The Management of Pregnancies Complicated by Immune Thrombocytopenic Purpura: A Retrospective Analysis of 22 Patients

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Introduction

Immune Thrombocytopenic Purpura (ITP) is an autoimmune disorder resulting in damaged platelets. The acute form, which is self-limiting and the result of a viral infection, generally affects children. The chronic form generally occurs in the third decade of life and comprises 3% of thrombocytopenia cases observed during pregnancy [1,2]. Antibodies formed in ITP are directed to platelet membrane glycoproteins. In ITP patients, the platelet–antibody complexes are then sequestered and destroyed in the reticuloendothelial system, particularly the spleen [3]. ITP is generally diagnosed before pregnancy. The diagnosis is facilitated by the presence of bleeding and various symptoms, such as bruising, epistaxis, petechiae, a history of menorrhagia. The diagnosis of ITP is done when a platelet count<50,000/mm³. These verity is characterized by platelet count<50,000/mm³. It is recommended to refrain from administering specific treatment in the cases in which the platelet count exceeds the value of 50,000/mm³ [4,5].

The incidence of ITP has been reported to be 1–10/10,000 pregnancies [6]. Research has suggested that ITP is responsible for 3% of thrombocytopenia detected at birth [7]. Pregnant women have a risk of massive bleeding in the post-partum period, depending on the platelet count, with bleeding particularly common at levels below 20,000/mm³. The risk of neonatal thrombocytopenia is 9–15% and that of intracranial bleeding is 1% due to the trans-placental passage of anti-platelet antibodies in the maternal circulation [8,9]. Thus, pregnant women with ITP need to be monitored closely. Pregnancy does not affect the treatment of ITP. The most commonly used agent is prednisolone, which is intended to maintain the platelet count in the range of 30,000 to 50,000/mm³ [10–12].

The aim of the present study was to describe the clinical course and treatment protocols of 22 pregnant women with ITP who were managed in our clinic.

Material and Methods

This retrospective study was carried out at the Department of Gynaecology and Obstetrics at our university, and it complied with the Second Declaration of Helsinki (revised in 2008) and was approved by the local ethics committee.

We identified 22 patients by screening an electronic database for patients admitted to the obstetrics clinic with a diagnosis of ITP (ICD code D69.3) and delivery (ICD codes O80, O81, O82, O84) between 1 January, 2010 and 31 August, 2014.

The diagnosis of ITP was based on the presence of thrombocytopenia for at least 6 months, normal bone marrow findings, normal white blood cell and erythrocyte counts and the elimination of other aetiological factors that can cause thrombocytopenia.
Table 1: Demographic characteristics and laboratory data of the 22 pregnant women with immune thrombocytopenic purpura.

<table>
<thead>
<tr>
<th>Demographic characteristics</th>
<th>Minimum</th>
<th>Maximum</th>
<th>Mean±SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>18</td>
<td>34</td>
<td>26.27±4.79</td>
</tr>
<tr>
<td>Gravidity (n)</td>
<td>1</td>
<td>5</td>
<td>2.18±1.05</td>
</tr>
<tr>
<td>Parity (n)</td>
<td>1</td>
<td>6</td>
<td>3.09±1.47</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>23</td>
<td>34.11</td>
<td>28.50±3.27</td>
</tr>
<tr>
<td>Gestational week at delivery</td>
<td>34</td>
<td>41</td>
<td>38.22±1.72</td>
</tr>
<tr>
<td>Birth weight (gr)</td>
<td>2600</td>
<td>4250</td>
<td>3209±440.12</td>
</tr>
<tr>
<td>Hemoglobin (gr/dl)</td>
<td>9.6</td>
<td>13.5</td>
<td>11.25±1.13</td>
</tr>
<tr>
<td>Platelet at delivery (mm³)</td>
<td>15000</td>
<td>75000</td>
<td>47772.73±16523.96</td>
</tr>
</tbody>
</table>

Demographic characteristics and information on whether the patient received treatment for ITP, the maternal platelet count at birth, the administration of a platelet suspension and types of complications at birth were obtained from medical records.

Exclusion criteria

Patients with gestational thrombocytopenia, thrombocytopenic purpura, disseminated intravascular coagulation, systemic lupus erythematosus, drug-induced thrombocytopenia, pre-eclampsia, eclampsia and haemolytic uraemic syndrome were excluded who could have findings of thrombocytopenia in a complete blood count.

Results

(Table 1) summarises the demographic characteristics and laboratory data of the 22 pregnant women with ITP who were included in the present study. ITP was diagnosed during pregnancy follow-up in four (18.18%) of the women (Table 2) and in the remaining 18 (81.82%) women with a diagnosis of ITP before pregnancy who had been managed in the haematology department. These patients who are diagnosed ITP before pregnancy were not refractory to ITP treatment. The platelet count in the begging of pregnancy ranged between 34,000/mm³ and 74,000/mm³. Two of the patients with ITP who were diagnosed before pregnancy had previously undergone gonesplenectomy.

