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Ovarian Hyperstimulation Syndrome Complicated by Acute Hepatocellular Injury: A Diagnostic Crossroads in Early Pregnancy After Embryo Transfer

Srinisha Soundararajan [1], Herman Tournaye [2, 3]

1. Obstetrics and Gynaecology, Vrije Universiteit Brussel, Universitair Ziekenhuis Brussel, Brussels, BEL 2. Brussels IVF, Universitair Ziekenhuis Brussel (UZ Brussel), Brussels, BEL 3. Biology of the Testis Laboratory, Genetics, Reproduction and Development (GRAD) Research Group, Vrije Universiteit Brussel (VUB), Brussels, BEL

Corresponding author: Srinisha Soundararajan, srinisha_6@yahoo.co.in

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Abstract

Ovarian hyperstimulation syndrome (OHSS) is the principal iatrogenic complication of controlled ovarian stimulation and has a characteristic but under-recognised sonographic appearance. Transient elevation of liver enzymes is a less familiar feature of the syndrome and remains a diagnosis of exclusion. We report a nulligravid woman in her early 30s who developed moderate OHSS six days after fresh embryo transfer, presenting with abdominal distension, tachycardia, and elevated haemoconcentration and leucocytosis. Transvaginal and transabdominal ultrasound demonstrated bilaterally enlarged multifollicular ovaries, peri-follicular hypervascularity on colour Doppler, and free fluid extending from the pelvis to the perihepatic and peri-splenic recesses, without sonographic signs of ovarian torsion. As the ascites and haemoconcentration regressed, a marked increase in hepatocellular transaminases occurred, peaking at an aspartate aminotransferase of 195 U/L and an alanine aminotransferase of 242 U/L. Repeat ultrasound, including hepatic Doppler examination, demonstrated a morphologically normal liver and biliary tree with patent portal and hepatic veins, and an extensive serological and autoimmune workup was negative. The hepatocellular injury was attributed to OHSS-associated hepatic dysfunction and was managed expectantly. Enzymes normalised spontaneously, and a viable dichorionic diamniotic twin pregnancy was confirmed. This case illustrates the dual role of ultrasound in OHSS, establishing the diagnosis through the characteristic ovarian and peritoneal findings while assessing for torsion and, when liver enzymes rise, excluding structural and vascular hepatobiliary disease so that a benign, self-limiting complication is not mistaken for a hepatic emergency.

Categories: Obstetrics/Gynecology, Radiology
Keywords: assisted reproduction, colour doppler and ascites, liver function tests, ovarian hyperstimulation syndrome (ohss), ultrasound

Introduction

Ovarian hyperstimulation syndrome (OHSS) is the most significant iatrogenic complication of controlled ovarian stimulation for assisted reproductive technology (ART) [1]. In its mild form, it affects up to a third of in vitro fertilisation (IVF) cycles, whereas moderate to severe disease occurs in approximately 1-5% of cycles and rarely becomes life-threatening [2,3]. The syndrome is driven by human chorionic gonadotrophin (hCG), which triggers the ovarian release of vasoactive mediators, chiefly vascular endothelial growth factor (VEGF), thereby increasing capillary permeability and shifting protein-rich fluid from the intravascular compartment into the third space [4]. The resulting sequence of ovarian enlargement, ascites, mildly elevated haematocrit and a hypercoagulable state produces a recognisable clinical and radiological picture [5,6].

Ultrasound is central to the diagnosis and monitoring of OHSS, yet radiologists frequently encounter these patients on emergency studies without being given the fertility history. The enlarged cysts may be misinterpreted as neoplastic, or the presentation may be attributed to a surgical abdomen [7]. The most important and time-critical differential diagnosis is ovarian torsion, the incidence of which is substantially increased in stimulated, enlarged ovaries [8,9].

Less familiar to both radiologists and gynaecologists is the association of OHSS with abnormal liver tests, most commonly a hepatocellular pattern of transaminase elevation [10,11]. Because this is a diagnosis of exclusion, imaging plays an important role by demonstrating a structurally and vascularly normal liver, allowing thrombotic and other hepatobiliary emergencies to be confidently excluded [12,13]. We present a case in which ultrasound fulfilled both roles, and we use it to review the sonographic approach to OHSS and its hepatic complications.

