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CASE REPORT

Unusual ovarian hyperstimulation syndrome presentation: Pleural effusion without ascites. A case report

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Abstract

BACKGROUND

Ovarian hyperstimulation syndrome (OHSS) is a life-threatening complication that can occur in the luteal phase or early pregnancy after controlled ovarian stimulation. This case report highlights a unique manifestation of OHSS involving pleural effusion (PE) in a patient without identifiable risk factors.

CASE SUMMARY

A 39-year-old woman who underwent controlled ovarian hyperstimulation for an *in vitro* fertilization (IVF) cycle experienced dyspnea on the eleventh day of post oocyte retrieval. The diagnosis was severe OHSS with a unique manifestation of PE without ascites. Clinical management involved fluid balance and treatment with albumin, furosemide, thromboembolic prophylaxis, and thoracentesis. A continued drainage of the pleural cavity was performed. The patient had a favorable outcome, and a dichorionic diamniotic gestation passed without incident.

CONCLUSION

OHSS and its potential complications can include respiratory distress and PE, as well as thromboembolic disorders.

Key Words: Dyspnea; Infertility; *In vitro* fertilization; Ovarian hyperstimulation syndrome;

Pleural effusion; Thoracentesis; Case report

Core Tip: With the increasing use of assisted reproductive technologies, ovarian hyperstimulation syndrome (OHSS) should be a topic of continued research, and it is crucial to develop systematic guidelines for prevention, diagnosis, and management. The present case underscores the severe nature of OHSS and its potential complications, which can include respiratory distress and pleural effusion, as well as impact on the cardiac function. Furthermore, it highlights an atypical presentation of OHSS in a patient without identifiable risk factors, suggesting a need for reconsideration of both the criteria defining at-risk patients, and consequently identifying individuals who would benefit from preventive measures.

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INTRODUCTION

Ovarian hyperstimulation syndrome (OHSS) is a complication associated with controlled ovarian hyperstimulation during *in vitro* fertilization (IVF). In patients undergoing IVF cycles, it is crucial to identify potential risk factors for OHSS and to include biomarkers of ovarian reserve, particularly anti-Müllerian hormone (AMH) and the antral follicle count (AFC), in previous studies to plan stimulation cycles. Once treatment has commenced, characteristics linked to multifollicular growth become valuable in identifying patients at increased risk. Prophylactic measures must then be applied, such as triggering with gonadotropin hormone-releasing hormone (GnRH) agonists or embryo cryopreservation, to avoid pregnancy in the same cycle of IVF. The diagnosis of OHSS was based on clinical criteria, with typical symptoms including abdominal distension and discomfort. Hemodynamic changes associated with OHSS can manifest as hypotension, severe tachycardia, decreased renal perfusion and hemoconcentration[1]. The interplay of an elevated hematocrit and reduced serum osmolality and sodium serves as an indicator of the presence of OHSS[2]. An ideal approach for treating OHSS should focus on prevention and supportive care. For severe cases, morbidity prevention depends on monitoring clinical parameters, maintaining fluid balance, managing electrolytes, providing thrombosis prophylaxis, and addressing ascites[3].

CASE PRESENTATION

Chief complaints

A 39-year-old female presented with abdominal discomfort, twenty-four-hour evolution dyspnea, and three-day evolution bloating.

History of present illness

After nearly two years of infertility, controlled ovarian stimulation was initiated in 2021. In October 2022, ovarian stimulation was initiated for an IVF cycle using an antagonist protocol with recombinant follicle-stimulating hormone and human menopausal gonadotropin. Triggering was performed using recombinant human chorionic gonadotropin, and plasma estradiol levels were 850 pg/mL. Five oocytes were recovered after follicular puncture. After intracytoplasmic sperm injection, four embryos were obtained, and two embryos were transferred (after three days). With an AFC of 24 and an AMH concentration of 4.23 ng/mL, the patient exhibited a normal response to controlled ovarian stimulation.

