

Immature Platelet Fraction – A Simple and Useful Novel Marker in Dengue Hemorrhagic Fever

In dengue fever, thrombocytopenia is ascribed to destruction of platelets by antiplatelet antibodies, disseminated intravascular coagulation, marrow suppression and peripheral sequestration of platelets. Platelet counts vary considerably during the course of illness, and patients may require platelet transfusion when the counts are $<10000/\text{mm}^3$ [1]. Platelet count is expected to rise in the late critical or the recovery phase of the infection. Immature platelet fraction (IPF, the percentage of immature platelets) can be used to fairly predict the rise or fall of platelet count during the course of dengue fever [2]. IPF defines the immature and larger platelets that have been recently released from the marrow, and have much larger RNA content than the mature platelets. A high IPF is usually found in either consumptive or recovering thrombocytopenic disorders, while a low IPF is characteristic of bone marrow suppression [2]. IPF is identified by simple flow cytometry technique and the use of a nucleic acid specific dye (e.g., oxazine dye 0.0003%) in the optical platelet channel which is available in most hematology laboratories. The test is simple, inexpensive and reproducible [3]. An IPF reference range in healthy

neonates is 4.1 ± 1.8 , and in children is 2.7 ± 1.3 [4]. IPF has been shown to have a strong correlation with the recovery of platelet counts in patients with dengue fever [5]. Patients with no warning signs or symptoms but with NS1 positive and borderline platelet count keep the treating pediatrician under dilemma – whether to admit the patient or to observe. Performing IPF in dengue patients may help in decision for admitting or monitoring during recovery in dengue fever.

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Infantile Tremor Syndrome: A Syndrome in Search of its Etiology

Indian Pediatrics has done a great service by revisiting 'Infantile Tremor Syndrome' (ITS) [1]. Even though, ITS has existed for almost 60 years, it continues to be perceived as a syndrome of unknown etiology. However, there is now enough epidemiological, clinical, laboratory and therapeutic evidence in the literature to support vitamin B₁₂ deficiency as the cause of ITS.

Epidemiologically, ITS occurs in exclusively breastfed infants of strictly vegetarian mothers. As a

result, these infants are predisposed to develop vitamin B₁₂ deficiency. Clinically, symptoms and signs of ITS are similar to those of vitamin B₁₂ deficiency in infants. Many studies in the past excluded vitamin B₁₂ deficiency in ITS on the basis of mere absence of macrocytosis or megaloblastic bone marrow. Absence of these features does not exclude the diagnosis of vitamin B₁₂ deficiency which requires serum vitamin B₁₂ measurement. Several studies [2-4] have consistently demonstrated low serum vitamin B₁₂ in these infants. Study by Bajpai, *et al.* [5] is the only report to have found normal serum vitamin B₁₂, but only 20 (15%) of 134 infants were tested in this series. Additionally, in some studies from India, infants with megaloblastic anemia due to vitamin B₁₂ deficiency