

*Case Report***Platelet Satellitism - Rare or Infrequently Reported Phenomenon?**Swati Sharma¹, Sushama Belurkar¹¹Associate Professor, Department of Pathology, Kasturba Medical College, Manipal, Karnataka, India

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*Received: 28/02/2015**Revised: 24/03/2015**Accepted: 07/04/2015***ABSTRACT**

Platelet satellitism (PS) is said to be a rare phenomenon characterized by formation of platelet rosettes predominantly around polymorphonuclear (PMN) leukocytes which are seen in Wright stained peripheral blood smears prepared almost exclusively from ethylene-diamine-tetra-acetic acid (EDTA) anticoagulated blood samples. It is a rare cause of pseudothrombocytopenia. We describe a case of PS noticed in a patient of chronic renal disease along with the proposed possible pathogenetic mechanisms. We also emphasize the importance to critically analyse the automated machine values in the laboratories.

Key words- satellitism, rosette, pseudothrombocytopenia.

INTRODUCTION

PS is an in-vitro phenomenon, presenting with platelets rosetting around PMN and in rare cases around other blood cells. [1] It is observed in peripheral blood smears prepared from samples exclusively anticoagulated with EDTA, but not in blood samples treated with heparin or sodium citrate. [1,2] PS is not related to functional abnormalities of the blood platelets, clinical condition of the patient or to any specific drug intake. Awareness of this phenomenon is very important as it is a rare cause of pseudothrombocytopenia and if not recognized, can lead to unnecessary treatment and unrequired diagnostic work up. [1] Immune and non-immune pathogenesis for PS has been described in literature [3] although the precise underlying mechanism remains to be fully elucidated. In agreement with previously published data, we found PS

only around PMN. There are only about a 100 cases described in literature. [1] According to the authors probably PS is much more frequent and possibly underdiagnosed or infrequently recognized and reported.

CASE REPORT

A 38 year old male, known case of focal segmental glomerulosclerosis, on regular treatment with steroids for last 23 years came for routine check-up. Physical examination was normal. Routine hematological investigations showed
Hb 15.3gm/dL
TLC 10,000 cells/cu.mm
Platelet count- Automated 94,000 cells/cu.mm

Rest of the hematologic and biochemical parameters were within normal limits. As a routine practice in our laboratory for all new cases of

thrombocytopenia, a peripheral smear examination is done to cross check the platelet count. In our case the smear showed normal red cell and white blood cell morphology. Manual platelet count was 2,40,000 cells/cu.mm. Platelets were seen clumping around the neutrophils (Fig.1 & 2).

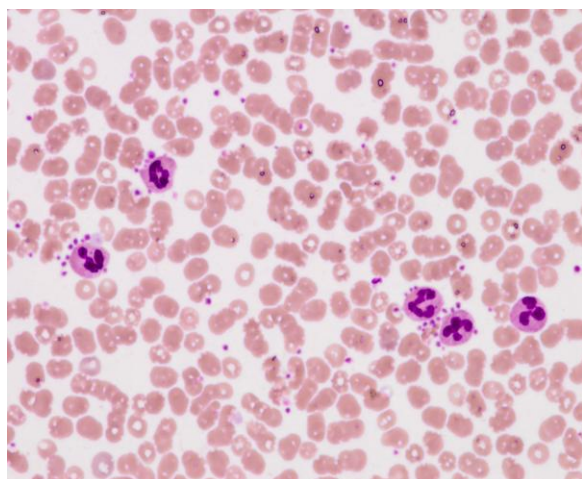


Fig.1- Platelet satellitism in the blood film prepared from EDTA anticoagulated whole blood with Leishman staining X200

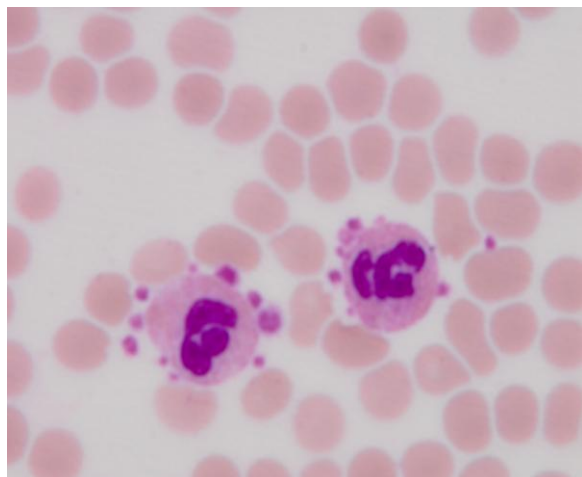


Fig.2- Platelet Satellitism Leishman staining X1000

DISCUSSION

Field and McLeod [4] were the first to report PS in the year 1963. PS is an infrequent and in-vitro phenomenon [2] with platelets rosetting around neutrophils, however, PS around neoplastic lymphoid

