

C A S E R E P O R T

A Parsonage-Turner Syndrome secondary to Parvovirus B19 infection

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Abstract. Parvovirus B19 (PVB19) is a small DNA virus that causes the fifth disease in children; however it can also affect adults. The infection can be asymptomatic in about a quarter of healthy subjects. Typical clinical manifestations are: short lived fever accompanied by asthenia, myalgias and pharyngodynia; symmetrical acute polyarthritides; megalocytosis in child; maculopapular rash and/or fleeting purpuric at the extremities in adult; adenopathies in the cervical area. Atypical manifestations can affect neurological system (both central and peripheral), heart and kidney. We describe a 37-year-old man with neuralgic amyotrophy (Parsonage-Turner syndrome) caused by Parvovirus B19 infection. (www.actabiomedica.it)

Key words: Parsonage-Turner Syndrome, Parvovirus B19

Introduction

Parvovirus B19 (PVB19) is a small non-capsulated icosahedral capsid linear monocatenary DNA virus that targets erythroid precursors cells. It is the cause of the fifth disease; however, it can also affect adults (1).

The PVB19 membrane receptor is the blood group P antigen found on the surface of red blood cells and erythroblast precursors, but also of megakaryocytes, endothelium, placenta, myocardium and hepatocytes (2).

The biological diagnosis of recent PVB19 infection is evidenced by the positivity of specific IgM. IgG appear approximately 2 weeks after infection, persist throughout life and are theoretically protective, although reinfection is possible in a minority of cases (3). The research of the viral DNA genome by PCR allows to confirm the presence of PVB19 (4).

The incubation period varies from 6 to 18 days. In the viremic phase the virus is detected in oral and nasal

secretions and the patient can transmit the infection. In the eruptive phase and articular manifestations, the patient is no longer contagious (5,6).

The virus is primarily spread by infected respiratory droplets, blood-borne transmission however has been reported (5).

The infection is asymptomatic in about a quarter of healthy subjects and in most cases, there is no characteristic symptom of PVB19 infection (2,5-7). Table 1 reports the typical and atypical clinical manifestations of PVB19 infection (8-25).

Case report

An immunocompetent 37-year old male, with positive familiarity for Rheumatoid Arthritis and Systemic Sclerosis, and otherwise negative pathological anamnesis, presented sudden onset of fever (38.5°C) and after 4-5 days appearance of arthralgias of hands, shoulders and elbows with paresthesia of the right hand.

Table 1. Parvovirus B19 clinical manifestation

Typical	Atypical
Short lived (24-48 hours) <i>fever</i> accompanied by <i>asthenia</i> , <i>myalgias</i> and <i>pharyngodynia</i> (8)	<i>Neurological manifestations:</i>
Symmetrical acute <i>polyarthritis</i> of hands, feet, ankles and knees (80%) that can persist beyond two months in 10-20% of patients (9-13)	- Central Nervous System (15)
<i>Cutaneous manifestations</i> (14):	o Encephalitis
- Child – megallo-erytema	o Meningitis
- Adult – maculopapular rash and/or fleeting purpuric at the extremities	o Stroke
	o Chorea
	o Cerebellar ataxia
	- Peripheral Nervous System (16-19)
	o Guillain-Barré syndrome
	o cranial and peripheral neuropathies
	o brachial plexus neuropathy
	o carpal tunnel syndrome
	<i>Cardiac manifestations</i> (20-24):
	- pericarditis
	- fulminant myocarditis
	- coronary syndrome
<i>Adenopathies</i> in the cervical area (8)	<i>Renal manifestations</i> (25):
	- Proliferative glomerulonephritis
	- collapsing glomerulopathy
	- thrombotic microangiopathy

The second and third right metacarpal joint capsules showed evidence on the ultrasound scan to be swollen.

Research for IgG and IgM antibodies for PVB19 was positive, as well as the DNA in the serum. ANA, ANCA, Rheumatoid Factor and anti-CCP antibodies were negative

Short term corticosteroid treatment with low dose methylprednisolone solved the paresthesias and obtained improvement of arthralgias, but the patient showed difficulties in moving his right shoulder and hand in addition to the appearance of right winged scapula.

On physical examination were observed: hypotrophy of the spinal muscles, deltoid, right brachial biceps; presence of right-wing scapula; deficit of right arm abduction and extension of fingers; non-sensory deficits; hyporeflexia of the upper right limb; hypostenia of the anterior serratus muscle.

The electrophysiological study showed suffering of the right suprascapular nerves and right radial with denervation in progress on the muscles which are innervated (m.infraspinatus and common extensor of the fingers).

The clinical and neurophysiological picture therefore confirmed a Parsonage-Turner Syndrome (neu-

ralgic amyotrophy) involving long, suprascapular, and radial thoracic nerves.

Discussion

The neuroalgic amyotrophy also called Parsonage-Turner syndrome or brachial plexus neuritis is a painful acute neuropathy that usually affects the upper brachial plexus region (26-29). It is a rare disease (incidence of 1/100 000 adults/year) which is not well known and takes a rather long time to make a diagnosis (30).

The typical form usually develops in three phases: a painful phase; a motor deficit phase associated with muscle weakness, amyotrophy and sensory disturbance; and a recovery phase (31).

Pain is the first symptom in 90% of the cases with a brutal, severe, neuropathic onset and affects the shoulder girdle. This phase that lasts from one day to two months is longer in men than in women. Muscle weakness arises in a short time from the moment pain occurs. Amyotrophy usually appears within 2 and 6 weeks and shows the importance of the axonal part. This is evident for superficial muscles (deltoid muscles and supra- and sub-spinous muscles) or totally invisible in deep muscles (big toothed muscles and prona-

tor quadratus). Muscle involvement can correspond to a nerve root (C5, C6, C8, T1), a portion of the brachial plexus (it is more common in the upper part), a nervous trunk or certain nerve bundles (32-36). Sensitive disorders are present in 66-78% of cases, however functional consequences are uncommon (37). In 75% of the cases a good or complete recovery is obtained in 6 months to 3 years. Quality and recovery time depend directly on the severity of the initial involvement and the speed of the nervous regrowth (38).

Since the first description of 1948 various triggering factors have been identified: traumatism, surgery, pregnancy, antitetanic serum therapy, vaccinations, bacterial and viral infections (including PVB19). Hereditary forms are also reported (39).

Further investigations are advisable in order to confirm the peripheral character of motor and sensory deficit and to exclude other possible diagnosis (40,41). In our case, the presence of fever and the subsequent development of arthritis and right upper limb paresthesia, according to previous reports (9-13, 16-19), raised the suspect of a PVB19 related disease.

The pathophysiology is still uncertain as it could be a reactive inflammation of disimmune origin or a direct effect of the virus on the nerve. However, the two could coexist (42).

The treatment is still rather empirical and varies according to the phase in which the patient is evaluated.

In the painful phase, a short corticosteroid treatment may be useful, and it seems to reduce the duration of the phase and favor recovery (43). Our case report confirms the positive effect of a short term corticosteroid treatment, even if the improvement was only partial and the motor and sensory deficit was not avoided.

Some authors reported that more severe forms could respond to pulse iv high dose corticosteroid and iv immunoglobulins (44,45).

A rehabilitation protocol can be established that involves an antalgic physiotherapy, a trophic massage and muscle strengthening.

The description of a case of PVB19 infection that preceded a Parsonage-Turner syndrome and the polymorphism of the clinical signs of PVB19 infection suggest a systematic search for the virus in case of Parsonage-Turner Syndrome.

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