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Case Presentation

A nulligravid woman in her early 30s was referred to the emergency department by the reproductive medicine service six days after a fresh embryo transfer. Eleven days earlier, she had undergone transvaginal oocyte retrieval, at which 13 oocytes were collected without complication. She had a gonadotrophin-releasing hormone (GnRH) antagonist protocol with an hCG trigger and a single day-5 embryo transfer. Luteal-phase support comprised vaginal progesterone, oral dydrogesterone and hCG. She reported one day of progressive abdominal distension and mild discomfort, without nausea, vomiting, dyspnoea or urinary symptoms. She had no personal or family history of liver, thrombotic or gynaecological disease and took no regular medication other than the stimulation and luteal support regimen. She did not drink alcohol and did not smoke. Her body mass index was 26 kg/m2, anti-Müllerian hormone was 1.2 µg/L, the antral follicular count was 10-12, and she did not meet the criteria for polycystic ovary syndrome.

On examination, she was alert and comfortable. She was tachycardic at 118 beats per minute and normotensive at 130/82 mmHg, afebrile and had normal oxygen saturation on room air. The abdomen was distended but soft and non-tender, and there was no peripheral oedema.

Transvaginal ultrasound showed an anteverted uterus with a thickened secretory endometrium of 20 mm and a moderate volume of free fluid in the Douglas pouch. Both ovaries were enlarged and contained multiple peripherally arranged, thin-walled follicular cysts surrounding central echogenic stroma. The following day, transabdominal ultrasound performed by the radiology service confirmed markedly enlarged ovaries, the right measuring max 65 mm and the left 72 mm, with the same multi-follicular architecture (Figure *1*). Colour Doppler demonstrated preserved, symmetrical arterial and venous flow to both ovaries with conspicuous perifollicular vascularity. There was no sonographic evidence of stromal oedema, no whirlpool sign of the vascular pedicle and no side-to-side asymmetry to suggest ovarian torsion. As preserved flow does not exclude torsion, the absence of these features, together with the clinical findings, was taken as evidence against it. The volume of the free fluid had increased, extending from the pelvis into the perihepatic and peri-splenic spaces.

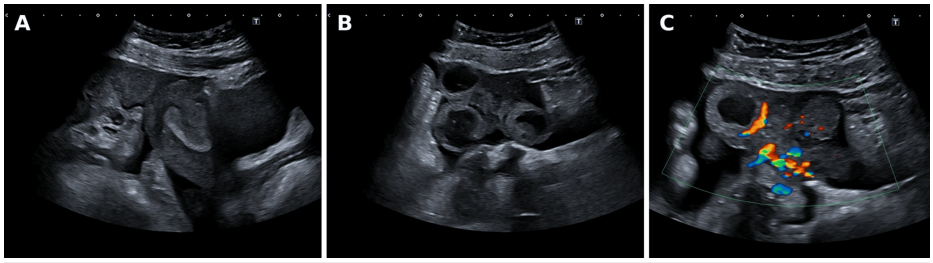


FIGURE 1: Transabdominal ultrasound findings in moderate ovarian hyperstimulation syndrome.

(A) Moderate free fluid in the pelvis. (B) Both ovaries are enlarged, exceeding 5 cm, with multiple peripherally arranged follicular cysts and echogenic central stroma. (C) Colour Doppler shows peripheral hypervascularity of the ovarian follicles with surrounding free fluid.

Initial laboratory investigation demonstrated mildly elevated haemoglobin and leucocytosis with otherwise preserved organ function (Table *1*). The haemoglobin was 15.2 g/dL with a haematocrit of 42.8%, the white cell count was 16.5 10^3/mm^3, and C-reactive protein was mildly elevated at 12.9 mg/L. Serum creatinine was normal, sodium was marginally low (134 mmol/L), potassium was marginally elevated (4.7 mmol/L) and the serum hCG confirmed early pregnancy. A diagnosis of moderate OHSS was made according to the Royal College of Obstetrics and Gynaecology (RCOG) Green-top Guideline No. 5 classification, based on ultrasound evidence of ascites extending beyond the pelvis in the presence of abdominal distension and tachycardia; haemoglobin was mildly elevated, whereas the haematocrit remained within the reference range. The patient was managed conservatively as an outpatient with encouragement of oral fluid intake, paracetamol for analgesia, strict avoidance of non-steroidal anti-inflammatory drugs, prophylactic low-molecular-weight heparin (LMWH) (nadroparin) for thromboprophylaxis, and close clinical and biochemical surveillance by the fertility service.

