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# Severe Ovarian Hyperstimulation Syndrome Complicated by *Stenotrophomonas maltophilia* Peritonitis: A Case Report and Literature Review

Abdul Rehman<sup>1</sup>, Noor Ul-Ain Baloch<sup>1</sup> and Muhammad Awais<sup>2</sup>

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## Abstract

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Ovarian hyperstimulation syndrome (OHSS) is an iatrogenic disorder resulting from ovulation induction. Although the occurrence of this disorder is rare, it can be potentially life-threatening in its most severe forms. We herein present the case of a young nulliparous woman who presented with features of abdominal compartment syndrome and was subsequently diagnosed with severe OHSS. All physicians, in particular critical care doctors, must be aware of this rare, but potentially life-threatening iatrogenic disorder.

**Key words:** ovulation induction, ovarian hyperstimulation syndrome, ascites, intra-abdominal hypertension

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## Introduction

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Ovarian hyperstimulation syndrome (OHSS) is a well-recognized, but rare complication of ovulation induction (1). This iatrogenic complication results from a loss of control of ovarian stimulation and can occur from any agent used for ovulation induction (2). The severe form of OHSS is reported to occur in 0.5-5% of stimulated ovarian cycles with a mortality rate of about 1 in 50,000 (3). We herein report the case of a young woman who presented with features of abdominal compartment syndrome and was diagnosed with severe OHSS.

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## Case Report

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A 28-year-old nulliparous woman presented to the emergency department with complaints of abdominal distension and shortness of breath. Her medical history included polycystic ovarian syndrome and she has had primary infertility for the past few years. She could not conceive despite receiving multiple courses of clomiphene citrate and multiple injections of human chorionic gonadotropin. On the physical examination, she was obese with marked tachycardia (150

beats/minute), tachypnea (25 breaths/minute) and dry mucous membranes. She also had a markedly distended abdomen with prominent shifting dullness. Initial investigations revealed a serum creatinine level of 2.2 mg/dL, suggestive of acute kidney injury, along with a leukoerythroblastic picture (total leukocyte count of 53,000/mm<sup>3</sup>). Ultrasonography of the abdomen confirmed the presence of ascites along with enlarged polycystic ovaries (Fig. 1). Based on the clinical picture and ultrasonographic evidence, a diagnosis of OHSS was made.

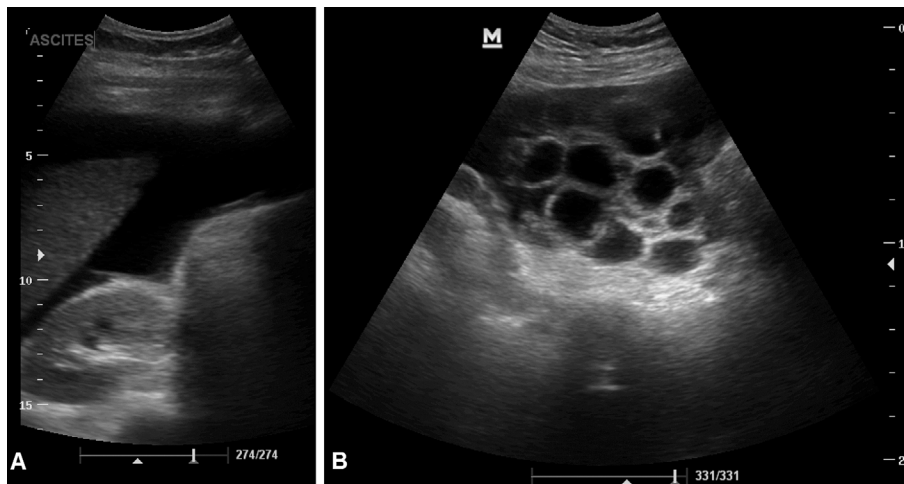
Foley's catheter and a central venous line were inserted and the patient was administered intravenous fluids judiciously. The intra-abdominal pressure was measured indirectly and was found to be significantly elevated. An arterial blood gas revealed hypoxemia along with hypercarbia for which she was started on non-invasive positive pressure ventilation. Blood products were arranged and an ultrasound-guided therapeutic paracentesis was performed, six hours after arrival to the emergency room, which drained 800 mL of hemorrhagic fluid. The patient was admitted to the high-dependency unit and monitored closely. Her condition initially improved after ascitic drainage; however, the patient then began to spike a fever approximately 20 hours after arrival. She was started on an empirical, renal-adjusted dose

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**Figure 1.** A: The longitudinal view of an ultrasonographic image of the right upper quadrant demonstrates the presence of free fluid within the perihepatic and hepatorenal spaces. B: The longitudinal view of an ultrasonographic image of the left ovary shows an enlarged, polycystic ovary surrounded by free fluid containing echogenic material, suggestive of hemorrhagic ascites.