During the pregnancy follow-up, intrauterine growth retardation was detected in two patients, and mild pre-eclampsia was detected in another patient. No complications were detected in the remaining 19 patients during pregnancy.

Nine (40.9%) of the pregnant women with ITP did not receive any treatment during pregnancy, and their platelet count at birth was higher than 50,000/mm³. Seven (31.8%) of the patients received steroid therapy, and two (9.1%) received intravenous immunoglobulin (IVI g) therapy. A platelet suspension was given to four of the pregnant women with ITP who had a platelet count<30,000/mm³ and underwent an emergency caesarean section (The indications for caesarean section were previous caesarean, fetal malpresentation and placenta previa).

Of the patients, 15 (68.2%) gave birth by vaginal delivery, and seven (31.8%) underwent a caesarean section.

Discussion

The platelet count decreases by approximately 10% during pregnancy, especially in the third trimester. This reduction is a benign condition and does not require treatment [13]. It is thought to be caused by haemodilution and increased platelet consumption during pregnancy. The platelet count was reported to be below 150,000/mm³ in 6–15% of all pregnancies, representing asymptomatic thrombocytopenia [14]. In general, a reduced platelet count is detected by chance during routine complete blood counts, and it is the second most common haematological abnormality, with anaemia being the most common [15]. When thrombocytopenia is detected during pregnancy, a meticulous evaluation should be performed to detect maternal and neonatal complications, as well as systemic diseases [8,15].

Gestational thrombocytopenia (70%) is the most common cause of thrombocytopenia diagnosed during pregnancy; followed by hypertensive disorders of gestation (21%) and idiopathic thrombocytopenic purpura (3%). Other less common abnormalities include disseminated intravascular coagulation, thrombotic thrombocytopenic purpura, haemolytic uraemic syndrome, systemic lupus erythematosus, congenital thrombocytopenia, hypersplenism and drug-induced thrombocytopenia [14-17].

ITP is an autoimmune disorder characterized by a decreased platelet count due to anti-platelet factors in the structure of immunoglobulin G [1,6]. Antibodies formed in ITP are directed to platelet membrane glycoproteins, and the platelet-antibody complex of the reticuloendothelial system, particularly the spleen, is destroyed. Although ITP can occur at any age, it generally affects women of reproductive age [11]. Both maternal and foetal problems can occur in pregnancies complicated by ITP. Maternal risks include haemorrhages and increased rates of caesarean sections, and foetal risks include intrauterine growth retardation, prematurity, foetal distress, neonatal thrombocytopenia and intracranial bleeding [8,11, 18]. Diagnostic criteria for ITP include the onset of thrombocytopenia before the second trimester, the presence of thrombocytopenia before pregnancy, a platelet count<75,000/mm³ and the persistence of thrombocytopenia after birth [1,15].

ITP is often confused with gestational thrombocytopenia. Gestational thrombocytopenia is the most common cause of thrombocytopenia in pregnancy [14]. Diagnostic criteria for gestational thrombocytopenia include a normal platelet count in the pre-conception and early gestational periods, no history of
spontaneous bleeding, a platelet count >70,000/mm³ and spontaneous resolution of the platelet count within 2–12 weeks after birth [15].

It is important to distinguish gestational thrombocytopenia from ITP for both the mother and infant. Pregnant women with immune thrombocytopenia have a risk of maternal bleeding, depending on their platelet count, with bleeding particularly common at levels below 20,000/mm³. The risk of neonatal thrombocytopenia is 9–15% and that of intracranial bleeding is 1%. These risks are not observed in gestational thrombocytopenia [8].

The American Society of Hematology and the British Society of Haematology recommend treating pregnant women with severe thrombocytopenia or those with haemorrhages accompanied by mild thrombocytopenia. Treatment is recommended when the platelet count is below 10,000/mm³ at any time during pregnancy and when it is below 30,000/mm³ in the second and third trimesters because of the risk of an emergency delivery. Treatment modalities include steroids, immunoglobulin and splenectomy. Due to the rapid destruction, a platelet transfusion is preferred only before surgery and emergency deliveries to decrease the risk of bleeding [13].

In conclusion, pregnant women with ITP should be managed in collaboration with a haematologist. Methyl prednisolone should be given during pregnancy to improve the platelet count. In cases refractory to steroid therapy, IVIg therapy can be given if the delivery is impending. In urgent cases, a platelet suspension can be lifesaving as an adjunct to other treatment modalities.

References