



Catamenial Pneumothorax (CP)

Leonard Ranasinghe^{1*}, Anne Liao² and Michelle Nguyen²

¹College of Medicine, California Northstate University, USA

²Medical Student, California Northstate University, USA

*Corresponding author: Leonard Ranasinghe, College of Medicine, California Northstate University, 9700 West Taron Dr. Elk Grove, CA 95757, USA

To Cite This Article: Leonard Ranasinghe. Catamenial Pneumothorax (CP). Am J Biomed Sci & Res. 2019 - 5(6). AJBSR.MS.ID.000969. DOI: 10.34297/AJBSR.2019.05.000969.

Received: 📅 October 17, 2019; Published: 📅 October 22, 2019

Abstract

Catamenial pneumothorax (CP) presents as a recurrent respiratory emergency due to failure to identify and treat the underlying gynecological condition. As a rare cause of pneumothorax, CP is unique in its temporal relationship with the onset of menses. Its nature of recurring during time of menstruation makes it less likely to be a separate spontaneous pneumothorax coinciding with menstruation. Several mechanisms have been proposed and although the exact pathophysiology of CP remains elusive, advances in surgical interventions have suggested diaphragmatic defects and thoracic endometriosis to play a fundamental role in the pathogenesis. In addition to technological advances, increased medical awareness and research have suggested that CP may be more common than previously thought. This review article will discuss the proposed mechanism of pathophysiology, evaluation, and acute and long-term management.

Introduction

Catamenial pneumothorax was first described by [1], as a pneumothorax associated with diaphragmatic endometriosis [2]. was the first to coin the term catamenial pneumothorax. Catamenial describes a temporal relationship with the onset of menses, and CP usually presents 1 day to 72 hours within the onset of menses in an ovulating woman. CP is considered a very rare clinical condition. However [3], suggests that CP may be more common than previously suspected in their retrospective review, identifying 8 CP patients out of 32 total pneumothorax female patients (25%) referred to their surgical center. According to [4], CP may have been missed before 1990 due to lack of proper visualization of the diaphragm that video-assisted thoracoscopy (VATS) now allows today in pneumothorax patients. Although the exact etiology of CP is unknown, most proposed theories involve the presence of ectopic endometrial tissue in the diaphragm or thorax cavity, known as thoracic endometriosis. CP is the most common presentation of thoracic endometriosis syndrome, over hemothorax, hemothysis, and parenchymal nodules. In addition, CPs are predominantly right sided (>90%) as described in current literature. CP should be considered in any woman of child-bearing age with recurrent pneumothoraces. Acute management of CP should include strong clinical suspicion for a pneumothorax and appropriate placement of chest tube and decompression therapy for the affected lung. Evaluation of a suspected CP includes video-

assisted thoracoscopy with a histological confirmation. Prevention of recurrences requires removal of ectopic endometrial tissue and repair of diaphragmatic defects.

Proposed Theories of Etiology

Prostaglandin theory

During menstruation, prostaglandins play a critical role in triggering the contraction of uterine muscles to expel menstrual products. Prostaglandin F₂ is a particularly potent vasoconstrictor of bronchioles and vascular structures. It is proposed to cause constriction of the bronchioles especially during the expiratory phase, leading to lung collapse and pneumothorax [5]. The research done by Rakhila et al (2015) further supports the prostaglandin theory in the context of endometriosis. They found increased mRNA expression of prostaglandin E₂ receptors, prostaglandin F_{2a} receptors, and prostaglandin transporter in ectopic endometrial tissue, increasing cell reactivity and uptake of prostaglandins during menses [6]. This increases the potential for prostaglandins to affect the lungs, although warrants more research.

Coelomic metaplasia

The pleura, peritoneum, and gonads develop from a common embryologic origin, the coelomic epithelium. Under the influence of estrogen, the pleura and peritoneum has been hypothesized to

differentiate into endometrial cells, leading to lung damage and pneumothorax [7]. This theory explains endometriosis in patients who have had a hysterectomy or bilateral salpingectomy and in men on high-dose estrogen therapy. However, this theory does not explain why catamenial pneumothoraces are predominantly right sided or intrapulmonary pathologies.

Diaphragmatic theory of air passage

According to Carter et al (1990) [8], endometrial implants in the pleura and diaphragm creates fenestrations and diaphragmatic pores through cyclical necrosis with menses, allowing air to enter the thorax. Bagan et al (2003)³ identified endometrial tissue around the diaphragmatic perforations, which is further evidence of ectopic endometrial tissue to causing diaphragmatic holes. In studies by [3,5], they found underlying diaphragmatic defects in every CP patient, suggesting the fundamental role of the diaphragm in CP. The air that enters the thorax is hypothesized to originate from the fallopian tubes due to a lack of cervical mucus plug during menstruation, then travels through the peritoneum and past the diaphragm. However, this theory does not describe CP in patients that lack fallopian tubes. In addition, air could enter the thorax with every menstrual cycle, but CP is not observed with every menstrual cycle.

