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Parsonage-Turner Syndrome rather than Zoster Neuritis?

Karim Gariani^a Michel R. Magistris^b Mathieu R. Nendaz^a

^aDivision of General Internal Medicine, Department of Internal Medicine, and

^bDivision of Neurology, Department of Clinical Neurosciences, Geneva University Hospitals, Geneva, Switzerland

Key Words

Acute brachial plexus neuritis · Differential diagnosis · Electromyography · Herpes zoster neuropathy · Neuralgic amyotrophy

Abstract

We report the case of an 86-year-old man with acute left shoulder pain, followed by left limb monoparesis and a herpetic rash on the left upper limb and thoracic region. This situation presented a diagnostic challenge because of the simultaneity of symptoms attributable to Parsonage-Turner syndrome and herpes zoster neuropathy. A detailed clinical history, physical examination and electroneuromyography were essential to distinguish the neurological structures involved and to ascertain the diagnosis.

Introduction

Parsonage-Turner syndrome (PTS), also known as neuralgic amyotrophy or acute brachial plexus neuritis, is a disorder characterized by the sudden appearance of severe shoulder pain, usually unilateral, followed a few days to a week later by progressive motor weakness. At times, some dysesthesiae and numbness may coexist. The key to establishing the diagnosis is the sequence of symptoms and confirmation by electroneuromyography.

We report the case of an 86-year-old man referred to our internal medicine department for severe left shoulder pain. In this patient, diagnosis was complicated by the occurrence of herpetic lesions on the upper left arm and thoracic region.

Case Presentation

An 86-year-old right-handed man presented to his general practitioner with complaints of acute left shoulder pain that awoke him one night. There was no history of previous shoulder trauma. The patient had suffered from a low-grade stage IV non-Hodgkin B-cell lymphoma 4 years earlier that was treated with rituximab chemotherapy alone without radiotherapy, which led to remission. An ultrasound of his

shoulder revealed a scapulohumeral periarthrititis. An intra-articular corticoid infiltration was performed on an outpatient basis. He was referred to our department 7 days after the onset of the symptoms because of persistent shoulder pain.

At admission, the patient presented a hypertensive crisis that was attributed to the severe shoulder pain and was managed with nitroglycerin and calcium-channel blockers. The patient denied any neck pain, headache, fever, chills or weight loss. Neurologic examination was normal at the time, with preserved sensory and motor functions of the left arm. The patient was oriented and showed no signs of meningeal irritation. Routine laboratory tests were normal.

Two days following hospital admission, i.e. 9 days after the beginning of the left shoulder pain, he presented a vesicular rash over the left D1 and D2 dermatomes. An immunofluorescence test of a skin sample was positive for herpes varicella-zoster virus. A 10-day treatment of intravenous acyclovir in conjunction with prednisone was started. Pain was managed with paracetamol, morphine, pregabalin and physiotherapy. Two days after the vesicular eruption, i.e. 11 days after the beginning of the shoulder pain, he developed a marked weakness of the left arm. This weakness was severe on elbow flexion (1/5), shoulder abduction (1/5) and arm external rotation (1/5), whereas elbow extension and handgrip remained normal (5/5). There was no sensory loss. Deep left bicipital tendon reflex was absent.

Needle electromyography ([table 1](#)) showed evidence of partial denervation of the left biceps, brachioradialis and deltoid muscles, consistent with peripheral motor nerve involvement of the C5 and C6 myotomes. As the rhomboid and serratus anterior muscles were not affected, it is possible that the denervation was related to motor axonal lesions within the upper trunk of the brachial plexus. Neurography showed responses of small but symmetric amplitudes, in particular for the sensory nerve conductions ([table 2](#)). A CT scan of the cervical spine showed mild cervical osteoarthritis without spinal compromise. A thoracic CT scan showed no adenopathy or mass associated with the lymphoma and ruled out a local root or plexus compression. MRI of the brain showed a global cerebral atrophy that was attributed to the age of the patient. MRI of the brachial plexus was not performed.

The diagnosis of troncular, motor neurological lesions due to herpes zoster infection was initially suspected. However, the symptoms, their timing, as well as the neurological territories affected – upper brachial plexus for the motor deficit, dermatomes of the lower brachial plexus D1 and upper thoracic region D2 for the vesicular rash – casted doubts on this hypothesis. The symptoms seemed more probably related to PTS rather than to herpes zoster infection of the upper left arm. The clinical presentation, the results of the CT scan of the cervical and thoracic spine, and the motor-evoked potentials made cervical myelopathy unlikely.

Over the course of the following days, the weakness persisted. The patient was transferred to a rehabilitation unit to continue physiotherapy. A second electroneuromyography was performed 24 days after the first, i.e. 6 weeks after the onset of the shoulder pain ([tables 1, 2](#)). It showed signs of severe denervation of the left biceps, deltoid, supraspinatus and brachioradialis muscles. The neurological findings and normal transcranial motor-evoked potentials were not suggestive of cervical myelopathy.

