**CLINIC CASE**

# SINDROME DE PARSONAGE TURNER, A PROPÓSITO DE UN CASO.

## PARSONAGE TURNER SYNDROME, A CASE REPORT.

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## RESUMEN

El síndrome de Parsonage-Turner (SPT) , plexitis braqueal o neuritis braquial es una patología poco frecuente, con una incidencia de aproximadamente 3 casos por cada 100.0000 habitantes de etiología es desconocida, aunque en algunos casos se han encontrado factores desencadenantes como infecciones bacterianas y se ha relacionado con enfermedades sistémicas[2]

Su característica clásica es la omalgia unilateral, de aparición súbita, incapacitante, con posterior debilidad muscular, que se acentúa en movimientos como elevación, abducción y rotación externa de la extremidad afectada. Se ha observado que en el SPT los nervios periféricos más comprometidos son el torácico largo, el axilar, musculocutáneo y el supraescapular. Se han descrito varias fases en el SPT. Fase de neuritis, Fase de parálisis y atrofia muscular, y la fase de recuperación. En la mayoría de los casos no se han reportado secuelas, sin embargo, en algunos pacientes se puede observar déficit motor permanente. [2,3,4,5,6]

Se trata de un caso clínico de una paciente femenina de 67 años de edad, con antecedente de DM2, quien consulta por dolor en hombro izquierdo, de inicio agudo asociado a pérdida de la fuerza, limitación funcional del hombro y alteraciones de la sensibilidad, el cual estuvo precedido de infección periodontal en maxilar inferior ipsilateral, que drenó espontáneamente para lo cual no recibió tratamiento antibiótico, y que luego de la realización de RMN, se encontró alteraciones en la intensidad de los músculos supraespinoso, infraespinoso y tendinopatía subescapular, además de electromiografía con signos crónicos de reinervación crónica y fibrilación en reposo de los músculos supraespinoso, deltoides y bíceps izquierdos, sin compromiso motor de los mismos, llegando así al diagnóstico de un síndrome de Parsonage Turner, el cual respondió positivamente al tratamiento con estimulación nerviosa transcutánea (TENS).


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**PALABRAS CLAVE:** Síndrome de Parsonage-Turner **,** Dolor Agudo, Electromiografía, Debilidad Muscular, Atrofia Muscular.

**SUMMARY**

Parsonage-Turner syndrome (PTS), brachial plexitis or brachial neuritis is a rare pathology, with an incidence of approximately 3 cases per 100,000 inhabitants of unknown etiology, although in some cases triggering factors such as bacterial infections have been found and it has been related to systemic diseases.[2]

Its classic characteristic is unilateral omalgia, of sudden onset, disabling, with subsequent muscle weakness, which is accentuated in movements such as elevation, abduction and external rotation of the affected limb. It has been observed that in PTS the peripheral nerves most involved are the long thoracic, axillary, musculocutaneous and suprascapular nerves. Several phases have been described in PTS.

The neuritis phase, the paralysis and muscle atrophy phase, and the recovery phase. In most cases no sequelae have been reported, however, permanent motor deficits may be observed in some patients. [2,3,4,5,6]

This is a clinical case of a 67-year-old female patient, with a history of DM2, who consults for acute onset left shoulder pain associated with loss of strength, functional limitation of the shoulder and alterations of sensitivity, which was preceded by periodontal infection in the ipsilateral lower jaw, which drained spontaneously for which she did not receive antibiotic treatment, and after performing an MRI, it was found alterations in the intensity of the supraspinatus, infraspinatus and subscapularis tendinopathy, in addition to electromyography with chronic signs of chronic reinnervation and fibrillation in chronic reinnervation, After performing an MRI, alterations in the intensity of the supraspinatus and infraspinatus muscles and subscapularis tendinopathy were found, in addition to electromyography with chronic signs of chronic reinnervation and fibrillation at rest of the supraspinatus, deltoid and left biceps muscles, without motor involvement of the same, thus reaching the diagnosis of a Parsonage Turner syndrome, which responded positively to treatment with transcutaneous nerve stimulation (TENS).

