mm^3, plaquetas 538 mil/mm^3, DHL 518 U/L, TAD negativo e HbS de 24,8%. Optado por observação e IGIV sob suspeita de SHH visto queda rápida de Hb. Após 5 dias de IGIV ele recebeu nova transfusão com Hb pré de 6,8 g/L e após transfusão 5,9 g/L e HbS 52,1% confirmando o diagnóstico de SHH. Paciente recebeu rituximabe 1000 mg, 2 infusões com intervalo de 2 semanas entre as mesmas com resolução de quadro hemolítico e independência transfusional (HbA1 65,6% e HbS de 29,8%). **Discussão:** SHH é diagnosticada quando ocorre anemia grave paradoxalmente após uma transfusão de sangue (Hb pós < Hb pré-transfusão). Resulta em hemólise concomitante das hemácias transfundidas e autólogas. Acredita-se que o mecanismo subjacente, a esta rara e geralmente fatal complicação, seja secundário a resposta imune contra os fosfolipídios expostos da membrana eritrocitária. A predisposição para SHH na AF também é variada e a busca por um padrão ou valor de predição tem sido evasiva. Pode ser dividida em 2 formas baseadas no tempo de transfusão até o desenvolvimento dos sintomas e formação de aloanticorpos. Aguda: sintomas clínicos são observados dentro de 7 dias pós transfusão. Os aloanticorpos geralmente não são formados nesse período e, portanto, um teste direto de antiglobulina (TAD) para detectar aloanticorpos provavelmente seria negativo. Tardia: sintomas clínicos são observados além de 7 dias após transfusão, e formação de aloanticorpos é mais provável. O TAD neste caso provavelmente seria positivo. Neste relato, a forma aguda foi observada porque os sintomas foram observados 5 dias após a transfusão e o TAD foi negativo. O tratamento visa suporte e evitar transfusões. IGIV e esteroides em altas doses mostram bons resultados em casos leves por suprimir a ativação macrofágica, encurtando a duração da hemólise. O Rituximabe também pode ser usado nestes casos de SHH e AF, como neste relato, com bons resultados. **Conclusão:** SHH é rara e necessita de pesquisas mais extensas para determinar indivíduos com maior predisposição a essa complicação de alta mortalidade. O alto índice de suspeição pode ser fundamental no manejo bem como monitorar pacientes com múltiplas transfusões.

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TWO CASE REPORT OF PARVOVIRUS B19 INFECTION IN HIV-INFECTED PATIENTS: DIFFERENT CLINICAL PRESENTATIONS FOLLOWING ACUTE AND PERSISTENT B19V INFECTION

DPM Almeida, J Bokel, ADR Alves, AG Vizzoni, ICF Tavares, JDSB Netto, B Grinsztejn, LAA Leon

Fundação Oswaldo Cruz (Fiocruz), Rio de Janeiro, RJ, Brazil

Introduction: *Parvovirus B19* (B19V) infection could cause clinical manifestations depending on the host's immunologic and hematologic status. In immunocompetents individuals, clinical manifestations range from asymptomatic, or flu-like symptoms in childhood including a viral exanthem (fifth disease) to a fatal hydrops fetalis in pregnant women. B19V infect red blood cell (RBC) precursors and in immunocompromised hosts, such as HIV-infected patients, it could be manifested as pure red cell aplasia and chronic anemia. B19V may cause transient aplastic crisis (TAC) in patients with increased RBC destruction, including hereditary spherocytosis (HS). **Objective:** To report cases of B19V infection in HIV-positive adults in follow-up at Instituto Nacional de Infectologia Evandro Chagas. **Results:** Case 1: A 27-year-old black male with HIV since childhood on irregular use of antiretroviral therapy (ART) complained of vertigo, headache, vomiting, fever. In April 2021 he had Hb 1,7 g/dL, Ht 6.5%, reticulocytes 0%, HIV viral load 40,305 cps/mL and $CD4^+$ count 6 cells/μL. Up to October 2021 he required multiple RBC transfusions. Myelogram showed giants proerythroblasts with nuclear inclusions, B19V real time polymerase chain reaction (qPCR) detected 4.3×10^{10} IU/mL, and B19V anti-IgM was negative. He received intravenous immunoglobulin (IVIG) and stabilized RBC counts. Nine months after treatment, he remained asymptomatic but had 11 cells/ μL CD4+ count and 27,642 HIV cps/mL due to poor adherence to ART. B19V was still detectable in qPCR (1.8×10^6 IU/mL), but he did not receive any additional treatment because blood counts were normal. Case 2: A 61-year-old white male with HIV since 2009 on regular use of ART, undetectable HIV viral load, but historically low $CD4^+$ count, presented four weeks of lethargy, and an acute incident of dizziness, diarrhea, and syncope. He exhibited pancytopenia (Hb 5.6 g/dL, Ht 15.2%, WBC 1.48×10^9/L, and platelets 41,000 per mm^3), non-immune hemolytic anemic features, jaundice, and palpable spleen. Reticulocyte counts were unavailable. He received transfusions of RBC, the HIV viral load at admission was undetectable, and $CD4^+$ count 127 cells/ μL. The myelogram presented giant proerythroblasts with nuclear inclusions, and bone marrow had 95% cellularity and mild erythroid hyperplasia. B19V anti-IgM was indeterminate, while B19V qPCR detected 8.9×10^3 IU/mL, in August 2021. The patient received IVIG and nine months later, showed resolution of pancytopenia, but maintained elevated indirect bilirubin, indicative of chronic hemolysis. B19-DNA was no longer detected by qPCR, hematoscopy showed spherocytes, and the osmotic fragility test suggested RBC membranopathy, revealing an undiagnosed HS. **Discussion:** Anemia is a common finding in the HIV setting, is often multifactorial, and represents a significant risk factor for mortality in AIDS. Our study highlights the importance of B19V infection be considered as a part of the differential diagnosis of anemia in this group. Clinicians should search for typical bone marrow and virologic findings. Identification of B19V DNA by qPCR was essential herein because of low sensitivity of serologic tests. **Conclusion:** Our findings showed that adherence to ART and the status of immunosuppression were crucial to the parvovirus clearance after IVIG. These cases highlight the challenging management of refractory parvovirus disease in immunosuppressed patient.

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CARCINOMA CELULAR SECUNDÁRIO A ÚLCERA CRÔNICA NA DOENÇA FALCIFORME

SMCBP Jesus, P Vicari, MS Figueiredo

Universidade Federal de São Paulo (UNIFESP), São Paulo, SP, Brasil