Benign obstetric and gynaecological diseases associated with pleural effusion: a narrative review

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This review provides a comprehensive and detailed overview of obstetric and gynaecological diseases that are associated with pleural effusion https://bit.ly/3Apcw7A

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Abstract

Certain obstetric and gynaecological diseases are associated with pleural effusion, including benign peripartum pleural effusion, endometriosis, ovarian hyperstimulation syndrome and Meigs syndrome. This review provides a comprehensive and detailed overview of this group of rare diseases. A thorough understanding of their unique characteristics is required to ensure early identification, correct diagnosis and appropriate management.

Introduction

Pleural effusion (PEf) is a common clinical problem with an estimated prevalence of 400 cases per 100 000 population [1]. Over 60 entities have been associated with PEf [1, 2], including some obstetric and gynaecological diseases [3, 4]. The low incidence of the latter may cause a delay in the aetiological diagnosis of PEf, sometimes due to lack of knowledge of the clinician. The objective of this article is to address different aspects of obstetric and gynaecological diseases causing PEf through a narrative review.

Benign peripartum pleural effusion

Small PEfs may remain asymptomatic and an imaging study may be required for diagnosis. Initial studies based on chest radiographs have shown that small PEfs may occur during labour and resolve spontaneously without complications [5, 6]. Hughson *et al.* [5] reviewed the records of 112 women who had a chest radiograph (postero-anterior and lateral) within 24 h after vaginal delivery. They found that 46% (51 out of 112) had a PEf, which was bilateral in 75% of cases. Subsequently, they conducted a prospective study including 30 women that revealed that 20 (67%) subjects developed a PEf, of which 11 were bilateral. The authors applied a scoring system to establish the degree of costophrenic sinus occupancy. They found that most of the PEfs were small in size with no predominance of one side over the other. They also incorporated a control group of 30 non-pregnant women, of whom only three (10%) had a small PEf [5]. No correlation was found between PEf and the presence of postpartum obstetric complications or adverse fetal outcomes.

These authors hypothesised that the physiological conditions of labour could favour fluid accumulation in the pleural space, due to increased blood volume and reduced oncotic pressure during pregnancy. Thus, changes in hydrostatic and oncotic pressures would favour transudation of fluid into the pleura. In addition, Valsalva manoeuvres during labour would increase intrathoracic pressure, which would decrease lymphatic drainage of the pleura by increasing systemic venous pressures. A pregnant uterus could also favour the formation of atelectasis by reducing diaphragmatic movement (especially in the last trimester of pregnancy), thereby facilitating the accumulation of fluid in the pleural space.

In a retrospective study, Stark and Pollack [6] evaluated 45 women presenting with fever or respiratory symptoms within the first 48 h after delivery to find that 44 (98%) had a small PEf. However, this series included women who had a caesarean section or cardiopulmonary complications; therefore, the authors were unable to draw definitive conclusions about the incidence of asymptomatic PEf in women with an uncomplicated delivery [6].

Udeshi *et al.* [7] used ultrasound to assess postpartum PEf in 50 women, as it was more specific than radiography in detecting PEf. Delivery was vaginal in 29 and by caesarean section in 21. Only a patient with severe pre-eclampsia and pulmonary oedema requiring treatment with diuretics had ultrasound evidence of PEf. Another study of 34 women with moderate/severe pre-eclampsia who underwent ultrasonography within 24 h of delivery showed that nine (26.5%) had PEf, although six had undergone an emergency caesarean section [8]. Between the group with PEf (n=9) and the group without PEf (n=25), there was no difference in the incidence of emergency caesarean section, severity of eclampsia, pulmonary oedema or fluids administered. The only significant difference between the two groups was that the incidence of perinatal fetal mortality was 10-fold higher in the group with PEf (possibly due to preterm delivery or short gestation periods) [8].

A few years later, Gourgoulianis *et al.* [9] studied the postpartum period by performing a thoracic ultrasound in the first 24 h postpartum on 31 women who had a vaginal delivery. As a control group, they included 22 non-pregnant women, all of Greek origin and of the same age group. The amount of fluid was subjectively classified as small (depth <1 cm on ultrasound), moderate (depth 2–3 cm) and large (depth >4 cm). Seven women had postpartum PEf (23%; three moderate and four small), as compared to none of the non-pregnant women ($p<0.001$). No women had complications such as pre-eclampsia, pulmonary embolism or cardiomyopathy.

