Pseudothrombocytopenia in cardiac surgical practice

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Abstract

Pseudothrombocytopenia is observed occasionally in post-cardiac surgical patients. It is commonly due to EDTA-mediated immunological mechanisms, which lead to agglutination of functionally intact platelets. This condition is harmless and does not warrant platelet transfusion. We describe an instance of pseudothrombocytopenia in our practice and discuss its clinical relevance.

Keywords: Platelet; Anti-coagulation

1. Introduction

Pseudothrombocytopenia results in a spuriously low platelet count (<10,000/μl) with no associated haemorrhagic tendencies [1]. Normal in vivo platelet function and number account for the absence of haemorrhagic manifestations. In pseudothrombocytopenia, platelet aggregation occurs in vitro and is generally associated with the use of EDTA in the blood-collection tube. Apart from EDTA, other anticoagulants namely citrate, oxalate or heparin have also been implicated [2].

Shreiner and Bell in 1973 described this condition initially [1]. Subsequently, both EDTA-dependent and independent variants of pseudothrombocytopenia have been described in the literature [2]. The condition results from agglutinating antibodies that cause platelet clumping in vitro.

In this condition, EDTA induces antibody production against platelet glycoprotein gpIIb-IIIa [3]. Most of the agglutinins react strongly at room temperature or below, but some are temperature-independent or react best at 37°C. Most are IgG, but IgM, IgA and combinations of IgG and IgM or IgG and IgA also have been described [3–6]. These antibodies bind to the platelet adhesive receptor gpIIb-IIIa, leading to clumping of platelets in a peripheral smear [7].

We recently observed this phenomenon in a patient who underwent aortic valve replacement. Though the patient had a very low platelet count in routine blood tests, he did not show any bleeding tendency and had normal bleeding and clotting times. Peripheral smear and full blood counts in EDTA and heparin-mixed blood along with the clinical scenario helped us come to the diagnosis. There are previous reports of unnecessary platelet transfusions being performed on patients with pseudothrombocytopenia [8]. We feel that knowledge of this entity in clinical practice would be useful for practicing cardiac surgeons and intensivists.

2. Case report

A 65-year-old, Caucasian male patient underwent mechanical aortic valve replacement for symptomatic calcific aortic stenosis. He recovered well from his operation. Though he had platelet counts within the normal range (169,000/mm³) before the operation, blood checks on the fourth postoperative day revealed a low platelet count (9000/mm³) in routine EDTA-anticoagulated blood. A peripheral smear performed concurrently is shown in Fig. 1. This smear does not show any platelets in the centre of the slide while platelet clumps are seen towards the margins of the peripheral smear. The patient did not exhibit any spontaneous bleeding tendency at this point.

Repeat blood checks on EDTA-anticoagulated blood confirmed severe thrombocytopenia. Heparin-induced thrombocytopenia (HIT) was excluded by a negative HIT screen test. Pseudothrombocytopenia was a possible explanation as the patient did not manifest any bleeding tendency. Full blood count was performed in a sample of heparin-anticoagulated blood, which confirmed normal platelet count and distribution as shown in Fig. 2. As this patient was not on gpIIb-IIIa inhibitors at any time, we assume that he did not have antibodies to gpIIb-IIIa complex on the platelet surface. We did not screen the patient to confirm this.

3. Discussion

Severe thrombocytopenia after cardiac surgery generally triggers platelet transfusion. In the absence of any bleeding tendency, the possibility of pseudothrombocytopenia should...
be considered. EDTA-associated pseudothrombocytopenia can be easily diagnosed by repeating platelet counts in citrate or heparin-anticoagulated blood samples. A peripheral smear alongside the automated complete blood counts will be helpful in arriving at the diagnosis. This could potentially avoid mismanagement in the form of unnecessary platelet transfusions.

Usually EDTA-dependent pseudothrombocytopenia tends to appear in hospitalised patients after an initially normal platelet count. As the antibody levels drop, this phenomenon becomes less prominent by the third or fourth week. Information is lacking in the literature regarding the recurrent observation of this phenomenon in patients in a temporal fashion. The combination of appropriate history, clinical examination excluding haemorrhages and normal platelet count and function in citrate or heparin-anticoagulated blood helps to diagnose the condition. Confirmatory tests include IgG assays for antibodies to gpIIb-IIIa complex on the platelet surface. Anti-platelet medications should be continued in pseudothrombocytopenia to prevent thrombotic complications, as the platelets are normal in number and function in vivo.

References