Global Science Research Journals

Vol. 4 (1), pp. 075-077, January, 2016

http://www.globalscienceresearchjournals.org/

Medical Case Studies and Case Reports

Case Report

Ovarian hyperstimulation syndrome in a 35-year old woman: A case report

Sulieman M.A. Sharafeldin[1] and Bahaeldin E. ELawad[2*]

[1]Department of Obstetrics and Gynecology. Faculty of Medicine. University of West Kordofan. Elnihood (Sudan).
[2]Department of Physiology. Faculty of Medicine. University of Umm-ALqura. Makkah (Kingdom of Saudi Arabia)

Accepted 18 January, 2016

Ovarian hyperstimulation syndrome is almost exclusively associated with exogenous gonadotropin stimulation and is very rarely observed after clomiphene citrate therapy or spontaneous ovulation. This report demonstrates a rare case on spontaneous ovarian hyperstimulation syndrome which gave rise to a full-term healthy baby. A-35-year old woman who was gravida III para II + 0 presented with amenorrhoea for three months and recent attack of vomiting and abdominal pain. She denied any exposure to ovulation induction therapy. Abdominal examination demonstrated tender abdomen and pelvic abdominal mass up to the level of umbilicus. Possible differential diagnoses were excluded. There was no clinical evidence of ascites. The patient was pale with hemoglobin of 5.1 g/dL.

Key words: Ovarian hyperstimulation syndrome; spontaneous ovarian hyperstimulation syndrome.

INTRODUCTION

Ovarian hyperstimulation syndrome (OHSS) is an iatrogenic complication of supraphysiologic ovarian stimulation during assisted reproductive technology (ovulation induction therapy) for the treatment of infertile patients. The syndrome is almost exclusively associated with exogenous gonadotropin stimulation and is only rarely observed after clomiphene citrate therapy or spontaneous ovulation (1). Most cases of OHSS are mild and of little clinical concern. However, when OHSS is severe, it is occasionally associated with severe morbidity, and fatalities have been reported (2). With rare exceptions, OHSS occurs only after a lutenizing hormone surge or exposure to human chorionic gonadotropin (3). After gonadotropin superovulation for invitro fertilization, the reported incidence of moderate OHSS is 3% to 6%, and for severe form is 0.1% to 2% (4). The mild form, which has little clinical consequence, occurs in about 20% to 33% of invitro fertilization cycles (5). The pathophysiology of OHSS is not well understood. Severe OHSS is a systemic condition thought to result from granulosa cell secretion of vasoactive substances into follicular fluid within hyperstimulated ovaries (1). Clinically the fundamental physiologic change in severe OHSS is an increase in the vascular permeability resulting in a fluid shift from intravascular to third space compartments such as peritoneal and thoracic cavities (6). Studies have shown serum vascular endothelial growth factor (VEGF) levels to correlate with the severity of OHSS (6).

*Corresponding author. E-**mail**: bahaelawad@gmail.com, **Tel**: 00966535548549 (KSA), 00249916085907 (Sudan), **Fax**: 00966125582711

Additionally, human chorionic gonadotropin has been shown to increase VEGF gene expression in granulosa cells, which in turn raises serum VEGF concentration (7). Other mediators such as angiotensin II, insulin-like growth factor-1, and inerleukin-6 have also been implicated in the disease process (8). Several factors increase the risk of developing severe OHSS. These include the following: age less than 30 years, Polycystic ovaries or high basal antral follicle count on ultrasound, and low body weight (9); and Previous history of OHSS (10).

Case Report

A 35-year- old woman who was gravida III para II + 0 presented to the outpatient clinic of obstetrics on 29th of April 2015 with amenorrhoea for three months and recent attack of vomiting and abdominal pain. The patient conceived naturally and denied any exposure to ovulation induction therapy. Her age at menarche was 11 years. She used to have a regular cycle length of 28 days and menstrual flow of 3 to 5 days duration.

