The Potential of Immature Platelet Fraction as a Prognostic Marker in Dengue Infection

Sir,

Dengue is a viral infection caused by dengue virus (DENV) and characterised by thrombocytopenia and platelet dysfunction. The exact mechanism by which DENV leads to clinical outcomes is not well understood. It is hypothesised that thrombocytopenia caused by DENV may be due to both underproduction and hyper-destruction. The virus particle itself may infect the hematopoietic progenitors leading to inhibition of production or may trigger the formation of antiplatelet antibodies leading to peripheral destruction.

Dengue infection begins as a febrile illness progressing either to a critical phase characterised by hemodynamic instability or a recovery phase characterised by the resolution of symptoms.

Owing to high patient admission rates with dengue infection due to fear of progression of the disease, immature platelet fraction (IPF) can be vital to predict recovery in dengue-related thrombocytopenia and thereby reduce admission rates, admission durations, and need for unnecessary platelet transfusions risking infections and fluid overload. The direct cost per patient of dengue infection in Pakistan in 2011 was Rs. 35,823 (equivalent to US$358). A total of 48,906 cases were reported in 2021, with 183 fatalities.

Identifying prognostic markers that can predict disease progression and outcomes in dengue patients is of utmost importance for timely intervention and optimal patient care. Moreover, in light of the yearly large outbreaks of dengue infection in Pakistan, the need for prognostic markers becomes even more critical.

Various studies have discussed the role of IPF as a prognostic indicator. According to a study by Looi et al., an increase in IPF occurs three days before platelet recovery. IPF >10% was observed in studies by Dadu et al. and Wayez et al. predicting platelet recovery within 48-72 hours.

Utilising IPF as a prognostic marker, we could improve the efficiency of risk stratification in dengue patients, allocating resources effectively. It can help us understand the pathophysiology of the disease better as to whether the cause of thrombocytopenia is hyper-destruction or hypo-production. More importantly, it can catalyse research efforts to improve clinical guidelines for managing dengue and contribute towards evidence-based medicine.

We believe this topic has important implications for managing dengue disease in Pakistan and warrants further research. We hope this letter contributes to the ongoing dialogue in the field and encourages future research collaborations and initiatives.

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REFERENCES