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Case Report

Pulmonary Alveolar Microlithiasis-like Process in Pregnancy with Concurrent Pneumothorax: A Case Report

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ABSTRACT

Background: Pulmonary alveolar microlithiasis (PAM) is a rare autosomal recessive lung disease caused by biallelic pathogenic mutations in the SLC34A2 gene, leading to calcium phosphate microlith accumulation in alveoli. **Methods:** We report a case of suspected PAM-like disease during pregnancy with postpartum histopathologic and genetic evaluation. **Results:** A 26-year-old pregnant woman presented with spontaneous left-sided pneumothorax at 16 weeks' gestation. Imaging revealed bilateral reticulonodular opacities with a characteristic sandstorm appearance. High-resolution CT confirmed widespread intra-alveolar calcifications. Lung biopsy showed intra-alveolar ossification with focal marrow elements and hemorrhage, without classic laminated microliths. Genetic testing identified a heterozygous missense SLC34A2 mutation (c.1110A>G; p.Ile370Met), previously reported in patients with PAM. Although a second pathogenic allele was not identified, the genetic findings were considered supportive but not diagnostic. **Conclusions:** This case highlights spontaneous pneumothorax as a rare initial presentation of a PAM-like phenotype, particularly in pregnancy. It underscores the diagnostic challenges posed by atypical histology and incomplete molecular confirmation.

1. Introduction

Pulmonary alveolar microlithiasis (PAM) is a rare, autosomal recessive lung disorder characterized by the widespread accumulation of calcium phosphate microliths within the alveolar spaces. This progressive condition is most attributed to inactivating mutations in the SLC34A2 gene, which encodes the type IIb sodium-phosphate cotransporter [1]. Over time, these deposits disrupt normal lung architecture, resulting in restrictive lung disease, reduced gas exchange, and, in advanced stages, pulmonary hypertension and respiratory failure [2]. Although classically described as an autosomal recessive disorder, PAM demonstrates substantial clinical, radiologic, and histopathologic heterogeneity, and atypical presentations and incomplete molecular confirmation have been reported.

The intersection of PAM and pregnancy is exceedingly rare and not well characterized in the literature [3]. Physiological changes during pregnancy, including increased oxygen demand, altered lung mechanics, and immune modulation, can exacerbate pre-existing pulmonary conditions or unmask underlying pathology. However, the clinical implications of PAM during gestation, for both mother and fetus, remain poorly understood [4].

The few available reports describe maternal ages ranging from 21 to 37 years, with most cases occurring sporadically and only occasional reports of familial disease. Diagnosis has been made before pregnancy, during gestation, or shortly after delivery, and pulmonary function has ranged from near-normal in asymptomatic individuals to severe restrictive impairment. Some patients remained stable throughout pregnancy, while others developed respiratory symptoms in the late second or third trimester. Delivery was most commonly by cesarean section, often preterm, although successful full-term vaginal delivery has also been described. Reported outcomes have varied widely, from stable maternal disease to progressive respiratory deterioration and even postpartum mortality, reflecting the unpredictable clinical course of PAM in pregnancy [4].

Despite these reports, important knowledge gaps remain. In particular, there is limited information regarding the presentation of PAM early in pregnancy, the occurrence of spontaneous pneumothorax as an initial manifestation during gestation, and the diagnostic challenges posed by atypical histopathologic findings and incomplete molecular confirmation.

In this report, we describe a rare and diagnostically informative case of a 26-year-old woman who presented with a spontaneous

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pneumothorax at 16 weeks' gestation associated with atypical histopathologic findings and a heterozygous SLC34A2 variant identified in the postpartum period. This case underscores the importance of considering uncommon genetic lung diseases in the differential diagnosis of pneumothorax in young patients. It also highlights the diagnostic uncertainty that can arise from non-classical pathology and incomplete molecular findings, as well as the challenges of managing rare diffuse lung disease in pregnancy.