Twelve days after embryo transfer, she presented with abdominal discomfort, dyspnea, and bloating.

History of past illness

Deep endometriosis located in the umbilical area and other locations was diagnosed with a right ovarian 4-centimeter endometrioma in 2014. She was treated with oral contraceptives until her gestational desire in May 2019.

Personal and family history

No significant family history was reported.

Physical examination

Upon admission, she presented with tachycardia and hypotension, and physical examination revealed that the patient

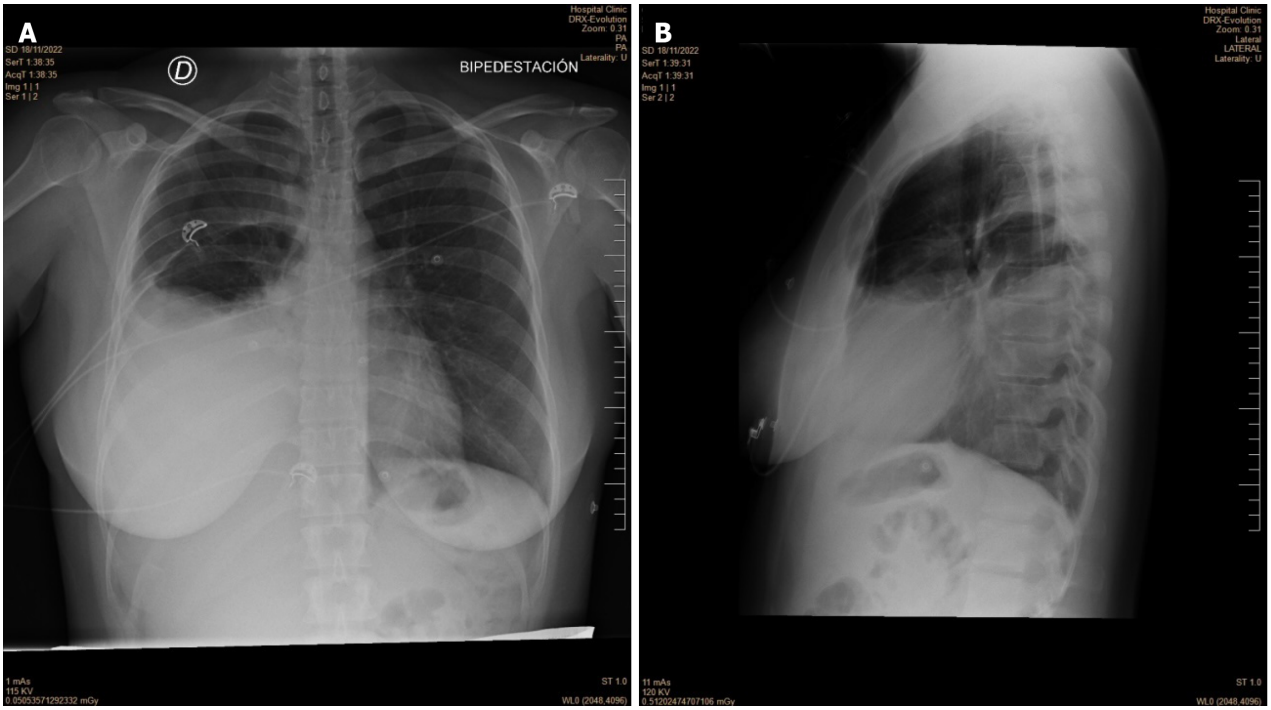

Figure 1 Chest X-ray upon admission (November 18, 2022) showing right pleural effusion. A: Erect anteroposterior view; B. Erect lateral view.

was conscious and oriented, had a normal skin color, was adequately hydrated, was hemodynamically stable, was tachypneic, and had dyspnea upon exertion. Abdominal examination revealed light distention, preserved hydroaerial sounds, and no pain on palpation. Pulmonary auscultation revealed right hypophonesis of the inferior two-thirds without crackling or other added sounds. The left thoracic auscultation was normal, indicating normal respiratory sounds. Cardiac auscultation revealed tachycardia and rhythmic sounds, with no cardiac murmurs or friction sounds. Her body mass index was 28.03 kg/m^2.

