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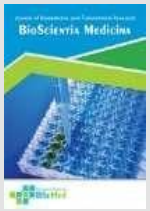

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Catamenial Pneumothorax in a Patient with Adenomyosis: A Case Report on a Successful Multidisciplinary Approach with Pleurodesis and Hormonal Therapy

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A B S T R A C T

Background: Catamenial pneumothorax, a rare manifestation of thoracic endometriosis syndrome (TES), presents a significant diagnostic and therapeutic challenge. It is characterized by recurrent spontaneous pneumothorax occurring in temporal relation to menstruation in women of reproductive age. The underlying pathophysiology is complex, often involving the ectopic presence of endometrial tissue within the thoracic cavity. Coexisting pelvic pathologies, such as adenomyosis, may be associated, further complicating the clinical picture. **Case presentation:** We present the case of a 38-year-old woman with a four-month history of recurrent, right-sided pneumothorax, with symptoms consistently commencing 24 to 48 hours prior to the onset of her menstrual cycle. Initial investigations, including high-resolution computed tomography of the thorax performed between menstrual cycles and microbiological analysis for tuberculosis, were unremarkable. The diagnosis of catamenial pneumothorax was established based on the distinct cyclical pattern of her symptoms. A subsequent gynecological evaluation, prompted by a history of secondary dysmenorrhea and menorrhagia, revealed uterine adenomyosis via transvaginal ultrasonography. The patient was managed through a collaborative, multidisciplinary approach involving pulmonology, thoracic surgery, and gynecology. Treatment consisted of chemical pleurodesis with doxycycline, administered via a chest tube, followed by continuous hormonal suppression therapy with oral progestin (2 mg/day). **Conclusion:** This case highlights the critical importance of maintaining a high index of suspicion for catamenial pneumothorax in women of reproductive age presenting with recurrent pneumothorax. A successful outcome was achieved through a coordinated, multidisciplinary strategy combining definitive pleural symphysis via pleurodesis with systemic hormonal therapy to suppress the underlying endometriotic process. This dual approach effectively prevented pneumothorax recurrence over a 12-month follow-up period, underscoring its efficacy in managing this complex condition.

1. Introduction

Spontaneous pneumothorax in women of reproductive age is a clinical entity that, while uncommon, necessitates a broad differential diagnosis.[1] Among the rarer causes is catamenial pneumothorax, a condition defined by the recurrent collapse of the lung in temporal association with menstruation, typically occurring within 72 hours before or after the onset of menses. It represents the most frequent manifestation of thoracic endometriosis syndrome (TES), a spectrum of disorders characterized by the presence of functioning endometrial tissue within the thoracic cavity, including the pleura, lung parenchyma, and diaphragm.[2] Despite being recognized for over six decades, its precise incidence remains difficult to ascertain, though it is estimated to account for 3-6% of spontaneous pneumothorax cases in menstruating women.[3]

The pathophysiology of catamenial pneumothorax is multifaceted and not yet fully elucidated. Several hypotheses have been proposed, with the most widely accepted being the theory of retrograde menstruation and peritoneal-pleural migration. This theory posits that endometrial cells travel from the pelvic cavity to the subdiaphragmatic space and subsequently enter the pleural cavity through congenital or acquired diaphragmatic defects.[4] Once implanted on the pleural surfaces, this ectopic endometrial tissue undergoes cyclical hormonal stimulation, leading to proliferation, bleeding, and shedding in synchrony with the menstrual cycle. This process can induce inflammation, tissue breakdown, and the formation of fenestrations, ultimately culminating in alveolar rupture and the entry of air into the pleural space.[5] Other proposed mechanisms include the hematogenous or lymphatic metastasis of endometrial cells and the prostaglandin-mediated bronchoconstriction and vasospasm leading to alveolar rupture.