2. Case Presentation

A 26-year-old woman at 16 weeks of gestation, with no significant past medical or surgical history, presented to the Emergency Department (ED) with a four-day history of left-sided pleuritic chest pain, associated with dyspnoea and palpitations. She denied any history of trauma, cough, hemoptysis, or wheezing. She was a non-smoker with no history of alcohol or illicit substance use. There was a significant family history of multiple myeloma in her mother.

On presentation, her vital signs were unremarkable, maintaining oxygen saturation at 100% on room air. Physical examination revealed decreased air entry on the left side of the chest. Cardiovascular examination was unremarkable with normal heart sounds. Abdominal examination was benign, and there were no clinical signs of deep vein thrombosis in the lower limbs.

A chest radiograph done in the ED demonstrated a large left-sided pneumothorax, measuring 8.3 cm from the apex, with associated flattening of the left hemidiaphragm and contralateral mediastinal shift (**Figure 1**). Routine blood tests, including a complete blood count, renal profile, and serum glucose, were within normal limits.

A 12 Fr chest drain was inserted into the left hemithorax using the Seldinger technique. After 48 hours, the chest drain was removed with no complications. Repeat chest radiographs following drainage confirmed resolution of the pneumothorax. However, bilateral reticular markings were noted, prompting further outpatient evaluation (**Figure 2**).

High-resolution computed tomography (HRCT) of the chest revealed multifocal nodular and branching calcifications with basal predominance. A cluster of cystic airspaces in the posterior segment of the right upper lobe was also noted. There was absence of interstitial fibrosis, ground-glass opacities, or additional cysts (**Figure 3**).

Serum calcium, phosphate, renal function, and parathyroid hormone levels were normal, arguing against metastatic pulmonary calcification. There was no clinical or radiologic evidence of chronic kidney disease, granulomatous infection, or occupational exposure.

The patient experienced no recurrence of pneumothorax and remained clinically stable throughout the remainder of pregnancy, with routine obstetric monitoring demonstrating no fetal compromise. She was evaluated in the obstetric clinic every 4 weeks up to the 30th week of gestation, and thereafter every two weeks. She was also evaluated at the respiratory clinic closely up to delivery.

Given the atypical presentation and pregnancy status, multidisciplinary discussion was undertaken. After careful consideration of maternal and fetal risks, a video-assisted thoracoscopic (VATS) lung biopsy of the right middle lobe was suggested weeks following childbirth to minimize any potential risks.

At 36+5 weeks, obstetric ultrasonography demonstrated a positive fetal heartbeat but reduced amniotic fluid volume, consistent with oligohydramnios. The estimated fetal weight was 2,478 g, below the 50th percentile, indicating a small-for-gestational-age fetus. Umbilical artery Doppler findings were normal. The obstetric team decided to proceed with an elective cesarean section at 37+5 weeks, and the patient was hospitalized pending delivery. The cesarean section was performed under spinal anesthesia. A male infant weighing 2,850 g was delivered. The Apgar scores were 9 at 1 minute and 9 at 5 minutes. To date, the child has shown no signs of respiratory distress or other pulmonary pathology.

Postpartum histopathological analysis of the VATS biopsy demonstrated fresh intra-alveolar hemorrhage, very focal anthracosis at the periphery, and several smaller fragments of woven bone containing marrow spaces, all intimately associated with the alveolar structures. These features were consistent with osseous metaplasia. Although classic laminated intra-alveolar microliths were not identified, the presence of intra-alveolar ossification closely associated with alveolar structures, in combination with the characteristic radiologic pattern, was considered supportive of a PAM-like process. Alternative diagnoses, including diffuse pulmonary ossification, were considered but felt less likely given the imaging distribution and clinical context (**Figure 4**).

