CASE REPORT

Triplets with Two IUFD with IUGR of Live Fetus with DIC in a Patient with BOH and Septate Uterus

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Introduction

Multiple pregnancies account for 3 % of all births. The incidence of triplet pregnancy is 1 in 8,100 pregnancies. However, use of in vitro fertilization and ovulation induction has increased the incidence of multiple pregnancies. The incidence of Mullerian defects in women with recurrent miscarriage is 5–10 % [1] and the incidence in late miscarriage and preterm births is more than 25 % [1]. The occurrence of triplet pregnancy in patients with a septate uterus and previous recurrent pregnancy losses is rare. The occurrence of intrauterine fetus death (IUFD) of two fetuses of the triplets with development of chronic disseminated intravascular coagulation (DIC) in the mother with survival of one fetus is all the more rare. Heparin has an important but controversial role in treatment of such cases.

Case Report

A 30-year-old lady married for the past 8 years, Gravida 6 Abortion 5, at 30 weeks of gestation was referred to our

hospital with triplet pregnancy (triamniotic dichorionic) with IUFD of two fetuses (monochorionic diamniotic), which had occurred at 16 weeks of gestation. The patient had developed chronic DIC and the live fetus developed intrauterine growth restriction (IUGR) at 28 weeks of gestation with Color Doppler studies suggestive of fetoplacental insufficiency.

Prior to this pregnancy, the patient had five pregnancy losses at 8 weeks of gestation. Hysteroscopy revealed a complete midline septum in the uterine cavity arising from the fundus and extending up to the cervix. Hysteroscopic resection of the uterine septum was done.

She then conceived with a triplet pregnancy on treatment with an ovulation-inducing drug (clomiphene citrate). Cervical encirclage was done at 15 weeks of gestation. USG done at 16 weeks of gestations showed IUFD of two fetuses. Thereafter, her coagulation studies were done weekly. She developed DIC at 29 weeks of gestation. After taking the opinion of the hematologist, injection heparin, 1,000 U/h continuous infusion, was started. Her coagulation profile improved 2 days after starting heparin.

However, she had preterm premature rupture of membranes at 30 + 5 weeks of gestation. An emergency LSCS was done (injection protamine 1 mg IV was given prior to LSCS). Two papyraceous fetuses (Fig. 1) weighing 70 and 110 g and a live preterm fetus with a birth weight of 940 g were delivered along with a dichorionic placenta weighing 370 g. The baby was admitted to the NICU for prematurity. Both the mother and baby had an uneventful recovery and were discharged.

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Fig. 1 Two papyraceous fetuses

Discussion

The incidence of septate uterus is 35 % [2]. The miscarriage rate in women with a septate uterus is between 25 and 47 %. The incidence of IUGR in dichorionic triplets is 33.3 %. The growth rate begins to slow in triplets at 27–28 weeks of gestation. There is increased risk of prematurity in multiple pregnancies and for triplets, it is at 31 weeks. The incidence of IUFD in dichorionic triplets is 8.8 %.

Several weeks after IUFD, one-third of the patients may exhibit laboratory signs of DIC [3, 4]. Tissue factor from the retained dead fetus or placenta slowly enters maternal circulation and initiates DIC which is sometimes accompanied by significant fibrinolysis.

IUFD and DIC can occur following the demise of one of the multiple gestations. The role of heparin in such cases is

controversial. None of the clinical reports have shown a decrease in mortality in patients with DIC treated with heparin. At best, heparin has improved the levels of hemostatic factors in the treated patients [5].

In chronic DIC, due to IUFD, continuous infusion of heparin, 500–1,000 U/h, may be sufficient to maintain the benefit.

In contrast, heparin can seriously aggravate bleeding in such patients, especially when they have severe hemostatic failure because of consumption [6]. In fact, heparin may have reduced the anticoagulant effect in DIC as anti-thrombin (AT) is commonly depleted and fibrin monomers which are produced during DIC protect thrombin from inactivation by heparin AT complex.

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