of piperacillin-tazobactam. A repeat paracentesis was performed, which revealed a neutrophil count of  $800/\mu\text{L}$  suggestive of bacterial peritonitis. The patient's fever persisted for the next 48 hours and she was switched to meropenem and vancomycin after sending blood and urine cultures to the clinical laboratory. The ascitic fluid culture subsequently revealed heavy growth of *Stenotrophomonas maltophilia* which was sensitive to levofloxacin, minocycline and ceftazidime. The patient was started on intravenous ceftazidime (1 g/12 hours) and continued on supportive care. Gradually, her fever subsided, intra-abdominal pressure reduced, serum creatinine level returned to baseline, urinary output increased and her overall condition began to improve. Her blood and urine cultures did not grow any bacterial pathogens. After being moved to the general ward, the patient was switched from intravenous ceftazidime to oral levofloxacin (750 mg, once daily). She was discharged after a two-week stay in the hospital following a repeat ultrasound, which documented the resolution of the ascites. At a 6-month follow-up visit, the patient remained asymptomatic and now leads a normal life.

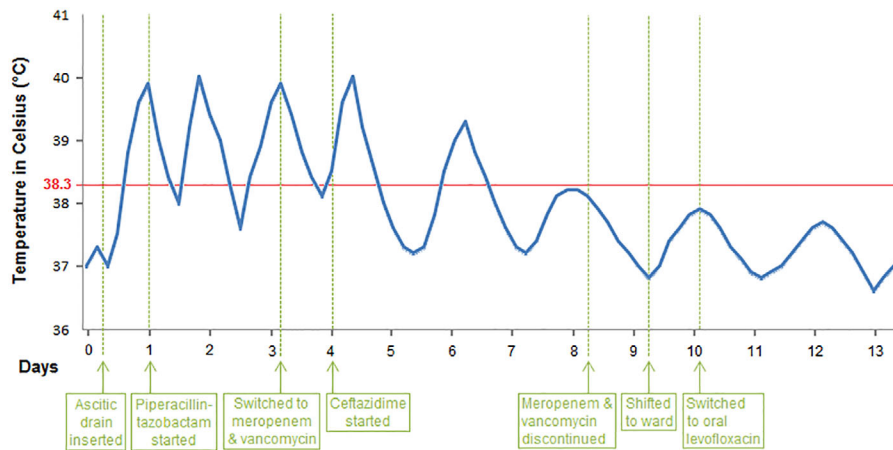
## Discussion

OHSS is a purely iatrogenic disorder resulting from uncontrolled ovarian stimulation due to exogenous means of ovulation induction. The first case of this syndrome was reported back in 1943 (4) and since then, the incidence of this disorder has increased steadily due to widespread availability and use of assisted fertility techniques (5). Although fertility is essential for the propagation of families, it is not vital for sustaining life. Therefore, the perpetuation of a life-threatening complication as a consequence of ovulation induction must be avoided. Current evidence suggests that OHSS may be prevented if ovarian hyperstimulation is recognized early by the treating obstetrician (6). In such cases,

many preventive measures can be taken, such as cancelling the stimulated cycle, coasting (withholding gonadotropins), aspirating or cauterizing ovarian follicles, or administering progesterone and steroids (7-10).

Of all possible methods of ovulation induction, The use of human chorionic gonadotropin is most strongly associated with the development of OHSS (11). The published literature suggests that human chorionic gonadotropin plays a central role in the pathogenesis of OHSS by inducing the secretion of cytokines and vasoactive mediators, which leads to increased vascular permeability and third spacing (12). Intra-abdominal hypertension is also thought to be a key component in the development of this syndrome and accounts for a number of consequences (13). Additional risk factors for this syndrome include young age (younger than 35 years), lean build, polycystic ovarian syndrome, history of atopy or allergies and a past history of OHSS (14-18).

Abdominal distension, abdominal discomfort, nausea, vomiting and diarrhea are common clinical features of this syndrome (19). These symptoms result as a consequence of ovarian stimulation, which results in ovarian enlargement and the accumulation of exudative ascites (20). Gastrointestinal symptoms are believed to occur due to the effects of human chorionic gonadotropin (21). Morbidity and mortality in OHSS results from its complications, which include massive ascites, pleural effusions, pericardial effusions, electrolyte derangements, hemoconcentration, hypovolemia, acute renal failure, thromboembolic events, neurologic complications, secondary infections and ovarian hemorrhage, torsion or even rupture (22-26). The presence of ascites along with enlarged polycystic ovaries is highly suggestive of the diagnosis (27). The differential diagnosis of this disorder includes all other causes of ascites such as hepatic disorders (Budd-Chiari syndrome, chronic liver disease, portal vein thrombosis), renal disorders (nephrotic syndrome), congestive cardiac failure, ovarian tumors, peritoneal metastases



**Figure 2.** The graph depicts the timeline of the patient's fever and events during the course of admission.

and abdominal infections (tuberculosis) (1, 28, 29).