After appropriate genetic counseling, genetic testing was performed postpartum, revealing a heterozygous missense mutation in the *SLC34A2* gene (c.1110A>G; p.Ile370Met), resulting in an isoleucine-to-methionine substitution at position 370. While this variant has been reported as pathogenic in association with autosomal recessive PAM, no second pathogenic allele or copy number variation was detected. Therefore, molecular confirmation of PAM remains incomplete and best interpreted as contributory but not definitively confirmatory. At follow-up, the patient remains stable from a respiratory standpoint with no deterioration compared to previous months (**Figure 5**). She is not on any regular medications and reports no dyspnoea at rest. She can carry out routine activities of daily living independently but continues to experience exertional shortness of breath when walking uphill or climbing two flights of stairs, which is unchanged from her previous baseline. Annual CT thorax imaging demonstrates stable findings with no radiological evidence of progression. Physiologically, there has been an apparent improvement in pulmonary function, with FVC increasing from 1.64 L (43%) recorded in the late third trimester in 2022 to 2.59 L (68%) in 2024. A diffusing capacity of the lungs for carbon monoxide (DLCO) performed in 2024 showed DLCO of 54% and diffusing capacity per liter of lung volume (DLCO/VA) of 84%, suggesting reduced overall gas transfer with relatively preserved transfer per unit lung volume. Her oxygen saturation is 98% on room air, and there are no clinical signs of right-sided heart failure. She has received routine vaccinations against pneumococcus and influenza.

3. Discussion

PAM is a rare, inherited lung disease caused by inactivating mutations in the SLC34A2 gene, which is characterized by progressive intra-alveolar deposition of calcium phosphate due to impaired phosphate transport by alveolar type II pneumocytes [1]. Although PAM is classically inherited in an autosomal recessive manner, the disease demonstrates clinical, radiological, and histopathological heterogeneity.

Spontaneous pneumothorax is an uncommon presentation of PAM, reported in 1.6% of patients in one large review, and it is thought to result from rupture of subpleural cysts [1]. Presentation during pregnancy is particularly rare, with fewer than ten cases reported in the literature to date, limiting evidence-based guidance for diagnosis and management [4].