Laboratory examinations

On admission, human chorionic gonadotropin blood test result was 723.5 mIU/mL, indicating pregnancy. An initial blood test (Table 1) revealed elevated D-dimer levels (2550 ng/mL; normal range: < 500 ng/mL), hemoconcentration (hematocrit 50%; normal range: 36%-46%), leukocytosis (23000 leukocytes/mL; normal range: 4-11 × 10^6 leukocytes/mL), and thrombocytosis (431000 platelets/mL; normal range: 130-400 × 10^6 platelets/mL).

Imaging examinations

Chest radiography performed on admission (Figure 1) revealed right pleural effusion (PE) without ascites. The abdominal ultrasound showed enlarged ovaries of approximately 5 cm in size, with a slight amount of fluid in the pouch of Douglas, and absence of ascites. Ultrasound-guided thoracentesis was performed for the patient's right-sided PE on the day of admission. The procedure yielded 40 mL of yellow liquid fluid with transudate characteristics.

FINAL DIAGNOSIS

OHSS manifesting as a right PE without ascites.

TREATMENT

The management of this patient included the control of inputs and outputs, balance, and daily weight. Albumin (20%, 20 g/8 hours) and furosemide (20 mg/8 h) were administered, as well as thrombosis prophylaxis with enoxaparin (60 mg/24 hours). Analgesia with paracetamol was prescribed for pain and oxygen therapy on demand through the use of a nasal cannula.

Although the clinical outcome of the patient was favorable, with a significant decrease in dyspnea on oxygen therapy, the tendency for tachycardia persisted together with light dyspnea on exertion and oxygen saturation between 95% and 96%.

A screening electrocardiogram (ECG) showed sinus tachycardia, a slight right axis deviation of approximately 90°, and negative T in V1 and inferior leads (Figure 2). An arterial blood gas test revealed hypoxemia (partial pressure of oxygen