**KEYWORDS:** Parsonage- Turner Syndrome, Acute Pain, Electromyography, Muscle Weakness, Muscular Atrophy

## Introducción.

Parsonage-Turner syndrome, also known as brachial plexitis or brachial neuritis, is a neuropathy first described in 1897 by Feinberg in a case associated with influenza. Over time, it was referred to by several names, including “serratus anterior paralysis,” “localized shoulder neuritis,” and “acute brachial radiculitis.” It was Parsonage and Turner who, in 1948, described the clinical presentation in 136 soldiers and later in 82 additional patients under the name “amyotrophic neuralgia of the shoulder.” [6]

This is a rare syndrome, with an incidence of approximately 3 cases per

100,000 inhabitants, its etiology remains unknown, although certain triggering factors have been identified in some cases, such as bacterial infections, and it has been associated with systemic diseases including lupus and diabetes mellitus. While some reports in the literature have documented bilateral presentation in up to 25% of cases, its classical presentation is characterized by sudden-onset, disabling unilateral shoulder pain (omalgia), followed by muscular weakness, particularly exacerbated by movements such as elevation, abduction, and external rotation of the affected limb. In Parsonage-Turner syndrome (PTS), the most commonly affected peripheral nerves are the long thoracic, axillary, musculocutaneous, and suprascapular nerves. Several clinical phases have been described in PTS. The initial **neuritic phase** is associated with acute, sudden-onset pain of moderate to severe intensity. This is followed by the **paralysis and muscle atrophy phase**, during which patients often report improvement in pain; the most frequently affected muscles include the deltoid, supraspinatus, infraspinatus, and/or serratus anterior. The final **recovery phase** may extend over several months and up to one year. Although most cases do not result in long-term sequelae, some patients may experience permanent motor deficits. [7.]

## Case presentation

A 67-year-old female presented with a one-day history of left shoulder pain radiating to the left lateral neck and lower submandibular region, rated 8/10 on the visual analog scale (VAS), accompanied by ipsilateral paresthesia and inability to elevate the arm, with preserved wrist and finger mobility. The patient reported having had a spontaneously drained abscess on molar 37 four days earlier, which did not require antibiotic treatment. She denied any prior trauma, dyspnea, or dysarthria. Her medical history included type 2 diabetes mellitus managed with once-daily metformin. On physical examination, vital signs were within normal limits, with no focal neurological signs. The left upper limb showed decreased strength and transient paresthesias, without evidence of deformity. Initial management focused on ruling out precordial pain versus cerebrovascular accident, with a normal EKG, negative troponin, normal cervical spine and left shoulder X-rays, and a normal non-contrast head CT scan. The internal medicine team later evaluated the patient, suggested left shoulder bursitis, raised suspicion of brachial plexus impingement, and requested orthopedic evaluation. Orthopedics recommended outpatient management and a non-contrast MRI of the left shoulder, followed by reevaluation. The MRI revealed thickening and altered signal in the supraspinatus and infraspinatus tendons consistent with moderate tendinopathy, without abnormalities in the long head of the biceps tendon. Distal subscapularis tendinopathy and subacromiodeltoid bursitis were also noted. Orthopedics

proceeded with a nerve block for the shoulder. Due to persistent symptoms, the patient consulted a physiatrist, who found complete deltoid weakness (grade 0 strength in all three heads), with no resistance or movement against gravity, and inability to perform abduction, adduction, flexion, or extension, as well as internal and external rotation of the left shoulder. Sensory function was preserved. An electromyography (EMG) was ordered to rule out brachial plexus motor involvement, which revealed an abnormal study compatible with left brachial plexus injury affecting the suprascapular, axillary, and musculocutaneous branches, showing chronic reinnervation changes and fibrillation at rest in the left supraspinatus, deltoid, and biceps muscles, without complete motor denervation. The patient was referred to a hand orthopedics service, where she was diagnosed with Parsonage-Turner syndrome. Treatment with electrical stimulation therapy was initiated, and follow-up was advised. At present, after five sessions of transcutaneous electrical nerve stimulation (TENS), the patient reports significant improvement in symptoms, is able to hold up her arm, and has regained considerable strength.

