Unilateral Isolated Pleural Effusion in Ovarian Hyperstimulation Syndrome

Ali Sami GÜRBÜZ1, Emel Ebru ÖZÇİMEN2, Necati ÖZÇİMEN3
Konya, Turkey

ABSTRACT

Ovarian hyperstimulation syndrome (OHSS) is an iatrogenic complication with symptoms ranging from mild to severe. Pleural effusion occurs in severe forms of OHSS; however, isolated pleural effusion is rare. Here, we report a case of OHSS with isolated pleural effusion at presentation and discuss OHSS in light of the literature.

Keywords: Ovarian hyperstimulation syndrome, Ovulation induction, Pleural effusion


Introduction

Ovarian hyperstimulation syndrome (OHSS) is an iatrogenic complication that occurs in 33% of ovarian stimulation cycles, with clinical manifestations ranging from mild to severe (1). The pathogenesis of OHSS is unknown; however, increased vascular permeability leading to hemoconcentration and inadequate end-organ perfusion is considered to be the primary pathophysiology (2,3). Pleural effusion has been reported in 10% of severe OHSS cases and is usually associated with marked ascites (4,5). Isolated pleural effusion is rare in OHSS cases (5). Here, we report a case of OHSS with isolated pleural effusion and discuss the condition in light of the literature.

Case Report

A 30-year-old nulligravid woman with 5 years of unexplained infertility and no significant past medical history or physical findings was referred to our in vitro fertilization clinic. On day 2 of her menstrual cycle, 11 antral follicles were observed on each ovary; her follicle-stimulating hormone (FSH), luteinizing hormone, and estradiol (E2) values were 5.28 mIU/mL, 2.12 mIU/mL, and 20 pg/mL, respectively. Her male partner’s spermiogram parameters were normal.

Ovarian stimulation was initiated with 225 IU of recombinant FSH for 5 days. The dose was increased to 300 IU on day 5 because of low E2 levels and a poor ovarian response. On day 10 after induction, two follicles reached 20 and 18 mm in diameter. The patient’s peak E2 level was 1733 pg/mL. Oocyte pick-up (OPU) was performed 36 h after the administration of 10000 IU of urinary human chorionic gonadotropin (hCG). A total of 12 oocytes were retrieved, and one embryo transfer (ET) was performed on day 3 after OPU.

The patient’s β-hCG level was 278 IU/mL on day 12 after ET. The patient complained of dyspnea 2 days after β-hCG positivity was established. She was afebrile and tachycardic, and her weight had increased by 2 kg. Her oxygen saturation was 97% on room air. She had shortness of breath, a cough, and chest pain, tachypnea, and decreased breathing sounds. The results of echocardiography and electrocardiography were normal. She did not have nausea, vomiting, or abdominal distension.

An abdominal ultrasound revealed no evidence of intraperitoneal fluid; however, the patient’s ovaries were enlarged bilaterally (right: 50x85 mm; left: 52x89 mm). Her liver and renal function tests and electrolytes were normal, and her hematocrit was 42%.

The patient and her family refused a chest x-ray because of her pregnancy. An ultrasound examination revealed no evidence of pleural effusion and minimal pleural effusion on the left side. Approximately 1500 cc of yellow-colored fluid was aspirated from the right side by thoracentesis, and an additional 3000 cc of fluid was drained via a pigtail catheter over 3 days. The fluid was exudative with 40 g/L protein. A pleural fluid culture was negative. Low-molecular-weight heparin was administered to prevent deep vein thrombosis.

An abdominal ultrasound was performed daily and no fluid was observed. After 3 days, the patient’s dyspnea and cough
resolved, her β-hCG value was 1285 IU/mL, and the pigtail catheter was removed without complications. The patient was discharged 2 days after the pigtail catheter was removed and followed closely as an outpatient undergoing serial ultrasound surveillance for pleural effusion and dyspnea. She recovered fully without sequelae. At present, the patient is at 9 weeks gestation in an uncomplicated singleton pregnancy.

Discussion

OHSS is a serious iatrogenic complication of controlled ovarian hyperstimulation. The condition is usually self-limiting, but it may be life-threatening (6). OHSS is classified as mild, moderate, or severe. Mild OHSS symptoms are relatively common in induced cycles and include abdominal distention, mild nausea, vomiting, and diarrhea (7). Pleural or pericardial effusion is a manifestation of severe OHSS (8). Severe OHSS has been reported in fewer than 2% of patients. Early OHSS is related to the ovarian response to stimulation and is an acute effect of exogenous hCG administration, which generally occurs within 9 days of oocyte retrieval. In contrast, late OHSS occurs after the initial 10-day period and is related more to the endogenous hCG produced by an implanted embryo than to the ovarian response (9). The primary goal of ovulation induction is to induce pregnancy; however, if pregnancy occurs, OHSS tends to be more severe with a longer duration.

Herein we report a case of isolated unilateral pleural effusion as a symptom of severe OHSS. Isolated pleural effusion associated with OHSS was first described in 1975 (4); however, few cases have been reported since (10-12).

The pathogenesis of isolated pleural effusion without ascites is unknown; however, negative intrapleural pressure may pull fluid from the abdomen into the thoracic cavity through the holes in the diaphragm (13).

Previous studies have shown that pleural effusions may be exudative, as in our case, or transudative (8). Because our patient was pregnant, a chest x-ray could not be performed; however, we used ultrasonography to detect pleural effusion. Ultrasound techniques have been shown to be useful for the diagnosis of pleural effusion (14).

Our patient did not have abdominal distension or ascites; thus, the pleural effusion may have been overlooked with an abdominal examination alone. The view that dyspnea is only a direct result of increased intra-abdominal pressure in patients with OHSS can mislead physicians.

Dyspnea, pleural effusion, ascites, and hemoconcentration are observed in severe cases of OHSS. Our patient had no significant markers of severe OHSS aside from massive pleural effusion. Although massive pleural effusion may be caused by several systemic diseases, we suspected OHSS because our patient was pregnant, and she improved following thoracentesis and supportive therapy. Thus, a thorough examination of patients with OHSS coupled with effective supportive therapy may lead to a favorable prognosis. In OHSS cases without abdominal ascites, pleural effusion should be considered in the presence of dyspnea.

References