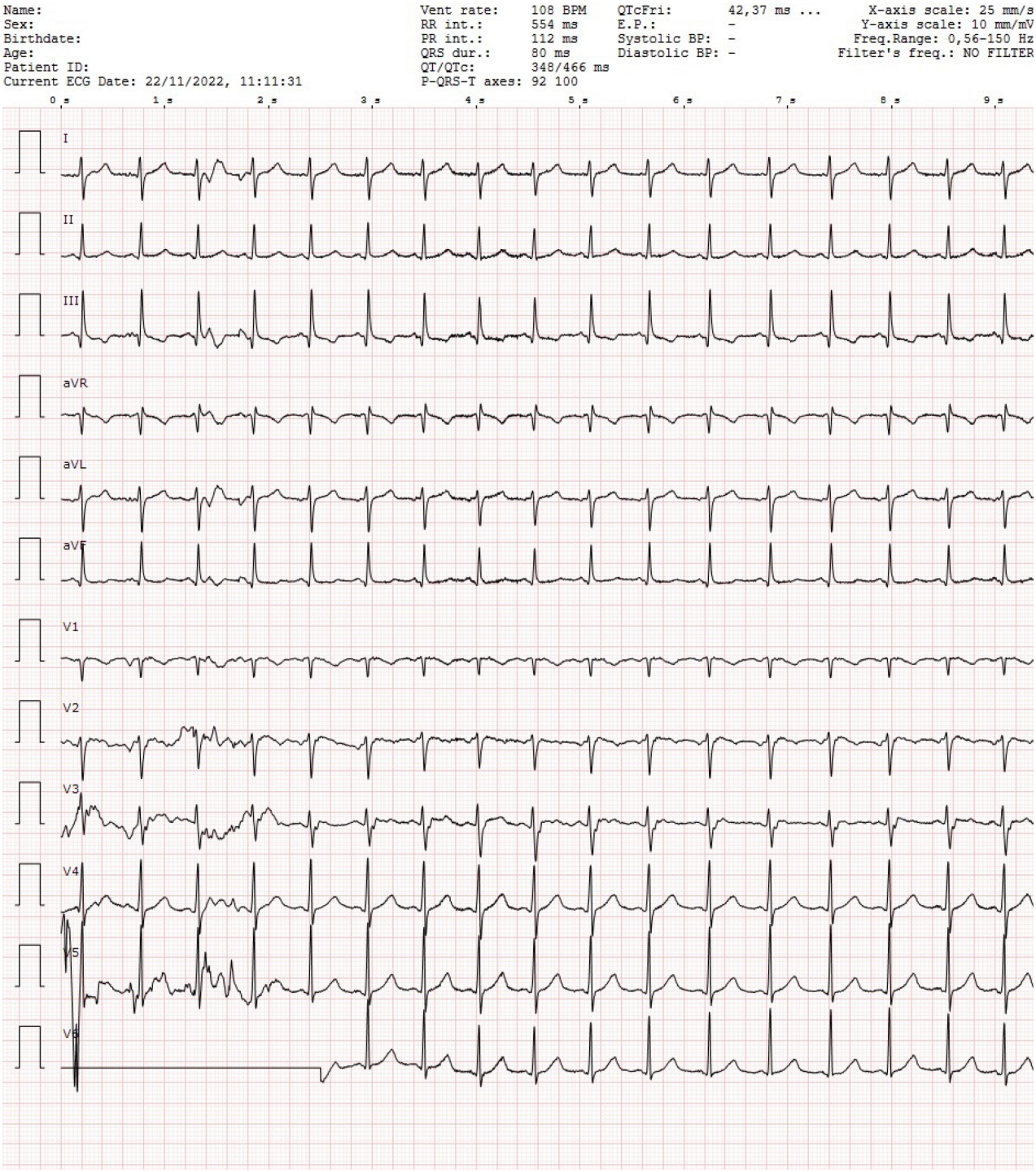

Figure 2 Screening electrocardiogram (November 22, 2022) showed sinus tachycardia, a slight right axis deviation of approximately 90°, and negative T in V1 and inferior leads.

in arterial blood of 59 mmHg), hypocapnia (partial pressure of carbon dioxide in arterial blood of 27 mmHg), and alkalemia (bicarbonate, 22 mmol/L) (Table 1).

Based on the results of the ECG and arterial blood gas tests, thoracic and cardiac ultrasound was performed, in which a large PE was observed, along with secondary passive atelectasis of the right lung. The function of the right ventricle was normal, with no evidence of dilatation, while the function of the right atrium was compromised. Ultrasound-guided thoracentesis was then performed. This procedure yielded a volume of 1200 mL. A pleural drainage catheter was left in place for continued drainage (Figure 3A).

The hypoxemic respiratory failure of the patient could be caused by a large PE; however, considering the clinical picture of tachycardia, hypocapnia, and major risk factors such as ovarian stimulation, pulmonary embolism (PE) was also mandatory for inclusion in the differential diagnosis. Compression Doppler ultrasound of both lower extremities revealed no thrombosis, thus ruling out deep vein thrombosis. Computed tomography angiography was not required for the screening of PE because there was no worsening of dyspnea or a significant increase in D-dimer levels.

