A Case of Severe Ovarian Hyperstimulation Syndrome: A Case Report

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INTRODUCTION
Ovarian hyperstimulation syndrome (OHSS) involves an increase in vascular permeability resulting in a fluid shift from intravascular to third space compartments such as the peritoneal and thoracic cavities. First described in 1943, the first fatal cases were documented in 1951.¹ This syndrome occurs almost exclusively during assisted reproductive technology (ART) cycles, although OHSS might also occur during ovarian stimulation using gonadotropins and clomiphene citrate

CASE PRESENTATION
A 27 yrs women was hospitalized on 08/09/2016 at civil hospital B.J. Medical College Ahmedabad presented to emergency department with chief complaints of pain abdomen, abdominal distension since 7 days. The patient was managed symptomatically with no complications. Although ovarian hyperstimulation is a rare entity, it is important that the physician recognizes this condition. Prompt diagnosis and successful management is likely to avoid serious and rapid development of complications.

Patient is a 27 yrs P1 L1 A0 woman ovum donor since last 5 months. Her pulse was 92/min, BP 130/8mmHg oxygen saturation 94% with venti mask. The physical examination revealed reduced bilateral air entry into lungs and severely distended abdomen with evidence of ascites but without any palpable mass in the abdomen.

The patient was on regular gonadotropin injections first for the treatment of infertility, later for ovum formation as OVUM DON0R. Which she continued even after she developed moderate free fluid in abdomen. Patient undergoes TVS–pelvis at Isanpur Pvt Hospital every month followed by some injection (inj HCG) deep im, this is followed by ovum pick up at different places (Ovagen Hospital). Last pick up done 6 days back.

Her menarche was at 13 yrs of age and her cycles were regular(4-5days/28-30days). Her last menstrual period was on 10/08/2016. Married 10 yrs back she had conceived as a result of infertility treatment after 6yrs of marriage. Laboratory tests revealed renal function tests and liver function tests within normal range. Other laboratory tests at admission were hemoglobin 8.4g% (9-11g%), total

CASE REPORT
ABSTRACT
BACKGROUND: To report a case of ovarian hyperstimulation syndrome following controlled ovarian hyperstimulation. CASE PRESENTATION: A 27 years women had severe OHSS as a complication of gonadotropin stimulation. Clinical picture showed enlarged ovaries, massive ascites, pleural effusion, abdominal pain. Conservative medical treatment showed massive improvement without any invasive measures being done. CONCLUSION: Physicians can reduce the risk of OHSS by monitoring gonadotropin therapy and by withholding human chorionic gonadotropin medication. In in vitro fertilization protocols it can be advantageous to postpone the embryo transfer by freezing the embryos.

Key words: Human chorionic gonadotrophin, ovarian hyperstimulation, intravascular depletion

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A Case of Severe Ovarian Hyperstimulation Syndrome

Immediately after admission, patient was started on symptomatic therapy with close monitoring. Infusion therapy was started, consisting of normal saline infusion, 5% dextrose, injectable antibiotics, and injection dexamethasone. Daily examination with Body weight, abdominal circumference, intake and output, breath sounds continued for 4-5 days. Laboratory studies were monitored strictly daily. Renal function was supported using diuretics.

Followup Ultrasonography: both ovaries bulky in size & shows multiple varying sized follicular cysts & echogenic stroma. Few of them showing internal septations and echoes. Both ovaries show raised internal vascularity.

- left ovary measures 70 x 56 x 57 mm (110 cc)
- right ovary measures 88 x 54 x 86 mm (210 cc)

Laboratory parameters were normalised and pleural effusion and ascites resolved and she was discharged after 2 weeks of hospital stay, in a stable condition.

