A Case Study of Thrombocytopenia in Pregnancy

Charlotte Goodman
Logan Hospital, QLD

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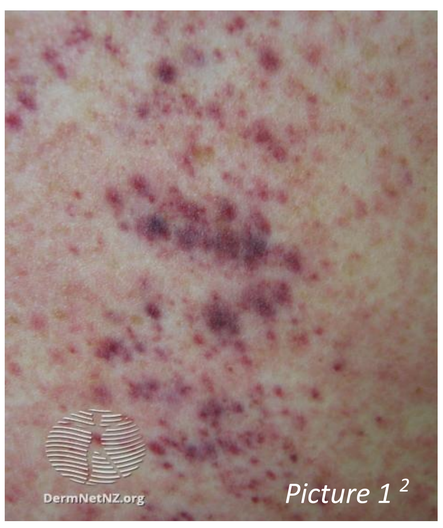
Introduction

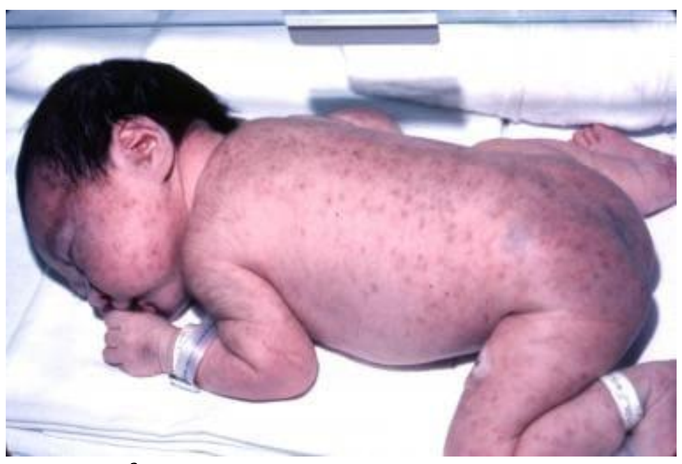
Thrombocytopenia affects 5-10% of pregnancies. There are a range of causes but the most common is gestational thrombocytopenia, which is responsible for 75% of cases. Immune thrombocytopenic purpura (ITP) is another important cause. It is not affected by pregnancy but will occur in 1-2 in 10,000 pregnancies and 3% occur during pregnancy.

Objectives

To review the investigation and management of thrombocytopenia in pregnancy. This case looks at this in an antenatal patient with compounding liver transaminase derangement and an evolving clinical picture.



Picture 1 [2]



Picture 2 [3]

Case

A 38 year old woman with IVF pregnancy, a background of congenital myotonic dystrophy, one previous Caesarean section and early gestational diabetes requiring insulin. Mild thrombocytopenia and liver transaminase derangement noted from booking. She presented with right abdominal pain at 30 weeks gestation with a platelet count of 87 and worsening transaminases, prompting subsequent investigation and management. Haematology and gastroenterology were involved. She developed obstetric cholestasis at 32 weeks which was medicated. She was admitted with new pre-eclampsia (PET) at 34+4 weeks and commenced on labetalol. She was delivered by Caesarean section at 34+6 under spinal anaesthetic due to worsening biochemistry and BP control with new onset contractions. This was complicated by a 3.5L postpartum haemorrhage (PPH) secondary to an adherent placenta. She was discharged at day 3 postpartum.

Management

The differential diagnoses for this case included ITP, gestational thrombocytopenia, PET/ HELLP syndrome, microangiopathic haemolytic anaemia syndromes (such as thrombotic thrombocytopenic purpura), acute fatty liver of pregnancy and diagnosis related to underlying chronic liver disease (CLD). Initial investigations included a blood film, coagulation studies, liver screen including virology (HIV, hepatitis B/ C, EBV, CMV) and autoantibody screen, haematinics, haptoglobin and LDH. ITP was favoured as the diagnosis. The patient was commenced on prednisolone 25mg OD at 33 weeks with platelet levels of 80. Her levels fell to 71 and the plan was for IV Ig 2mg/kg over 3 days. Prior to this she developed PET with a urine PCR of 308. She had platelets of 77 at the time the decision was made for delivery; anaesthetics proceeded with a first pass spinal and covering platelet transfusion (2 units peri-operatively). She received 2 units of blood post PPH. Steroids were weaned postpartum. Postpartum platelets remained low (129 at last review) and ITP is still the favoured diagnosis. She has been advised to monitor for signs of bleeding (picture 1). Transaminases improved but ALP remained elevated and hepatology are investigating for CLD.

Discussion

ITP was favoured over gestational thrombocytopenia in this case. The latter typically occurs in the third trimester with a gradual fall in levels. It is a benign condition with no adverse consequences for mother or baby and therefore requires no treatment. Platelet levels will return to normal after delivery. ITP is due to autoantibodies which cause destruction of platelets, mainly in the spleen. It can be idiopathic or due to secondary causes. Antiplatelet IgG can cross the placenta and cause thrombocytopenia in the neonate (picture 2). Treatment may be with oral prednisolone, IV immunoglobulin, anti-D and in some cases a splenectomy or azathioprine may be required. Platelet transfusions are usually ineffective as they increase antibody titres.[1] This was an interesting case as her clinical picture evolved with multiple diagnoses. The main focus for this patient was safe delivery planning in a timely manner given her evolving PET. Neuraxial analgesia is generally safe if platelet count is >80. IV Ig needs time to have effect so a platelet transfusion was used in this case as a transient measure to allow for a safe spinal insertion.

References

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