Table 1 Analytical evolution of the patient

Parameter	November 18	November 20	November 21	November 22	November 23	November 28	Units	Reference interval
Hemogram								
Leukocytes	23.86	15.21	12.75	-	11.64	11.77	10^9/L	4.00-11.00
Red blood cells	5.25	4.98	4.75	-	4.35	3.61	10^{12}/L	3.80-4.80
Hemoglobin	160	156	147	-	137	115	g/L	120-150
Hematocrit	0.500	0.470	0.440	-	0.410	0.340	L/L	0.360-0.460
Mean corpuscular volume	94.8	94.8	93.2	-	93.7	94.9	fl	80.0-100.0
Mean corpuscular hemoglobin	30.6	31.3	31.0	-	31.4	31.8	pg	26.7-33.3
Mean corpuscular hemoglobin concentration	322	330	333	-	335	335	g/L	310-350
Red cell distribution width	14.4	13.4	13.3	-	13.0	13.4	%	10.5-17.2
Hemoglobin distribution width	22.8	-	-	-	-	19.9	g/L	22.7-28.1
Platelets	431	432	374	-	371	380	10^9/L	130-400
Mean platelet volume	8.9	7.6	7.0	-	6.9	9.6	fl	6.2-11.0
Platelet distribution width	48.3	-	-	-	-	35.9	%	40.0-90.0
Automatic differential count								
Neutrophils	79.5	77.9	76.6	-	82.3	70.8	%	45.0-75.0
Eosinophils	0.1	0.2	0.3	-	0.1	1.9	%	< 5.0
Basophils	0.7	0.2	0.2	-	0.2	0.4	%	< 2.0
Lymphocytes	14.1	14.8	16.0	-	11.1	20.7	%	17.0-55.0
Monocytes	4.6	5.8	5.5	-	5.2	5.3	%	2.0-10.0
Unidentified cell	1.0	1.2	1.5	-	1.0	0.9	%	< 4.0
Neutrophils	19.0	11.8	9.8	-	9.6	8.3	10^9/L	2.0-7.0
Eosinophils	0.0	0.0	0.0	-	0.0	0.2	10^9/L	< 0.5
Basophils	0.2	0.0	0.0	-	0.0	0.0	10^9/L	< 0.2
Lymphocytes	3.4	2.3	2.0	-	1.3	2.4	10^9/L	0.9-4.5
Monocytes	1.1	0.9	0.7	-	0.6	0.6	10^9/L	0.1-1.0
Unidentified cells ABS	0.2	0.2	0.2	-	0.1	0.1	10^9/L	< 4.0
Hemostasis								
Prothrombin time	97.5	-	98.9	-	-	100.0	%	80.0-100.0
Prothrombin time (seconds)	12.2	-	11.8	-	-	12.0	sec	9.9-13.6
International normalized ratio	1.05	-	0.99	-	-	1.03		
Prothrombin time ratio	1.03	-	1.01	-	-	1.02		0.85-1.15
Activated partial thromboplastin time	24.2	-	26.5	-	-	26.3	sec	23.5-32.5
Activated partial thromboplastin time ratio	0.9	-	1.0	-	-	1.0		0.8-1.2
Quantitative D-dimer	2550	1120	-	-	-	-	ng/mL	< 500
General biochemistry								
C reactive protein	-	-	2.29	-	-	-	mg/dL	< 1.00
Glucose	87	92	94	-	115	66	mg/dL	65-110
Creatinine	0.84	0.86	0.92	-	0.93	0.68	mg/dL	0.30-1.30