Secondary infection is a recognized complication of OHSS and bacterial peritonitis in association with OHSS has been reported in the literature (1). *Escherichia coli*, *Pseudomonas aeruginosa*, *Klebsiella pneumoniae*, *Proteus mirabilis* and *Proteus vulgaris* have been reported to be the most common pathogens in such cases (1, 19, 30). However, our patient developed bacterial peritonitis secondary to *Stenotrophomonas maltophilia*, a very uncommon pathogen. This gram-negative rod is commonly implicated in nosocomial infections; however, it may rarely be community-acquired (31). In the present case, this organism was either acquired from the hospital (likely source being the ascitic drain) or from the community. Although fever developed early in our patient (Fig. 2), the presence of an ascitic drain made a nosocomial infection more likely as the route of infection.

The management of OHSS is primarily supportive and consists of close monitoring of volume status (urinary catheter and central venous line), judicious fluid therapy, prophylactic or therapeutic anti-coagulation, ventilatory support (if needed) and therapeutic paracentesis (to relieve intra-abdominal pressure) (1, 19, 32, 33). To facilitate the management of this disorder, OHSS has been classified in a number of ways. One commonly used classification divides OHSS into moderate (abdominal distension with enlarged, polycystic ovaries), severe (hemoconcentration or the presence of large ascites with pleural or pericardial effusions) or life-threatening (renal failure, tense ascites, neurologic and/or thromboembolic complications) (34, 35). Prompt recognition of secondary infections, especially bacterial peritonitis, is important to institute early antibiotic therapy and prevent the development of septic shock (36). With adequate treatment of this condition, the mortality is only 1 in 450,000 patients (1).

**The authors state that they have no Conflict of Interest (COI).**

Written informed consent was obtained from the patient for the preparation of this manuscript.

## References

- Budev MM, Arroliga AC, Falcone T. Ovarian hyperstimulation syndrome. *Crit Care Med* **33**: S301-S306, 2005.
- Kumar P, Sait SF, Sharma A, Kumar M. Ovarian hyperstimulation syndrome. *J Hum Reprod Sci* **4**: 70-75, 2011.
- Schenker JG, Weinstein D. Ovarian hyperstimulation syndrome: a current survey. *Fertil Steril* **30**: 255-268, 1978.
- Varma TR, Patel RH. Ovarian hyperstimulation syndrome: a case history and review. *Acta Obstet Gynecol Scand* **67**: 579-584, 1988.
- Delvigne A. Epidemiology of OHSS. *Reprod Biomed Online* **19**: 8-13, 2009.
- Humaidan P, Quartarolo J, Papanikolaou EG. Preventing ovarian hyperstimulation syndrome: guidance for the clinician. *Fertil Steril* **94**: 389-400, 2010.
- Delvigne A, Rozenberg S. Epidemiology and prevention of ovarian hyperstimulation syndrome (OHSS): a review. *Hum Reprod Update* **8**: 559-577, 2002.
- Aboulghar MA, Mansour RT. Ovarian hyperstimulation syndrome: classifications and critical analysis of preventive measures. *Hum Reprod Update* **9**: 275-289, 2003.
- Mansour R, Aboulghar M, Serour G, Amin Y, Abou-Setta AM. Criteria of a successful coasting protocol for the prevention of severe ovarian hyperstimulation syndrome. *Hum Reprod* **20**: 3167-3172, 2005.
- Mathur R, Kailasam C, Jenkins J. Review of the evidence base of strategies to prevent ovarian hyperstimulation syndrome. *Hum Fertil* **10**: 75-85, 2007.
- Alper MM, Smith LP, Sills ES. Ovarian hyperstimulation syndrome: current views on pathophysiology, risk factors, prevention and management. *J Exp Clin Assist Reprod* **6**: 3, 2009.
- Garcia-Velasco JA, Pellicer A. New concepts in the understanding of the ovarian hyperstimulation syndrome. *Curr Opin Obstet Gynecol* **15**: 251-256, 2003.
- Grossman LC, Michalakis KG, Browne H, Payson MD, Segars JH. The pathophysiology of ovarian hyperstimulation syndrome: an unrecognized compartment syndrome. *Fertil Steril* **94**: 1392-1398, 2010.
- Tummon I, Gavrilova-Jordan L, Allemann MC, Session D. Polycystic ovaries and ovarian hyperstimulation syndrome: a systematic review\*. *Acta Obstet Gynecol Scand* **84**: 611-616, 2005.

