

CASE REPORT

Critical pain in the armpit of a 75 year old woman Parsonage-Turner syndrome

Dolor crítico en la axila de una mujer de 75 años. Síndrome de Parsonage-Turner

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Abstract

Parsonage-Turner (PT) syndrome is a painful disease with unknown aetiology. It often presents with acute pain in the shoulder, upper arm, the side of the neck and over the scapula. We describe the concurrence of PT in a 75-year-old woman with intense shoulder pain. PT is a diagnose of exclusion after clinical elimination of other frequent diseases and should be considered when the pain in the shoulder is not improved with medical treatment and/or physical therapy.

Keywords: Parsonage-Turner Syndrome, brachial neuritis, brachial amyotrophy.

Resumen

El síndrome de Parsonage-Turner (PT) es una enfermedad dolorosa de etiología desconocida. A menudo se presenta con dolor agudo en el hombro, la parte superior del brazo, el lado del cuello y sobre la escápula. Describimos la concurrencia de TP en una mujer de 75 años con intenso dolor en el hombro. La TP es un diagnóstico de exclusión tras la eliminación clínica de otras enfermedades frecuentes y debe considerarse cuando el dolor en el hombro no mejora con tratamiento médico y/o fisioterapia.

Palabras clave: Síndrome de Parsonage-Turner, neuritis braquial, amiotrofia braquial.

We describe the case of a 75-year-old-woman, who has hypercholesterolaemia, hypertension and depression (all well controlled and treated with atorvastatin and enalapril). She was referred to us from her primary care physician, who described her as having intense pain and functional limitation of the right upper limb some weeks after pain started. Symptoms appeared some months ago and were treated initially with oral NSAIDs without significant response. On examination, there was pain with the right arm mobilization and constant pain in the right axillary area with irradiation to the chest. We conducted a neurological examination, which was normal.

There was no damage to the joints and no mass lesion was found in the radiographic examination. Laboratory tests were normal (serum proteins, PCR, anti-DNA, anti-ENAs, ANCA, ACE levels, immunoglobulins, hepatic and tumour markers, and rheumatoid factor were negative). Other biochemical and blood tests (white/red cell count, sodium, potassium, troponins) and ECG were normal.

A posterior MR scanning showed oedema in supra and infraspinatus muscles. These results were completed

with electromyography resulting in an intense denervation pattern. With all these findings and after a meeting with doctors from orthopaedics, neurology, and geriatrics the diagnose of Parsonage-Turner Syndrome was decided.

The Parsonage-Turner Syndrome (PTS), also known as brachial neuritis or amyotrophic neuralgia, is a clinical syndrome of infrequent presentation and unknown aetiology. PTS often presents with acute pain in the shoulder, upper arm, the side of the neck and over the scapula. The pain lasts four weeks on average, followed by a rapid multifocal weakness and atrophy of the affected upper limb. The symptoms are often unilateral but can appear as a bilateral condition¹.

PTS is a diagnose of exclusion with an average delay of three to nine months before the diagnosis is made. The incidence of PTS is 1.64 per 100.000 in the United States but is expected to be higher due to the difficulty of recognition. Other conditions, like Rotator Cuff Injury, arthrosis in the shoulder joint or Adhesive Capsulitis, are often considered before the diagnose of PTS is made¹. Some of the features of PTS compared to other conditions are shown in **table I**:

Table I: Classification of Parsonage-Turner Syndrome and associated conditions (own elaboration)^{1,3,4}.

	Parsonage-Turner	Traumatic Plexopathy	Rheumatoid Arthritis (RA)
Onset	Sudden	Sudden	Slowly progressive
Sex	Men > Women (Age 20-69 years)	Men > Women (Age 15 – 25 years)	Women > Men (age often > 65 years)
Aetiology	Unknown, but underlying predisposition, susceptibility to mechanical injury and immune-mediated response in the brachial plexus.	Penetrating injuries. Elongation, traction, or compression of the brachial plexus.	An inflammatory cascade of unknown origin, leading to persistent synovial inflammation, damage to the cartilage and underlying bone and extra-articular disease.
Symptoms	Intense pain, which can be difficult to localize (shoulder and proximal part of the upper limb) – 25% bilateral, followed by weakness and muscle atrophy, often in combination with paresthesia and sensory disturbances.	Depending on the involvement of the plexus.	Weak pain, swelling and heat sensation in the fingers, jaw, and neck. Often seen in combination with fatigue, depression, and loss of appetite.
Physical Examination	Muscles commonly affected is the Deltoid, supraspinatus, infraspinatus, serratus ant., biceps and triceps. Nerve affection is seen in isolated nerves or multiple nerves both inside and outside the brachial plexus.	Manual motor testing, range of motion and sensation examination is used to differentiate preganglionic plexopathy from postganglionic plexopathy, (i.e. Involvement of proximal innervated muscles (i.e. rhomboids) suggest preganglionic plexopathy).	Numbers of swollen joints and serology-test (Rheumatoid Factor and ACPA) to meet the ACR/EULAR 2010 criteria.
Electromyography (EMG)	Early EMG is not recommended. EMG after 4-6 weeks shows acute denervation and reduction of motor unit recruitment in a non-myotomal pattern.	Variable axonal plexopathy	Is not used to diagnose/manage RA.
Radiological Findings	Radiographic imaging is often used to exclude other disorders. MR: After 4-5 weeks neurogenic oedema (diffuse hyperintense muscle injury) is seen. Later, atrophy and fatty infiltration are seen.	MR: plexus lesions, compression, and nerve oedema. However, CT myelography is considered the benchmark of radiologic evaluation for nerve root avulsion	Juxta-articular erosions.
Remission	Usually reversible (years)	Unusual without treatment	Unusual
Treatment	NSAIDs/opioids, oral corticoids, and physical therapy.	Physical therapy and/or surgery.	NSAIDs, Corticoids, Disease-modifying Antirheumatic Drugs (DMARDs) and Biological Agents.

There is no specific treatment, that has been proved to reduce the neurologic impairment, nor improves the prognosis of PTS¹. There have been no randomised control trials and the evidence to support a treatment option is anecdotal. In the acute stage, severe pain is often treated with a combination of NSAIDs and opioids. In one study, 60 mg/day oral prednisone was given in the first week and then tapered to 10 mg per day in the second week, which seems to shorten the duration of symptoms. Antiepileptic medications and tricyclic antidepressants are often used to treat neuropathic pain that often persists after the acute painful attack, however, antiepileptic medications are not as effective at treating the acute severe pain of PTS because of their delayed onset^{1,2}.

Nonpharmacologic treatments used in the treatment of PTS include physical therapy, osteopathic manipulation,

therapeutic modalities, and acupuncture. Despite a proposed role of physical therapy for preventing loss of range of motion and further disability, physical therapy does not seem to speed up recovery. Goals of physical therapy should include maintenance of a range of motion and prevention of loss of function. Depending on the level of pain, range-of-motion exercises for the shoulder may be started immediately².

Finally, in some cases, PTS are refractory to conservative pharmacologic and nonpharmacologic treatments. In these cases, surgery is often considered. Surgical procedures for PTS include neurolysis, nerve grafts, and nerve transfers¹.

Interests conflict

The authors declare no conflict of interest.

Patient/ research participant consent

Written informed consent for publication of their clinical details was obtained from the patient.

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