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Ovarian Hyperstimulation Syndrome an Unusual Case After Embryo Transplant

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KEYWORDS :

INTRODUCTION

OVARIAN HYPERSTIMULATION SYNDROME (OHSS) IS A RELATIVELY COMMON COMPLICATION OF OVARIAN STIMULATION AND CAN BE LIFE THREATENING.It is an iatrogenic complication of assisted reproductive technology.The syndrome is characterized by cystic enlargement of ova and the fluid shift from the intravascular space to the third space.[1].The patho-physiology of OHSS is characterized by, increased capillary permeability, leading to leakage of fluid from the vascular compartment , with third space fluid accumulation and intravascular dehydration.The increased intra-abdominal pressure indicated thatOHSS may be considered a compartment syndrome[2].Today, due to aggressive treatment protocols including the development of invitro fertilization and cryopreservation with the goal of obtaining sufficient number of oocyes and embryos, an increased risk of developing OHSS is present.

CASE REPORT

A 36 years old female, came to NIMS Medical College with known case of primary infertility (inability to conceive even after repeated IUI and oral ovulation induction drugs)with husband semen analysis normal.Patient attended IVF centre and was advised for induction of ovulation after following investigation

INVESTIGATION:Patient was investigated with her HB-13.2 gm% , blood group –B +ve , rest complete blood profile within normal limits. RBS- 73 mg/dl, HIV, HBsAG, VDRL, HCV nonreactive. Her LFT , RFT ,BT, CT within normal limits.All her hormonal profiles were normal wherein her AMH- 8.9 ng/ml ,FSH-3.38 mIU/ml, LH-1.54mIU/ml , TSH-2.2ng/ml, PRL-7ng/ml.

On Diagnostic Hystero –Laproscopy , her uterus and adnexa were normal and chromopertubation test negative showing bilateral tubal blockage.

After going through detailed history ,patient was counseled and advised for IVF. She was then taken for long protocol step down for induction, with recombinant FSH and followed by recombinant HMG. Her E2 on day 2 of stimulation was <20pg/ml progesterone -0.9ng/ml, on day 5 E2- 21.6 pg/ml, on day 8 E2- 461 pg/ml . On day 10 patient had 8 follicles of 18mm on right and left side and rest four were small in both the ovaries measuring around 16mm with E2 of >2000pg/ml. Decision to give trigger with recombinant HCG was taken and ovum pick up was planned after 35 hours. On ovum pick up, 10/10 follicles were retrieved , all were M2. The patient was given prophylactic infusion of albumin and cabergolin was started to prevent OHSS.

On the day of transferher ovaries were bulky, hence patient was explained and counseled about OHSS,and two blastocysts were transferred considering her age.Patient was discharged on request after three days due to some personal reasons.

After four days patient came with chief complain of pain in abdomen, distension of abdomen.On USG, there was moderate ascitis with ovarian enlargement. Patient was admitted in MICU, strictly monitored for vitals, daily input output, abdominal girth charting,USG and serum creatinine. Proper fluid management with adequate input output, albumin infusion given, all luteal phase support medications were stopped.Patient became apparatly alright ten days after admission and discharged.

CONCLUSION: OHSS can be thought of, as the loss of control over hyperstimulation of the ovaries. Although the prevalence of the severe form of OHSS is small , it is important to remember that OHSS is usually an iatrogenic complication of a non vital treatment that has the potential for a fatal outcome.

DISCUSSION:

Ovarian hyperstimulation syndrome (OHSS) is a rare complication of ovulation induction therapy. The syndrome was first described in 1941 and first fatal case of OHSS with renal failure and death was described in 1951.familial cases of recurrent OHSS are seen in person with mutation in follicle stimulating hormone (FSH) receptors. [4].

