Letter to The Editor

Persistent Thrombocytopenia Following Dengue Shock Syndrome

Sir,

We read with great interest the clinical brief by Kohli *et al* on "Persistent Thrombocytopenia Following Dengue Shock Syndrome".[1] The case report provides an excellent overview of the pathogenesis of thrombocytopenia in dengue hemorrhagic fever. It also rightly emphasizes the importance of monitoring post- recovery platelet counts, in order to anticipate and prevent the serious complications of thrombocytopenia.

We would like to share our views in this regard.

The pleural effusion in dengue fever is due to capillary leak mechanism and is a transient, self-limiting phenomenon. Hence there is no need for thoracocentesis routinely. But in the case presented, where the patient was having increased respiratory distress secondary to pleural effusion thoracocentesis may have helped.

There are some unusual features which are seen in this patient. Despite the presence of hypotension and fever, the patient had a disproportionately low heart rate. Bradycardia is a known phenomenon in the convalescent phase in dengue fever.

Repeated platelet and fresh frozen plasma transfusion are known to cause acute respiratory distress syndrome (ARDS) and acute lung injury. Hence transfusions should be given only when it is indicated and the possibility of acute lung injury should be kept in mind.[2]

It has not been mentioned anywhere in the case report whether this patient had retinal bleeds. The presence of retinal bleeds alerts the physician to the possibility of intracranial bleeding and this is known as Terson syndrome, which is the presence of any intraocular hemorrhage occurring with intracranial haemorrhage and elevated intracranial pressures.[3] Hence in patients having thrombocytopenia, regular fundus examination is an easy way to screen for intracranial bleeding.

The persistence of thrombocytopenia in the case presented could be attributed to alloimmunisation secondary to repeated platelet transfusions. Refractoriness may lead to fatal bleeding complications like intracranial bleeding as in this patient. Alloimmune platelet refractoriness can be prevented by using leucocyte-depleted blood products. Steroids do not have much of a role in the management of platelet refractoriness secondary to alloimmunisation. Intravenous gamma globulin has been proposed as a modality of treatment in such cases.[4]

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Author's Reply

Sir,

We would like to thank the authors for the interest in the report[1] and raising several important issues. The authors have rightly pointed out that relative bradycardia is a known phenomenon in the convalescence phase of dengue. The fact that the child responded to antimicrobials and recovered from ARDS even when PRP infusions were continued due to extremely low platelet counts (~10,000/mm^3), would clearly indicate that multiple PRP infusions were not the cause of ARDS in this patient. Several factors led us to decide in favor of PRP administration in this patient. The patient presented with spontaneous clinical bleeding (epistaxis). Thoracocentesis is an invasive procedure and PRP was administered during the acute phase to decrease the risk of bleeding during that procedure. During convalescence, the platelet counts were in the range (~10,000/mm^3) where risk of spontaneous intracranial bleeding was believed to be significant and PRP was administered to prevent this complication. We did not find retinal hemorrhages on multiple funduscopic examinations and not all patients with intracranial hemorrhage have concomitant retinal hemorrhage. However, we agree with the authors that retinal hemorrhage, if present, can alert the physician to the possibility of the presence of intracranial hemorrhage.

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