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Case report

Parvovirus B19 infection as a cause of acute myositis in an adult

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ABSTRACT

Parvovirus B19 infection is often asymptomatic, but clinical expressions may include transient aplastic crisis, erythema infectiosum, non-immune hydrops fetalis, and chronic red cell aplasia. This virus has also been associated with rheumatoid arthritis and other autoimmune connective tissue diseases; however, we could not identify any acute adult myositis case developed after a Parvovirus B19 infection in the literature. For this reason, we would like to present a rare case of acute myositis developed after Parvovirus B19 infection. In patients presenting with symptoms of fever, rash on the legs and myositis, viral infections such as Parvovirus B19 should be kept in mind.

Infecção por parvovírus B19 como causa de miosite aguda em um adulto

Palavras-chave:
Parvovírus B19
Miosite aguda do adulto
Febre

RESUMO

A infecção pelo Parvovírus B19 costuma ser assintomática, mas as expressões clínicas podem incluir crise aplástica transitória, eritema infeccioso, hidropisia fetal não imune e aplasia crônica da série vermelha. Esse vírus também se associa à artrite reumatoide e a outras doenças autoimunes do tecido conjuntivo; entretanto, não conseguimos identificar na literatura nenhum caso de miosite aguda em adulto desenvolvida depois de infecção pelo Parvovírus B19. Por essa razão, gostaríamos de apresentar um caso raro de miosite aguda desenvolvida depois de infecção pelo Parvovírus B19. Nos pacientes que apresentam sintomas de febre, *rash* nas pernas e miosite, devem ser consideradas as infecções virais, como a causada pelo Parvovírus B19.

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Introduction

Parvovirus B19 (B19), a non-enveloped 22–26 nm icosahedral single-stranded DNA virus discovered in 1975, is the only member of the family Parvoviridae known to be pathogenic to humans.[1] This infectious disease is generally seen during childhood, but rarely in adults. Infection appears to confer lifelong immunity in immunocompetent hosts. Although the seroprevalence is high, viremia or presence of viral DNA in peripheral blood is rare in healthy individuals.[2] B19 is transmitted through the respiratory route, but can also be transmitted vertically from mother to fetus, through bone marrow and organ transplantations, and via transfused blood products.[2] Most cases of B19 infections are asymptomatic and resolve spontaneously in adults. But sometimes B19 causes a transient aplastic crisis, erythema-infectiosum (fifth disease), non-immune hydrops fetalis and chronic red cell aplasia. This virus has also been associated with rheumatoid arthritis and other autoimmune connective tissue diseases;[3] however, we could not identify any acute adult myositis cases developed after B19 infection. In the literature, for that reason, we would like to present a rare case of acute myositis developed after B19 infection.

Case report

A 38-year-old male patient was admitted to the internal medicine polyclinic with complaints of high fever and malaise. These, plus diffuse pain, had started 3 days before and became increasingly severe. He had no history of diseases or medicine use. In the physical examination, his auxiliary body temperature was 39 °C, and other systemic examinations were normal. The laboratory tests revealed increased levels of alanine aminotransferase (ALT), aspartate aminotransferase (AST), and C-reactive protein (CRP). Leukopenia, thrombocytopenia, and monocytosis were found in his hemogram (Table 1). A slight increase in monocytes was found in his peripheral blood smear. Blood culture taken during his high fever period showed no growth. Heterophile antibody (monospot) was negative.

Maculopapular rashes developed under both knees but his fever decreased within the next 2 days. B19 antibody IgM tested positive, and thus B19 was considered to be the cause of the rashes. On the third day of the disease, arthralgia developed in both ankles, and also in both hands. On the sixth day, the rashes remitted, replaced by pain in both calves so severe that it prevented him from walking.

In the investigations performed for the follow-up, AST, ALT and creatine kinase (CK) were found to have decreased to within normal ranges, but CRP and erythrocyte sedimentation rates (ESH) had increased (Table 1). The patient was hospitalized, and naproxen sodium 1100 mg/day was initiated. The bilateral cruris MRI taken due to continuing severe pain on his bilateral calf muscles revealed thickening and edema in both gastrocnemius and the semimembranosus muscle group, an increased signal in T2, and an edematous appearance. The minimal contrast involvement in the post-contrast series was considered to be myositis (Fig. 1). Follow-up on the 10th day of the treatment revealed reduced pain, and ESH and CRP had returned to within their normal ranges.

Discussion

B19 is common worldwide, and the seroprevalence increases with age, so that 15% of preschool children, 50% of younger

Table 1 – Test results of the patient.