Parameter	Result	Reference range
Haemoglobin	15.2	11.7-15.1 g/dL
Haematocrit	42.8	35.4-46.1%
White blood cells	16.5	4.3-9.64 ×10³/mm³
C-reactive protein	12.9	<5 mg/L
Creatinine	0.77	0.51-0.95 mg/dL
Sodium	134	136-145 mmol/L
Potassium	4.7	3.4-4.5 mmol/L

TABLE 1: Laboratory findings at presentation, day 6 after embryo transfer.

Over the subsequent 10 days, the abdominal distension, ascites and haematocrit gradually regressed. However, serial biochemistry revealed a progressive, initially unexplained rise in serum aminotransferases, which were already elevated on day 9 (aspartate aminotransferase (AST) 95 U/L, alanine aminotransferase (ALT) 99 U/L). These peaked on day 15 after transfer, with an AST of 195 U/L and an ALT of 242 U/L, approximately six and seven times the upper limit of normal, respectively (Table *2*). On day 17, total bilirubin and alkaline phosphatase were within normal limits, and gamma-glutamyl transferase was mildly elevated (62 U/L). The biochemical pattern was hepatocellular.

Parameter	Day 9	Day 15	Day 17	Day 26	Reference
Aspartate aminotransferase	95	195	100	46	0-32 U/L
Alanine aminotransferase	99	242	180	87	0-33 U/L
Alkaline phosphatase	-	-	75	86	35-104 U/L
Gamma-glutamyl transferase	-	-	62	62	<40 U/L
Total bilirubin	-	-	0.17	<0.15	0-1.2 mg/dL

TABLE 2: Course of liver enzymes after embryo transfer.

Because the hepatocellular injury required explanation, the patient was referred for hepatological assessment and repeat imaging. Abdominal ultrasound demonstrated no hepatomegaly, smooth liver contour and homogeneous liver parenchyma. The bile ducts were not dilated, and the gallbladder was collapsed; the only abnormal ultrasound finding was a persistent mild perihepatic fluid (Figure *2*). Colour and spectral Doppler confirmed a patent portal vein with hepatopetal flow and patent hepatic veins, excluding occlusive portal vein thrombosis and hepatic venous outflow obstruction. A comprehensive workup for alternative causes of hepatitis, including viral serology (hepatitis A, B, C and E, cytomegalovirus), immunoglobulins and an autoimmune panel comprising antinuclear, antimitochondrial, liver-kidney microsomal and antineutrophil cytoplasmic antibodies, together with tissue transglutaminase antibodies, was negative or normal.

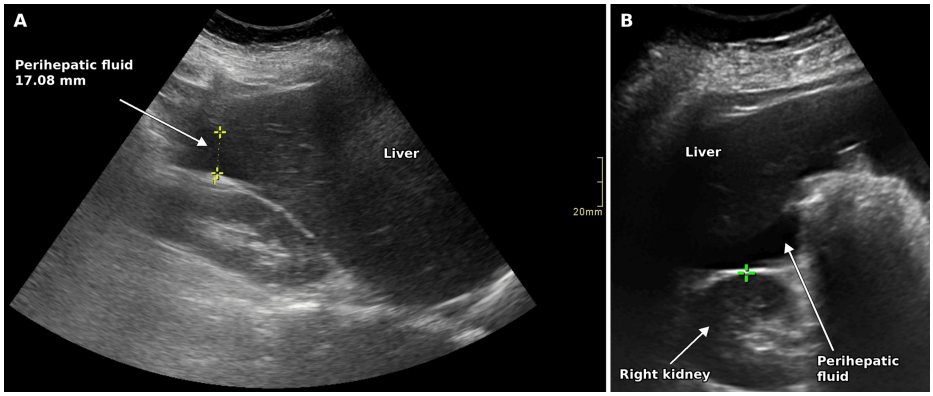


FIGURE 2: Hepatic ultrasound excluding structural liver disease in ovarian hyperstimulation syndrome.