**Diagnóstico.**

The suspected diagnosis of PTS is established through a detailed patient history and physical examination, initially being a clinical diagnosis. Typical findings on physical examination include intense pain, present in 90-95% of patients [5], followed by decreased muscle mass in the affected limb (atrophy), and in some cases, paralysis of the limb, followed by a slow and progressive recovery. Paraclinical examinations in most patients show no abnormalities and are indicated only in cases where a systemic disease is suspected. Other diagnostic aids, such as shoulder X-rays, will also not show significant alterations, but should be considered in order to rule out pathologies that compromise the joint and can guide the physician towards a differential diagnosis. [2]

Nerve conduction studies are considered important to rule out conditions such as mononeuropathies, while electromyography is vital for documenting denervation, which may reveal involvement of a specific nerve or multiple nerves comprising the brachial plexus. In Parsonage-Turner syndrome (PTS), the suprascapular nerve is most commonly affected; however, other nerve branches originating from the brachial plexus, such as the axillary and musculocutaneous nerves, may also be involved. In our patient, findings were consistent with those reported in the literature, including denervation and chronic reinnervation. [3, 7.]

There are diagnostic tools that can be highly useful; shoulder MRI may reveal nerve demyelination and T1 hyperintensities, findings that are more commonly observed during the acute phase. However, in some cases, MRI may appear normal in the early days of the disease, with detectable

abnormalities emerging 2 to 3 weeks later.[3] T1-weighted images may also show atrophy of the affected muscles, along with T2 signal intensity alterations. [5]

## PTS phases

Clinically, the condition is classified into three phases: painful, weakness, and recovery. The painful phase is characterized by an acute onset, present in approximately 90% of cases, with an average intensity of 7/10 on the numerical pain scale, typically localized in the shoulder girdle. This phase may last from 1 day to 2 months, although in some cases, chronic neuropathic pain may persist. The weakness phase may precede pain in 5% of cases, appear 24 hours later in 34%, between 1 to 7 days in 39% (which corresponds to the case described in this report), and between 1 and 4 weeks in 27% of cases. The most commonly affected muscles include the infraspinatus, serratus anterior, supraspinatus, biceps, rhomboid, and pronator teres; the trapezius and sternocleidomastoid muscles are less frequently involved. Sensory disturbances may be present in up to 66% of patients; however, motor weakness remains the cardinal feature for diagnosing this condition. Lastly, the recovery phase may take 6 months to 1 year, and in some cases up to 3 years, with a recurrence rate of approximately 74%. The quality and duration of recovery largely depend on the extent of axonal damage.[4]

## Treatment

Initial treatment usually involves corticosteroid therapy, with a prednisone dose of 60 mg/day for the first week, followed by 10 mg/day for the second week, resulting in good patient outcomes. It can be combined with opioids if the pain is very intense in the initial phases, or with NSAIDs. Immunoglobulin has been proposed, although its efficacy has not been fully proven.[4]

Transcutaneous electrical nerve stimulation (TENS) therapy and cryotherapy have demonstrated high effectiveness in reducing pain and improving muscle weakness, promoting the restoration of normal muscle trophism. These therapies are the most recommended for recovering shoulder function and mobility.[3.]

In the case of the patient mentioned in this article, the diagnosis was not made early, so corticosteroid administration did not show benefits according to scientific evidence. However, TENS was indicated, with successful recovery after treatment.

## Discussion

In the described case, it can be observed that the early diagnosis of Parsonage-Turner syndrome is indeed a clinical challenge. This is due to the multiple differential diagnoses coupled with its low incidence, which makes it a rare diagnosis and one that medical

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personnel may take days or even weeks to detect.

As described in the literature, the suspicion of Parsonage-Turner syndrome is based on the patient's anamnesis, which revealed a recent episode of bacterial infection associated with a history of systemic disease (type II diabetes mellitus), as well as physical examination findings such as sudden, severe pain in the left upper limb without neurological deficits. Imaging studies, including magnetic resonance imaging and electromyography, demonstrated changes suggestive of brachial plexus neuritis, supporting the diagnosis of this uncommon clinical condition.

## Conclusions

The diagnosis of Parsonage-Turner syndrome can be challenging for healthcare professionals, which underscores the imperative need to be familiar with this pathology. This includes the importance of a detailed anamnesis that considers the patient's clinical history, possible differential diagnoses, and a thorough physical examination assessing strength, sensation, and mobility of the upper limb.

Early diagnosis can increase the chances of a successful recovery and a shorter recovery time, improving the prognosis and reducing possible sequelae. This reiterates the importance of considering this diagnosis when the patient's clinical picture cannot be attributed to other pathologies.

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