International Journal of Reproduction, Contraception, Obstetrics and Gynecology
Thathira S et al. Int J Reprod Contracept Obstet Gynecol. 2026 Jun;15(6):2286-2289
www.ijrcog.org
pISSN 2320-1770 | eISSN 2320-1789

DOI: https://dx.doi.org/10.18203/2320-1770.ijrcog20261651

Case Report

Ovarian hyperstimulation syndrome in a spontaneous pregnancy: a diagnostic dilemma

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Received: 25 April 2026
Revised: 20 May 2026
Accepted: 21 May 2026

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ABSTRACT

Spontaneous ovarian hyperstimulation stimulation (sOHSS) is an extremely rare condition reported in a spontaneously/ naturally conceived pregnancy. It is most commonly associated with multiple gestation, hypothyroidism, and polycystic ovarian syndrome (PCOS). Severe form is a rare entity in a singleton pregnancy with spontaneous ovulation. These cases can very well be misdiagnosed as an acute abdomen in pregnancy and may lead to unnecessary exploratory laparotomy or a malignancy. The following case reports a 20-year-old female with a spontaneous normal singleton pregnancy presenting with acute abdomen, vomiting and distention of the abdomen. Ultrasonography reveals a 5.5-week viable intrauterine single fetus with bilateral multilocular cystic ovarian masses with ascites. The patient was managed conservatively with oral cabergoline and resolution of symptoms with eventual uncomplicated pregnancy.

Keywords: Ovarian hyperstimulation stimulation, Pregnancy, Dopamine agonist, Ascites

INTRODUCTION

Ovarian hyperstimulation syndrome (OHSS) is typically a form of hyperreaction luteinalis.[1] Mild cases may be asymptomatic or present with nausea, vomiting, and abdominal distension. Moderate-to-severe OHSS occurs in 3 to 8% of in vitro fertilisation cycles.[2] However, OHSS in a spontaneous natural pregnancy is extremely rare, with incidence rates ranging from 0.2 to 1.2%.[3] Herein this case reports a rare case of moderate OHSS in a patient with spontaneous conception of pregnancy with no antecedent cause identified, creating a diagnostic dilemma and delaying appropriate management and further treatment.

CASE REPORT

A 20-year-old female presented to our emergency department with complaints of severe abdominal pain, tense distention of the abdomen and vomiting for 1 day and insidious in onset with 1.5 months of amenorrhea.

On examination, a urine pregnancy test was done, which was positive. The patient was unaware of pregnancy as it was a spontaneous, naturally conceived pregnancy with no history of ovulation induction or exogenous estrogen administration.

On examination, the patient was a well-nourished female weighing 62 kg with stable vitals. Systemic examination revealed a moderately distended abdomen, tense with positive shifting dullness on percussion. On auscultation of the abdomen, bowel sounds were present. On pelvic examination, bilateral cystic masses could be felt. Uterine size could not be appreciated.

An urgent ultrasound was done, suggestive of an intrauterine pregnancy of 5 weeks, with a gestational sac with no fetal pole and yolk sac seen, with gross ascites with multiple enlarged follicles each measuring around 35×40 mm. The bilateral ovaries measure approximately 17×15×12 cm (Figure 1).

The preliminary blood investigations revealed haemoglobin of 12.1 g/dl and hematocrit of 35.5%, and serum electrolytes, liver function test, renal function tests and coagulation profile were normal. The TSH being 5.12, the patient was started on levothyroxine 50 micrograms for treating hypothyroidism.

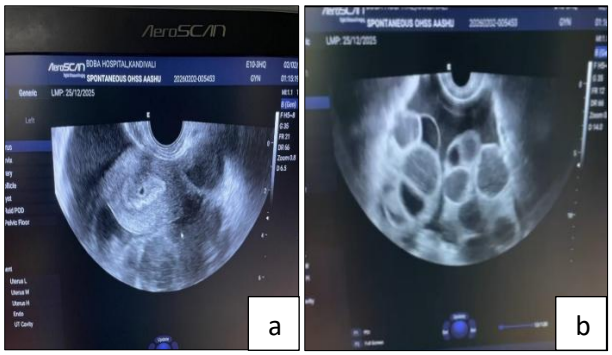

Figure 1 (a and b): Ultrasonographic imaging revealed bilateral ovarian enlargement with multiple contiguous thin-walled cysts containing anechoic fluid.

Tumour markers like Ca 125, carcino embryonic antigen (CEA), lactate dehydrogenase (LDH), alpha feto protein (AFP) and Ca 19.9 were sent, which were within normal limits to rule out ovarian malignancy. Serum estradiol was 10,200 picograms/ml. Chest X ray was within normal limits. A provisional diagnosis of severe spontaneous OHSS was made and the patient was admitted for supportive therapy and monitoring.

Liberal oral intake was encouraged. Medications included cabergoline 0.5 mg oral for 14 days and oral metoclopramide and pantoprazole. Daily monitoring of abdominal girth and weight was taken. Both the parameters showed an increase from 58 to 63 kg and 90 to 104 cm, respectively till day 5. Hematocrit was monitored daily and showed a rising trend till day 5. There was no respiratory difficulty or any significant symptoms. Patient started improving clinically by day 5 with supportive therapy. There was spontaneous resolution of symptoms and hence was discharged on day 8 of hospitalization on with blood parameters showing improvement in hematocrit and rest of the routine investigations in normal limits.

The patient was followed up in OPD every fortnightly. Resolution of the ascites and decrease in follicular size occurred gradually, as demonstrated by clinical and ultrasonographic studies. The patient's pregnancy proceeded normally and delivered vaginally at 39 weeks with a 3.4 kg male baby.