Retrograde menstruation

This theory is based on the movement of endometrial tissue into the peritoneum, up the right paracolic gutter, and through the diaphragm preferentially on the right side due to the "piston" action of the liver against the diaphragm. The piston effect transmits peritoneal pressure through the diaphragm and allows passage of air and endometrial tissue into thoracic cavity [3]. This explains the occurrence of right sided catamenial pneumothoraces but not left sided. In addition, clockwise peritoneal circulation sweeps endometrial products from the pelvis up the right side of the peritoneal cavity. The falciform ligament halts migration to the left leading to predominantly right sided pneumothoraces.

Clinical Presentation

Diagnosis of CP can be challenging but should be suspected in women in their fourth decade of life with recurrent right sided pleuritic chest pain in close temporal relation to onset of menstrual bleeding [9,10]. Chest pain the day preceding or within 72 hours of menstrual bleeding is typical of CP. Pain and associated pneumothorax typically occurs on the right side [9,10]. Catamenial pneumothorax has late onset, generally in women 30 to 40 years old. A history of pelvic endometriosis may or may not be present, and diaphragmatic defects ranging from 1 to 10 mm are frequently observed. A subset of CP, termed juvenile catamenial pneumothorax (JCP) has been described in the literature [11]. This is typically earlier in onset, from age 19 and below. JCP has the usual features of close temporality with menses but does not appear to have preferential right sided laterality. A higher frequency

of left sided PTX has been observed in this population, and as such has been hypothesized to have a different etiology than usual CP. Furthermore, diaphragmatic defects observed in UCP are generally not seen in JCP.

Evaluation

Imaging by chest x ray and CT has limited use in diagnosis of CP [10]. Findings on CXR are typically consistent with pneumothorax, generally without mediastinal shift. Findings of pneumoperitoneum may be present¹⁰. MRI may show hyperintensities representing endometrial deposits with T1 or T2 weighted images¹⁰. CT may also be useful in imaging endometrial lesions [10]. However, as previously discussed, endometrial deposits may not be present in cases of CP. Therefore, the gold standard in diagnosis of CP remains video-assisted thoracoscopy (VATS) [10] in combination with histologic confirmation. Diaphragmatic defects and other lesions within the thoracic cavity may be easily visualized and repaired with VATS.

Management & Complications

For acute management of pneumothorax, appropriate treatment with placement of a chest tube or decompression to reinflate the lung may be indicated [12]. Historically, axillary thoracotomy was the mainstay for treatment of CP [13]. However, axillary thoracotomy presented several difficulties, particularly in that visualization of the diaphragm was virtually impossible. VATS provides an effective means of visualizing the diaphragm and thoracic cavity, and allows good access to repair any perforations or other defects observed¹³. The literature describes several different methods of repair, including removal of defects by stapler, simple suturing of defects, and plication [5,13,14]. A retrospective study by Bagan et al advocates for the use of the application of glue in combination with polyglactin mesh to the diaphragm to treat perforations which may not be visualized, and promote fibrotic adhesion of the diaphragm with the lung to prevent recurrence⁴. Repair of defects is typically followed by pleurectomy or pleurodesis by mechanical or chemical means (Talc powder) to promote fibrosis and adhesion between lung and diaphragmatic surfaces³. Surgical intervention is followed with medical treatment to prevent recurrence [15].

Medical treatment for CP aims to suppress ovulation [10], and options for treatment are similar to those used in management of endometriosis. These include gonadotropin releasing hormone (GnRH) analogs, danazol, oral contraceptive agents, and progestin drugs. Pregnancy, and consequent anovulation, has also been observed to suppress episodes of CP [9]. Use of GnRH analogs is typically limited to 6 to 12 months due to side effects¹⁴ of hypoestrogenism, particularly decreased trabecular bone density, vaginal dryness, and hot flashes. Bone density is typically restored after 2 years of GnRH analog cessation. Recurrence of CP has been observed in some cases with delayed initiation of medical treatment following surgical intervention [14], but there is some evidence in

the literature that appropriate surgical treatment alone may be effective in prevention of recurrence [16].

Acknowledgements

We would like to thank Dr. Leonard Ranasinghe for his assistance and mentorship.

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