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Scientific comment

Comments on the clinical and laboratory characteristics of patients with dengue hemorrhagic fever manifestations and their transfusion profile☆

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Transmitted by mosquitoes of the genus *Aedes*, dengue fever is a viral disease caused by one of four serotypes: DENV1, DENV2, DENV3 and DENV4.[1] It is considered a public health problem worldwide. It is estimated that about 40 million symptomatic infections occur annually, of which about 2 million require hospitalization,[2] resulting in about 20,000 deaths.[3]

The infection is characterized by a broad spectrum of clinical presentations, ranging from mild foot swelling to death.[4] Thrombocytopenia is a prominent feature of infection.[5] A platelet count less than 100×10^9/L is a diagnostic criterion for dengue hemorrhagic fever (DHF).[6] However, severe thrombocytopenia may be observed both in dengue fever (DF) and DHF. There is a significant negative correlation between disease severity and platelet count.[7] Thrombocytopenia and hypofibrinogenemia are responsible for changes in hemostasis but are not considered predictive markers of hemorrhage.[8] The cause of thrombocytopenia in dengue is multifactorial and not fully understood; the mechanisms involved are bone marrow suppression and peripheral platelet destruction. Peripheral immune complex mediated destruction is probably the main contributor to thrombocytopenia in dengue infection.[8]

The characteristic clinical manifestations of dengue are fever, headache, retro-orbital pain, general malaise, arthralgia, rash, pruritus, diarrhea, nausea, respiratory distress, dry cough, painful hepatomegaly, continuous abdominal pain, vomiting, postural dizziness, sweating, hypothermia and bleeding.[9] Hemorrhagic manifestations, such as epistaxis, gingival bleeding, menorrhagia, gastrointestinal bleeding, intracranial bleeding, hematuria, effusions and spontaneous bleeding in places of venipuncture[10] can be used as warning signs in the evolution of dengue.[11] Among laboratory findings, an increase in hematocrit, leukopenia, relative lymphocytosis, atypical lymphocytes, low platelet count, prolonged prothrombin time (PT) and activated partial thromboplastin time (aPTT) and increased serum aspartate aminotransferase (AST), serum alanine aminotransferase (ALT), alkaline phosphatase and serum gamma-glutamyl transferase (GT) range and decreased serum albumin may occur.[9-11]

Prophylactic transfusion of platelet concentrate (PC) is indicated in patients without any history of bleeding but with a platelet count lower than 20×10^9/L, and transfusion therapy in patients with chronic active bleeding and platelet count lower than 50×10^9/L.[12] Several studies have shown that there is a correlation between the bleeding degree and the platelet count.[13-15] The prevention of bleeding in DHF should be directed to the early recognition of shock and immediate correction with PC of and/or fresh frozen plasma (FFP) transfusions.[13,16,17] The monitoring of hematocrit in series, which reflects the degree of plasma leakage, at the expense of monitoring thrombocytopenia and coagulopathies results in a reduction in the use of blood products.[17] Fugimoto & Koifman, based on secondary data from patients with DHF admitted to hospitals in Rio Branco, Acre, reported that the

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clinical and laboratory profile of patients is similar to those described in the literature.[18] As the transfusion of FFP and PC proved ineffective, there is currently limited support for the use of prophylactic PC transfusions in cases of dengue, despite its inclusion in some national guidelines. Therefore, it is necessary to conduct clinical trials to build an evidence base to guide the appropriate use of platelets in dengue. In summary, in the opinion of many authors prophylactic platelet transfusions in patients with stable dengue and without risk of bleeding can be avoided without affecting patient safety factors.

Conflicts of interest

The author declares no conflicts of interest.

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