Use of gestational surrogacy for a patient with recurrent pregnancy loss from early onset of severe pre-eclampsia: Case report.

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ABSTRACT

Introduction: The use of a gestational carrier deserves consideration as a treatment option in patients with poor reproductive histories because of early onset severe pre-eclampsia (PET) and hemolysis, elevated liver enzymes, and low platelets syndrome (HELLP). The case report is of 33 years old woman and 35 years old husband who presented with bad obstetric outcome of 7 pregnancies losses in the second trimester due to severe PET. After the 7th pregnancy loss, the couple opted for gestational surrogacy. In a traditional surrogacy arrangement, the surrogate mother provides the oocyte and the uterus to foster a pregnancy. With a gestational surrogate IVF cycle, the gestational surrogate is not the genetic mother because she does not provide the oocyte. In Vitro Fertilization (IVF) was undertaken using the patients oocytes and her husband's sperm with the transfer of three embryos to a gestational surrogate where conception occurred with subsequent twin delivery.

The objective of this case report is to report a case of gestational surrogate treatment in the prevention of severe early onset pre-eclampsia (PET) with subsequent term twin delivery.

Keywords: Severe Pre eclampsia, HELLP syndrome, In Vitro fertilization, Gestational surrogacy.

INTRODUCTION

Hypertensive disorders are leading cause of maternal and mortality and complicates 12%–22% of all pregnancies (1). Severe PET is one of the most devastating complications of the hypertensive disorders with profound maternal and neonatal morbidity and mortality, especially when the disease onset is in the second trimester (2,3). Hemolysis, elevated liver enzymes, and low platelets (HELLP) syndrome is related to about 20% of severe pre-eclampsia cases and HELLP has an increase of 30%–40% risk of adverse outcomes such as placental abruption, renal failure, subcapsular hepatic hematoma, preterm delivery, fetal or maternal death, and recurrent pre-eclampsia (4,5). The presence of HELLP syndrome is an indication for delivery, regardless of gestational age (4,5,6,7).

Multiple mechanisms have been postulated to explain why pre-eclampsia develops, and these have centered on maternal factors, fetal or embryologic factors, and paternal factors. Genetic predisposition, immunologic-mediated mechanisms, coagulation abnormalities, vascular endothelial damage, cardiovascular maladaptation, interference by various growth factors and dietary deficiencies are maternal factors that have been implicated as possible causes of PET (4,5,8). Abnormalities in trophoblast invasion have been the focus of much of the literature on the etiology of PET; there is defective decidualization and suboptimal modification of the subplacental vasculature in PET (8,9). Development of PET and to what degree is determined by the factors that influence trophoblast invasion control and normal placental vasculature development (8,9).

Gestational surrogate is an acceptable reproductive strategy, which has been used to assist women who lack a functional uterus because of mullerian anomalies or gynecologic disease requiring hysterectomy or women with poor reproductive

history without known anatomical abnormality (10). This report describes a patient with a very poor obstetric history secondary to recurrent severe PET who used IVF and a gestational surrogate to obtain a successful obstetrical outcome.

CASE REPORT

A 33-year-old woman and her husband with seven consecutive pregnancies complicated by early onset severe PET causing fetal demises between 21 and 24 weeks gestation; and she had one incidence of life-threatening maternal hemolysis, elevated liver enzymes, and low platelets. The couple opted for gestational surrogate to avoid recurrent pregnancy losses. The surrogate was explained on the risk of her acquiring PET due to paternal factor and transmissible diseases; she accepted all risks and signed an informed consent. Medical and psychological counseling was provided to gestational surrogate. Counseling included the fact that the surrogate had potential risk of developing severe PET and HELLP syndrome from paternal or fetal origin.

The appropriate transmissible disease testing (Hepatitis B, Human immunodeficiency virus, Syphilis) was performed on all three individuals involved. Cycle synchronization between the genetic mother and the gestational surrogate was achieved by down-regulation with leuprolide acetate. The gestational surrogate endometrial priming for the preparation of implantation was undertaken with progynova 2mg three times and intramuscular gestone 100mg was administered after oocyte retrieval. The patient had ovarian stimulation utilizing a long protocol (down-regulation with lupride 3.6mg (GnRH agonist), followed by 225IU of Gonal F (gonadotropins) daily, and human Chorionic Gonadotropin on day 11 of stimulation. In Vitro Fertilization (IVF) was undertaken using the patients oocytes and her husband's sperm. Sixteen oocytes were obtained and fertilized normally. Nine good quality embryos were obtained and three were transferred to the gestational carrier on day 3, six embryos were cryopreserved.