Clinically, catamenial pneumothorax classically presents as right-sided chest pain and dyspnea, with over 90% of cases affecting the right hemithorax. This right-sided predominance is thought to be related to the preferential clockwise flow of peritoneal fluid along the right paracolic gutter towards the right hemidiaphragm. The diagnosis is often delayed due to a lack of awareness among clinicians and the cyclical, transient nature of the symptoms.[6] While imaging such as chest radiography confirms the pneumothorax, definitive evidence of thoracic endometriosis, such as pleural implants, is identified in less than half of the cases during video-assisted thoracoscopic surgery (VATS). Therefore, the diagnosis frequently relies on a high index of clinical suspicion based on the characteristic temporal relationship with menstruation.[7]

The management of catamenial pneumothorax is aimed at two primary goals: treating the acute pneumothorax and preventing its recurrence.[8] While initial management involves chest tube thoracostomy, the high rate of recurrence necessitates a more definitive strategy. This typically involves a multimodal approach combining surgical intervention to address the pleural space and hormonal therapy to suppress the underlying endometriosis. Surgical options include pleurodesis (chemical or mechanical) and repair of any identified diaphragmatic defects, often performed via VATS.[9] Hormonal suppression, typically with gonadotropin-releasing hormone (GnRH) agonists or progestins like Dienogest, aims to induce a state of amenorrhea and prevent the cyclical stimulation of ectopic endometrial tissue. The association between TES and pelvic endometriosis is well-established, with concurrent pelvic disease found in a significant proportion of patients.[10] However, the specific link with adenomyosis-a condition where endometrial glands and stroma are present within the myometrium-is less frequently reported but pathophysiologically plausible.

This case report details the diagnosis and successful management of a 38-year-old woman with catamenial pneumothorax and concurrent uterine adenomyosis. The novelty of this report lies in its detailed description of a successful, minimally invasive, multidisciplinary management strategy combining chemical pleurodesis with a modern, well-tolerated progestin in a patient with a confirmed uterine pathology that shares a common pathophysiological basis with endometriosis. The aim of this study is to underscore the importance of a collaborative approach between pulmonologists, thoracic surgeons, and gynecologists and to highlight the efficacy of this combined treatment modality in achieving long-term remission, thereby contributing to the clinical evidence base for managing this rare and complex disorder.

2. Case Presentation

A 38-year-old G2P2A0 woman presented to the emergency department of Dr. Achmad Mochtar Regional General Hospital, Bukittinggi, Indonesia, in December 2024, with a chief complaint of acute-onset, severe right-sided pleuritic chest pain, and progressive dyspnea. The patient reported that these symptoms

had started abruptly approximately 24 hours prior to the expected onset of her menstruation. She described the pain as sharp and stabbing, localized to the right lateral chest wall with radiation to the right shoulder, and exacerbated by deep inspiration and movement (Table 1).

Her medical history was significant for three previous, similar episodes over the preceding four months. The first episode occurred in August 2024, when she was diagnosed with a right-sided spontaneous pneumothorax and managed with chest tube thoracostomy. The lung re-expanded successfully, and the chest tube was removed after three days. Subsequent episodes in October and November 2024 were clinically milder and managed conservatively with observation and analgesia, as she was hesitant to seek hospital care. Crucially, the patient had meticulously tracked her symptoms and noted a consistent pattern: the onset of chest pain and breathlessness invariably occurred 1-2 days before her menstrual bleeding began, with symptoms gradually resolving over the subsequent 5-7 days.

Her gynecological history was notable. She had experienced menarche at age 13 with regular 28-day cycles. For the past five years, she had developed secondary dysmenorrhea and menorrhagia (heavy menstrual bleeding). She had two children, aged 10 and 8, conceived after a four-year period of primary infertility, for which she had not sought extensive investigation. She had no history of pelvic surgery. There was no family history of endometriosis or pneumothorax. She was a non-smoker and had no history of chronic lung disease, trauma, or recent air travel.

On physical examination at the current presentation, the patient was in moderate respiratory distress. Her vital signs were as follows: blood pressure 112/69 mmHg, heart rate 92 beats per minute, respiratory rate 28 breaths per minute, and oxygen saturation of 94% on room air, which improved to 100% with a non-rebreathing mask at 10 L/min. Her temperature was 36.4°C. She had a Body Mass Index (BMI) of 25 kg/m^2. Examination of the respiratory system revealed diminished chest wall movement on the right side, a hyperresonant percussion note over the right hemithorax, and markedly decreased to absent breath sounds on auscultation on the right side. The trachea was centrally located. Cardiovascular examination was unremarkable, with normal heart sounds and no murmurs. The remainder of the systemic examination was normal.

Initial laboratory investigations, including a complete blood count, coagulation profile, and basic metabolic panel, were all within normal limits. Her hemoglobin was 12.8 g/dL, leukocyte count was 6,620/mm^3, and platelet count was 258,000/mm^3. A D-dimer test was negative, ruling out significant pulmonary embolism. A posteroanterior chest radiograph confirmed a large right-sided pneumothorax, with an estimated 50% collapse of the right lung and a visible visceral pleural line. There was no evidence of tension physiology, pleural effusion, or parenchymal abnormalities.