15. Delvigne A, Rozenberg S. Systematic review of data concerning etiopathology of ovarian hyperstimulation syndrome. *Int J Fertil Womens Med* **47**: 211-226, 2001.
16. Maheshwari A, Stofberg L, Bhattacharya S. Effect of overweight and obesity on assisted reproductive technology--a systematic review. *Hum Reprod Update* **13**: 433-444, 2007.
17. Beerendonk CCM, Van Dop PA, Braat DDM, Merkus JMWM. Ovarian hyperstimulation syndrome: facts and fallacies. *Obstet Gynecol Surv* **53**: 439-449, 1998.
18. Vloeberghs V, Peeraer K, Pexsters A, D'Hooghe T. Ovarian hyperstimulation syndrome and complications of ART. *Best Pract Res Clin Obstet Gynaecol* **23**: 691-709, 2009.
19. Delvigne A, Rozenberg S. Review of clinical course and treatment of ovarian hyperstimulation syndrome (OHSS). *Hum Reprod Update* **9**: 77-96, 2003.
20. Gómez R, Soares SR, Busso C, Garcia-Velasco JA, Simón C, Pellicer A. Physiology and pathology of ovarian hyperstimulation syndrome. *Semin Reprod Med* **28**: 448-457, 2010.
21. Elchalal U, Schenker JG. The pathophysiology of ovarian hyperstimulation syndrome--views and ideas. *Hum Reprod* **12**: 1129-1137, 1997.
22. Abramov Y, Elchalal U, Schenker JG. Pulmonary manifestations of severe ovarian hyperstimulation syndrome: a multicenter study. *Fertil Steril* **71**: 645-651, 1999.
23. Balasch J, Carmona F, Llach J, Arroyo V, Jove I, Vanrell JA. Acute prerenal failure and liver dysfunction in a patient with severe ovarian hyperstimulation syndrome. *Hum Reprod* **5**: 348-351, 1990.
24. Binder H, Dittrich R, Einhaus F, et al. Update on ovarian hyperstimulation syndrome: Part 1--Incidence and pathogenesis. *Int J Fertil Womens Med* **52**: 11-26, 2006.
25. Yoshii F, Ooki N, Shinohara Y, Uehara K, Mochimaru F. Multiple cerebral infarctions associated with ovarian hyperstimulation syndrome. *Neurology* **53**: 225, 1999.
26. Balasch J, Arroyo V, Fabregues F, et al. Neurohormonal and hemodynamic changes in severe cases of the ovarian hyperstimulation syndrome. *Ann Intern Med* **121**: 27-33, 1994.
27. Brinsden PR, Wada I, Tan SL, Balen A, Jacobs HS. Diagnosis, prevention and management of ovarian hyperstimulation syndrome. *BJOG* **102**: 767-772, 1995.
28. Mathur RS, Akande AV, Keay SD, Hunt LP, Jenkins JM. Distinction between early and late ovarian hyperstimulation syndrome. *Fertil Steril* **73**: 901-907, 2000.
29. Schenker JG. Clinical aspects of ovarian hyperstimulation syndrome. *Eur J Obstet Gynecol Reprod Biol* **85**: 13-20, 1999.
30. Abramov Y, Barac V, Nisman B, Schenker JG. Vascular endothelial growth factor plasma levels correlate to the clinical picture in severe ovarian hyperstimulation syndrome. *Fertil Steril* **67**: 261-265, 1997.
31. Falagas ME, Kastoris AC, Vouloumanou EK, Dimopoulos G. Community-acquired *Stenotrophomonas maltophilia* infections: a systematic review. *Eur J Clin Microbiol Infect Dis* **28**: 719-730, 2009.
32. Casals G, Fábregues F, Pavesi M, Arroyo V, Balasch J. Conservative medical treatment of ovarian hyperstimulation syndrome: a single center series and cost analysis study. *Acta Obstet Gynecol Scand* **92**: 686-691, 2013.
33. Fiedler K, Ezcurra D. Predicting and preventing ovarian hyperstimulation syndrome (OHSS): the need for individualized not standardized treatment. *Reprod Biol Endocrinol* **10**: 32-41, 2012.
34. Golan A, Weissman A. A modern classification of OHSS. *Reprod Biomed Online* **19**: 28-32, 2009.
35. Zivi E, Simon A, Laufer N. Ovarian hyperstimulation syndrome: definition, incidence, and classification. *Semin Reprod Med* **28**: 441-447, 2010.
36. Gera PS, Tatpati LL, Allemand MC, Wentworth MA, Coddington CC. Ovarian hyperstimulation syndrome: steps to maximize success and minimize effect for assisted reproductive outcome. *Fertil Steril* **94**: 173-178, 2010.