Transabdominal ultrasound demonstrated a normal-sized liver with a smooth contour and homogeneous parenchyma. Perihepatic free fluid was measured between the callipers (17.08 mm) at the level of the hepatorenal recess.

With structural, vascular, obstructive, infective, autoimmune and toxic causes excluded, the elevated transaminases were attributed to hepatic dysfunction associated with OHSS. The enzymes were followed expectantly and normalised without specific hepatic treatment. By day 20 after transfer, the ovaries had regressed towards normal size with only trace residual free fluid. Serial obstetric ultrasound showed an ongoing twin pregnancy. At six weeks of gestation, there were two separate gestational sacs, each containing an embryo with normal cardiac activity and concordant crown-rump lengths (Figures *3A*-*3C*). At 12 weeks, the lambda sign at the insertion of the intertwin membrane confirmed a dichorionic diamniotic twin pregnancy, with concordant crown-rump lengths in both foetuses (Figures *3D*-*3F*). The patient remained clinically stable throughout the course of pregnancy.

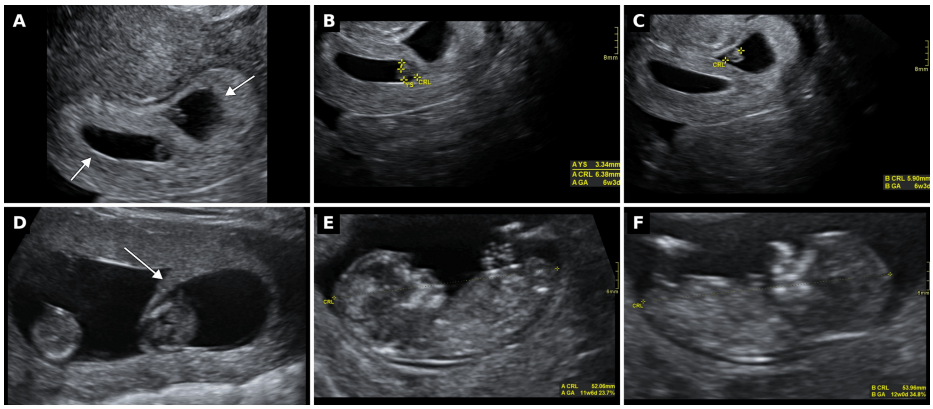


FIGURE 3: Serial transvaginal and transabdominal obstetric ultrasound confirming a dichorionic diamniotic twin pregnancy.

(A) Transvaginal ultrasound at six weeks of gestation showing two separate gestational sacs (arrows), each containing an embryo. (B) Twin A with a crown-rump length of 6.3 mm corresponding to six weeks three days. (C) Twin B with a crown-rump length of 5.9 mm corresponding to six weeks three days. (D) Transabdominal ultrasound at 12 weeks showing the lambda (twin peak) sign (arrow) at the insertion of the intertwin membrane, confirming a dichorionic diamniotic twin gestation. (E) Twin A with a crown-rump length of 52.06 mm corresponding to 11 weeks six days. (F) Twin B with a crown-rump length of 53.96 mm corresponding to 12 weeks 0 days.

Discussion

OHSS is a systemic disorder that follows an exaggerated ovarian response to gonadotrophin stimulation. Its pathophysiology centres on hCG-driven overexpression of VEGF, which increases vascular permeability, leading to ovarian enlargement, mildly elevated haemoglobin and hypercoagulability that define the syndrome [1,4]. It is conventionally classified by severity into mild, moderate, severe and critical forms and by timing into early-onset disease, which follows the exogenous hCG trigger (usually within nine days), and late-onset disease, which is precipitated by the endogenous hCG from an established pregnancy and tends to

be more severe and protracted [1]. Recognised risk factors include young age, low body mass index, polycystic ovarian syndrome, a high antral follicle count, a high number of oocytes retrieved, the use of hCG for luteal phase support and, in particular, fresh embryo transfer resulting in conception, as in our patient, whose twin pregnancy would have sustained a rising hCG stimulus [5]. Given symptom onset 11 days after oocyte retrieval in a conception cycle with hCG luteal-phase support, our case is consistent with late-onset OHSS.