In short, there is much discrepancy in determining the incidence of benign postpartum PEf in women with uncomplicated pregnancy and delivery. This inconsistency lies in the methods used to detect PEf. What is certain is that the incidence of PEf was higher in the studies that had used non-specific procedures for PEf diagnosis (radiography) and had not positioned patients in lateral decubitus due to the association of this technique with false positive results. When ultrasound is used, the observed frequency of PEf is much lower, unless the patient has pre-eclampsia. In these cases, PEf may correlate with increased perinatal mortality. If the PEf is secondary to physiological changes that have occurred during normal pregnancy and delivery, it will be small and asymptomatic. If the PEf is middle-sized or symptomatic, it should be studied as any PEf unrelated to pregnancy or delivery.

Endometriosis

Endometriosis is defined as the presence of endometrial tissue outside the uterus, and affects 5–10% of women of childbearing age [10]. Although extra-uterine locations are more frequent in the pelvis, thoracic endometriosis has been extensively described. It can manifest as pneumothorax (73%), haemothorax (14%), haemoptysis (7%) or pulmonary nodules (6%) [11]. As it occurs in hepatic hydrothorax, pelvic endometriosis may also induce PEf secondary to ascites, thereby resulting in fluid reaching the pleural space through diaphragmatic defects [12].

There are several theories about the aetiology of thoracic endometriosis. The first is coelomic metaplasia, where endometrial tissue and pleural and peritoneal mesothelia share the same embryological origin. Thus, pathogenic stimuli could induce pleural or peritoneal precursor cells to differentiate into endometrial cells. However, this theory would not explain intrapulmonary endometriosis. The second theory is lymphatic or haematogenous embolisation from the uterus or pelvis, which could explain both intrapulmonary endometriosis and endometriosis in other extrapelvic locations. The predisposing factor for microembolisation would be trauma or manipulation of uterine tissue. The last theory is that of retrograde menstruation with subsequent peritoneal implantation of endometrial tissue, which is currently the most plausible explanation. It is based on the retrograde migration of endometrial cells from the fallopian tube into the peritoneal cavity and the right paracolic space; there, endometrial cells reach the pleural space through right hemidiaphragm defects, since access to the left pleural cavity would be hindered by different anatomical structures. Implantation in the thorax would follow lymphovascular embolisation or transabdominal-transdiaphragmatic migration. However, none of these theories alone explains all cases of thoracic endometriosis and this condition probably has a multifactorial aetiology [13].

The series published to date about PEf secondary to endometriosis are limited. Consequently, the clinical characteristics of this entity and the optimal diagnostic and therapeutic procedures for PEf have not yet been conclusively established. However, two systematic reviews contributed recently to clarifying some of

these aspects [14, 15]. We will refer to data provided by Porcel *et al.* [15], as they conducted the largest review (142 articles published in the last 70 years, including 176 patients from all parts of the world, with the largest series including only 11 patients). The most relevant characteristics of their series were the following: median age was 33 years, 78% were nulliparous and PEf was synchronised with menstruation in 87% [15, 16]. The most frequent symptoms were dyspnoea (67%), chest pain (55%) and abdominal pain (40%). Ascites was present in 42% of patients. PEf was right-sided in 88.5% and bilateral in 11% of patients. Only one case out of 174 was left-sided. PEf was small in 18%, moderate in 26% and massive in 56%. Pleural fluid was almost always bloody (99%) and always fulfilled the criteria for an exudate (100%). As many as 73% had >100 000 red blood cells per mm^3, only 9% had a neutrophil count >50%, and 50% had adenosine deaminase values >35 $U \cdot L^{-1}$. These clinical findings, a bloody effusion, association with menstruation and demonstration of pelvic endometriosis are sufficient to establish diagnosis [16].

In the Porcel *et al.* [15] series, pleural biopsy yielded findings consistent with endometriosis in 74% of cases (79 out of 107), with no difference in performance between closed pleural biopsy and thoracoscopy (18 out of 26 (69%) *versus* 48 out of 58 (82%), respectively; p=0.16). This finding is striking, since diagnostic yield should improve when the pleural cavity is visualised during biopsy, as it enables the selection of the biopsy site (local anaesthetic thoracoscopy and video-assisted thoracic surgery). However, the authors do not provide any explanation for this. Clusters of endometrial glands supporting this diagnosis were observed in 9% of cytologies [15]. Cytological examination can occasionally help confirm diagnosis [17].

