CASE REPORT

Transient EDTA-Dependent Pseudothrombocytopenia Phenomenon in a Patient with Antiphospholipid Syndrome

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SUMMARY

Antiphospholipid syndrome (APS) is an autoimmune disorder associated with arterial/venous thrombosis and pregnancy loss; thrombocytopenia is another common manifestation of APS. In the present study, we discovered a transient ethylenediaminetetraacetic acid-dependent pseudothrombocytopenia (EDTA-PTCP) phenomenon in APS, which has not yet been reported in the literature.


KEY WORDS

ethylenediaminetetraacetic acid, pseudothrombocytopenia, antiphospholipid syndrome

INTRODUCTION

Vessel thrombosis and pregnancy loss are two main clinical manifestations of antiphospholipid syndrome (APS). Additionally, APS patients can also present as cardiac valvular disease, thrombocytopenia, hemolytic anemia, renal thrombotic microangiopathy, and cognitive impairment [1]. A former study had reported the prevalence of thrombocytopenia in APS was 23.4% [2]. The ethylenediaminetetraacetic acid-dependent pseudothrombocytopenia (EDTA-PTCP) occurrence rate is approximately 0.1%, which can cause erroneous reporting of low platelet counts by automated analyzers [3]. The EDTA-PTCP phenomenon has been described in variable diseases, such as acute hepatitis [4], sepsis [3], cytomegalovirus infection [5] and so on. To our knowledge, transient EDTA-PTCP phenomenon has not yet been described in patients with APS. In the present study, we observed the transient appearance of EDTA-PTCP in a patient with APS.
Figure 1. Changes in platelet counts in EDTA anticoagulated blood.
Platelet count was measured by an automated analyzer.

Figure 2. Blood smear with EDTA-anticoagulant.

Figure 3. Blood smear with sodium citrate anticoagulant.
CASE REPORT

A 62-year-old man was admitted to our hospital suffering from fever for 11 days. The body temperature fluctuated between 37.5 and 38.5°C. Physical examination did not find any abnormalities. His past medical history included cholecystolithiasis and chronic non-atrophic gastritis. Anti-cardiolipin (ACL) antibody IgG and IgM isotypes results were > 120 (0 - 10 GPLU/mL) and > 80 (0 - 7 MPLU/mL), respectively, and anti-β2 glycoprotein I (β2GPI) antibody was 21.10 (0 - 10 U/mL) (Alegria Analyser, Germany). Doppler vascular ultrasound of bilateral lower limbs showed no obvious thrombus changes; however, nuclear magnetic resonance (MRI) angiography showed obvious stenosis changes in the straight, left transverse, and sigmoid sinus.

PLT counts, detected by automated hematology analyzer (SYSMEX 2100, Japan) with EDTA-anticoagulants, was displayed in Figure 1. On the 10 day, PLT counts dropped to 42 x 10⁹/L and automated hematology analyzer prompted PLT clumps? Subsequently, we made a blood smear and PLT aggregations were discovered under the microscope, especially in the tail and both sides (Figure 2). Finally, we detected PLT counts again with sodium citrate anticoagulant. The PLT count was 100 x 10⁹/L and blood smear showed no PLT aggregation (Figure 3). This confirmed the EDTA-PTCP phenomenon.

Based on the above-mentioned evidence, physicians considered that the patient had a great possibility of suffering from APS and prescribed aspirin, methylprednisolone, and hydroxychloroquine for the patient. About 8 days later, the EDTA-PTCP phenomenon disappeared. After 2 years of follow-up, this phenomenon did not appear again.

DISCUSSION

The EDTA-PTCP phenomenon can be observed in various autoimmune diseases, such as systemic lupus erythematosus (SLE) [6] and Sjögren’s syndrome (SS) [7]. To our knowledge, there is no literature about the EDTA-PTCP phenomenon in patients with APS. Despite extensive research, the pathogenesis of APS remains unclear. However, it is widely accepted that thrombosis is the result of a hypercoagulable state caused by antibodies directed against β2-GPI. PLTs may be involved in APS and its thrombotic manifestations, especially arterial, in several ways [8]. Thrombocytopenia is a usual manifestation of APS [1], which is possibly caused by direct binding of anti-β2-GPI antibodies or anti-β2-GPI-β2-GPI complexes [8].

In the present study, the PLT counts of the patient with APS experienced a transient EDTA-PTCP phenomenon when anticoagulated with EDTA. EDTA-PTCP should be considered as a differential diagnosis and can be confirmed by peripheral blood smear showing PLT clumps.

Use of citrate as an anticoagulant could confirm the EDTA-PTCP phenomenon.

CONCLUSION

We speculate that a part of thrombocytopenia in patients with APS may be caused by the EDTA-PTCP phenomenon, so when a patient with APS shows thrombocytopenia, we should exclude the possibility of EDTA-PTCP.

Declaration of Interest:
The authors report no declarations of interest and there was no funding support for this study.

References: