Case Report

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Complicated Pregnancy in Polycystic Ovarian Syndrome after Moderate Ovarian Hyper-Stimulation Syndrome (OHSS) Followed by Peripartum Cardiomyopathy

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Abstract

Pregnancy is a wonderful experience. But not always is the experience as picturesque as we would plan it to be. Often times unexpected complications arise that can foul up from trying to have pregnancy to actually having a healthy pregnancy. We report a 35 year old nullipara presented with history of 2 year infertility with polycystic ovary syndrome (PCOS). Ovulation induction done with gonadotrophins and intrauterine insemination done. Moderate ovarian Hyperstimulation syndrome (OHSS) developed that was conservatively managed. In same cycle patient became pregnant. Bed rest and progesterone support advised in view of threatened abortion in early pregnancy. At around 28 weeks betamethasone coverage and tocolysis started due to preterm labor pains, continued up to 35 weeks. At 36 weeks emergency caesarean section done in view of absent diastolic flow in fetal umbilical artery. A live healthy male baby weighing 2.4 kg delivered. On post operative day 7, symptoms of congestive cardiac failure appeared and diagnosis of Peripartum cardiomyopathy (PPCM) was made, admitted in ICU and managed actively by multidisciplinary team.

Keywords: Ovulation Induction; Gonadotrophins; Ovarian Hyperstimulation Syndrome; Peripartum Cardiomyopathy.

Introduction

Anovulation and infertility are the predominant problems in the majority of women PCOS. It constitutes a high risk for the development of OHSS due to unacceptable rate of excessive follicle development. PPCM is idiopathic heart failure occurring in the absence of any determinable heart disease during the last month of pregnancy or the first 5 months postpartum. Both of them may be life threatening to any pregnant female needed multidisciplinary management and treatment.

Case History

The 35 years age patient, married life two years, P_{0010} (History of one previous abortion), diagnosed as a case of infertility with PCOD on USG and history of prolonged menstrual cycles. Rest of the investigation was normal and patient was advised oral metformin therapy due to raised fasting blood sugar levels. Down regulation of the ovaries were done for three months with estrogen and progesterone oral combined pills in view of raised luteinising hormone (LH). Tubal patency checked with Hysterosalpingography (HSG). Three cycles with clomiphene citrate tried upto 100 mg per day for 5 days from day 2 of cycle but there was no adequate follicular growth. The ovulation induction was done with gonadotropins along with follicular monitoring. Eight to nine follicles formed in each ovary. The follicular rupture was documented on transvaginal ultrasound. Intrauterine insemination (IUI) was done with prepared husband semen sample on day 13 of cycle. She was advised progesterone support and to wait for overdue of periods. On day 22 of cycle the patients felt some discomfort and distension of abdomen, dyspnoea on exertion and nausea vomitting. Ultrasound was done,

showing enlarged bilateral ovaries, moderate ascites and pleural effusion of both the lungs. On investigations the liver function test, electrolytes were slightly deranged. PCV was 42% and WBC 18000/cumm. On these clinical and investigations picture diagnosis of moderate OHSS was made [1]. Conservative treatment in form of bed rest, input output monitoring, plenty of fluids, high protein diet, tablet Cabgolin 0.5 mg daily for 10 days and antiemetics along with strict vital monitoring was done in the HDU. The symptoms and signs improved in one week. In the same cycle patient conceived, pregnancy confirmed by urine pregnancy test and serum β- HCG. The intrauterine gestational sac was confirmed on ultrasound at 5 weeks. At 7 weeks fetal cardiac activity appeared on ultrasound with small subchorionic bleed. So this threatened abortion was managed conservatively with bed rest, plenty of fluids, progesterone support and folic acid.

At 12 weeks fetal USG to see nucal translucency and presence of nasal bone was done along with double marker screening. All other routine antenatal investigations were normal. At 28 weeks patient had mild preterm labor pains. On ultrasound the cervical length was shorter than normal (2.5 cm) and decreased amniotic fluid. Injection betamethasone coverage and bed rest, tocolysis and plenty of fluids were started. In tocolysis beta agonist tablet isoxuperine 40 mg twice a day given for 5 weeks and switched over to calcium channel blockers Nifedepine retard 20 mg twice a day. On regular antenatal follow-up all parameters were normal. At 35 weeks on ultrasound the fetus had intrauterine growth restriction and reduced liquor. Patient developed mild respiratory symptoms like mild breathlessness on walking and mild pedal edema which were considered usual due to normal pregnancy. At 36 weeks 3 days there was increased resistance in umbilical artery blood flow on Doppler ultrasound and on next day umbilical artery flow became absent. Emergency caesarean section was done under combined epidural-spinal anesthesia. A male live baby weighing 2.4 kg was delivered. Peri-operative and immediate postoperative period was uneventful. On 4th postoperative day the patient was discharged with usual advice.

On 5th day the patient developed cough, breathlessness and pedal edema. On 7th postoperative day the pedal edema increased and patient developed severe dyspnoea (NYHA III/IV), orthopnea, severe dry cough and heaviness in chest. On physician consultation pitting pedal edema, raised JVP, BP-100/70, PR-120/min, RR-34/min, bilateral course crepitations, S3 gallop, with SPO2 74% were found. So her urgent admission was done in ICU. ECG showing sinus tachycardia, chest X-ray heart size was increased with pulmonary edema and bilateral pleural effusion. On Echocardiography dilated left ventricle, global hypokinesia, EF- <35%, severe MR and left ventricular dysfunction was present. The diagnosis of dilated PPCM was made and managed conservatively on high flow oxygen therapy, antibiotics, diuretics, vasodilators, beta blockers, digoxin, low molecular weight Heparin (LMWH) therapy, and urine input/output monitoring. After 5 days of ICU stay patient was discharged in stable condition on diuretics, vasodilator and beta blocker. On discharge the ECHO findings were dilated and hypokinetic LV with EF-38% moderate MR. On advised regular medical treatment the patient improved symptomatically with EF 45% at 6 months follow-up.

Discussion

OHSS is an iatrogenic complication of assisted reproduction technology. This syndrome is characterized by cystic enlargement of the ovaries and increased capillary permeability leading to fluid shift from the intravascular to the third space. Accumulation of free fluid in peritoneal cavity and enlargement of the ovaries causes abdominal pain, nausea and vomiting. Due to massive fluid-shift to the third compartment results in intravascular hypovolemia, hemoconcentration causing hypercoagulable state, electrolytes imbalance and development of edema, ascites, hydrothorax and hydropericardium [2]. Treatment of mild to moderate OHSS is supportive consists of observation, bed rest, adequate fluids and USG monitoring of the size of cysts and ovaries. Hospitalization is mandatory for the treatment of severe OHSS where multidisciplinary team can treat and follow-up. Patient with critical OHSS should be admitted in an intensive care unit.

The intravascular blood volume is maintained by correction of the disturbed fluid and electrolyte imbalance, management of secondary complications of ascites and hydrothorax and preventing thromboembolic phenomena. The development of OHSS can be prevented by using low dose gonadotrophins in PCOS, antagonist protocol,[3] prophylactic administration of albumin and cabergolin, and use of LH and agonist for ovulation trigger [4]. The use of hCG in luteal phase support should be avoided to decrease the risk of OHSS [5]. Subcutaneous heparin 5000-7500 U/d is started on the first day of admission to prevent thrombo-

embolism due to hemoconcentration.

PPCM is a rare and life-threatening disease that occurs most frequently in the last trimester of pregnancy and in the first months after delivery [6]. Diagnostic echocardiographic criteria include left ventricular ejection fraction < 45% or M-mode fractional shortening <30% (or both) and end-diastolic dimension >2.7 cm/m^2[7]. These risk factors include increased age, gravidity or parity, African origin, toxaemia or hypertension of pregnancy, use of tocolytics, twin pregnancy, obesity and low socioeconomic status [8]. As in our case patient belongs to increased age with history of tocolysis for long duration. The treatment is standard heart failure therapy (diuretics, vasodilators, and digoxin, anticoagulant as needed).

Therapy regimens include diuretics to diminish volume overload (preload) after load reduction with angiotensin-converting-enzyme inhibitors (postpartum only) and beta-blockers after signs and symptoms of congestion have improved. Treatment options during pregnancy include hydralazine and nitrates [9]. ACE inhibitors should be considered a mainstay of treatment for PPCM after delivery [10]. Calcium channel blockers can be used during pregnancy to control blood pressure (and decrease uterine contractility), but most have negative inotropic properties. Patients with significantly depressed left ventricular function (ejection fraction ≤ 35%) may benefit from anticoagulation therapy to prevent thrombosis and emboli. Arrhythmias should be treated according to standard protocols. Atrial arrhythmias may be treated with digoxin, which may also be used for its positive inotropic effect. Salt and water restriction are important, particularly in women with symptoms and signs of heart failure. If this treatment is ineffective, more aggressive ventricular support such as intra-aortic balloon counter pulsation, and left ventricular assist device or heart transplantation may be considered.

When cardiomyopathy occurs during pregnancy, delivery of the fetus reduces the hemodynamic stresses on the heart. As cardiomyopathy manifests in the final trimester, the fetus is usually mature and can be delivered safely before or at the time of commencement of medical therapy. The mode of delivery for patients with peripartum cardiomyopathy is generally based on obstetric indications. The advantages of vaginal delivery are minimal blood loss, greater hemodynamic stability, avoidance of surgical stress, and less chance of postoperative infection and pulmonary complications. Effective pain management is a necessity to avoid further increases in cardiac output from pain and anxiety. Caesarean delivery is reserved for indications such as fetal distress or failure to progress. Even after complete recovery from peripartum cardiomyopathy, the risk of recurrence in subsequent pregnancies remains high, and LVEF, once improved, can worsen again [11].

In earlier time the OHSS was unavoidable but in today's modern era of ART the OHSS is not only avoidable but also treatable with timely diagnosis and management the severe cases may have optimal outcome. PPCM may be a life threatening condition in any pregnant female that needed a multidisciplinary team approach. With recent knowledge and pro-active team complicated high risk pregnancy can be turned into good maternal and fetal outcome.

Key Messages

In today's modern era of ART the OHSS is not only avoidable but also treatable with timely diagnosis and management. PPCM may be a life threatening condition in any pregnant female that needed a multidisciplinary team approach. With recent knowledge and pro-active team complicated high risk pregnancy can be turned into good maternal and fetal outcome.

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