The sonographic diagnosis of OHSS rests on a reproducible triad [7]. First, the ovaries are bilaterally enlarged, commonly exceeding 5 cm and, in more severe disease, reaching 10 cm or more because of multiple stimulated follicles and corpora lutea. Second, these follicles are characteristically thin-walled and peripherally distributed around compressed echogenic central stroma, producing the frequently described spoke-wheel or wheel-within-a-wheel appearance. Third, free fluid accumulates, beginning in the pelvis and, with increasing severity, extending to the paracolic gutters and the perihepatic and peri-splenic recesses. Large-volume ascites, pleural effusion and, rarely, pericardial effusion characterise severe and critical disease. Colour Doppler typically shows increased perifollicular vascularity, and ultrasound is used for quantitative measures of ascites volume and ovarian size to grade severity and guide management [9]. In our patient, the constellation of enlarged multi-follicular ovaries, perifollicular hypervascularity and ascites was diagnostic.

The single most important differential diagnosis to address actively is ovarian torsion. The risk of torsion is increased in the enlarged, oedematous ovaries of stimulated cycles, and the clinical presentation of abdominal pain and distension in a patient after oocyte retrieval overlaps substantially with uncomplicated OHSS [8,9]. Crucially, the demonstration of preserved arterial and venous flow on colour Doppler does not exclude torsion. The dual ovarian blood supply from the ovarian and uterine arteries and the frequently intermittent nature of torsion may maintain a detectable signal even in a compromised ovary [9]. The radiologist should therefore look beyond simple flow, seeking ovarian oedema and stromal heterogeneity, an abnormally positioned or midline ovary, the whirlpool sign of a twisted vascular pedicle and disproportionate unilateral pain. Any residual uncertainty should be communicated rather than offering false reassurance. In our patient, the symmetry of flow, absence of ovarian oedema and lack of whirlpool sign, taken together with a non-tender abdomen, argued strongly against ovarian torsion, and the subsequent uneventful course supported this. The principal lesson of this case concerns communication and interpretation across two specialities. When the radiologist is informed that an emergency patient with enlarged ovaries and ascites has recently undergone embryo transfer, a confident diagnosis of OHSS can be made, torsion can be actively assessed and, if liver enzymes are abnormal, the ultrasound examination can be directed to the hepatic vasculature. The gynaecologist who understands what ultrasound can and cannot exclude can manage transaminase elevation conservatively, avoid unnecessary investigation and reassure the patient. A dichorionic diamniotic twin pregnancy after single blastocyst transfer most likely reflects concurrent spontaneous conception, although rare early monozygotic splitting cannot be excluded because zygosity was not determined. The dichorionic twin pregnancy that followed single-embryo transfer also highlights the importance of counselling patients to use barrier contraception or abstain from intercourse during ovarian stimulation cycles, because spontaneous conception may result in an unplanned multiple pregnancy, which itself increases the risk of late-onset OHSS.

The second, less familiar contribution of imaging concerns the liver. Abnormal liver function tests are reported in a substantial minority of OHSS cases and typically take the form of a hepatocellular transaminase rise of the magnitude seen here, appearing during or shortly after the peak of the OHSS and resolving spontaneously as it settles [10,12]. In our patient, the transaminases peaked while the ascites was regressing, which may reflect the continued rise in endogenous hCG from the twin pregnancy sustaining hepatic effects beyond clinical regression of the ascites. The mechanism is incompletely understood but is thought to reflect oestrogen-mediated alterations in hepatic sinusoidal permeability and hepatocyte function, together with the systemic effects of increased vascular permeability and possible microvascular changes, rather than direct hepatotoxicity [14]. Cholestatic and mixed patterns, and more severe hepatic dysfunction, are described but are less common [11,13]. Importantly, OHSS-related liver injury remains a diagnosis of exclusion. In early pregnancy after IVF, the differential for a hepatocellular transaminitis is broad and includes viral and autoimmune hepatitis, drug-induced liver injury, intrahepatic cholestasis of pregnancy and, critically, the vascular complications of the hypercoagulable OHSS state, namely portal vein thrombosis and Budd-Chiari syndrome (hepatic venous outflow obstruction) [14,15]. Thromboembolism is the most feared complication of OHSS, and although the classic sites are the deep veins of the upper body and the cerebral circulation, splenic and hepatic venous thrombosis would produce liver dysfunction and demand urgent anticoagulation or intervention [15]. It is precisely here that ultrasound is decisive: a normal-sized liver with homogeneous parenchyma, a patent portal and hepatic veins, as demonstrated in our patients, reliably excludes occlusive portal and hepatic venous thrombosis at the bedside, although small non-occlusive thrombi may be missed. Ultrasound cannot, however, establish the cause of the liver injury, and in the absence of liver biopsy and with concurrent nadroparin exposure, the attribution to OHSS remains one of association rather than proven causation.