Given the history of recurrent pneumothorax, further investigations had been conducted during her previous admissions. A sputum GeneXpert MTB/RIF test performed in August 2024 was negative for *Mycobacterium tuberculosis*. A high-resolution computed tomography (HRCT) scan of the thorax, performed with contrast in November 2024 (during a symptom-free interval between menstrual cycles), was reported as normal. Specifically, it showed no evidence of bullae, blebs, parenchymal lung disease, mediastinal lymphadenopathy, or diaphragmatic abnormalities. Based on the unequivocal temporal correlation between the recurrent right-sided pneumothorax and her menstrual cycle, a clinical diagnosis of catamenial pneumothorax was made. To investigate the associated gynecological symptoms and a potential underlying pelvic pathology, the patient was referred for a gynecological consultation.

Table 1. Clinical Summary of the Patient on Admission

A comprehensive overview of findings for a 38-year-old female with catamenial pneumothorax.

CATEGORY	PARAMETER	FINDING	REFERENCE RANGE / CLINICAL SIGNIFICANCE
Patient Demographics	Age	38 years	Falls within the typical reproductive age range for catamenial pneumothorax.
	Obstetric History	G2P2A0 (Gravida 2, Para 2, Abortus 0)	History of two successful pregnancies.
History	Chief Complaint	Acute-onset severe right-sided pleuritic chest pain and progressive dyspnea.	Classic symptoms of spontaneous pneumothorax.
	History of Present Illness	Recurrent episodes (4 in 4 months), consistently starting 24-48 hours before menstruation.	**Pathognomonic cyclical pattern for catamenial pneumothorax.**
	Gynecological History	Secondary dysmenorrhea, menorrhagia, and a 4-year history of primary infertility.	Suggests an underlying gynecological disorder like endometriosis or adenomyosis.
Physical Examination	Vital Signs	BP: 112/69 mmHg HR: 92 bpm RR: 28 breaths/min SpO_2: 94% (Room Air)	Tachypnea and mild hypoxemia are consistent with respiratory distress from pneumothorax.
	General Appearance	Moderate respiratory distress.	Indicates a significant lung collapse.
	Respiratory Exam (Right Hemithorax)	Diminished chest wall movement	Classic triad of physical findings for pneumothorax.
		Hyperresonant percussion note	
		Absent breath sounds	
Laboratory & Imaging	Complete Blood Count	Hb: 12.8 g/dL WBC: 6,620/mm^3 Platelets: 258,000/mm^3	Within normal limits, ruling out anemia or an infectious process.
	Chest Radiograph	Large right-sided pneumothorax (~50% collapse) with visible visceral pleural line.	Confirms the primary diagnosis.
	HRCT Thorax (inter-menstrual)	Normal; no bullae, blebs, or diaphragmatic defects identified.	Common finding in catamenial pneumothorax, as abnormalities can be subtle or transient.
	Transvaginal Ultrasound	Globular, enlarged uterus with heterogeneous myometrium.	**Highly characteristic of uterine adenomyosis, providing a link to the systemic condition.**

A transvaginal ultrasound was performed, which revealed a globular, enlarged uterus (measuring 6.5 x 4.2 cm) with a heterogeneous myometrial echotexture, myometrial cysts, and indistinctness of the endometrial-myometrial junction. These findings were highly characteristic of diffuse uterine adenomyosis.

Both ovaries appeared normal. The patient's management was planned and executed by a multidisciplinary team. The immediate priority was the management of the acute pneumothorax. A 24-French chest tube was inserted into the right pleural space in the 5th intercostal space, mid-axillary line, under local anesthesia (Table 2). This resulted in the immediate evacuation of air and prompt re-expansion of the right lung, confirmed by a post-procedure chest radiograph. The patient reported significant relief of her dyspnea and chest pain. Notably, within two hours of insertion, approximately 100 mL of dark, non-clotting blood drained from the chest tube, suggestive of catamenial hemothorax, another manifestation of TES.

Table 2. Summary of Therapeutic Interventions

A step-by-step overview of the multidisciplinary management strategy.