cells in a patient of mantle cell lymphoma has been described. [5] It occurs in blood samples anticoagulated only with EDTA but not seen when other anticoagulants such as heparin, acid-citrate dextrose, or citrate are used. [2,5] We found PS only around PMN and in samples anticoagulated with EDTA, as described in literature. Since PS has been found in normal individuals and in association with a variety of disease conditions, [1] so it is said to be independent from any pathologic processes occurring in the patient. Earlier studies described this phenomenon as temperature and time dependent [2] but according to the recent reports PS was seen in fresh and several hours old blood samples and in all areas of the blood smear. Wet and unstained blood smears made from EDTA anticoagulated blood revealed similar features as seen in fixed and stained smears. Variation of the temperature of the blood sample also had no apparent effect on the ability of the platelets resetting. [6]

The pathogenetic mechanisms of PS involving platelet adhesion to PMN are not completely understood yet. It is seen that PS can be transferred by incubating PS-patient EDTA-plasma or serum, with EDTA anticoagulated blood from subjects with no PS. Also, PS activity in plasma can be annulled by prior incubation with anti-IgG serum, but not with anti-IgM or anti-IgA. This demonstrates that PS is probably mediated by an IgG antibody and requires presence of EDTA. [1] It is also postulated that autoantibodies IgG directed to the glycoprotein IIb/IIIa complex of the platelet membrane, as well as against the neutrophil Fc gamma receptor, are involved. Possibly the epitopes for the antibodies which are usually hidden in leukocyte and platelet membranes are exposed by changes induced by EDTA, [2] probably by chelation of calcium ions by EDTA. [5] This may explain why PS is observed exclusively in blood samples with

EDTA anticoagulant. [2] Another possibility suggested is that changes induced by EDTA in the plasma membrane of platelets and neutrophils are responsible in the formation of molecular bridges which cross link platelet with the neutrophils. [7] An alternative, nonimmunologic pathogenetic mechanism states that thrombospondin, or some other alpha-granule platelet protein, has a role in platelet adherence to the PMN. [5] Since these antibodies are neutrophil specific, probably that's why eosinophils, lymphocytes, and monocytes do not show PS. [3] These EDTA-dependent anti-platelet and anti-neutrophil antibodies can be demonstrated in the serum of patients showing PS. [3] However, in contrary to the above explained mechanisms, PS was reported in the unpreserved heel-stick peripheral blood smear [5] which would argue against an EDTA-mediated mechanism as been proposed in the literature so far. We need further studies for complete elucidation of this phenomenon. In-vivo and in-vitro platelet function tests are normal in the cases of PS, demonstrating that platelets are physiologically normal in these cases. [5] In a case report, Lee et al [8] have hypothesized that the cause of spurious thrombocytopenia could be due to associated neutrophilic thrombophagocytosis.

PS is a very important phenomenon to be aware of as it can cause pseudothrombocytopenia. Failure to recognize this phenomenon in the laboratory can lead to unnecessary laboratory testing, delay of surgery and unrequired transfusions, bone marrow aspirations and other invasive or expensive investigations. [3] With increased automation in hematology and clinical laboratories, and falling blood smear review rates, the importance of identifying these sources of spurious results has risen. In our case, thrombocytopenia was picked up on blood smear examination

because of the laboratory policy of reviewing samples by blood smears if parameters are below the critical values. Automation is a loyal helper of an intelligent master, hence we need to analyse the machine values and review smears more critically and regularly. We believe that PS is much more frequent than reported, and few other authors have also suggested the same. [1,2]

Summary

PS is an infrequently reported in-vitro phenomenon of platelets rosetting around neutrophils observed in blood treated with EDTA as an anticoagulant. The proposed pathogenesis includes immunologic and non immunologic mechanisms. In-vivo and in-vitro platelet function tests are normal. PS is an important cause of pseudothrombocytopenia and hence laboratory scientists and technicians should be aware to avoid unnecessary and unrequired laboratory work up and treatment. Also the automated machine values should be critically analysed and regularly reviewed.

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