Estimated glomerular filtration rate (Chronic kidney disease epidemiology collaboration)	88	85.33	78.65	-	77.63	>90	mL/min/1.73m²	
Aspartate aminotransferase	19	31	-	-	51	37	U/L	5-40
Alanine aminotransferase	16	42	-	-	93	81	U/L	5-40
Gamma-glutamyl transferase	25	38	-	-	61	63	U/L	5-40
Total bilirubin	1.3	1.3	-	-	2.10	0.5	mg/dL	< 1.2
Direct bilirubin	0.4	0.4	-	-	0.90	-	mg/dL	< 0.6
Indirect bilirubin	0.9	0.9	-	-	1.2	-	mg/dL	< 0.6
Alkaline phosphatase	55	56	-	-	-	101	U/L	46-116
Lactate dehydrogenase	165	106	-	-	-	123	U/L	< 234
Albumin	45	50	-	-	60	46	g/L	34-48
Sodium	134	132	132	-	129	131	mEq/L	135-145
Potassium	4.4	4.0	3.6	-	3.8	4.5	mEq/L	3.5-5.5
Calcium	-	-	-	-	-	9.0	mEq/L	8.5-10.5
Recent urine sodium	21	-	-	-	-	-	mEq/L	
Urine potassium	61.3	-	-	-	-	-	mEq/L	
High-sensitivity troponin I (urgent)	-	-	-	12.5	-		ng/L	< 45.2
Gonadal axis								
Beta human chorionic gonadotropin, serum	-	-	-	-	3392.70		mUI/mL	< 5.00
Arterial blood gas								
pH, arterial blood	-	-	-	7.508	7.479			7.350-7.450
pCO_2, arterial blood	-	-	-	27.0	30.9		mmHg	35.0-45.0
pO_2, arterial blood	-	-	-	59.6	103.2		mmHg	75.0-100.0
Actual bicarbonate, arterial blood	-	-	-	21.0	22.4		mmol/L	21.0-26.0
Total CO_2, arterial blood	-	-	-	21.8	23.4		mmol/L	23.0-29.0
Base excess, arterial blood	-	-	-	-0.80	-0.20		mmol/L	-3.00-3.00
Hematocrit, arterial blood	-	-	-	39.0	-		%	36.0-51.0
Hemoglobin, arterial blood	-	-	-	13.1	-		g/dL	12.0-17.0
Carboxyhemoglobin, arterial blood	-	-	-	0.90	-		%	< 4.00
Methemoglobin, arterial blood	-	-	-	0.3	-		%	< 1.5
Ionized calcium, arterial blood	-	-	-	1.08	-		mmol/L	1.03-1.23
Sodium, arterial blood	-	-	-	126	-		mmol/L	135-145
Potassium, arterial blood	-	-	-	4.0	-		mmol/L	3.5-5.5
Chloride, arterial blood	-	-	-	94	-		mmol/L	98-107
Glucose, arterial blood	-	-	-	103	-		mg/dL	65-110
Lactate, arterial blood	-	-	-	12.2	-		mg/dL	5.0-20.0
Bilirubin, arterial blood	-	-	-	< 2	-		mg/dL	
Arterial standard bicarbonate	-	-	-	-	24.3		mmol/L	21.0-28.0
Oxygen Hb arterial saturation	-	-	-	-	98.3		%	95.0-99.0
Ionized calcium; arterial blood	-	-	-	-	1.11		mmol/L	1.03-1.23
Arterial oxyhemoglobin fraction	-	-	-	-	97.2		%	> 95.0

