

Although moderate bleeding is seen in severe factor V deficiency, less than 1% of patients experience bleeding. Cases in which thrombosis is prominent in the presence FV Leiden mutation and FV deficiency have been reported. Here, we present a patient with FV deficiency with FV Leiden heterozygous mutation in the etiology of recurrent abortion. **Case report:** A 41-year-old female patient who applied to her primary care physician with bilateral lumbar pain upon finding INR: 1.43 (0.8-1.2) and APTT: 37.6 seconds (25-36.5), the patient was recommended to apply to our out patient clinic. The patient who described two spontaneous abortions (at the age of 25, the first in the 2nd trimester and the other in the 3rd trimester), also had a history of ecchymosis in the extremities caused by minor trauma at intervals. **Methodology:** PT, INR and APTT returned to normal with the mixing test performed on the patient (12.1 sEC, 1.03 and 28.6 sec, respectively). Afterwards, FV, which is one of the factors in the common pathway of coagulation, was found low in the examination repeated twice (12.3% and 10.2%) (N: 62-139%). The APCR studied twice in screening for thrombophilia was 1.4 and 2.4 (N: 2.61-3.32) Protein C, protein S, antithrombin III levels were within normal limits, LAC and APA were negative. **Results:** According to this result, FV Leiden heterozygous mutation was detected in the genetic thrombophilia panel. Also the patient had FV deficiency. **Conclusion:** Authors termed the coexistence of heterozygous FV Leiden mutation and type1 FV deficiency as pseudohomozygous FV Leiden mutation. In our and other studies, we concluded that thrombosis was clinically significant, where as bleeding was rare and mild. We think that prolonged PT and APTT results in patients with a history of thrombosis with FV Leiden mutation are also stimulating in evaluating FV activity.

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##### BLEEDING MANAGEMENT DURING DELIVERY AND POSTPARTUM PERIOD IN GLANZMANN THROMBASTHENIA: EXPERIENCES FROM TWO CASES

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**Objective:** Glanzmann thrombasthenia (GT) is a hereditary bleeding disorder. The platelets lack  $\alpha IIb\beta 3$  integrin and fail to aggregate. Pregnancy can also lead to isoantibody formation when fetal cells with  $\beta 3$  integrins pass into the circulation of a mother lacking them; a consequence is neonatal thrombocytopenia and a high risk of mortality. We here present our experience with two GT patients, in which rFVIIa was our choice for bleeding prophylaxis and/or control during delivery and postpartum period. **Case report:** Case 1: A 28-year-old woman with GT was hospitalized. She was on 38th gestational week (GW).

Vaginal delivery was completed with rFVIIa prophylaxis. Postpartum 5th day rFVIIa stopped. The patient discharged with a minimal vaginal bleeding. Postpartum 10th day, she developed severe bleeding. GT seemed to be the only related factor. rFVIIa restarted with tranexamic acid and misoprostol. Two apheresis units of platelets were transfused. That time, rFVIIa continued 7 days. **Methodology:** Case 2: A 26-year-old woman with GT developed hematuria on 30th GW. No urinary system pathology was found. With rFVIIa treatment, hematuria was ceased. On 39th GW, during labor she developed massive bleeding. As urgent management, 8 random units of platelet and 5 units of packed red blood cell were transfused with local vaginal compress. rFVIIa treatment was initiated. On 10th days of rFIIa with minimal vaginal bleeding the patient was discharged from the hospital. **Results:** In both of the patients, no major neonatal bleeding problem was experienced. **Conclusion:** GT patients are at risk for heavy bleeding during labor, deliver or postpartum. Platelet transfusion is simple and easy option for bleeding control. In alloimmunized patients pooled platelet should be used. The use of rFVIIa appears to be safe and relatively effective.

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##### CASE REPORT OF MARGINAL ZONE LYMPHOMA DETECTED WHILE INVESTIGATING ETIOLOGY FOR HEMOSTASIS DISORDER

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**Case report:** In this article, we wanted to present our case in which we detected SMZL during examining for defects in coagulation tests and correlated the PT and aPTT elevation with the development of inhibitors against coagulation factors related to this disease. The PT and aPTT values of the patient diagnosed with MZL did not improve in the mixing test, and no other etiology was found. With the second course of chemotherapy, the patient's values improved.

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##### ACQUIRED FACTOR XIII DEFICIENCY WITH RUNX1 MUTATION, A REPORT OF TWO CASES TREATED WITH FACTOR XIII CONCENTRATE

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