DISCUSSION

Ovarian hyperstimulation syndrome may rarely be associated with a spontaneous ovulatory cycle, usually in the case of multiple gestations, hypothyroidism or polycystic ovary syndrome. Primary risk factors for the development of OHSS include young age, polycystic ovary...
syndrome (PCOS), prior OHSS, high basal anti mullerian hormone levels, while secondary risk factors are high or rapidly rising estradiol (E2) levels, high number of follicles, high vascular endothelial growth factor (VEGF) and high Inhibin B levels. As many as one in three (33%) women develop mild OHSS. About one in 20 (5%) women develops moderate or severe OHSS. A serum estradiol (E2) level of 12,315 pmol/L (3,354 pg/mL) on day 11 of ovarian stimulation gives a sensitivity and specificity of 85% for the detection of women at risk for OHSS. The syndrome is associated with the process of ovulation induced by luteinizing hormone or human chorionic gonadotropin. Following ovulation, one or more substances produced by the ovary are liberated in excess, increasing capillary permeability, resulting in the clinical features of the syndrome. It may well be that the syndrome is not triggered by a single mechanism but by the production and secretion of several substances acting in concert. These may include prostaglandins, cytokines, interleukins, the ovarian renin angiotensin system, vascular endothelial growth factor, and nitric oxide. Enlargement of the ovaries causes abdominal pain, nausea and vomiting. When OHSS is classified as severe, the subsequent clinical picture is the result of increased vascular permeability and ascites, leading to dehydration with haemoconcentration. The extravascular albumin-rich exudate accumulates in the peritoneal cavity, occasionally in the pleura, and rarely in the pericardiac space. Intravascular hypovolemia with concomitant development of edema, ascites, hydrothorax and/or hydropericardium can lead to life-threatening complications including thromboembolic events and even death. Proposed RCOG classification of severity of OHSS

Category Features

- **Mild OHSS** Abdominal bloating Mild abdominal pain Ovarian size usually < 8 cm.
- **Moderate OHSS** Moderate abdominal pain Nausea ± vomiting Ultrasound evidence of ascites Ovarian size usually 8–12 cm
- **Severe OHSS** Clinical ascites (±hydrothorax) Oliguria (< 300 ml/day or < 30 ml/hour) Haematocrit > 0.45 Hyponatraemia (sodium < 135 mmol/l) Hypo-osmolality (osmolality < 282 mOsm/kg) Hyperkalaemia (potassium > 5 mmol/l) Hypoproteinaemia (serum albumin < 35 g/l) Ovarian size usually > 12 cm.
- **Critical OHSS** Tense ascites/large hydrothorax Haematocrit > 0.55 White cell count > 25 000/ml Oliguria/anuria Thromboembolism Acute respiratory distress syndrome

Antiphospholipid antibodies (APLA) are a family of immunoglobulins that recognize a variety of plasma proteins in association with anionic phospholipids. These antibodies may lead to a number of clinical syndromes including venous and arterial thromboses, thrombocytopenia, and recurrent fetal loss. Ovarian stimulation in infertile women with PCOS poses a particular challenge, as many of these women exhibit exaggerated response to clomiphene citrate, gonadotropins, ovarian drilling, insulin sensitizing agents or assisted reproductive technology (ART), resulting in an increased risk of ovarian hyperstimulation syndrome (OHSS) and multiple gestations. The recent implementation of four new modalities, the GnRH antagonist protocol, GnRH agonist (GnRHa) triggering of ovulation, blastocyst transfer and embryo/oocyte vitrification, renders feasible the elimination of OHSS in connection with ovarian hyperstimulation for IVF treatment.

**CONCLUSION**

If the OHSS develops during the treatment of infertility, it may be prudent to postpone the treatment since the establishment of pregnancy can lengthen the recovery time or contribute to a more severe course as in our case. Physicians can reduce the risk of OHSS by monitoring gonadotropin therapy and by using this medication cautiously. The patients with OHSS in early stage must be identified and treated urgently and
A Case of Severe Ovarian Hyperstimulation Syndrome

with multidisciplinary management. If left untreated, OHSS can result in serious health complications and even death.

REFERENCES