INTERVENTION PHASE	PROCEDURE / MEDICATION	DETAILED DESCRIPTION & RATIONALE	IMMEDIATE OUTCOME / RESPONSE
1. Acute Pneumothorax Management	**Chest Tube Thoracostomy**	Insertion of a 24-French chest tube into the right 5th intercostal space (mid-axillary line). **Rationale:** To evacuate air from the pleural space, allow for lung re-expansion, and relieve respiratory distress.	• Prompt and full re-expansion of the right lung. • Significant subjective relief of dyspnea and chest pain.
	Observation of Drainage	Within 2 hours of insertion, ~100 mL of dark, non-clotting blood was drained. **Rationale:** This finding was noted and considered highly suggestive of a concurrent catamenial hemothorax.	Confirmed the presence of bleeding from ectopic endometrial tissue, strengthening the diagnosis of TES.
2. Definitive Pleural Treatment	**Chemical Pleurodesis**	Instillation of a slurry containing 500 mg Doxycycline in 50 mL normal saline via the chest tube. **Rationale:** To create an inflammatory reaction leading to adhesion of the visceral and parietal pleura, thereby obliterating the pleural space and preventing pneumothorax recurrence. Chosen as a less invasive alternative to VATS at the patient's request.	• Procedure was well-tolerated with effective pain management. • Chest tube successfully removed 48 hours post-procedure.
	Post-Procedure Monitoring	Patient was monitored for 48 hours post-pleurodesis before chest tube removal. **Rationale:** To ensure no significant air leak recurred and that the lung remained fully expanded.	No residual pneumothorax on follow-up chest radiograph.
3. Systemic Hormonal Therapy	**Initiation of Dienogest**	Oral Dienogest 2 mg administered once daily, initiated concurrently during admission. **Rationale:** To systemically suppress the underlying hormonal driver of the disease. Dienogest induces atrophy of ectopic endometrial tissue and prevents cyclical proliferation and bleeding.	Patient was discharged with instructions to continue this therapy indefinitely.
	Long-Term Follow-up (12 Months)	Regular follow-up in pulmonology and gynecology clinics. **Rationale:** To monitor for treatment efficacy, side effects, and any signs of recurrence.	• **Complete absence of pneumothorax recurrence.** • **Amenorrhea achieved within 2 months.** • **Resolution of both thoracic and pelvic symptoms.** • **Marked improvement in quality of life.**

Given the high recurrence rate of catamenial pneumothorax, a definitive procedure to achieve pleurodesis was deemed necessary. The patient was consulted by the thoracic surgery team. While VATS with talc pleurodesis and diaphragmatic inspection/repair is often considered the gold standard, the patient expressed a strong preference for a less invasive approach if possible. After a thorough discussion of the risks, benefits, and alternatives, a decision was made to perform chemical pleurodesis via the existing chest tube.

Table 3. Follow-up and Outcomes (12-Month Period)

An overview of the patient's progress following the combined therapeutic strategy.

FOLLOW-UP TIMEFRAME	THORACIC OUTCOMES (PULMONOLOGY)	GYNECOLOGICAL / HORMONAL OUTCOMES	OVERALL STATUS & QUALITY OF LIFE
Discharge to 3 Months	• **Pneumothorax:** No recurrence. • **Symptoms:** Mild, intermittent right-sided chest discomfort noted around the time of expected menses. Described as minor and non-limiting.	• **Hormonal Therapy:** Continuous daily use of Dienogest 2 mg. • **Menstruation:** Amenorrhea (cessation of menses) achieved by the end of the second month. • **Pelvic Symptoms:** Gradual reduction in dysmenorrhea-like pelvic discomfort.	Significant improvement. Patient able to resume normal daily activities with increased confidence. Mild thoracic discomfort caused some initial anxiety but did not require medication.
3 to 6 Months	• **Pneumothorax:** No recurrence. • **Symptoms:** Complete resolution of the intermittent chest discomfort. • **Imaging (at 6 months):** Chest radiograph showed clear lung fields with minor pleural thickening, consistent with successful pleurodesis.	• **Hormonal Therapy:** Continued Dienogest 2 mg daily with good tolerance. • **Menstruation:** Sustained amenorrhea. • **Pelvic Symptoms:** Complete resolution of dysmenorrhea and menorrhagia.	Excellent. Full resolution of all cyclical symptoms. Patient reported feeling "back to normal" and was free from the fear of monthly recurrence.
6 to 12 Months	• **Pneumothorax:** No recurrence. • **Symptoms:** Remained completely asymptomatic from a respiratory standpoint.	• **Hormonal Therapy:** Maintained on Dienogest 2 mg daily. No significant side effects reported. • **Menstruation:** Sustained amenorrhea.	Stable and positive. Patient engaged in all life activities without limitation. Expressed high satisfaction with the treatment outcome.
Overall 12-Month Summary	**Complete success in preventing pneumothorax recurrence.**	**Effective suppression of underlying hormonal drivers and resolution of associated pelvic symptoms.**	**Dramatic and sustained improvement in overall health and quality of life.**

