Unnecessary transfusion in a patient with pseudothrombocytopenia

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Introduction

Pseudothrombocytopenia (PTCP) is a condition which is secondary to anticoagulant activated platelet agglutination that results in a low platelet count by automated analyzer.

Case Report

A 46 year old African American male with Past History of HIV with unknown CD4, Ex: IPVDA on Methadone Maintenance Program, Hepatitis C, Chronic Renal Insufficiency and Latent TB (treated) was admitted to the medical floor with progressive swelling of both lower extremities and genitalia for 4 weeks. The patient denied having any orthopnea, paroxysmal nocturnal dyspnea, palpitation, exertional dyspnea, chest pain, diarrhea, nausea, vomiting, any rash or bleeding.

On Physical examination vital signs were stable. Skin examination was normal with no petechiae or ecchymosis or purpura. Cardiac exam revealed Grade 3-4/6 diastolic murmur at the aortic area without radiation and there was no gallop and jugular venous distention but had 3+ pitting edema of bilateral lower extremities and edematous scrotum and penis. Abdominal examination was within normal limits and gastric was negative.

Laboratory examination showed WBC of 6700/cubic mm with Hemoglobin of 8g/dL, Platelet of 19000/cu mm with clumping and Reticulocyte count of 2.1 %. Urinalysis showed trace protein with 2 WBCs per high power field and no RBCs. His uric acid was 26 and creatinine was 2.9 with other electrolytes within normal range. Proteinuria was not in the nephrotic range.

At admission, many differentials were thought of as Rule out nephropathy sec to HIV or HCV or Congestive heart failure. Anemia and thrombocytopenia was attributed to his medical problems and was thought as multifactorial. In view of his abnormal renal function and thrombocytopenia, Hematology evaluation was requested and after evaluating peripheral blood smear, Thrombotic Thrombocytopenic Purpura (TTP) was ruled out.

The third day the patient had platelet count of 8000/cu mm. As TTP was ruled out we made an assumption that his thrombocytopenia related to HIV vs. HCV and patient was transfused with 6 units of platelet as his platelet counts were below 10,000. After transfusion his platelet even went further down and led us to think about other causes. One of our differentials was idiopathic Thrombocytopenic Purpura (ITP) and steroid was started. On the fourth day in view of repeated platelet clumping in peripheral blood smear pseudothrombocytopenia was considered. Peripheral blood smear was reviewed by hematologist again and actual platelet count interpreted as 140,000/cu mm after which steroid was discontinued.

Thoracic echo was done that revealed 2 mobile masses at aortic valves which were confirmed later by transeosophageal echocardiogram as 10mm x 7mm and another smaller one. Vancomycin and Cefazidine were empirically started but serial blood cultures were negative and even we considered fastidious organisms as cause of endocarditis and work up done but all were negative. Nephrology evaluation was done and suggested renal biopsy. Patients' ischemic loops were obtained from his PMD at another hospital and was found that patient had Streptococcus Bovis endocarditis and was treated with ceftriaxone for 4 weeks and was discharged 5 days before coming to our hospital which patient never revealed. Patient was then discharged on the 5th day of hospitalization with advice to follow with his PMD.

Discussion

Pseudothrombocytopenia is an in vitro phenomenon where anticoagulant activated platelet agglutination results in spuriously low platelet count by automated analyzer. This usually occurs with EDTA but this phenomenon is still possible with heparin or citrate though rate with these anticoagulants (1). PTCP is induced by agglutinating antibodies that cause platelet clumping by binding GP IIb/IIIa receptors on the platelets(2). Most of the agglutinins act at room temperature and are IgG but IgM and IgA are also implicated (2). EDTA dependent PTCP is reported to occur in 0.2% of asymptomatic individuals but the incidence as high as 1.9 % of hospitalized patients (1). PTCP was the second most common cause for low platelet count with an incidence of 17 % in one study (3). This phenomenon has led to unnecessary invasive tests like bone marrow examination, unnecessary treatment with platelet transfusion, steroids, IVIG etc. Our patient also got platelet transfusion and steroids. The diagnosis of PTCP should be suspected in patients without symptoms and signs of low platelet although they have blood count with low platelet. Pseudoleukocytosis may be seen sometimes as automated analyzer may pick platelet clumps as leukocytes (4). Whenever PTCP is suspected, peripheral blood smear should be reviewed and platelet estimated manually or another anticoagulant should be used. PTCP can also occur after use of abciximab or Reopro which is a monoclonal antibody directed against GPIIb/IIIa. This is also reported to be associated with valproic acid use(7). No consistent association has been found with any particular pathological condition.

CONCLUSIONS

A clinician should thus think of this diagnosis in patients presenting with low platelet and this will avoid unnecessary transfusions, expensive and invasive work up and cancellation of urgent and important surgeries.

BIBLIOGRAPHY