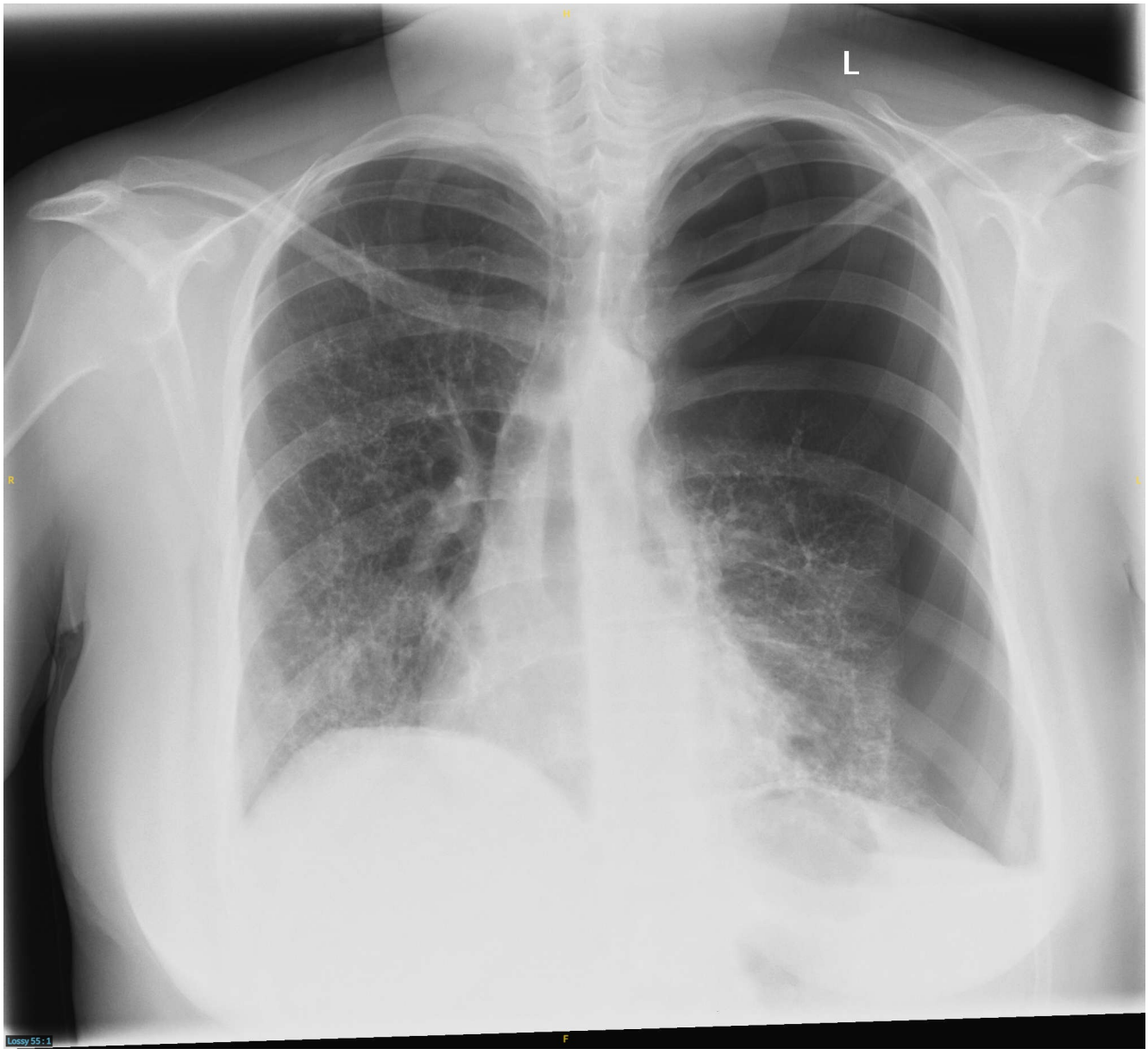

Figure 1: A posteroanterior chest X-ray showing a large left-sided pneumothorax with evidence of mediastinal shift. L, left.

A broad differential diagnosis for diffuse pulmonary calcifications was systematically considered. Metastatic pulmonary calcification, seen in end-stage renal disease or hyperparathyroidism, and milk-alkali syndrome were considered unlikely in the setting of normal renal function and normal serum calcium and phosphate levels. Talcosis and other inhalation or embolization of contrast agents were excluded based on the absence of occupational exposure, intravenous drug use, or prior contrast embolization. Amiodarone toxicity was ruled out given no history of exposure. There were no radiological or occupational findings to suggest pneumoconioses such as silicosis. Granulomatous diseases such as sarcoidosis and histoplasmosis were not supported clinically or radiologically, with no typical distinct features suggesting these underlying diseases. Pulmonary hemosiderosis and healed viral pneumonias were also considered less likely given the absence of recurrent hemoptysis, anemia, or compatible imaging findings [5–7].

Diagnosis is often straightforward with classic imaging features, especially when supported by a positive family history [2, 8]. Nevertheless, lung biopsy continues to be used in practice, possibly reflecting unfamiliarity with the disease. Biopsy should generally be reserved for ambiguous cases where non-invasive diagnostics are inconclusive [1]. A notable aspect of the present case is the atypical histopathologic pattern. Although the hallmark histological feature of PAM is the presence of concentric, lamellated calcium phosphate microliths within alveolar spaces, our patient's biopsy demonstrated an atypical histopathological pattern, including osseous metaplasia with marrow elements and fresh intra-alveolar hemorrhage. Such findings have been reported only sporadically and may represent advanced or reactive remodeling related to chronic microlith deposition rather than a distinct pathological entity. Although the biopsy demonstrated ossification with marrow elements rather than classic laminated microliths, several features favored a PAM-like process over diffuse pulmonary ossification. Radiologically, the