The gestational surrogate was initiated on progesterone supplementation (sustain 400mg twice a day) on the day of embryo transfer and continued on progesterone until 12 weeks gestation.

IVF was successful with twin gestation and the pregnancy progressed without complication of pre eclampsia in the surrogate. The surrogate's pregnancy progressed without other obstetrical complications and she had delivery of healthy twins, a boy 2.7kg and a girl 2.2kg at 38 weeks gestation by caesarean section. Post operatively the surrogate mother had uneventful recovery.

DISCUSSION

Severe PET in the second trimester is connected with life-threatening maternal complications such as HELLP syndrome leading to maternal morbidity and mortality, and fetal mortality (2,3,4,5). Recurrent PET has a risk of 65% in 21 % in subsequent pregnancies among women who have experienced severe PET in the second or early third trimester (3). Currently, PET has few interventions that can prevent its recurrence. Low dose aspirin is linked to only 19% reduction in the risk of PET while treatment with vitamins E and C, and calcium has little impact (11,12,13,14). The patient presented had recurrence of PET despite the use of low dose aspirin, vitamin E and calcium and hence had poor prognosis for future pregnancy.

The successful birth of healthy twin babies in this particular case implies that utilization of a surrogate may provide an alternative reproductive strategy for women with recurrent severe PET. The successful pregnancy achieved by using a gestational surrogate, without altering paternity or the maternal contribution to the embryo, indicates that maternal factors rather than paternal or embryologic factors may have been the determining factor in this patient's previous recurrences of severe early onset PET.

In women for whom PET impedes reproductive success, use of gestational surrogate can serve as a viable solution and more so, with the fact that this particular gestational surrogate did not incur any morbidity. The use of a gestational surrogate is a viable reproductive alternative for women with adverse pregnancy outcomes secondary to recurrent severe PET/HELLP syndrome. The case presented suggests that development of PET may have a large maternal component rather than embryological or paternal factors.

Gestational surrogate utilization warrants attention as a treatment option in patients with poor obstetric histories due to early onset severe PET and its attendant complications. However, gestational surrogate must be exhaustively counseled concerning the potential medical complications, and informed consent must be obtained.

REFERENCES

1. ACOG committee on practice bulleting-Obstetric. Diagnosis and management of preeclampsia and eclampsia. Obstet Gynecol 2002; 99:159-167.
2. Sibai BM, Tashmi M, Abdell TN et al. Maternal and perinatal outcome of conservative management of severe preeclampsia in midtrimester. Am J Obstet Gynecol 1985; 152:32-37.
3. Sibai B, Mercer B, Scrinoghi C. Severe preeclampsia in the second trimester: recurrence risk and long-term prognosis. Obstet Gynecol 1991; 165:1408-1412.
4. Egerman RS, Sibai BM. HELLP syndrome. Clin Obstet Gynaecol 1999; 42:381-389.
5. Baxter JK, Weinstein C. HELLP Syndrome: the state of the art. Obstet Gynaecol Surv 2004; 59:838-845.
6. Sibai BM, Mercer BM, Schiff E et al. Aggressive versus expectant management of severe preeclampsia at 28 to 32 weeks' gestation: a randomized controlled trial. Am J Obstet Gynecol 1994; 171:818-826.
7. Thomas T, Jophy R, Mhasker A et al. Are we increasing serious maternal morbidity by postponing termination of pregnancy in severe pre-eclampsia/eclampsia? J Obstet Gynaecol 2005; 25:347-351.
8. Redman I.L. Latest advances in understanding preeclampsia Science 2005; 308; 1592- 1594.
9. Raijmakers E.M, Roes R.H. Morsche E.A et al. Haptoglobin and its association with HELLP syndrome. J Med Genet 2003; 40:214-216.
10. Wright V, Schieve LA, Reynolds MA et al. Assisted reproductive technology surveillance - United States, 2002 MMWR Surveill Summ 2005; 54:1-24.
11. Duley L, Henderson-Smith DJ, Knight M. Antiplatelet agents for preventing pre-eclampsia and its complications. Cochrane Database Syst CD004659
12. Sibai BM. Prevention of preeclampsia: a big disappointment. Am J Obstet Gynecol 1998; 179:1275-1276
13. Levine RJ, Hauth JC, Curet LB, Trial of calcium to prevent preeclampsia N Engl J Med 1997; 337:69-76.
14. Caritis S, Sibai B, Hauth J. Low-dose aspirin to prevent preeclampsia in women at high risk. N Engl J Med 1998; 338:701-705.

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