Three types of treatment were administered to these patients: hormone therapy, thoracic surgery and different combinations of abdominal surgery. Hormone therapy is administered to suppress ectopic endometrial tissue by interfering with ovarian oestrogen production using agents such as oral contraceptives, progestogens, gonadotropin-releasing hormone agonists or danazol [13, 18]. In those who received some form of hormone therapy (127 patients), the median duration of treatment was 6 months; there was no difference in terms of positive response or recurrence of PEf between the different combinations of hormone therapy. Treatment will depend on the size of the PEf, with large PEfs requiring drainage. As many as 80% of patients underwent a procedure (surgical or non-surgical) on the pleural cavity and 26% experienced PEf relapse (median follow-up 2 years; quartiles 1–5 years). If haemothorax recurs (about 50% of cases), video-assisted thoracoscopic surgery is indicated to visualise and remove pleural implants [19]. If haemothorax recurs, treatment must be individualised on the basis of the patient's age, desire for fertility and symptomatology. Total hysterectomy with bilateral salpingo-oophorectomy may be necessary. The recurrence rate was lower when hormonal treatment was combined with thoracic surgery or with abdominal surgery, or when PEf alone was treated with thoracic surgery [15].

In short, although PEf secondary to endometriosis is rare, it should be considered if it occurs in a woman of childbearing age, is right-sided, moderate to large in size and bloody in appearance. A pleural biopsy is usually required to confirm diagnosis, and treatment includes a combination of hormone treatment and thoracic and/or abdominal surgery. Despite treatment, a quarter of cases may relapse with PEf.

Ovarian hyperstimulation syndrome

Ovarian hyperstimulation syndrome (OHS) is a serious iatrogenic complication that occurs when women undergo ovarian stimulation to induce ovulation and facilitate pregnancy, donor egg collection or *in vitro* fertilisation with exogenous chorionic gonadotropin hormone (CGH) and, less frequently, clomiphene citrate [20, 21]. The early form of the disease appears within the first 10 days after stimulation and may improve after a few days, when serum CGH levels fall. This syndrome can be life-threatening, as it causes a significant increase in vascular permeability, resulting in massive extravascular displacement of protein-rich fluid. This may lead to acute respiratory distress, haemoconcentration, hypovolaemia, thromboembolic events, electrolyte imbalance, ascites, hydrothorax and multi-organ failure in selected cases [22, 23]. There are several theories to explain increased vascular permeability. A hypothesis attributes it to increased levels of cytokines and vasoactive substances produced by ovarian hyperstimulation, especially interleukin-6 and vascular endothelial growth factor (VEGF) [24, 25]. Another theory posits that induced elevation of oestradiol would increase the expression of cystic fibrosis transmembrane conductance regulator, leading to massive fluid shifts through epithelial ion channels [26]. Finally, OHS may also occur late, 10–17 days after CGH administration, due to endogenous secretion of CGH following a successful pregnancy. Severity is not associated with early or late presentation, as a similar number of cases complicated with thrombosis has been reported for the two forms [22].

Fluid may reach the pleural space by two different routes. Bilateral PEf is probably caused by generalised capillary extravasation. In contrast, right-sided PEf is probably caused by fluid travelling from the peritoneal

cavity and crossing the diaphragm through the pores/defects it contains, which are more numerous on the right side than on the left [27].

Reportedly, OHS may occur spontaneously (at the beginning of a natural pregnancy and in the absence of any assisted reproductive treatment) [28]. In contrast, moderate–severe OHS occurs in 3–10% of cases of ovulation induction, which may reach 20% in high-risk women. Risk factors include age younger than 30–35 years, asthenic build, multigestational pregnancy, previous episodes, polycystic ovaries, rapidly rising oestradiol levels and excessive follicular response to stimulation drugs [21, 29, 30].

Initial clinical manifestations usually include abdominal pain and swelling followed by vomiting, diarrhoea, dyspnoea and reduced urine output [23]. In severe forms, hypotension, renal failure, ascites, PEf, pericardial effusion, acute respiratory distress syndrome and thrombotic events (including stroke) may be observed. PEfs are frequently unilateral with right-sided predominance (77%), although they are bilateral in 15% of cases [23, 31]. Biochemically, pleural fluid can be either a transudate or an exudate [22].

Irani *et al.* [32] recently conducted a systematic review of PEf as the only extra-ovarian manifestation of severe OHS. The study included 30 patients from 24 research studies: their mean age was 31.5 years; 29% had polycystic ovary syndrome; all had received CGH for oocyte maturation; and the mean±SEM level of oestradiol was 3110±330 $pg \cdot mL^{-1}$ on the day of the ovulatory trigger. The presenting symptom was dyspnoea in 86.6%; the PEf was right-sided in 80%; the fluid was an exudate in 66.7% and a transudate in 33.3% of cases. Fluid initially accumulated in the peritoneal cavity and then entered the pleural space.

Diagnosis must inevitably include a history of previous ovarian induction, and pleural fluid must be analysed to exclude other causes of PEf. The key aspect of treatment is volume repletion. Albumin administration neutralises mediators of vascular permeability and rapidly increases intravascular volume, reducing haemoconcentration and hypovolaemia. Fluid expanders (including crystalloids) decrease plasma aldosterone and renin levels, which improves renal function and contributes to CGH clearance [33, 34]. If the patient is dyspnoeic because of the size of the PEf, therapeutic thoracentesis should be performed.