The abdominal pain was in form of bilateral lower colicky nature of three days duration. The patient was afebrile, pale, and looking ill. Pulse, 92 bpm of low volume; arterial blood pressure, 90/40 mm Hg; respiratory rate, 20 breaths per minute; core body temperature, 37 degree celcius; body weight, 68 Kg; and body mass index, 24.

Abdominal examination demonstrated tender abdomen and pelvic abdominal mass up to the level of umbilicus. There was no rebound tenderness. There was no clinical evidence of ascites. Examination of other systems was unremarkable.

On complete blood count: Hemoglobin was 5.1 g/dL; packed cell volume, 32%; white blood cell count, 6800/µL; and platelet count 180000/µL. Blood coagulation tests were normal; liver function tests: total bilirubin 0.3 mg/dL, Direct bilirubin 0.2 mg/dL, albumin 3.5 g/dL, alkaline phosphatase 276 U/L, SGOT 36.6 U/L, SGPT 20U/L; renal function tests; thyroid function tests; and blood electrolytes were normal. Urinalysis was not specific.

Ultrasound scanning revealed a gravid uterus with a viable fetus of 12 weeks gestation and huge ovaries with multiple large loculated cysts replacing the entire ovarian parenchyma. The right ovary measuring 11 cm X 8cm with the largest cyst measuring 6 cm in diameter. The left ovary measuring 7cm X 6 cm with the largest cyst measuring 4.5 cm in diameter. Mild intraperitoneal fluid collection (ascites) was demonstrated. There was neither pleural effusion nor pericardial effusion detected. All other organs were normal. Acute appendicitis, pelvic infection, ectopic pregnancy, ovarian cyst torsion or hemorrhage, intra-abdominal hemorrhage, and malignancy were excluded.

The patient received three units of blood and analgesia in form of diclophenac given in a dose of 75 mg IM. Within two weeks of admission pain was relieved and tenderness had disappeared. A second ultrasonographic examination done on 14th week of gestation revealed regression of both ovaries (7 cm X 3 cm). Laboratory results were standstill and the patient was well discharged. Pregnancy continued normally, and the lady delivered a full-term healthy normal boy of 3.1 Kg body weight.

DISCUSSION

Spontaneous OHSS is extremely rare in naturally conceived pregnancies. OHSS in the absence of exogenous goandotropins is very rare, and only a few cases have been reported in the literature (11). This report demonstrates a rare case of spontaneous OHSS associated with pregnancy which gave rise to a healthy full-term baby. The patient attended to the outpatient clinic at 12th week of gestation. Spontaneous OHSS is likely to occur at 8-14 weeks of gestation, while iatrogenic OHSS occurs earlier at 3-8 weeks of gestation (12). OHSS is a clinical diagnosis and is divided into four grades (mild, moderate, severe, and critical) according to the classification proposed by Navot et al (10) and modified by Mathur et al (1). The complaint of vomiting and abdominal pain and ultrasonic evidence of ascites with ovarian size less than 12 cm categorized our case as moderate OHSS. The pathophysiology of OHSS is not well understood, but the progression of moderate OHSS to severe OHSS is thought to be the result of increased vascular permeability in the region surrounding the ovaries and their vasculature (13). Loss of albumin from intravascular compartment leads to decreased plasma oncotic pressure. Consequently, intravascular fluid shifts to third space extravascular compartment leading to ascites and very occasionally pleural effusion or pericardial effusion. The intravascular volume depletion results into hemoconcentration with consequent hypercoagulability and its adverse complications (14). Smisha and Sridev (15) reported a case on spontaneous OHSS following natural conception and associated with primary hypothyroidism. In their case there was no ultrasonological evidence of ascites, pleural effusion or pericardial effusion; hemoglobin concentration was 9.6 g/dL; white blood cells and platelets were within normal range. They demonstrated that, unlike in iatrogenic OHSS, in spontaneous OHSS extravascular fluid retention may not occur and usually a hemodilution rather than hemoconcentration is seen (the blood picture showed anemia instead of hemoconcentration). Smisha and Sridev claimed that, this may be due to the difference in the etiopathogenesis of iatrogenic and spontaneous OHSS (15). In our case, there was ultrasonic evidence of ascites and the hemoglobin concentration was, extremely low, only 5.1 g/dL, that is to say very much dilution. Thus, despite retention of fluid in the extravascular compartment, hemocncentration did not occur. Therefore,