The management of OHSS is largely supportive and, for mild and moderate disease, can be delivered on an outpatient basis, as it was here [16,17]. It comprises symptom control with paracetamol, avoidance of non-steroidal anti-inflammatory drugs that may compromise renal perfusion, oral hydration,

thromboprophylaxis with LMWH in view of the hypercoagulable state, and structured clinical and biochemical monitoring, with escalation to inpatient care, paracentesis and intravenous fluids reserved for severe or critical disease [16,18]. LMWHs are associated with asymptomatic, reversible aminotransferase elevation, typically five to 10 days after initiation. The timing in our patient is compatible with this, and LMWH-associated hepatotoxicity cannot be excluded. Preventive strategies at the time of stimulation, including a GnRH antagonist protocol, a freeze-all approach and dopamine agonists, have reduced the incidence and severity of the syndrome but have not abolished it, and late-onset disease in a conception cycle remains an important scenario [17,19,20].

This report has limitations. Attribution of the liver injury to OHSS rests on exclusion and temporal association, and concurrent LMWH exposure precludes definitive causal attribution. Liver biopsy was not performed given the benign course.

Conclusions

OHSS is a systemic, iatrogenic complication of assisted reproduction in which ultrasound serves two complementary roles. It supports the diagnosis through the characteristic combination of enlarged multifollicular ovaries, perifollicular hypervascularity and ascites, and it looks for features of ovarian torsion, although preserved flow alone does not exclude torsion. When a hepatocellular transaminase rise complicates the course, ultrasound with Doppler can show the absence of structural, obstructive and occlusive major venous disease of the liver, supporting expectant management, but it cannot by itself establish the cause of the liver injury. In this patient, the course was compatible with self-limiting OHSS-associated hepatic dysfunction, although a contribution from LMWH could not be excluded. Close collaboration between reproductive medicine and radiology, with the fertility history stated on the imaging request, allows targeted imaging and avoids unnecessary intervention.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Srinisha Soundararajan

Acquisition, analysis, or interpretation of data: Srinisha Soundararajan, Herman Tournaye

Drafting of the manuscript: Srinisha Soundararajan

Critical review of the manuscript for important intellectual content: Herman Tournaye

Supervision: Herman Tournaye

Disclosures

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References

1. Nastri CO, Teixeira DM, Moroni RM, Leitão VM, Martins WP: Ovarian hyperstimulation syndrome: pathophysiology, staging, prediction and prevention. Ultrasound Obstet Gynecol. 2015, 45:377-93. 10.1002/uog.14684
2. Timmons D, Montrief T, Koyfman A, Long B: Ovarian hyperstimulation syndrome: a review for emergency clinicians. Am J Emerg Med. 2019, 37:1577-84. 10.1016/j.ajem.2019.05.018
3. Palomba S, Costanzi F, Nelson SM, Caserta D, Humaidan P: Interventions to prevent or reduce the incidence and severity of ovarian hyperstimulation syndrome: a systematic umbrella review of the best clinical evidence. Reprod Biol Endocrinol. 2023, 21:67. 10.1186/s12958-023-01113-6
4. Nastri CO, Ferriani RA, Rocha IA, Martins WP: Ovarian hyperstimulation syndrome: pathophysiology and prevention. J Assist Reprod Genet. 2010, 27:121-8. 10.1007/s10815-010-9387-6
5. Rosenberg H, Rangwala S, Magee B: Ovarian hyperstimulation syndrome. CMAJ. 2024, 196:E1012. 10.1503/cmaj.240213
6. Practice Committee of the American Society for Reproductive Medicine: Prevention of moderate and severe ovarian hyperstimulation syndrome: a guideline. Fertil Steril. 2024, 121:230-45.

