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Case Report

Systemic Lupus Erythematosus with Repeated Abortions

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Summary

A case is described of a pregnant patient with systemic lupus erythematosus (SLE). Three previous pregnancies had ended in abortion. Attempts to reverse the thrombocytopenia with steroids, plasmapheresis failed,and platelet count returning to normal immediately after the death of the fetus at 20 weeks gestation. It is known that patients suffering from SLE may show a deterioration of their disease during pregnancy and those spontaneous abortions are frequent. This report describes a case in which for several years the only significant symptom of SLE was mild thrombocytopenia; however, during this time 4th pregnancy also ended in spontaneous abortion.

Introduction:

Systemic lupus erythematosus is a chronic inflammatory disease with multi system involvement in which the tissues are damaged by auto antibodies and immune complexes and primarily affects young female at childbearing age[1]. Pregnancy is an important matter in every woman's life. However, different maternal diseases can complicate pregnancy. One of them is SLE, which can turn the life miserable during pregnancy if it is not treated properly. There is no standard statistical data in SLE for Bangladesh. Incidence and prevalence of SLE is still very low in India. A prevalence study in India (carried out in a rural population near Delhi) found a point prevalence of 3 per 100,000[2]. Sex specific SLE prevalence in the UK: Females: 49.6/100,000 (ie 1 in 2000 adult women have SLE) and Males: 3.6/100,000. Ninety percent of cases of SLE affects women, the incidence of SLE during the child bearing age being 1 in 500. The fetal effects are mainly prematurity, intrauterine growth restriction, neonatal lupus, and in extreme cases stillbirth. Congenital heart blocks result as a consequence of diffuse myocarditis and fibrosis[1].

Case Report

A 30 year old hypertensive woman who was a housewife and gave a history of 3 previous pregnancies all of which ended in spontaneous abortion between 18 and 24 weeks. The second pregnancy was complicated by episodes of purpura and thrombocytopenia with platelet counts of around 20 x 109/l. These responded to oral prednisone. The platelet counts did not fall significantly during the first and third pregnancies. When first seen in Diabetic Association Medical College Hospital in 2017, SLE had already been diagnosed on the basis of a positive anti-nuclear factor and a DNA binding of 70%. She had been prescribed 10 mg prednisone on alternate days with 100 mg azathioprine daily. Then azathioprine was stopped without any ill effects, and her only symptoms were occasional attacks of arthralgia. Platelet count varied between 70 and 140 x 109/l with occasional episodes of mild purpura. Then patient was admitted to hospital in 15, October 2017, during the 14th week of fourth pregnancy, because of a steadily deteriorating thrombocytopenia, which had failed to respond to raising the prednisone dose to 80 mg daily. On admission she was found to be in excellent physical health except hypertension, there was no bruising, purpura or bleeding from any source in spite of a platelet countof 20 x 10^9/l. It appeared from notes that the platelet count 3 weeks earlier had been in the region of 200 x 10^9/l, On examination, anaemia absent, BP 140/90 mm of Hg, pulse 80/min, temp 99^0F, Oedema absent, reduced flexion deformity of 5th finger of left hand, sensation normal. Other system reveals no abnormality.

Investigations

Investigations showed a hemoglobin (Hb) of 10.5gm/dl, normal urea, electrolytes and liver function tests, ESR 20 mm in 1st hr, WBC-10300/dl, Platelet count-20 x 10^9/l, Urine R/E-Albumin(+++), Anti-dsDNA-13.8U/ml, Anti-Cardiolipn IgM-7.1U/ml, IgG-6.23U/ml, Anti-SSA-0.52U/ml, DAT 1/32 and an active bonemarrow with abundant megakaryocytes. Complement fixing platelet antibodies were also found although, no cryoprecipitateor anti-complementary activity could be detected at any time. The chances of a successful pregnancy in the presence of severe thrombocytopenia and high steroid dosage were considered to be very small. One further methods was therefore proposed to reverse the thrombocytopenia; plasmapheresis to remove the platelet autoantibodies. Plasmapheresis was tried, as it has been successfully used in pregnancy for the removal of anti- Rhesus antibodies with no side effects on the fetus[3]. The procedure was carried out 3 times at 4-day intervals, each exchange consisting of 41. Platelet counts, complement factors, and platelet antibodies were monitored before and

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after each exchange but apart from a slight improvement in the platelet count after the first episode, there was changed. For the next 5 days there was no change in any haematological or biochemical parameter, except for a slight rise in platelets due to transfusion. The fetal heart sounds remained audible. Thereafter, the fetal heart sounds could no longer be heard and simultaneously a rise in the platelet count was observed. This continued and reached 250 x 109/l after a week. The complement and anti-platelet antibody levels also returned to normal during this time, and within 10 days. The pregnancy was considered to be non-viable,and a therapeutic abortion was performed. The fetus was small and macerated. The platelet count remained at around 250 x 109/l for the remainder of her stay in hospital, but fell to 160 x 109/l on discharge on 1st November 2017. Since then, it has fluctuated between 90 and 200 x 109/l. The subject remains in good health, taking only 7.5 mg of prednisone daily.

Discussion

Apart from minor improvements all three methods used to reverse the thrombocytopenia, ie; high dose steroids, plasma pheresis were without effect. Since the platelet autoantibodies that had been detected during pregnancy on several occasions were shown to fix complement, it was concluded that their destruction was occurring primarily within the circulation. A hypothesis supported by the findings of a normal sized spleen at splenectomy, and reduced levels of complement in the serum. It was also assumed that the mild thrombocytopenia that existed in the non-pregnant state was due to the same antibodies at a much lower titre. In 25% of samples from their patients with SLE[4] reported that, platelets were capable of fixing complement even in the absence of serum, suggesting that the platelets were already coated with antibody. This did not occur in normal controls. The failure of plasmapheresis to remove these antibodies implies a high rate of synthesis. This procedure is often disappointing in the treatment of SLE, possible for this very reason, but it has been used with success in acute life threatening situations when there is good evidence of circulating immunecomplexes[5,6]. Assuming the platelet count inversely reflected autoantibody activity, the effect of the pregnancy on the rate of autoantibody production could be closely monitored. The platelet count was seen to fall steadily over a period of about five weeks between the 9th and 14th week of the pregnancy, and to rise rapidly at a time coincident with the apparentdemise of the fetus. Presumably, therefore, the autoantibody production increased steadily from its non-pregnant level to its maximum over this period and stopped suddenly when the pregnancy ended. The cause of the repeated abortions, during the trimester generally considered to be the safest is unclear.

Lympho cytotoxic antibodies which have been shown to be present in 80% of patients with SLE, are also present in the majority of patients suffering repeated miscarriages. However usually it was absent in patients with SLE who have successful pregnancies[7]. This case in history, had only slightly raised levels of lympho cytotoxic antibodies. In cerebral lupus, lymphocytotoxic antibodies have been shown to cross react with brain tissue[7,8], but it is not yet clear whether a similar cross reactivity between such antibodies and placental tissue exists in patients who suffer from repeated abortions.

Conclusion

SLE causes an increase rate of spontaneous abortion (miscarriage). Overall live-birth rate in SLE patient has been estimated 72%. Pregnancy outcome appears to be worse in SLE patients whose disease flares up during pregnancy.

Miscarriages in the first trimester appear either to have no known cause or to be associated with signs of active SLE.
Later lupus appear to occur primarily due to the antiphospholipid syndrom, in spite of treatment with heparin and aspirin. All women with lupus are recommended to be screened for antiphospholipid antibodies, both the lupus anticoagulant and anticardiolipin antibodies

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