International Journal of Reproduction, Contraception, Obstetrics and Gynecology
Sehgal C et al. Int J Reprod Contracept Obstet Gynecol. 2026 Feb;15(2):739-741
www.ijrcog.org
pISSN 2320-1770 | eISSN 2320-1789

DOI: https://dx.doi.org/10.18203/2320-1770.ijrcog20260201

Case Report

Spontaneous ovarian hyperstimulation syndrome following evacuation of a partial mole: a case report

Chandni Sehgal, Sekhar Chakrabarti, Ravmeet K. Sareen*

Department of Obstetrics and Gynecology, MGM Medical College, Kishanganj, Bihar, India

Received: 01 December 2025
Revised: 06 January 2026
Accepted: 07 January 2026

***Correspondence:**
Dr. Ravmeet K. Sareen,
E-mail: dr.ravmeet@gmail.com

ABSTRACT

Ovarian hyperstimulation syndrome (OHSS) is usually an iatrogenic complication of ovarian stimulation, while spontaneous OHSS is rare and linked to conditions with high human chorionic gonadotropin (hCG), such as molar pregnancy. Herein, this case reports an 18-year-old woman who developed spontaneous OHSS two months after suction evacuation of a partial mole, presenting with abdominal pain, vomiting, and distension. Her β-hCG had fallen from 59,027 to 1,463 mIU/ml. Ultrasonography showed bilaterally enlarged multicystic ovaries (~8–9 cm), ascites, and an empty uterus; laboratory parameters were normal. Pregnancy, ovarian torsion, and persistent mole were excluded, confirming spontaneous OHSS. She was treated conservatively with cabergoline 0.5 mg daily for 10 days, with resolution of symptoms in 4–5 days, and discharged on combined oral contraceptives. β-hCG remained normal over 2 years of follow-up. This case underscores the rarity of spontaneous OHSS post-molar evacuation and the importance of early recognition and conservative management for favorable outcomes.

Keywords: Ovarian hyperstimulation syndrome, Human chorionic gonadotropin

INTRODUCTION

Ovarian hyperstimulation syndrome (OHSS) is a potentially serious condition characterized by ovarian enlargement and increased capillary permeability, leading to fluid shift into the third space. It is most commonly an iatrogenic complication of ovulation induction using exogenous gonadotropins during assisted reproductive techniques.[1,2] In contrast, spontaneous OHSS is exceedingly rare and occurs in the absence of fertility treatment, usually in association with conditions that result in elevated endogenous human chorionic gonadotropin (hCG) levels such as hydatidiform mole, multiple gestation, and, less commonly, hypothyroidism.[3-5]

The pathophysiology of OHSS is primarily mediated by hCG-induced upregulation of vascular endothelial growth factor (VEGF) in granulosa lutein cells, resulting in increased vascular permeability and subsequent development of ascites, pleural effusion, hemoconcentration, renal dysfunction, and a hypercoagulable state.[6,7] While the syndrome is well described in the context of assisted reproduction, spontaneous OHSS remains poorly understood and underreported, particularly following evacuation of molar pregnancies.[8,9] Reports of OHSS occurring after partial molar pregnancy are especially scarce, and the diagnosis may be challenging due to overlapping clinical features with ovarian torsion, ruptured ovarian cysts, or persistent gestational trophoblastic disease.[10]

We present a rare case of spontaneous OHSS developing after suction evacuation of a partial hydatidiform mole, emphasizing the importance of clinical awareness, early diagnosis, and conservative management.

CASE REPORT

An 18-year-old woman with a history of partial hydatidiform mole underwent suction and evacuation

(confirmed by histopathology). Pre-evacuation serum β-hCG measured 59,027 mIU/ml and chest radiography was normal. Two months later, she presented with abdominal pain, vomiting, and progressive abdominal distension for 3 days. She denied sexual activity since evacuation.

On examination

On examination, the patient was hypotensive with a blood pressure of 90/60 mmHg and tachycardic with a low-volume pulse of 118/min; chest examination was unremarkable, while abdominal examination revealed diffuse tenderness with distension and preserved bowel sounds.