Subsequently, the patient experienced a general deterioration and sepsis caused by *Staphylococcus aureus* infection. Despite antibiotic therapy, he died 2 months after admission to the rehabilitation unit, i.e. 3 months after the onset of the symptoms.

Discussion

The clinical presentation of a herpetic eruption and a motor deficit in the same limb may lead to diagnostic pitfalls. The first diagnosis that may come to mind in this situation is a motor monoparesis due to herpes zoster infection. However, this early impression may be challenged in this situation. Our hypothesis is that the patient presented two different successive conditions. First, left shoulder pain followed 11 days later by left arm weakness, possibly related to PTS, then a vesicular eruption of the left arm caused by varicella-zoster virus reactivation, as confirmed by immunofluorescence. The time elapsed between the eruption and the weakness, only 2 days, is rather short to be

explained by a herpes zoster motor monoparesis. Moreover, the distance between the lesions of the sensory ganglions causing the skin vesicular rash (dermatomes D1 and D2) and the motor axonal lesions causing denervation within the upper myotomes of the brachial plexus (C5–C6) also makes the diagnosis of herpes zoster monoparesis unlikely. Sensory findings, if present, are usually much less prominent than motor deficits in PTS. The contrary is true in zoster neuropathies, in which sensory lesions are usual and motor deficits rare (with the notable exception of zoster facial palsy).

Although all weak muscles observed to exhibit denervation belonged to the C5 and C6 myotomes, a radiculopathy appears unlikely in this patient since (1) it should have very seriously affected both C5 and C6 roots simultaneously in order to explain the complete or subcomplete denervation of the muscles of the anterior arm, and, in such a case, it would then probably have affected the spinatus and brachioradialis muscles with a similar severity; (2) a mechanical lesion of the roots would have caused sensory symptoms in the C5 and C6 dermatomes; (3) denervation would have concerned the rhomboid and serratus muscles. The severe, incomplete axonal lesion was thus more likely localized within the upper trunk. Symmetrical sensory neurography (from the C6 dermatome; digit I) without clinical sensory loss (in particular in the C5–C6 dermatomes), points to a lesion that essentially affected the motor axons. This is in line with the possibility of a selective targeted immunologic lesion, and thus in good agreement with the suspected causal mechanism of PTS. We hypothesize, therefore, that the development of herpes zoster was a consecutive phenomenon, possibly favored by the PTS itself, the lymphoma and the intra-articular corticoid infiltration.

PTS is a neuritis of unknown cause affecting the brachial plexus with an overall incidence of 1.64 cases per 100,000 individuals [1–4]. The classic description of PTS is a condition in which the patient first develops an abrupt, severe and constant unilateral shoulder pain that can extend proximally to the neck but also distally to the upper arm, forearm and hand. Pain lasts from a few hours to several weeks, with an average duration of 4 weeks. The weakness appears within 24 h of the onset of pain in approximately one-third of patients but can take up to 4 weeks to occur [5]. A sensory deficit may also occur but its prevalence varies, depending on studies, from 66% to only a minority of patients [6–8]. The upper trunk of the brachial plexus is the most frequent site of the lesion [6]. Clinical observation and electromyogram studies suggest that the lesions are often multifocal within the plexus or in the individual branches [9, 10]. Electroneuromyography is helpful to confirm the diagnosis, to rule out other possible causes of painful weakness of the upper limb (radiculopathies, thoracic outlet syndrome or mononeuropathies), and to define the prognosis. Frequently, the syndrome is initially mistaken for an arthropathy of the shoulder, as was the case in this patient. Blood and cerebrospinal fluid analyses are generally unhelpful. A chest radiograph or CT scan can be performed if there is any suspicion of malignancy.

The treatment is based on pain management, NSAIDs, prednisolone and neuroleptics [11, 12]. This condition generally carries a good prognosis, as about 75% of all patients recover completely within 2 years, and 89% by the end of the third year. Patients with predominantly lower plexus involvement have a slower recovery. The rate of recurrence varies among studies, from 5 to 26%, and the second episode typically turns out less severe [4].

Herpes zoster can cause segmental paresis with a reported incidence of 3–5% of limb weakness [13]. The paresis affecting the same zone typically occurs 2–3 weeks after the herpetic rash. The prognosis of functional recovery with zoster paresis is good, with a complete or partial recovery reported in 75% of patients within 1 or 2 years [14]. The administration of antiviral therapy may have a positive effect on the course of the paresis. In both PTS and zoster paresis, prevention of muscle contracture and atrophy by physiotherapy is an important part of the treatment [15].

