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Case Report

Parsonage-Turner Syndrome Caused by Parvovirus B19: A Case Report and Review of Literature

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Abstract

Parsonage-Turner Syndrome (PTS), is a rare brachial plexopathy that is typically characterized by several different clinical phases as: neuritis phase; motor deficit phase; muscular atrophy phase; and recovery phase. It has an incidence rate of 1.6-3 cases/100,000 inhabitants, predominantly in males during the 2nd and 5th decades of life. It is difficult to distinguish from other neuropathies. We describe an unusual case of a 40-year-old man who was diagnosed with PTS secondary to Parvovirus B19 infection and responded effectively to intravenous lidocaine and amitriptyline.

Keywords: Parsonage-Turner Syndrome; Parvovirus; Plexopathy

Introduction

Parsonage-Turner Syndrome (PTS), also called neuralgic amyotrophy or brachial neuritis, is a rare plexopathy of the brachial plexus, typically characterized by different phases: neuritis phase with shoulder pain which radiates to the upper extremity; motor deficit phase; the muscular atrophy phase, generally in ipsilateral shoulder and the area proximal to the elbow; and finally, the recovery phase that can last up to more than a year, and the patient may be left with several sequelae. It has an incidence rate of 1.6-3 cases/100,000 inhabitants, predominantly in males during the 2nd and 5th decades of life. We present an unusual case of a 40-year-old man who was diagnosed with PTS secondary to Parvovirus B19 infection and responded effectively to intravenous lidocaine and amitriptyline.

Case Presentation

A 40-year-old male patient with a history of asthma and who didn't undergo the usual treatments, presented with symptoms that included lumbar pain and band paraesthesia in the left knee. After 24 hours he presented with neuropathic pain in the proximal left upper limb, burning and predominantly at night, with fasciculation's in the deltoid and pectoral muscles and 4/5 weakness in the distal muscles. He mentioned that the week prior to the onset of those symptoms, he had a fever of 38.5ºC and macular rash on the torso, coinciding with confirmed Parvovirus B19 infection in his daughter and wife.

Several tests were performed; serology for Parvovirus B19 IgM and IgG, which were positive; Electromyogram (EGM) that showed data of peripheral motor axonal polyneuropathy predominantly in the lower limbs and left extremities; and Magnetic Resonance Imaging (MRI), that showed indirect signs suggestive of left brachial plexitis, such as elevation of the left hemi diaphragm, a hyper signal of the left hemi diaphragm (phrenic nerve paresis) and a hyper signal of the left infraspinatus muscle; which is another sign of acute denervation.

Based on the results of the tests performed and the patient`s symptoms, he was diagnosed with Parsonage-Tuner Syndrome secondary to Parvovirus B 19 infection.

Treatment with steroids and analgesics was started, requiring a Fentanyl patch of 25 mcg every 72 hours, pregabalin 75 mg every 12 hours and tramadol 100mg. Due to poor pain control with that combination of medication, a daily cycle of intravenous lidocaine had to be started, which was effective during the days of administration. After finishing the lidocaine cycle the fentanyl patch was increased to 50mcg every 72 hours and amitriptyline 10mg was added. With these changes in treatment, he was discharged two weeks after admission, reporting an improvement of up to 50% with a maximum Visual Analogue Scale (VAS) of 7 and a minimum VAS of 4. He presented a first review in outpatient consultation where he required an increase in amitriptyline to 75 mg and a new cycle of lidocaine. He also required rehabilitation treatment during his admission, which continued at discharge. He was currently under follow-up by the pain management unit, reporting less intensity of pain (VAS minimum 3, maximum 6).

Discussion

Parsonage-Turner syndrome is an acute brachial neuritis; consisting of shoulder pain, followed by motor deficit and amyotrophy, predominantly affecting the muscles of the shoulder girdle. Parsonage and Turner described it in 1948 and they called this disease Amyotrophic Neuralgia.

The pain is usually worse at night and generally presents complete reversibility in a time that varies from weeks to prolonged periods [1]. There are some cases that have lasted for many years, some are recurrent and in others there are sequelae.

Regarding the aetiology, there are 2 forms; an autosomal dominant hereditary form with mutations in chromosome 17q [2] and another, more typical form that is idiopathic, in which more than 50% of patients present precipitating factors, such as, a previous surgery, an infection, systemic disease or a side effect to vaccination [3]. These causes can provoke an immune response against the brachial plexus in people with a genetic predisposition.

The diagnosis is made with a correct history, physical examination, electromyography in which pathological resting potentials are observed, indicating acute denervation; and an MRI, which shows hyper tense signals in the affected tissues.

The treatment consists of reducing the pain using tramadol, fentanyl, corticosteroids, lidocaine and amitriptyline, among others. Amitriptyline has been useful for neuropathic pain and as an additional treatment when the pain is incessant [4].

Rehabilitation to maintain joint mobility is very important as this, coupled with the medications, shortens the resolution period. Studies have shown that the administration of intravenous immunoglobulins linked to methylprednisolone produce a rapid improvement of the episode [5].

To control the pain in our patient we used IV tramadol and fentanyl patches, along with cycles of lidocaine, pregabalin, and amitriptyline, due to the neuropathic component of PTS. This therapy was administered with steroid treatments that were started early. Finally, rehabilitation was effective, using active and passive mobility exercises. It has been seen that the rehabilitation therapy is crucial to improve the strength and joint mobility in these patients [6].

Surgery must be considered in cases that have not responded to any other medical treatment, like tendon transfers or nerve grafting.

Conclusion

PTS is a rare entity that is difficult to diagnose in its acute condition and it must be considered in every painful shoulder. A differential diagnosis must be made with other injuries that affect the muscles of the shoulder girdle. To do this a correct clinical history, physical examination, and complementary tests such as EMG and MRI will help us to identify and treat early, which will speed up recovery.

Although there is not a specific treatment, the pain management continues to be a challenge for the anaesthetist. All of this, accompanied with rehabilitation treatment, is essential for a favourable outcome.

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