Case Report

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Continuous Abdominal Paracentesis for Management of Late Type Severe Ovarian Hyperstimulation Syndrome

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The ovarian hyperstimulation syndrome (OHSS) is often observed in patients undergoing assisted reproductive technology (ART). In severe form OHSS is a serious and potentially life-threatening. Here we report a 36-year-old woman with primary infertility due to endometriosis who underwent controlled ovarian hyperstimulation. Ten days later, severe late-onset ovarian hyperstimulation syndrome, severe ascites and pulmonary effusion, developed. Continuous abdominal paracentesis of 5000 mL/day was performed on the third day. With this procedure, ascitic fluid was drained efficiently and the patient's condition improved. This report suggests that early continuous abdominal paracentesis with drainage of ascitic fluid is an efficacious procedure for management of the severe ovarian hyperstimulation syndrome as soon as euvolemia is achieved clinically.

Key Words
continuous paracentesis;
euvolemia;
OHSS

The ovarian hyperstimulation syndrome (OHSS) is often observed in patients undergoing controlled ovarian hyperstimulation (COH) cycles. In its severe form, OHSS is a serious and potentially life-threatening complication resulting in respiratory failure, severe hemoconcentration, hypovolemia, and electrolyte imbalances. Ascites and hydrothorax causing respiratory difficulty are typical in severe OHSS. Treatment in severe OHSS is supportive and is aimed at correcting electrolyte imbalances and hemoconcentration. A recent study[1] showed that for women with severe OHSS, abdominal paracentesis significantly improves urinary output, regardless of medical treatment. We report a case of late severe OHSS occurring on the 10^{th} day after ovum retrieval. Conservative management such as abdominal tapping was unsuccessful. Subsequently, continuous abdominal paracentesis provided successful relief of OHSS symptoms.

CASE REPORT

A 36-year-old woman with primary infertility due to endometriosis for 8 years underwent ovulation induction for *in vitro* fertilization. The medication used for ovulation induction was 225 IU/day recombinant follicular stimulating hormone (FSH) (Gonal-F; Laboratories Serono Aubonne, Switzerland) for 10 days after the patient achieved adequate endogenous gonadotropin suppression by long protocol of Gonadotropin releasing hormone analog (GnRHa) (Leuprolide Acetate; Abbott Laboratories North Chicago, Illinois, USA). On the human chorionic gonadotropin (hCG) day, the serum estradiol (E_2), 1722 pg/ mL and follicle number 8 under ultrasound were monitored. A total of 4 oocytes were retrieved after a transvaginal oocyte aspiration. Four eight-cell embryos were transferred 3 days after retrieval. After retrieved, the medication used for luteal

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support was 1500 IU hCG on days 2, 5 and 8. Ten days after retrieval, the patient suffered from nausea, vomiting, general edema, abdominal bloating, shortness of breath, and weight gain. Upon admission, physical examination revealed a pale, ill-appearing woman in moderate distress suffering from abdominal pain. The body weight was 56 kg and the abdominal circumference was 87 cm. Supine blood pressure was 100/60 mmHg with a pulse of 78 beats/minute, but systolic blood pressure decreased to 50 mmHg and the pulse rate increased to 130 beats/minute on standing. She had an effortless respiratory rate of 19 breaths/minute. In addition, physical examination showed decreased breathing sounds at the right lung base and tense ascites accompanied by diffuse abdominal tenderness, rebound and percussion tenderness. The laboratory values were notable for hematocrit of 38.8% and a white blood cell count of $22.5 \times 10^3/mm^3$ with 85% neurophils and 0% band forms. Serum chemistry results included sodium, 130 mmol/L, potassium, 4.8 mmol/L, chloride, 101 mmol/L, aspartate aminotransferase (AST), 85 IU/L, alanine aminotransferase (ALT), 109 IU/L, creatinine, 0.7 mg/dL, total protein, 4.2 g/dL, and albumin, 2.8 g/dL. There were markedly enlarged cystic ovaries (right, 8 × 7 cm; lift, 8 × 5 cm) plus a voluminous ascites as demonstrated by abdominal sonogram. She received symptomatic treatment of intravenous isotonic fluid (normal saline 3000 mL/day) and albumin (25% in 50 mL, 4 bottles/day). In addition, abdominal paracentesis of 1000 mL was performed on the first day and 700 mL asutes was drained on second day, respectively followed by continuous abdominal paracentesis of 5000 mL in 4 hours on the third day. During the drainage time, the blood pressure and the pulse rate were kept in normal range. Thereafter, the symptoms subsided. Two days later, the patient's body weight was 53 kg and the abdominal circumference was 82 cm. CBC profile was hematocrit of 35.1%, white blood cell count of $9.2 \times 10^3/mm^3$, red blood cell count of $3.78 \times 10^6/mm^3$ and Hb of 12.2 g/dL. Serum chemistry result included sodium, 139 mmol/L, potassium, 3.3 mmol/L, chloride, 109 mmol/L, AST, 21 IU/L; ALT, 19 IU/L, creatinine, 0.7 mg/dL, total protein, 5.6 g/dL, albumin, 3.6 g/dL and urine output of 3700 mL/day. One week later, 3 intrauterine gestation sacs were noted by sonogram at our outpatient department.

