10754625.