Etiopathogenesis

Invitro fertilization techniques use GnRH agonist or antagonist and gonadotropin to stimulate the ovary. Following stimulation, human chorionic gonadotrophin is used to initiate ovulation and maintain luteal phase. These agents,alone or together produce a state of increased capillary permeability which is a hallmark of OHSS. Serum VEGF levels correlate with severity of OHSS .[5,6]

Clinical Features: Symptoms usually begin with sensation of bloating , abdominal discomfort, nausea, vomiting.As disease progress accumulation of fluid in third space leads to ascitis, pleural and pericardial effusion, oligouria, haemoconcentration, hypovolemia and electrolyte imbalance.[7,8,9]

Laboratory data is characterized by ,haemoconcentration >45%, and raised WBC and PCV[9]. Electrolyte disturbances in the form of hypernatremia and hyperkalemia may be seen. Abnormal liver function test are seen in 30 % of patients in OHSS.[10]. Hypoalbuminemia is universal. Raised serum creatinine points to severe OHSS . low levels of IgA & IgG are seen in patients with severe OHSS and predisposes to infection.[11]. The plasma levels of rennin, aldosterone, noradrenaline,antidiuretic hormone(ADH) and ANP are increased.[9,11]. Urinary sodium concentration is low in most patients. Ascitic fluid study reveals high protein and low cell counts.[11].

Complications:

Pulmonary complication of OHSS include hydrothorax, pulmonary embolism, ARDS, pulmonary edema, atelactasis and intra alveolar haemmorage [5].

Pleural effusion develops in approximately 20% of severe OHSS. Usually bilateral.[5]

Thromboembolism is common in OHSS due to high estrogen, haemoconcentration, reduce circulating blood volume and thrombocytosis. [12,13]

High incidence of infection is attributed to decreased IgG and IgA levels. Most common cause of fever is urinanry tract infection. Seen in 20.5% patient.

The causative organisms isolated includes klebseilla pneumonia, proteus merabillis, ecoli, pseudomonas areginosa, and proteus vulgaris. [14].

Classification:
Mild OHSS
GRADE I – abdominal distension and discomfort
GRADE II- grade I plus nausea , vomiting or diarrhea plus ovarian enlargement from 5 -12 cm .
MODERATE OHSS
GRADE III- features of mild OHSS plus USG evidence of ascites.\
SEVERE OHSS
GRADE IV- features of moderate OHSS plus clinical evidence of ascites and hydrothorax and breathing difficulties.

GRADE V- all of the above plus a change in blood volume , increased viscosity due to haemoconcentration, coagulation abnormalities and diminished renal perfusion. [15]

Management:
There is no specific treatment for OHSS . therapy is manily supportive.

The syndrome is self limiting and resolution parallels default in HCG levels.

Mild cases can be managed on out patient basis with daily meassurment of weight, urinary output, avoidance of stranious activity and sexual intercourse.

Oral fluid intake should be monitored. Patient should have serial meassurments of haematocrit , electrolytes and creatinine. [16,17,18]

Moderate OHSS usually subsides with bed rest for 2-3 weeks. [16]

Severe OHSS should be managed as in patient. Haemodynamic and respiratory status should be assessed, physical examination to rule out venous thrombosis should be done. And iv line ideally subclavian catheter should be placed to measure central venous pressure. [16]. Patient should be monitored daily with haematocrit, total count, electrolytes, LFT, RFT[16,19,20]

Ultrasound abdomen is done to measure the size of ovaries and ascites.

A chest radiograph after shielding the uterus should be done. Along with blood gas analysis and oxygen saturation. In case of respiratory failure endotracheal intubation and mechanical ventilaton may be required. [19,20]

In patient with haematocrit > 45% or hypoalbuminemia less than 30 gm/lt. or ascites, human albumin is the plasma expander of choice. Once sufficient volume expansion has been achieved and haematocrit is less than 36%, furosamide should be given to acess renal function. [16,19,21].

In severe cases of OHSS prophylactic anticoagulation should be used.

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