On the third day of admission, after ensuring full lung re-expansion and minimal air leak, chemical pleurodesis was performed. The patient was premedicated with intravenous analgesia. A slurry containing 500 mg of doxycycline mixed in 50 mL of normal saline was instilled into the pleural cavity through the chest tube. The tube was clamped, and the patient was rotated through various positions over the next two hours to ensure even distribution of the sclerosant. The chest tube was then unclamped and connected back to a water-seal drainage system. The patient experienced moderate pleuritic pain following the procedure, which was effectively managed with patient-controlled analgesia. The chest tube was removed 48 hours later after a chest radiograph showed continued full lung expansion and no residual pneumothorax.

Concurrently, the gynecological team initiated hormonal suppression therapy. The patient was started on oral progestin (Dienogest) 2 mg once daily. This specific progestin was chosen for its potent endometrial suppressive effects, favorable side-effect profile, and proven efficacy in managing endometriosis-related symptoms. The goal of this therapy was to induce amenorrhea and suppress the hormonal stimulation of any ectopic endometrial tissue, both in the pelvis and the thorax. The patient was discharged on the sixth day of admission with instructions to continue oral progestin indefinitely and to follow up regularly in both the pulmonology and gynecology clinics.

Over the subsequent 12-month follow-up period, the patient remained completely free of pneumothorax recurrence (Table 4). She achieved amenorrhea within

two months of starting oral progestin. While she initially reported some mild, intermittent right-sided chest discomfort around the time of her expected menses for the first three months, these symptoms were minor, did not limit her daily activities, and resolved completely thereafter. Her dysmenorrhea and other pelvic symptoms also resolved. A follow-up chest radiograph at six months showed clear lung fields and evidence of minor pleural thickening, consistent with successful pleurodesis. The patient reported a dramatic improvement in her quality of life and has been able to resume all normal activities without fear of recurrence.

3. Discussion

This case report illustrates a classic presentation of catamenial pneumothorax in a 38-year-old woman, whose diagnosis was secured by the pathognomonic cyclical nature of her symptoms. The successful management underscores the efficacy of a modern, multidisciplinary, and minimally invasive approach.[11] The discussion will focus on the intricate pathophysiology of this condition, linking the proposed theories to the clinical and diagnostic findings in our patient.

The pathophysiology of catamenial pneumothorax represents a fascinating intersection of gynecology, pulmonology, and cellular biology (Figure 1). It is a subject of ongoing research, with several compelling theories that are not mutually exclusive but rather likely act in concert to produce the clinical syndrome.[12] The specific clinical features of our patient—the strict right-sidedness, the perimenstrual timing, the concurrent adenomyosis, and the small hemothorax—provide a valuable framework.