DISCUSSION

The treatment for OHSS is predominantly supportive, with the correction of electrolyte imbalances and expansion of intravascular volume thereby reducing hemoconcentration and promoting diuresis. The primary indications for abdominal paracentesis are symptomatic (severe discomfort or pain and pulmonary compromise). Oligouria without such symptoms requires primarily intensive hydration, rather than immediate paracentesis of ascitic fluids. Paracentesis is then indicated when renal compromise does not respond to hydration. Continuous complete paracentesis can completely release the symptoms, but small tapping (< 1000 mL) only releases the symptom for a short time and needs repetitive tapping. In this case, the patient was intensively hydrated on the days following her admission, but required abdominal paracentesis subsequently. Paracentesis of ascitic fluids in women with severe OHSS has an isolated effect on renal function, as is proven by increased urinary output, improved renal function, creatinine clearance, and reduced blood urea nitrogen.[2] Such management results in euvolemia and massive ascites, which causes intra-abdominal pressure sufficient to impede renal blood flow. In this case, abdominal paracentesis significantly improved urinary output, regardless of medical treatment. In addition, improvement in hemoconcentration was supported by the reduction of BUN, hematocrit, and leukocyte concentration. The impact of continuous paracentesis on the ongoing pregnancy was unknown, but in this case, an uneventful twin pregnancy after fetal reduction at 11 weeks was noted. The mechanisms by which removal of ascitic fluid increases urine production in severe OHSS include reduction in intra-abdominal pressure compressing the vena cava, increase in venous return, improved urinary perfusion, output, and function in other situations involving ascities, such as cirrhosis[3] of the liver.

Despite knowledge of prediction and prevention of OHSS, 8% to 23% of patients undergoing ovulation induction experience mild symptoms; up to 7% experience moderate symptoms; and 1.8% develop severe symptoms.[4] Mild or moderate OHSS is usually self-limiting within 2 weeks and requires no therapy beyond evalua-

tion of daily weight to detect sodium retention. Severe OHSS is marked by tense ascites, respiratory distress, and hemoconcentration. Late OHSS is a more serious and potentially life-threatening physiologic complication than the early type.

The hCG is the most significant factor influencing the clinical course of moderate and severe OHSS. In the course of an assisted reproductive technology cycle, a patient can be exposed to hCG from many sources, both endogenous and exogenous. Exogenous hCG is universally given as an ovulatory trigger and occasionally used for luteal support. Those who conceive via assisted reproductive technology will also be exposed to endogenous hCG. The continuous and progressive production of hCG from the rapidly proliferating trophoblast appears to be a factor causing late OHSS. Several risk factors[5] for OHSS have been identified, including younger age, low body weight,[6] number of oocytes retrieved[7] and polycystic ovarian disease as a result of ovulation induction.[8] Careful monitoring of ovarian stimulation with follicle ultrasound and serum E_2 can be used as a best predictor for OHSS.[9] Prevention techniques are as following: lowest standard gonadotropin dosing regimen, lower than standard dose of hCG for oocyte maturation and luteal phase support, withholding medications at mid-cycle or delaying the administration of hCG (coasting),[10] prophylactic intravenous human albumin at oocyte retrieval, cryopreserving all embryos, and postponed embryo transfer.[11]

In this case, earlier continuous aspiration of the ascitic fluid would have improved the patients' condition as soon as euvolemia was reached. We suggest that abdominal paracentesis with continuous drainage should be performed earlier in such patients.

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