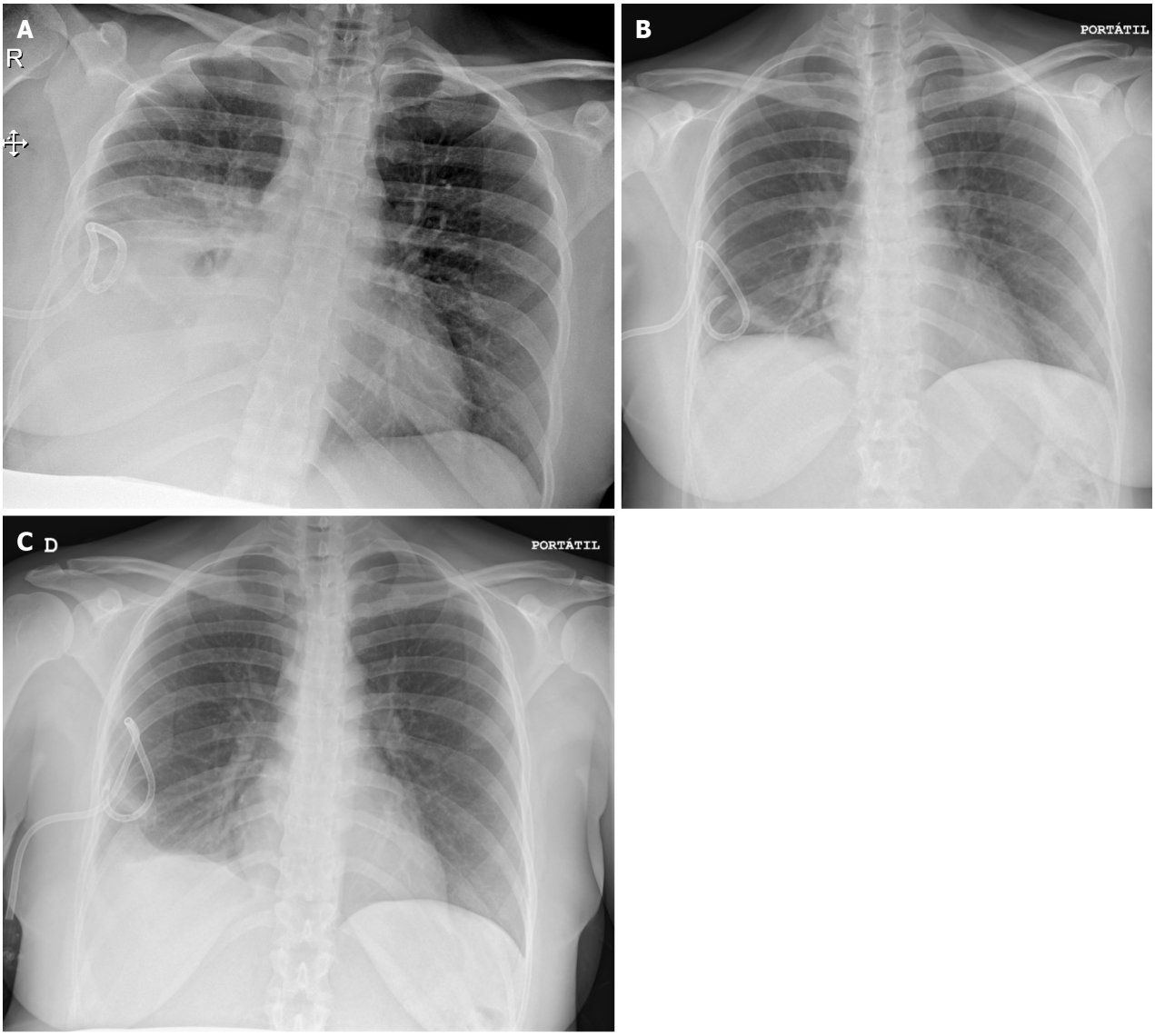

Figure 3 Chest X-ray. A: Chest X-ray after ultrasound-guided thoracentesis (yielding a volume of 1200 mL) and placement of the pigtail catheter for continuous pleural drainage of the right pleural effusion (November 22, 2022); B: Chest X-ray follow-up (November 23, 2022). The evolution of the patient was favorable, with periodic clamping of the drainage and treatment with furosemide and albumin; C: Chest X-ray follow-up (November 26, 2022). On this day, treatment with furosemide and albumin was discontinued. The pleural drainage was removed two days after, having drained up to 5 L during the whole hospitalization period.

OUTCOME AND FOLLOW-UP

The evolution of the patient was favorable, and periodic clamping was performed, draining up to 5 liters of pleural fluid during the entire hospitalization period. Treatment with furosemide and albumin was discontinued on the eighth day due to the significant decrease in pleural fluid drainage. Follow-up chest X-ray imaging was performed (Figure 3B and C), and on the tenth day of hospitalization, the pleural drainage tube was removed, with further ultrasound evidence of very little pleural fluid in the thoracic cavity.

Ultrasound revealed dichorionic diamniotic gestation. The pregnancy passed without incident and was completed at 37 weeks by cesarean section. Two children weighing 2484 and 2705 g were born with correct neonatal evolution.