DISCUSSION

Spontaneous OHSS is a rare, potentially life-threatening condition characterised by massive ovarian enlargement and third-space loss of fluid in the absence of external fertility drugs. Unlike iatrogenic OHSS, which occurs early, sOHSS typically develops later, usually between 8 and 14 weeks of pregnancy, or in cases of severe hypothyroidism or molar pregnancy.[4]

The pathophysiology of spontaneous OHSS has been explained in various literature as the structural similarity between beta hCG and the pituitary glycoproteins such as follicle stimulation hormone (FSH), thyroid stimulating hormone (TSH) and leutnising hormone (LH), leading to overstimulation of the FSH receptors of the ovary. The other proposed theory is mutated FSH receptor is being stimulated for overproduction of FSH and leading to s-OHSS.[5]

In case of primary hypothyroidism, the low levels of circulating T3 and T4 lead to stimulation of thyrotropin-releasing hormone (TRH) in the hypothalamus, leading to release of LH, FSH and prolactin that in turn leads to excessive release of FSH and hence the follicular growth. Similarly, the hypermutated FSH receptors lead to follicular growth due to excessive FSH release, due to structural similarities to beta hCG and the pituitary glycoproteins crosslinking at the binding sites.

The overstimulated, luteinized follicles release high amounts of vascular endothelial growth factor (VEGF) and other inflammatory mediators. VEGF increases capillary permeability, leading to a massive shift of protein-rich fluid from the intravascular space to the "third space". This shift results in hemoconcentration (high hematocrit), intravascular hypovolemia (low blood volume), ascites, and potential multi-organ failure.

The sOHSS has been classified into 3 types by DeLeneer et al which are type 1 corresponding to mutated FSH receptor, type 2 corresponding to high beta hCG levels seen in conditions like multiple pregnancy, and choriocarcinoma and type 3 associated with hypothyroidism.[6]

In our case, the type 2 sOHSS can be ruled out as the beta hcg levels were normal with a singleton pregnancy, hence our case will most probably be a type 3 or type 1 sOHSS. However, this cannot be proved in our case as the patient was not willing to undergo genetic testing for the mutated FSH receptor.

The symptoms of sOHSS can range from mild to moderate, such as abdominal pain, distension, nausea, vomiting, rapid weight gain characterised by an increase in weight by 2 pounds in 24 hours, respiratory distress, oliguria and ovarian enlargement.

The complications in severe sOHSS can present thromboembolic events, renal failure, acute respiratory distress, hypovolemic shock and rarely death.

As reported in our case, the patient presented with the characteristic finding of haemoconcentration found in this

syndrome, similar to Dieterich et al.[7] The case report published by Sridev et al and Edwards-Silva et al reported patients who also presented with anaemia, which was not the case in our patient.[8,9]

The diagnosis is often clinical and based on imaging findings on USG. Management is often conservative and includes supportive care, fluid retention, low molecular weight heparin, electrolyte correction and cabergoline.

Therapeutic paracentesis is reserved only for patients having significant pain, respiratory compromise or renal impairment. Most patients' symptoms resolve spontaneously and have good fetal-maternal outcomes.

All case reports had their patients managed by adequate levothyroxine treatment in varying dosages from 50 to 200 mcg, similar to our patient, who was treated with levothyroxine 50 mcg. In the cases described by Cardoso et al, Nappi et al, Borna et al, Dieterich et al, and Edwards Silva et al, the patients also underwent fluid replacement similar to our case report.[7,9,10-12]

Dieterich et al, Edwards-Silva et al, and Delabaere et al reported that their patients received antithrombotic prophylaxis and analgesia, which was not so in our patient.[5,7,9]

Surgical procedures were reported only for the patient reported by Dieterich et al, wherein the patient had to undergo paracentesis, and in the case described by Borna et al, wherein the patient underwent a laparotomy for a wedge biopsy of the cysts, no surgical procedure was performed on our patient during the antenatal period.[7,12]

Cardoso et al and Borna et al reported a similar period of hormonal normalisation as our patient, wherein TSH levels decreased after 12–15 weeks, while Nappi et al showed that hormone reduction occurred after 2 weeks of therapy.[10-12]

The case reports by Cardoso et al, Nappi et al, Borna et al, Delabaere et al, and Dieterich et al show rapid regression in symptomatology after levothyroxine treatment for 2–8 weeks.[5,7,10-12] However, in the case of Sridev et al, the patient started showing improvement from week.[14] In our case, the patient started to show improvement by day 5 and by day 8, the laboratory parameters came back to normal.[8]

Cardoso et al and Delabaere et al described the same period to ovarian size normalisation as our patient, i.e., 12 weeks, while Sridev et al and Borna et al reported no evidence of cysts only at 20 and 28 weeks, respectively.[5,8,10,12]

In contrast to our patient, Cardoso et al's patient went into preterm labour spontaneously, and Edward-Silva et al reported a preterm caesarean delivery.[9,10] The remaining pregnancies were delivered at term, and all infants were born healthy, similar to our case.

CONCLUSION

A differential diagnosis of sOHSS must be kept in mind in cases of presentation of acute abdomen in pregnancy. It is a life-threatening condition whether it was an exogenous or endogenous OHSS. An early recognition and appropriate supportive management will ensure a good outcome and avoid unnecessary life-threatening complications.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

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Cite this article as: Thathira S, Tutwala N, Vernekar R. Ovarian hyperstimulation syndrome in a spontaneous pregnancy: a diagnostic dilemma. Int J Reprod Contracept Obstet Gynecol 2026;15:2286-9.