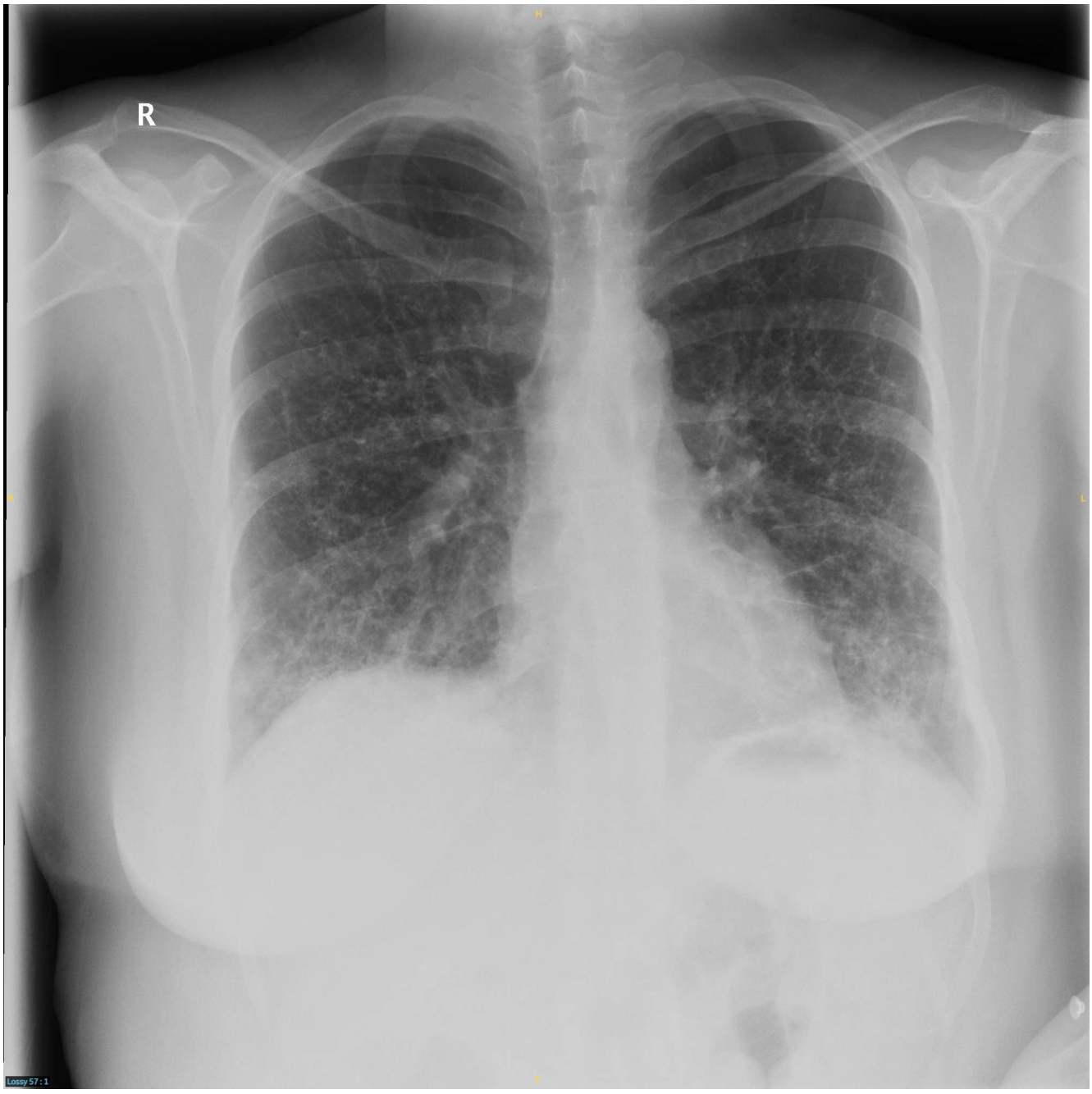

Figure 2: A posteroanterior chest X-ray showing bilateral, sand-like micronodules of calcific density with a sandstorm appearance predominantly in the mid to lower lung zones. R, right.