Investigations

Investigations revealed a positive urine pregnancy test with a serum β-hCG level of 1,463 mIU/ml, while complete blood count, liver function tests, renal function tests, serum electrolytes, and coagulation profile were within normal limits; thyroid-stimulating hormone and serum estradiol levels were also normal, and chest radiography showed no abnormalities.

Ultrasound findings

Ultrasonography demonstrated bilaterally enlarged multicystic ovaries measuring approximately 8–9 cm with associated ascites and free fluid in the pouch of Douglas, while the uterine cavity was empty, thereby ruling out a persistent molar pregnancy; Doppler evaluation showed normal ovarian blood flow, excluding torsion. In view of the ovarian enlargement, ascites, evidence of reduced intravascular volume, declining β-hCG levels, and absence of an ongoing pregnancy or persistent mole, a diagnosis of spontaneous ovarian hyperstimulation syndrome was made (Figure 1).

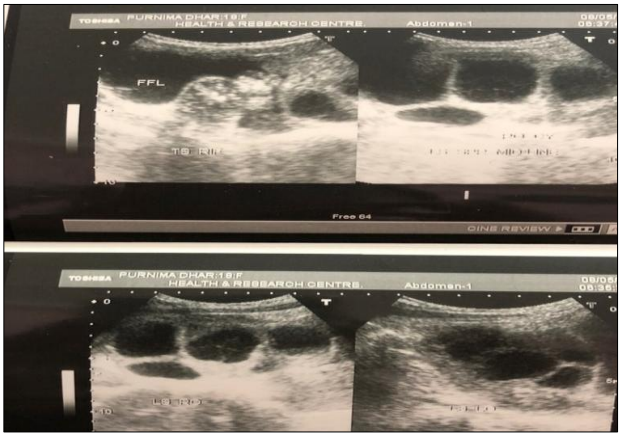

Figure 1: Ultrasonographic images.

Management

The patient was managed conservatively with cabergoline 0.5 mg once daily for 10 days, along with adequate hydration and close monitoring of vital signs and urine output, while being observed for potential complications. She showed rapid clinical improvement with complete resolution of symptoms within 4–5 days and was discharged after normalization of β-hCG levels, following which she was started on combined oral contraceptives.

Follow-up

Monthly β-hCG monitoring for 2 years remained normal. The last follow-up (27 February) showed normal β-hCG and complete recovery.

DISCUSSION

Spontaneous OHSS is a rare clinical condition, first described in 1943, and occurs in the absence of exogenous gonadotropin stimulation.[3] De Leener et al classified spontaneous OHSS into three pathogenic types: type 1 due to activating mutations of the follicle-stimulating hormone receptor, type 2 resulting from excessive endogenous hCG as seen in molar pregnancies and multiple gestations, and type 3 associated with hypothyroidism due to thyroid-stimulating hormone cross-reactivity with FSH receptors.[3,4]

The present case represents type 2 spontaneous OHSS, triggered by elevated hCG levels related to a partial molar pregnancy. Although β-hCG levels were declining at presentation, delayed onset of OHSS after molar evacuation has been previously reported and is attributed to sustained ovarian stimulation and prolonged VEGF activity.[5-7] hCG-mediated VEGF upregulation increases vascular permeability, resulting in capillary leak, ascites, hemoconcentration, and ovarian enlargement, all of which were observed in our patient.[5,6]

Young age and polycystic ovary syndrome (PCOS) are recognized risk factors for OHSS, even in spontaneous cases, due to increased ovarian sensitivity to gonadotropins.[7,8] The presence of PCOS in our patient likely contributed to her heightened susceptibility, consistent with previously reported cases of spontaneous OHSS.[8]

The clinical severity of OHSS ranges from mild abdominal discomfort to life-threatening complications. Classification systems proposed by Golan et al and Navot et al remain widely used to guide management and prognosis.[1,2] Our patient exhibited features consistent with moderate OHSS and responded well to conservative therapy.

Cabergoline, a dopamine agonist, reduces VEGF-mediated vascular permeability and has been shown to be effective in both prevention and treatment of OHSS.[11-13] In the present case, cabergoline therapy resulted in rapid symptomatic improvement without complications, in line with previous studies.[11,12]

Spontaneous OHSS may mimic conditions such as ovarian torsion, ruptured ovarian cysts, or persistent gestational trophoblastic disease, particularly following molar evacuation.[7,14] Awareness of this rare entity and early diagnosis are crucial, as timely conservative management can prevent serious morbidity and ensure favorable outcomes.

