Introduction

The main platelet disorders in pregnancy are divided into incidental thrombocytopenia, thrombocytosis, thrombocytopenia, thrombotic thrombocytopenic purpura, sticky platelet syndrome, aplastic anemia, immune thrombocytopenic purpura, preeclampsia, and HELLP syndrome (1). Thrombocytosis is the presence of high platelet blood counts (over 450,000 per mm³), and could be divided either as primary or reactive-secondary (2). It is characterised as a predisposing factor to thrombosis in some patients (1). The main causes of thrombocytosis - independently of pregnancy - include infection, hemolytic anemia, iron deficiency, autoimmune disorders, recent operations, and cancer (3). Moreover, different drugs or substances (such as cephalosporins) could be mentioned as possible causes of thrombocytosis (4). Less than 350 cases of pregnancies complicated with thrombocytosis are described in the current literature (5).

The aim of this report is to present a case of postpartum thrombocytosis and discuss the differential diagnosis of this condition through the presentation of its management.

Case report

This is a case of a 43-year-old primigravida primipara woman who presented in our Department in 36 weeks gestational age and underwent caesarean section due to preeclampsia. From her history, it was known that her pregnancy was an in vitro fertilization (IVF) result. She also received low molecular weight heparin because of thrombophilia (protein S insufficiency). Her first three postoperative days were uneventful, however the fourth day she had fever (up to 38.4°C), abdominal pain, and diarrhea. Her blood tests revealed increased white
A rare case of postpartum thrombocytosis. Differential diagnosis and management

During puerperium, hormonal changes lead to changes in blood coagulation, and fibrinolysis in order to create a hypercoagulability state and protect from haemorrhage (6). Platelets play a pivotal role in the initiation of the coagulation process. Moreover, they are implicated in the pathogenesis of preeclampsia (6). Stress platelets are produced under such conditions. The main difference between normal pregnancies and preeclamptic women is that the increase in platelet count is already seen in 2-5 days postpartum, whereas no difference is found in the preeclamptic group (3). Such an increase could be explained by persistent activation of the coagulation system with consumption of platelets during the first days postpartum. An increase in the number of platelets has been shown as a response to thrombopoietic stress. Later on, an increase in the number of megakaryocytes or colony-forming cells-megakaryocytes is also seen and could explain this rebound effect (7). It should be noticed that platelet reactivity does not return to normal even six weeks postpartum in preeclamptic women (8). Aune et al were the first to show that the platelet count is increased 2-3 fold in preeclamptic women compared to normotensive postpartum women (3). It should be noticed that the peak levels are seen between 6th-14th postpartum day when women have been already discharged (3). For this reason, such an increase in the platelets count is not identified and so it is underestimated. Because of this late rebound thrombocytosis, low molecular weight heparin is used for prolonged thromboprophylaxis. According to Attala et al the mean platelet count remains significantly higher at 12-16 days or even at the 24th day postpartum in women who deliver by caesarean section (526x10^9/L) compared to women delivering vaginally (369x10^9/L) (2). Furthermore, it has been shown that preeclamptic women – especially those delivering by caesarean section – have a significantly higher increase in platelets count (240 to 621x10^9/L at postoperative days 6-14) compared to normotensive women (214 to 351x10^9/L) (2). In our case, the patient had a history of preeclampsia, as well as she delivered by caesarean section. It should be stated that such an increase is mentioned independently of heparin use postpartum (2). In our case, the woman received low molecular weight heparin.

It is known that essential thrombocytosis is diagnosed when the platelet count is consistently over 600x10^9/L, the haematocrit is less than 40, there is stainable iron in the marrow, normal ferritin red blood count levels, absence of Philadelphia chromosome or marrow fibrosis, and/or myelodysplastic syndrome (1). Furthermore, any cause of reactive thrombocytosis such as infectious or inflammatory state, surgical procedure or tissue damage, malignancy, iron deficiency, hemolytic anemia, acute blood loss or renal disorder should be also evaluated (4). Moreover, Surgicel® use for haemostasis and postpartum fever could lead to the possibility of a thrombocytosis caused by a local infection. Topical hemostatic agents such as Surgicel® (Ethicon, Somerville, NJ, USA) are widely used in surgery to control bleeding at diverse sites (9). Surgicel® is a plant based product used in a variety of operations where hemostasis is difficult to be achieved. Its properties are based on the oxidized regenerated cellulose technology designed to ensure purity and consistency (9). This agent is fully absorbable within 7-14 days. However, it could be identified by ultrasound even in the 35th postoperative day (10). Macrophages play a significant role in the absorption process (10). The differential diagnosis of postoperative abdominal pain or fever in our case included abscess when gas was identified in the surgical bed. The ultrasound appearance of Surgicel® was characterized as an echogenic mass with posterior reverberation artifact. It is known that normal postoperative appearance of Surgicel® might mimic abscess as it can give a CT or MRI picture of gas in the operative site. In our case, foci of gas trapped within the gauze as it became saturated with blood was identified on postoperative CT or MRI. On CT, a low attenuation mass with linear foci of gas was found. CT scans usually show an unusual collection of gas surrounded by postoperative hematoma at the site of Surgicel® placement. When blood surrounds the air...
and gauze, the trapped air creates a linear or unifocal punctuate pattern. On MRI, it has a short T2 relaxation time leading to a hypointense mass on T2-weighted images.

In our case postpartum thrombocytosis was caused by preelampsia, infection (hematoma or Surgicel® collection) or pharmaceutical agents (antibiotics).

The differential diagnosis included preeclampsia, infection, anemia, and Surgicel® use.

Conclusion

Postpartum thrombocytosis is a rather rare diagnosis, it could lead to severe conditions such as thrombosis of different sites. Obstetricians should be aware of such a condition and close follow-up, as well as cooperation with radiologists and doctors specialized in hematological or infectious diseases is essential for the optimal management of such patients.

References