patient exhibited diffuse bilateral sand-like calcific micronodules, calcified interlobular septa, and subpleural linear calcifications with basal predominance, producing the characteristic sandstorm appearance typically described in PAM. In contrast, diffuse pulmonary ossification more commonly manifests as branching dendriform ossifications, often associated with chronic interstitial lung disease, chronic aspiration, or cardiac disease. Clinically, our patient lacked known predisposing conditions for diffuse pulmonary ossification and had normal serum calcium, phosphate, renal function, and parathyroid hormone levels. Furthermore, the identification of a heterozygous SLC34A2 variant, although insufficient for molecular confirmation, provided supportive evidence for disordered alveolar phosphate handling. We therefore considered the biopsy findings to represent an atypical ossification-dominant manifestation within a PAM-like clinicoradiologic phenotype rather than isolated diffuse pulmonary ossification.

Genetic testing for SLC34A2 mutations is now commercially available, although there is no known correlation between genotype and clinical severity [9]. In our patient, genetic testing identified a heterozygous missense SLC34A2 variant (c.1110A>G; p.Ile370Met), previously reported as pathogenic in association with pulmonary alveolar microlithiasis. While biallelic pathogenic variants provide the strongest molecular confirmation of this autosomal recessive disorder, an increasing number of reports describe patients with