CONCLUSION

Spontaneous OHSS following evacuation of a partial mole is rare but must be considered in patients presenting with abdominal distension and ovarian enlargement. Early diagnosis and conservative management, including cabergoline, can prevent severe morbidity. Long-term follow-up with β-hCG monitoring is essential.

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

REFERENCES

1. Golan A, Ron-El R, Herman A, Soffer Y, Weinraub Z, Caspi E. Ovarian hyperstimulation syndrome: an update review. Obstet Gynecol Surv. 1989;44:430-40.
2. Navot D, Bergh PA, Laufer N. Ovarian hyperstimulation syndrome in novel reproductive technologies: prevention and treatment. Fertil Steril. 1992;58:249-61.
3. De Leener A, Montanelli L, Van Durme J, Delbaere A, Smits G, et al. Presence and absence of follicle-stimulating hormone receptor mutations provide insights into the pathophysiology of spontaneous ovarian hyperstimulation syndrome. Hum Reprod Update. 2011;17(3):387-99.
4. Delbaere A, Smits G, De Leener A, Costagliola S, Vassart G. Understanding ovarian hyperstimulation syndrome. Endocrine. 2005;26(3):285-90.
5. McClure N, Healy DL, Rogers PA, Sullivan J, Beaton L, Haning RV Jr, et al. Vascular endothelial growth factor as capillary permeability agent in ovarian hyperstimulation syndrome. Lancet. 1994;344(8917):235-6.
6. Abramov Y, Barak V, Nisman B, Schenker JG. Vascular endothelial growth factor plasma levels correlate to the clinical picture in severe ovarian hyperstimulation syndrome. Fertil Steril. 1997;67:261-5.
7. Rachad M, Chaara H, Zahra Fdili F, Bouguern H, Melhouf A. Ovarian hyperstimulation syndrome in a spontaneous pregnancy with invasive mole: report of a case. Pan Afr Med J. 2011;9:23.
8. Razavi M, Hasanzadeh M. Ovarian hyperstimulation syndrome in two spontaneous pregnancies. Int J Med. 2014;2(2):45-8.
9. Sridev S, Rao KA. Spontaneous ovarian hyperstimulation syndrome associated with hypothyroidism: a rare entity. J Reprod Sci. 2013;20(2):89-92.
10. Chen CD, Wu MY, Chao KH, Ho HN, Yang YS. Update on prevention and management of ovarian hyperstimulation syndrome. Taiwan J Obstet Gynecol. 2011;50(1):2-10.
11. Alvarez C, Martí-Bonmatí L, Novella-Maestre E, Sanz R, Gómez R, Fernández-Sánchez M, et al. Dopamine agonist cabergoline reduces hemoconcentration and ascites in hyperstimulated women undergoing assisted reproduction. J Clin Endocrinol Metab. 2007;92(8):2931-7.
12. Youssef MA, van Wely M, Hassan MA, Al-Inany HG, Mochtar M, Khattab S, et al. Can dopamine agonists reduce the incidence and severity of OHSS in IVF/ICSI treatment cycles? A systematic review and meta-analysis. Hum Reprod Update. 2010;16(5):459-66.
13. Tang H, Hunter T, Hu Y, Zhai SD, Sheng X, Hart RJ. Cabergoline for preventing ovarian hyperstimulation syndrome. Cochrane Database Syst Rev. 2012;(2):CD008605.
14. Davies MJ, Fletcher TL, Cox Bauer CM. Spontaneous Ovarian Hyperstimulation Syndrome in a Partial Molar Pregnancy With Early Onset Severe Pre-eclampsia at 15 Weeks Gestation. Mil Med. 2020;185(9-10):e1836-9.

Cite this article as: Sehgal C, Chakrabarti S, Sareen RK. Spontaneous ovarian hyperstimulation syndrome following evacuation of a partial mole: a case report. Int J Reprod Contracept Obstet Gynecol 2026;15:739-41.