Although prognosis is usually good, pulmonary complications may occur, such as pulmonary embolism (in about 2% of patients) and pulmonary infiltrates on chest radiography, attributable to acute respiratory distress syndrome or volume repletion hypervolaemia.

Meigs, pseudo-Meigs and pseudo-pseudo Meigs syndromes

The initial definition of Meigs syndrome was “a benign solid ovarian tumour with ascites and PEf, which resolved when the tumour was removed” [35]. Subsequently, the term “pseudo-Meigs syndrome” or “atypical Meigs syndrome” was coined to include cystic ovarian tumours, fibromyomas, endometriomas and low-grade malignant ovarian tumours without metastases. In any case, the distinctive characteristic of this syndrome is that both ascites and PEf resolve upon tumour removal. Finally, the term “pseudo-pseudo Meigs syndrome” was coined to define the presence of ascites and PEf with elevated levels of blood carbohydrate or carbohydrate antigen 125 (CA-125) in patients with systemic lupus erythematosus (SLE) and without any gynaecological tumour [36].

Meigs syndromes are rare conditions, most frequently occurring in the presence of ovarian fibroma. It is worth noting that only 1–2% of women with ovarian fibroma develop Meigs syndrome, especially postmenopausal women [37]. Regardless of the type of tumour, the pathogenesis is the same [38], which has been explained by several theories. One of these theories posits that the primary tumour secretes fluid containing high levels of interleukins, tumour necrosis factor-α and VEGF into the peritoneum by different mechanisms [39]; as a result, vascular permeability increases, thereby favouring fluid accumulation in the peritoneal cavity. Another hypothesis postulates that the tumour inhibits drainage of fluid from the peritoneal cavity by exerting pressure on the abdominal and pelvic lymphatics. Due to the positive pressure in this cavity, fluid moves into the pleural space, where pressure is negative, through diaphragmatic pores [40]. In these cases, the PEf is usually right-sided for the reasons explained in previous sections (endometriosis and OHS).

Clinical symptoms (general syndrome) and imaging studies (pelvic mass) suggest metastatic ovarian cancer. The PEf is usually located on the right side and, when it is large, it may cause dyspnoea. Krenke *et al.* [41], in a systematic review including 541 patients (196 with Meigs syndrome, 113 with non-classical Meigs and 108 with pseudo-Meigs syndrome), described the most relevant features of PEf. PEfs were significantly more frequently located on the right side or bilaterally than on the left side ($p<0.001$).

Surprisingly, only seven patients met Light's criteria, and pleural fluid was an exudate in only six patients, with a significant correlation between pleural and ascitic proteins [41].

Pseudo-pseudo Meigs syndrome is a rare manifestation of SLE, defined by the presence of ascites, PEf, and elevated levels of CA-125. A differential diagnosis should be made with classical SLE [42, 43]. Although the pathophysiology of ascites is unclear, it is probably caused by severe uncontrolled inflammation involving the serous membranes. The deposition of immune complexes in the peritoneum may trigger a local inflammatory or vasculitis reaction in the peritoneal vessels that stimulates the production of cytokines, interferon-γ, VEGF and fibroblast growth factor. Inflammation of the omentum stimulates CA-125 synthesis, leading to an elevated serum concentration of this protein [44]. The characteristics of pleural fluid in this syndrome have not yet been appropriately defined.

To establish the diagnosis of Meigs or pseudo-Meigs syndrome, pleural and ascitic fluids should be analysed. If cytology is negative, the pelvic mass should be biopsied to rule out metastatic ovarian cancer. If there is no malignancy, the tumour should be removed. Diagnosis will be confirmed if both ascites and PEf resolve after 2 weeks [27, 45].

In the case of pseudo-pseudo Meigs syndrome, treatment is focused on SLE, including corticosteroids or azathioprine, or cyclophosphamide in refractory cases [42, 43]. The prognosis of Meigs syndrome is good, and <1% of fibromas will progress to fibrosarcoma [46].

Conclusions

Benign obstetric and gynaecological diseases with secondary PEf constitute a heterogeneous group of diseases. A thorough understanding of their manifestations and distinctive characteristics will ensure early identification, correct diagnosis and appropriate management. Many aspects of these diseases remain poorly understood, especially the mechanism by which fluid reaches the pleural space. Moreover, a definitive treatment for recurrent PEf has not yet been developed. Further large, prospective, multicentric, correctly designed studies are necessary to elucidate these and other aspects. As some of these diseases entail some risks, informed consent should be obtained from patients prior to inclusion in any research project.

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