DISCUSSION

Although rare, many cases of OHSS with massive PE without ascites have been reported in the literature[4,5]. A systematic review of 24 studies encompassing 30 clinical cases of OHSS with isolated PE conducted in 2018 revealed that the most common clinical complaint was dyspnea (86.6%). Most patients developed isolated right PE (80%), whereas some presented with bilateral PE (16.6%). The predominance of right PE has been related to anatomical defects on this side of the diaphragm as documented after laparoscopies, thoracotomies or post-mortem studies[4]. Laboratory investigations revealed hemoconcentration and elevated white blood cell counts. Among the clinical cases mentioned, 90% of the patients underwent thoracentesis[6].

In the present case, the patient experienced a severe form of OHSS, characterized by a large right PE along with associated atelectasis. In addition, the patient displayed compromised right cardiac function. In this clinical case, three important aspects deserve to be highlighted: The appearance of OHSS in a low-risk IVF cycle, the challenge of differential diagnosis with the presence of PE, and treatment with continuous aspiration of the pleural cavity with the administration of albumin.

First, the occurrence of OHSS in the context of IVF cycles has always been associated with a hyperresponse to ovarian stimulation, although sometimes OHSS also occurs in an unsuspected manner[7]. Recently, attempts have been made to reach a consensus on what hyperresponse means, especially for the application of effective prophylactic measures[8]. According to the latest Fertility and Sterility guidelines for the prevention of moderate and severe OHSS[9], the most notorious clinical finding that could have been considered a risk factor for our patient was an AMH concentration of 4.23 ng/mL at baseline. However, in our case, OHSS occurred in a situation of low response to ovarian stimulation; therefore, the final triggering of ovulation with GnRH analogs was not considered, nor was the strategy of "freezing all" the embryos. Nonetheless, cycle management was as expected. It must be considered that prophylactic measures do not completely prevent the occurrence of OHSS[10].

Second, a relevant aspect of this case was the suspicion of PE. The patient's clinical symptoms required us to rule out PE. Although there are clinical scores such as the Wells score, revised Geneva score or Years score whose objective is to avoid the use of computed tomography angiography[11], the determination of D-dimer increases the sensitivity of the scores[12]. In the case of our patient, there were some aspects that limited the effectiveness of the determination of D-dimer, on the one hand the fact of a recent oocyte recovery that could explain an increase in its levels and also the fact of a pregnancy that could also explain it[13]. However, D-dimer levels increase throughout pregnancy and in the first trimester it would not be expected to have levels as high as those observed in the patient[14]. Ultimately, the clinical presentation of the patient and the laboratory test results were attributed to an atypical, severe case of OHSS.

Third, this clinical case shows substantial improvement through the application of continuous drainage of the pleural cavity. Unlike other cases in which drainage was also applied, in our case, the effectiveness of this therapeutic measure accompanied by the administration of intravenous albumin was demonstrated. As in other pathological situations with similar pathophysiology, fluid drainage in the third space requires compensation for intravascular oncotic pressure to avoid serious hemodynamic disorders that could have occurred if intravenous albumin had not been administered during sustained drainage of the fluid in the pleural cavity[15].

CONCLUSION

This clinical case highlights the necessity for an accurate management in all patients undergoing IVF, as severe OHSS can occur unexpectedly even in patients with lack of risk factors. Our experience with aspiration of the pleural cavity combined with intravenous albumin administration demonstrates an effective treatment modality for managing massive PE. The critical challenge of differential diagnosis with PE, particularly in pregnant patients, reinforces the need for cautious interpretation of elevated D-dimer levels. Finally, future research should aim to refine prophylactic strategies against OHSS and further investigate optimal management techniques for severe presentations involving PE.

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FOOTNOTES

Author contributions: Solsona I and Peralta S collected the clinical information of the patient; Solsona I and Fàbregues F wrote the manuscript; Peralta S and Barral Y added information to the manuscript; Solsona I and Barral Y prepared the figures; Solsona I, Peralta S, Barral Y, Fàbregues F and Giménez-Bonafé P reviewed the manuscript; Giménez-Bonafé P supervised the data collection, organized the manuscript, corrected the English, and reviewed the final version of the manuscript.

Informed consent statement: The patient has given her consent for both the photos and her clinical information to be used anonymously for the publication of her case study.

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