**Pseudothrombocytopenia in Type 1 Diabetes**

A 5-yr-old girl diagnosed with Type 1 diabetes (T1D) since 1½ yr of age, and treated elsewhere with premixed insulins presented to our emergency department with complaints of vomiting, abdominal pain and altered sensorium for 1 day. She was treated for moderate diabetic ketoacidosis based on high blood glucose, positive urine ketones and metabolic acidosis (pH 7.107, HCO3 11.4 mEq/L). Her blood counts showed hemoglobin of 10.7 g/dL, platelet count of 86×10⁹/L and total leucocyte count of 6×10⁹/L. A repeat platelet count was 64×10⁹/L, and peripheral smear suggested clumping of platelets. There were no bleeding manifestations. Suspecting ethylene diamine tetra-acetic acid (EDTA) dependent pseudothrombocytopenia (PTCP), the sample was repeated in EDTA, heparin, and citrate vials which showed platelet counts of 78×10⁹/L, 408×10⁹/L and 416×10⁹/L, respectively. A diagnosis of EDTA - dependent PTCP was made and further workup for etiology of thrombocytopenia was withheld. The child was discharged after switching to basal bolus insulin regimen for a better glycemic control.

PTCP is a relatively uncommon laboratory phenomenon with estimated prevalence of 0.1%-0.3% in adults (1). Of the three types, namely EDTA-, heparin- and citrate-induced, the EDTA-PTCP is the most common [1,2]. The PTCP results from in vitro agglutination of platelets caused by IgG or IgM autoantibodies predominantly directed against epitopes on platelet surface glycoprotein (GP) IIb or IIIa [2]. EDTA induces a conformational change in GP IIb/IIIa, exposing these epitopes and resulting in platelet agglutination at low temperature [2]. This phenomenon is probably related to naturally occurring antibodies that cross react with platelet cryptoantigen exposed due to the effects of EDTA [2].

PTCP is an extremely rare condition in children and is described in association with autoimmune, neoplastic, chronic inflammatory and infectious diseases [3]. We could not find any previous reports of its occurrence in children with T1D; although, true thrombocytopenia of autoimmune and viral etiologies has been described in T1D [4]. There is a single report of an adult with T1D who developed PTCP after change in insulin therapy from premixed to basal bolus regimen [5]. However, he showed PCTP in both EDTA and heparinized samples unlike with only EDTA as in our patient [5]. Additionally, there was no recent change in insulin regimen in our patient. Thrombocytopenia without a bleeding diathesis should alert the attending physician to possibility of PTCP.

**REFERENCES**