Test	1st day	3rd day	8th day	10th day	17th day	Normal range
AST (U/L)	67	37	28	30	16	10–50
ALT (U/L)	98	77.5	57.7		34	15–38
LDH (U/L)	260		254	246	190	0–225
CK (U/L)	149			52	51	39–308
CRP (mg/dL)	2.4	1.74	1.76		5.9	0–0.5
ESH (mm/h)	10			42	90	<15
WBC ($\times 10^3/mm^3/\mu L$)	3.73	5.35	6		7.02	3.8–10
Neutrophils (%)	40.5	59	55		40	50–80
Lymphocytes (%)	26.8	25	29		43	10–48
Monocytes (%)	28	9.7	12		12.7	2–10
HGB (g/dL)	16.7	15.9	14.3		14.1	13–17.5
Platelets ($\times 10^3/mm^3/\mu L$)	123	124	274		321	142–424
Parvovirus B19 IgM		76.9 positive				<9 U
Parvovirus B19 IgG		11.8				<9 U
Herpes Simplex Type1 IgM			0.27			<1.1 ratio
Herpes Simplex Type1 IgG			>200			<22 RU
Herpes Simplex Type2 IgM			0.16			<1.1
Herpes Simplex Type2 IgG			<2			<22 RU/mL
Ebstein–Barr virus VCA-IgM			0.34			<1.1
Ebstein–Barr virus VCA-IgG			84.86			<22 RU/mL

ALT, alanine aminotransferase; AST, aspartate aminotransferase; CRP, C-reactive protein; CK, creatine kinase; ESH, erythrocyte sedimentation rates; WBC, white blood cell; HGB, hemoglobin.

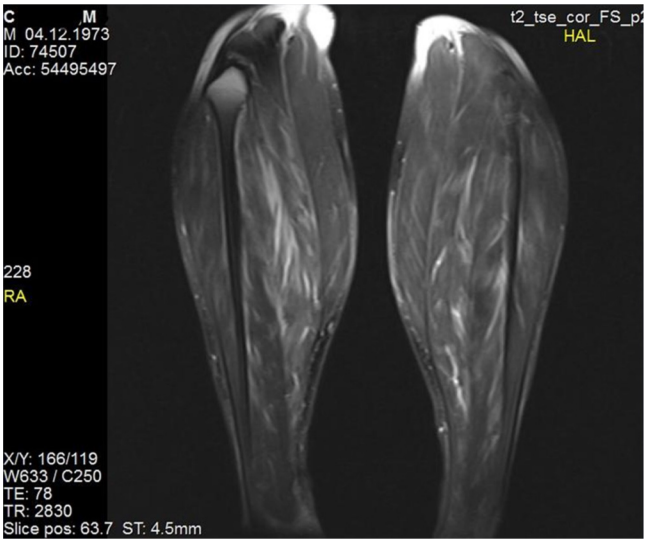

Fig. 1 – Both lower limbs' MRI images of the patient.

adults and about 85% of the elderly show serologic evidence of past infection.[2] This infectious disease is generally seen during childhood, but rarely in adults. Laboratory diagnosis of B19 can be performed using serology, PCR, histopathologic examination and immunohistochemistry.[2] The precise diagnosis of recent or past infection with B19 depends on the use of enzyme immunoassays to detect anti-B19 IgM and IgG in plasma. To confirm acute B19 infection, IgM antibodies must be detected in plasma or serum. These antibodies are synthesized approximately 7–10 days after the high-titer viremia.[4] B19 IgG appears shortly after IgM and persists lifelong with slowly decreasing titers unless boosted by subsequent encounters with the virus. The clinical significance of different serum-titers of B19 DNA is not fully established. In healthy individuals with acute B19 infection, viral titers as high as 10^{12} gEq/mL are detectable in blood.[3,4]

Benign acute myositis is a self-recovering [benign] condition characterized by acute bilateral calf pain and difficulty in walking that usually develops in school-age children after they have recovered from viral infections. It was first recognized in 1957 by Lundberg in 74 pediatric subjects.[5,6] Myositis cases coincide with dermatomyositis and systemic lupus erythematosus after Parvovirus B19 infection in adults.[7] Although the most common viral causes of acute myositis are Influenza A, B (including the H1N1 virus) and Coxsackievirus viruses in children, no case report was found about B19 associated with benign acute childhood myositis.[8] However, there is one case report about myofacitis associated with B19 in a healthy adult.[9] But, to our knowledge, literature reports no cases about myositis associated with B19 in an adult. As we know B19 infection occurs especially in transplant patients, patients with systemic diseases and immunocompromised patients.[4] In our case, although there was no history of illness and drug intake, history of intense pace of work, intense stress and a 12-h trip might have a negative impact on immune resistance and may cause myositis associated with B19.

In our patient, clinical and laboratory findings, magnetic resonance imaging and response to therapy support the diagnosis of acute myositis. The expected increased sedimentation and CRP were found; however, the CK level had not increased in our patient. The absence of increased CK levels may lead to confusion. From the patient's laboratory follow-up, we could see that CK level was three times higher in the first period. But CK level was normal in every period of the disease. Although most patients with inflammatory myopathies present with increased creatine kinase levels, reported series include some patients with normal creatine kinase levels.[9–11] In these cases, even though myopathy was diagnosed by methods such as biopsy and MRI, CK levels were normal. And concrete evidence was not submitted to explain this situation. In an article presenting seven patients with myositis, a lack of creatine kinase elevation in patients with myositis was found to be a poor prognostic sign.[10] Our case had a benign course and clinical findings improved quickly.

Despite the similarity with childhood benign acute myositis cases, our case was thought to be interesting because it was in an adult with no increased CK level. In patients who present with fever, rash on the legs, and myositis (such as in our case), viral infections such as B19 should be kept in mind.

Conflicts of interest

The authors declare no conflicts of interest.

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