either, the pathophysiology of spontaneous OHSS differs from that of iatrogenic OHSS or shifting of fluid to third space is not the only, if not the genuine, factor responsible for hemoconcentration that characterizes severe OHSS and its catastrophic sequences. These factors need to be clarified. Moderate OHSS is not incompatible with successful gestation. The clinical suspicion of this syndrome has to be raised in order to save pregnancies. Pathophysiology of OHSS remains to be elucidated to allow early diagnosis, preventive measures, and appropriate treatment of this potentially life-threatening condition.

REFERENCES

1. Marthur R, Kailasam C, Jenkins J. Review of the evidence base strategies to prevent ovarian hyperstimulation syndrome. Hum Fertil 2007;10:75-85
2. Semba S, Moriya T, Yousses EM et al. An autopsy case of ovarian hyperstimulation syndrome with massive pulmonary edema and pleural effusion. Pathol I*nt 2002;*50:549-52
3. Delvigne A and Rozenberg S. Epidemiology and prevention of ovarian hyperstimulation syndrome (OHSS):a review. Hum Reprod update 2002;8:559-77
4. Serour GI, Aboulughar M, Mansour R et al. Complications of medically assisted conception in 3,500 cycles. Fertil Sterlil 1998;70:638-42
5. Morris R.S., Miller C, Jacobs L, et al. Conservative management of ovarian hyperstimulation syndrome. J Reprod Med 1995;40:711-4
6. Geva E and Laffe RE. Role of vascular endothelial growth factor in ovarian physiology and pathology. Fertil Steril 2007;74:429-38
7. Neulen J, Yan Z, Racezek S et al. Human chorionic gonadotropin-dependent expression of vascular endothelial growth factor/vascular permeability factor in human granulosa cells: importance in ovarian hyperstimulation syndrome. J clin Endocrinol Metab 1995;80:1967-71
8. The practice committee of American society for reproductive medicine. Ovarian hyperstimulation syndrome. Fertil Steril 2006;86(suppl 4):S178-S183
9. Navot D, Relon A, Birkenfeld A. et al. Risk factors and prognostic variables in the ovarian hyperstimulation syndrome. Am J Obstet Gynecol 1988;159:210-15
10. Navot D, Bergh PA, Laufer N. Ovarian hyperstimulation syndrome in novel reproductive technologies: prevention and treatment. Fertil Sertil 1992;58:249-61
11. Ahmed Kamel RM. Spontaneous ovarian hyperstimulatio syndrome in a naturally conceived singleton pregnancy. Fertil Steril 2010;94:351.e.1-4
12. Smits G, Olatunbosun O, Delbaere A et al. Ovarian hyperstimulation syndrome due to a mutation in the follicle stimulating hormone-receptor. N Eng J Med 2003;349:760-6
13. Morris RS, Paulson RG. Ovarian derived prorenin angiotensin cascade in human reproduction. Fertil Steril 1994;62:1105-14
14. Vavilis D, Tzitzimikas S, Agorastos D et al. Postparacentesis bilateral massive vulval edema in a patient with severe ovarian hyperstimulation syndrome. Fertil Steril 2002;77:841-3
15. Smisha Sridev and Sridev Barathan. Case report on spontaneous ovarian hyperstimulation syndrome following natural conception and associated with primary hypothyroidism. J Hum Reprod Sci 2013;6(2):158-161