10.1016/j.fertnstert.2023.11.013
7. Kuang JY, Tang K: The application value of ultrasound in ovarian hyperstimulation syndrome and literature review. Br J Radiol. 2025, 98:987-92. 10.1093/bjr/tqaf064
8. Dawood MT, Naik M, Bharwani N, Sudderuddin SA, Rockall AG, Stewart VR: Adnexal torsion: review of radiologic appearances. Radiographics. 2021, 41:609-24. 10.1148/rg.2021200118
9. Garde I, Paredes C, Ventura L, et al.: Diagnostic accuracy of ultrasound signs for detecting adnexal torsion: systematic review and meta-analysis. Ultrasound Obstet Gynecol. 2023, 61:310-24. 10.1002/uog.24976
10. Younis JS, Zeevi D, Rabinowitz R, Laufer N, Schenker JG: Transient liver function tests abnormalities in ovarian hyperstimulation syndrome. Fertil Steril. 1988, 50:176-8. 10.1016/s0015-0282(16)60029-8
11. Chae-Kim JJ, Roman R, Hawkins K, Gavrilova-Jordan L: Severe late-onset ovarian hyperstimulation syndrome presenting with liver dysfunction after in vitro fertilization: a case report. Case Rep Womens Health. 2021, 31:e00332. 10.1016/j.crwh.2021.e00332
12. Singh N, Dogra Y, Saini M, Govindarajan M: Severe early-onset ovarian hyperstimulation syndrome with liver dysfunction in an IVF segmentation cycle. BMJ Case Rep. 2020, 13: 10.1136/bcr-2019-233379
13. Dumančić S, Mikuš M, Palčić Z, Habek D, Tešanović M, Mimica MD, Marušić J: Severe early-onset intrahepatic cholestasis of pregnancy following ovarian hyperstimulation syndrome with pulmonary presentation after in vitro fertilization: case report and systematic review of case reports. Life (Basel). 2024, 14:10.3390/life14010129
14. Nuñez Burbano MJ, Escobar SA, Pinto-Martínez D, Rangel VS, García Yepes R, Castaño Toro YM, Campo JL: Spontaneous ovarian hyperstimulation syndrome in early singleton pregnancy with hepatic involvement: two case reports of atypical presentations. Front Reprod Health. 2026, 8:1872608. 10.3389/frph.2026.1872608
15. Kasum M, Danolić D, Orešković S, Ježek D, Beketić-Orešković L, Pekez M: Thrombosis following ovarian hyperstimulation syndrome. Gynecol Endocrinol. 2014, 30:764-8. 10.3109/09513590.2014.927858
16. Hamoda H, Drakeley AJ, Brian K, Evbuomwan IO, Mathur R: The management of ovarian hyperstimulation syndrome green-top guideline no. 5. BJOG. 2026, 133:e50-69. 10.1111/1471-0528.70195
17. Buckett W, Sylvestre C, Liu K, et al.: Management of moderate and severe ovarian hyperstimulation syndrome: a Canadian Fertility and Andrology Society clinical practice guideline. Reprod Biomed Online. 2026, 52:105541. 10.1016/j.rbmo.2026.105541
18. Medwin C, Rozen G, Agresta F, Nassar N, Polyakov A: The ovarian hyperstimulation that truly matters: admissions, severity and prevention strategies. Aust N Z J Obstet Gynaecol. 2023, 63:583-7. 10.1111/ajo.13688
19. Tsampras N, Palinska-Rudzka K, Alebrahim Y, Craciunas L, Mathur R: Prevention of ovarian hyperstimulation syndrome (OHSS): British Fertility Society policy and practice guideline. Hum Fertil (Camb). 2025, 28:2441827. 10.1080/14647273.2024.2441827
20. Humaidan P, Haahr T: GnRHa trigger-the story of the ugly duckling . F S Rep. 2023, 4:15-9. 10.1016/j.xfre.2023.02.011