EDTA-DEPENDENT PSEUDOTHROMBOCYTOPENIA: CASE REPORT

EDTA YA BAĞLI PSÖDOTROMBOSİTOTENİ: OLGU SUNUMU

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EDTA-dependent pseudothrombocytopenia is the mismeasurement of the thrombocyte count in EDTA anticoagulated blood, due to aggregation of these cells. The situation may be mistaken with true thrombocytopenia and may lead to incorrect diagnosis and treatment, and to loss of power and time. The aim of this case report was to draw attention to EDTA-dependent pseudothrombocytopenia, which is one of the possible causes of thrombocytopenia. Case: isolated thrombocytopenia was detected in the EDTA blood sample sent to our laboratory, which had been obtained from a patient hospitalized with the diagnosis of epilepsy in the red area of the emergency medicine unit of our hospital. Another blood sample was collected into Na-citrate, in which the thrombocyte count was observed to be within the normal ranges. Furthermore, thrombocyte aggregations were observed in the peripheral blood smears prepared from the sample with EDTA, whereas no aggregation was observed from the sample with citrate.

Keywords: EDTA, peripheral blood smear, pseudothrombocytopenia, thrombocytopenia

No thrombocyte aggregation was observed in the smear obtained from the sample with Na citrate (Image-2). The situation was concluded to be EDTA-dependent PTP for the patient sample collected into the EDTA tube revealed a platelet count of 5x10^9/μL, which was 7x10^9/μL in the subsequent measurement, and the sample anticoagulated with heparin revealed a platelet count of 234x10^9/μL. Valproic acid may lead to hematopoietic toxicity and frequently affects thrombocytes. Its effect on thrombocytes may be thrombocytopenia, thrombocyte dysfunction or bone marrow suppression. The authors of the same study have reported that autoimmune thrombocytolysis may be due to the molecular configuration of valproic acid that mimics the fatty acids on the thrombocyte membrane and to the presence of anti-platelet antibodies. The pathophysiology of valproic acid-induced EDTA-dependent PTP has not been clearly understood yet; however, it has been related to the structure of valproic acid.14 History of medication with valproic acid is present in the case we presented as well. However, drug interaction is not the only cause of EDTA-PTP. Thus, other possibilities should be considered as well, and further studies should be conducted. Different methods may detect PTP. These include use of anticoagulants other than EDTA such as Na citrate, heparin or oxalate, and examination of the sample anticoagulated with EDTA brought to 37°C; blood samples with added kanamycin and peripheral blood smears. In our study, we used Na citrated samples in order to detect the presence of any EDTA-PTP. Furthermore, we investigated the peripheral blood smears of both EDTA and Sodium citrated samples. Different anticoagulants may be used instead of Na citrate. However, anticoagulants such as citrate, oxalate, acid-citrate dextrose and heparin may lead to PTP as well. The possible mechanism in the method of bringing the EDTA-anticoagulated sample to 37°C has been suggested as the inhibition of thrombocyte aggregation by unbinding of the glycoprotein IIb/IIIa complex at 37°C. However, aggregation may continue in some cases observed by this method as well, with unknown reasons.15 In conclusion, although EDTA-PTP is not meaningful clinically, the misdiagnosis may lead to further examination requests, unnecessary loss of power and high cost. Therefore, we would like to emphasize that in all situations of incompatible clinical and laboratory findings, clinicians should contact the laboratory specialist, which would be beneficial for the patient, the clinician and the laboratory specialist.

References