The etiology of PTS is unclear but many possible promoting factors have been proposed including trauma, recent surgery, infection, heavy exercise, immunization and autoimmune conditions. Although the literature reports a direct relationship between herpes zoster and plexus neuritis [6, 15], the causal relationship between this viral infection and PTS is not yet established. The hypothesis that a number of neuralgic amyotrophy cases may be caused by unrecognized herpes neuritis is tenable, in particular in case of zoster sine herpete.

Conclusion

PTS is a diagnostic challenge because it may mimic several conditions causing pain and weakness around the shoulder, such as rotator cuff tears, acute calcifying tendinitis, impingement syndromes, cervical radiculopathy, tumors of the brachial plexus and spinal cord and compressive nerve injuries. In the reported case, the presence of concomitant shingles was a pitfall, with the potential to mask the underlying PTS and to lead to a misdiagnosis of herpetic brachial plexopathy. A detailed clinical history and physical examination, as well as the help of electroneuromyography were essential to distinguish the neurological structures involved and to ascertain the correct diagnosis.

Table 1. Needle electromyography

	Fib.	PSW	Fasc.	Description and voluntary activity
Days 12 and 14 (left side)				
Supraspinatus	+/-	+/-	0	Simple recording –1 MUP firing at 25 Hz
Infraspinatus	+	+/-	0	Increased insertional activity; poor intermediate pattern
Deltoid	++	+/-	0	Intermediate pattern
Biceps brachii	+	+	0	No voluntary activity
Brachioradialis	+	+	0	Intermediate pattern
Rhomboid major	//	//	//	Clinically normal; no needle EMG
Day 38 (left side)				
Rhomboid major	0	0	0	Interference pattern; normal size MUPs
Serratus anterior (2 sites)	0	0	0	Interference pattern; normal size MUPs
Supra spinatus	++	++	0	Intermediate pattern; high frequencies (25 Hz or more audible)
Deltoid	+++	+++	0	Simple recording; 1 MUP firing at >25Hz
Biceps brachii	++++	+++	0	Increased insertional activity; no voluntary activity
Brachialis	++++	+++	0	No voluntary activity
Brachioradialis	++	++	0	Intermediate pattern with frequencies >25 Hz

Fib. = Fibrillation; PSW = positive sharp wave; Fasc. = fasciculation; MUP = motor unit potential.

0 = No spontaneous activity; +/- = 1 Fib. (or PSW) in 1 region of the muscle; + = 2 Fibs. (or PSWs) in 2 different regions; ++ = >2 Fibs. (or PSWs) in several regions; +++ = abundant Fibs. (or PSWs) in most regions; ++++ = abundant Fibs. (or PSWs) in all regions with no voluntary activity and no response to electrical nerve stimulation; // = not studied.

Table 2. Nerve conduction studies

Sensory	Latency ms	Amplitude μV	Duration ms	Area $\mu\text{V} \times \text{ms}$	Distance mm	Conduction velocity m/s	Temperature $^{\circ}\text{C}$
<i>Days 12 and 14</i>							
Median							
Wrist–digit II L	3.0	8	1.9	5	142	48	34.1
Wrist–digit II R	2.7	6	1.8	6	131	49	33.0
Palm–digit II R	1.5	9	1.5	4	76	51	33.0
Wrist–digit I L	2.3	10	2.2	5	105	46	34.2
Wrist–digit I R	1.8	11	1.6	6	83	45	33.0
Radial							
Wrist–digit I L	2.0	2	1.2	2	100	50	33.9
Wrist–digit I R	1.8	2	1.1	2	85	46	33.0
Ulnar							
Wrist–digit V R	2.3	10	1.7	6	117	52	33.0
<i>Day 38</i>							
Median							
Wrist–digit II L	2.7	8	2.1	4	135	50	31.9
Ulnar							
Wrist–digit V L	2.4	9	2.1	6	125	52	33.0
Motor	Latency ms	Amplitude mV	Duration ms	Area $\text{mV} \times \text{ms}$	Distance mm	Conduction velocity m/s	CMCT (ms)
Ulnar							
ADM L–wrist	2.5	6.8	7.2	21.4	55		
Wrist–elbow below	6.6	6.8	7.5	20.7	220	54	
Elbow below–above	8.2	6.4	7.9	21.8	70	44	
Elbow above–axilla	12.0	6.0	7.2	19.6			
Axilla–elbow	15.1	5.8	7.2	19.6			
F waves ADM L–Wrist	29.8						
Median							
ADM–elbow	9.4	(+)0.1	11.1	(+)0.6			
MEP (TMS)							
ADM L–cortex R	22.0	4.5	9.5	18.9			6.3

Sensory neurography is antidromic; for amplitude, peak-to-peak amplitude was measured; for duration, negative take-off to positive peak duration was measured. Motor neurography: for the compound muscle action potential, amplitude, duration and area of the negative peak were measured. ADM = Abductor digiti minimi; MEP = motor-evoked potential; TMS = transcranial magnetic stimulation; CMCT = central motor conduction time; (+) = positive peak.

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