Hematological and biochemical findings in patients with dengue fever: a current issue

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Dengue fever epidemics pose a serious issue in public health as they are conditioned to the existence of the so-called social determinants in the health-disease process, which potentiate the emergence of new mosquito breeding sites, limit the results of vector control measures and hamper access to health services. Among the social determinants in Brazil, the accelerated population growth in urban centers, the still considerable intermittence in potable water supplies and the collection and disposal of solid waste are worth mentioning. Although presenting significant improvements over the last decade, these social constraints will unfortunately last for many years, especially in the outskirts of most medium and large cities.

Since the reintroduction of dengue fever in 1986, about seven million cases of the disease have been reported in Brazil, thousands of which are considered dengue hemorrhagic fever according to the former classification of the World Health Organization. Since the epidemics recorded from 2007 to 2009, signs of worsening clinical forms have been seen, especially in under 15-year-old patients. The magnitude of the epidemics and the resulting deaths have had a powerful impact on the healthcare network, overcrowding health facilities with suspected cases and, in some instances, triggering real social upheaval. The absence of specific treatment and effective prevention reinforce the relevance of studies involving still unknown aspects of the severe forms of the disease. Thus, the article "Dengue: hematological and biochemical profile dynamics"[1] is an important contribution to the management of the disease.

Although not completely understood, the pathophysiology of dengue fever has organic and tissue impacts from the direct action of the virus to immune mechanisms triggered by the infected organism, mainly resulting from the presence of antibodies against viral proteins. Antibodies produced against nonstructural protein 1 (NS1) also react with platelets and endothelial cells and other viral proteins (core, prM, and E) present sequences homologous to diverse coagulation molecules.[2-4]

The variable degrees of bleeding seen in dengue fever arise from defects in the first phase of hemostasis as a result of thrombopathy and vascular abnormalities, but also from more serious flaws in blood coagulation, such as disseminated intravascular coagulation. Coagulopathy in dengue hemorrhagic fever has been associated with a reduction in antithrombin III and α-2-anti-plasmin. There are mild or moderate reductions in factors II, V, VII, VIII, IX, X and XII and a slight increase in D-dimer. In necropsies of patients with dengue shock, widespread fibrin thrombi were seen in the lungs, bone marrow, kidneys, adrenal gland and brain.[5]

Histopathological studies suggest that dengue-infected patients have some bone marrow suppression characterized by generalized hypoplasia by the fourth day after the

onset of fever which returns to normal cellularity between 7 and 10 days after onset.[6]

Several infectious diseases should be considered when laboratory changes similar to those found in dengue are observed. AIDS and other arbovirus infections should be listed as possible differential diagnoses; in addition, in the current epidemiological context, the overlapping of endemic areas for dengue fever and leishmaniasis in Brazil is worth considering.

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