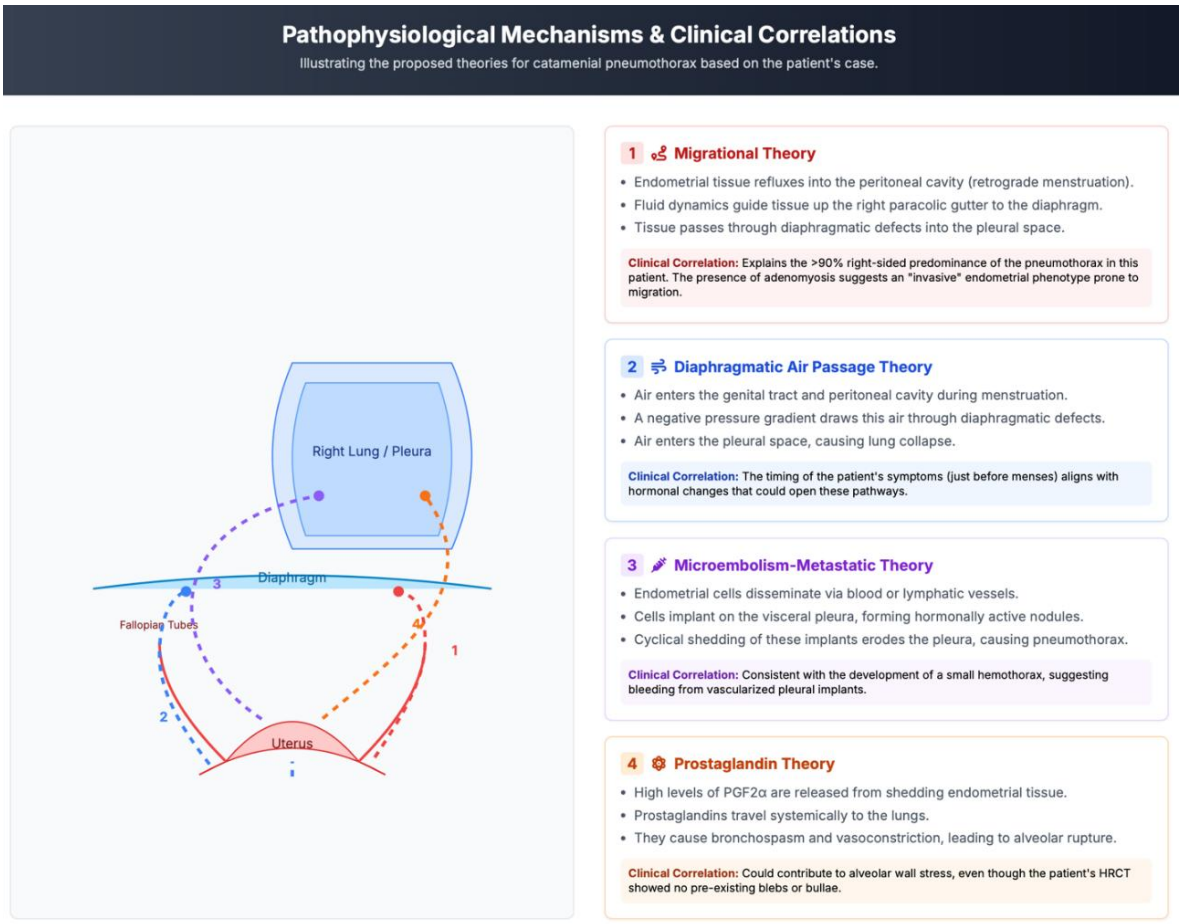

Figure 1. Pathophysiological mechanism of catamenial pneumothorax.

The foundational and most widely accepted explanation for thoracic endometriosis is the migrational theory, first conceptualized by John A. Sampson in the 1920s. This theory proposes a direct anatomical journey of viable endometrial cells from the uterus to the thorax.[13] During menstruation, not all endometrial debris is expelled; a portion is refluxed through the fallopian tubes into the peritoneal cavity. For these cells to establish distant colonies, they must possess remarkable resilience. They must survive the immunologically hostile environment of the peritoneum, adhere to new surfaces, invade tissues, and establish their own blood supply (angiogenesis).

The journey to the thorax is not random. The unique anatomy of the peritoneal cavity dictates the path. Peritoneal fluid, driven by respiratory movements of the diaphragm and intestinal peristalsis, circulates in a predictable clockwise pattern.[14] This flow directs fluid and its cellular contents up the right paracolic gutter—the space lateral to the ascending colon—towards the subphrenic space beneath the right hemidiaphragm. This anatomical preference provides a compelling explanation for the striking right-sided predominance (>90%) of catamenial pneumothorax, a hallmark feature seen in our patient. Once these ectopic endometrial cells reach the diaphragm, they must traverse this formidable musculotendinous barrier. This can occur through two primary means. First, congenital defects, such as persistent pleuroperitoneal canals from embryonic development, can offer a pre-existing conduit. Second, and perhaps more commonly, the endometrial cells themselves can create acquired defects. These ectopic implants, under cyclical hormonal stimulation, can produce matrix metalloproteinases and other enzymes that digest the diaphragmatic collagen and elastin, creating microscopic fenestrations or perforations.

In our patient, the diagnosis of adenomyosis is a crucial piece of this puzzle. Adenomyosis, the invasion of the myometrium by endometrial glands and stroma, signifies an inherently more aggressive or "invasive" endometrial phenotype.[15] This suggests that her endometrial cells may possess enhanced capabilities for migration, invasion, and ectopic survival, making the journey to the thorax more plausible. While her inter-menstrual HRCT did not reveal overt diaphragmatic defects, this is not surprising. Such fenestrations are often only millimeters in diameter, may be covered by fibrin during non-menstrual phases, and are frequently only identifiable during meticulous video-assisted thoracoscopic surgery (VATS), often requiring submersion of the diaphragm in saline to visualize air bubbles passing from the abdomen.[16]

The diaphragmatic air passage theory functions as a direct corollary to the migrational model. While the migrational theory explains how the pathway is created, the air passage theory explains how the pneumothorax itself occurs.[17] It posits that during the perimenstrual phase, the cervical mucus plug, which normally acts as a barrier, thins and disappears due to hormonal shifts. This allows air from the external environment to ascend from the vagina into the uterine cavity and through the fallopian tubes into the peritoneal space, creating a state of benign, transient pneumoperitoneum. This phenomenon is often asymptomatic and radiologically undetectable.

However, in a patient with pre-existing diaphragmatic defects, this intraperitoneal air is now positioned directly beneath a portal to the chest. The thoracic cavity maintains a negative pressure relative to the abdomen, a gradient that is amplified during inspiration. This pressure difference acts as a siphon, drawing the free peritoneal air through the diaphragmatic fenestrations and into the pleural space, leading to the collapse of the lung. The timing of our patient's symptoms, commencing 24-48 hours before the onset of menstrual flow, aligns perfectly with this mechanism. This is the precise window when progesterone levels plummet, triggering the breakdown of both the uterine endometrium and the ectopic endometrial implants on the diaphragm. This tissue breakdown could open up the previously sealed microscopic defects, just as air is potentially

entering the peritoneal cavity, creating a "perfect storm" for pneumothorax development.

While the migrational theory explains the majority of right-sided cases, it does not easily account for the rare instances of left-sided, bilateral, or parenchymal (lung nodule) forms of thoracic endometriosis. The metastatic theory proposes that, akin to cancer cells, benign endometrial cells can invade uterine venules and lymphatic channels during menstruation. These cells can then embolize and travel through the circulatory system, eventually lodging in the capillary beds of distant organs.[18] The lungs, being the first major capillary filter for venous return, are a prime target.

Once these cells have arrested in the pulmonary microvasculature, they can extravasate and implant on the visceral pleura or within the lung parenchyma itself. Here, they form hormonally responsive nodules. Under the influence of estrogen and progesterone, these implants proliferate and, during menstruation, they necrose and shed. When this occurs in an implant on the visceral pleura, it can cause localized inflammation and enzymatic degradation, eroding through the delicate pleural membrane and the wall of an adjacent alveolus. This breach creates a bronchopleural fistula, allowing air to leak from the lung into the pleural space. This theory is strongly supported by the clinical finding of a small hemothorax in our patient. The 100 mL of dark, non-clotting blood drained from her chest tube is characteristic of bleeding from these highly vascularized, hormonally active pleural implants, providing tangible evidence of ectopic endometrial tissue within her thorax.

The prostaglandin theory offers a physiological, rather than anatomical, explanation. During the breakdown of endometrial tissue (both uterine and ectopic), large quantities of prostaglandins, particularly PGF2α, are released. Prostaglandins are potent local hormones; in the uterus, PGF2α causes the intense vasoconstriction and myometrial contractions necessary for menstruation.[19] It is hypothesized that a "spillover" of these prostaglandins into the systemic circulation can have effects on distant organs. When they reach the lungs, they can cause intense bronchospasm of the small airways and vasoconstriction of the pulmonary arterioles. This could lead to localized ischemia and reperfusion injury of the alveolar walls, weakening them and making them susceptible to rupture, especially in the presence of pre-existing architectural abnormalities like blebs or bullae. While our patient's HRCT was normal, this mechanism could still act as a contributing factor or a "second hit," increasing alveolar wall stress and predisposing the lung to rupture from the pressure changes associated with other mechanisms.

In this patient, it is highly improbable that a single mechanism is solely responsible. Rather, a convergence of these theories provides the most comprehensive explanation. The long history of primary infertility and the eventual diagnosis of adenomyosis point to a decades-long process of endometrial dysfunction and aggressive cellular behavior. This likely facilitated the migrational journey of endometrial cells to the right hemidiaphragm, where they established implants and gradually created microscopic fenestrations. Concurrently, the diaphragmatic air passage theory explains the cyclical entry of air into the thorax through these defects. Furthermore, the metastatic theory accounts for the presence of visceral pleural implants, which, upon their cyclical breakdown, caused the observed hemothorax and may have created an independent site of visceral pleural weakness. Finally, surges of prostaglandins could have further compromised the integrity of the alveoli, lowering the threshold for rupture. This synergistic model, driven by the patient's underlying "invasive" endometrial phenotype, explains the eventual clinical manifestation in her late 30s, as the cumulative damage and cyclical insults finally reached a critical threshold.

The diagnosis of catamenial pneumothorax remains a significant clinical challenge, primarily because its definitive signs are often absent or invisible with standard diagnostic tools. As

demonstrated in this case, a high-resolution CT scan performed between menstrual cycles is frequently unrevealing. Ectopic endometrial implants may be too small to resolve, and diaphragmatic defects may be transiently sealed by fibrin or surrounding tissue. Even during VATS, the gold standard for inspection, implants can be subtle, appearing as small reddish-blue spots or simply areas of inflammation, and can be easily missed if the surgeon is not specifically looking for them. This reality underscores the paramount importance of a meticulous clinical history. The cyclical, recurrent nature of the symptoms, locked in time with the menstrual cycle, is the single most powerful diagnostic clue and should be considered virtually pathognomonic until proven otherwise.[20]

The management strategy employed in this case reflects a modern, patient-centered, and evidence-based approach that addresses both the local thoracic problem and the systemic underlying disease. The choice of doxycycline pleurodesis was a key decision. Doxycycline, when instilled into the pleural space, acts as a potent sclerosant. It triggers an intense local inflammatory cascade, recruiting neutrophils and macrophages, which in turn release cytokines and growth factors like TGF-β. This process leads to the proliferation of fibroblasts and the deposition of collagen, effectively creating a fibrous symphysis that fuses the visceral and parietal pleura together, obliterating the potential space and preventing air from re-accumulating. While potentially less effective than mechanical abrasion or talc poudrage via VATS, it is a significantly less invasive procedure that can be performed through an existing chest tube, avoiding the risks and recovery time of general anesthesia and surgery. Its success in this patient, particularly when buttressed by robust hormonal therapy, highlights its value as a frontline definitive treatment.

However, pleurodesis alone is insufficient as it only addresses the consequence (the pneumothorax) and not the cause (the hormonally active ectopic endometrium). The cornerstone of preventing recurrence is systemic hormonal suppression. The choice of Dienogest represents a targeted pharmacological intervention. As a fourth-generation oral progestin, Dienogest has a unique pharmacological profile perfectly suited for this condition. It exerts a powerful progestogenic effect on endometrial tissue, leading to a process called decidualization, followed by atrophy of both the uterine and ectopic implants. Crucially, its efficacy extends beyond simple suppression. Dienogest has demonstrated significant anti-proliferative effects by inhibiting DNA synthesis in endometrial cells, anti-inflammatory properties by reducing the production of prostaglandins and cytokines within the implants, and anti-angiogenic effects by inhibiting the formation of new blood vessels that are essential for the survival and growth of ectopic tissue. Furthermore, it effectively suppresses ovulation and moderately reduces systemic estrogen levels without inducing the profound, often poorly tolerated, hypoestrogenic state associated with GnRH agonists (which cause severe menopausal symptoms like hot flashes and bone density loss). The complete resolution of our patient's thoracic and pelvic symptoms, and the sustained absence of pneumothorax recurrence over a 12-month period, provide strong clinical evidence for the efficacy of Dienogest in controlling the entire disease process. The success of this case was fundamentally dependent on the seamless multidisciplinary collaboration: the pulmonology team stabilized the acute respiratory compromise, the thoracic surgery team provided the definitive local therapy, and the gynecology team diagnosed and managed the underlying systemic hormonal disorder.

4. Conclusion

This case of catamenial pneumothorax in a patient with adenomyosis highlights the complex interplay between pelvic and thoracic pathology driven by ectopic endometrial tissue. The diagnosis hinges on recognizing the pathognomonic temporal relationship between recurrent pneumothorax and the menstrual cycle, a clinical clue that should prompt a high index of suspicion in any woman of reproductive age. Our

experience demonstrates that a successful long-term outcome can be achieved with a carefully coordinated, multidisciplinary approach. The combination of a minimally invasive definitive pleural procedure, such as chemical pleurodesis, with effective and well-tolerated hormonal suppression therapy, like continuous Dienogest, provides a powerful dual strategy to both manage the acute thoracic event and control the underlying systemic disease, ultimately preventing recurrence and significantly improving the patient's quality of life.

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