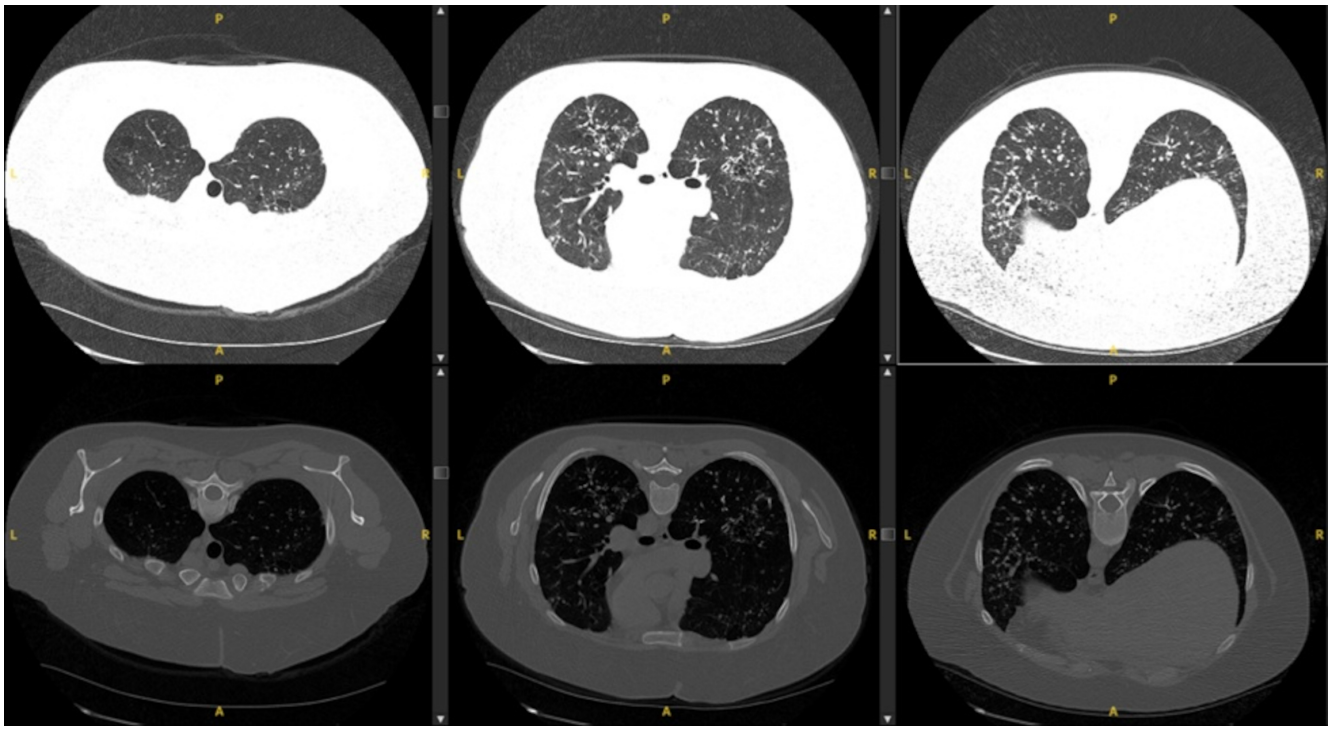

Figure 3: Upper row: Axial high-resolution CT thorax images as viewed in the lung window setting, showing calcified interlobular septa with widespread bilateral intra-alveolar deposition of sand-like calcifications throughout the lungs, predominantly in the lung bases. There is evidence of a cluster of small subpleural cysts in the right upper lobe. Lower row: Axial high-resolution CT thorax images as viewed in the bone window setting, showing fine, diffuse, basilar-predominant micronodular opacities and subpleural linear calcifications. L, left; R, right; A, anterior; P, posterior.

clinicoradiologic and histopathologic features consistent with PAM in whom only a single heterozygous SLC34A2 variant was identified [10–14]. Proposed explanations include incomplete detection of a second allele due to deep intronic or regulatory region variants, copy number variation, epigenetic mechanisms, or haploinsufficiency with modifying environmental or physiological factors [12–14]. These observations suggest that monoallelic SLC34A2 variants may contribute to disease expression in selected cases rather than representing incidental findings alone. In this context, the variant identified in our patient is best interpreted as contributory but not definitively confirmatory.

Diagnosis and management of suspected PAM during pregnancy require careful multidisciplinary collaboration. Imaging decisions must balance diagnostic yield against fetal radiation exposure, with high-resolution CT reserved for situations in which results are likely to influence management. Pneumothorax during pregnancy carries additional risks, including recurrence and compromised maternal ventilation, and may necessitate early intervention [14, 15]. In the present case, conservative management with intercostal chest drainage was effective, and pregnancy progressed without respiratory deterioration or obstetric complications.

In expectant mothers with PAM, respiratory function commonly deteriorates during the third trimester of pregnancy, attributing this deterioration to the upward displacement of the diaphragm secondary to uterine enlargement, resulting in reduced lung volumes and a restrictive ventilatory pattern [3]. In our patient, pulmonary function tests demonstrated a forced vital capacity (FVC) of 1.64 L (43% predicted) in 2022, which improved to 2.59 L (68% predicted) in 2024.

Being an orphan lung disease, management protocols for expecting mothers are uncertain. Historical data often cite premature cesarean sections; however, our patient successfully delivered a healthy, full-term infant via cesarean section at 37 weeks. This aligns with other reported cases where cesarean delivery was typically indicated by obstetric factors rather than the progression of PAM itself. Notably, all fetuses in the reviewed literature were born alive with appropriate weight for their gestational age, just like our patient [3, 16–21]. While severe outcomes have been reported elsewhere, including a case of maternal death due to restrictive disease four months postpartum, our findings suggest that PAM is not an absolute indication for abortion or the premature termination of pregnancy [22]. With careful monitoring, a typical disease evolution can still result in a successful, full-term maternal and fetal outcome (**Table 1**).

This case report is strengthened by its description of a rare presentation of suspected pulmonary alveolar microlithiasis during pregnancy, initially manifesting as spontaneous pneumothorax. It demonstrates a systematic diagnostic approach, including detailed imaging, exclusion of alternative causes of diffuse pulmonary calcification, histopathologic evaluation, and genetic testing identifying a heterozygous SLC34A2 variant. Multidisciplinary management and longitudinal maternal and fetal follow-up further enhance its clinical relevance. However, the report is limited by incomplete molecular confirmation, as only a single pathogenic allele was identified in a classically autosomal recessive condition, and by atypical histopathology lacking classic laminated microliths, which introduces diagnostic uncertainty.

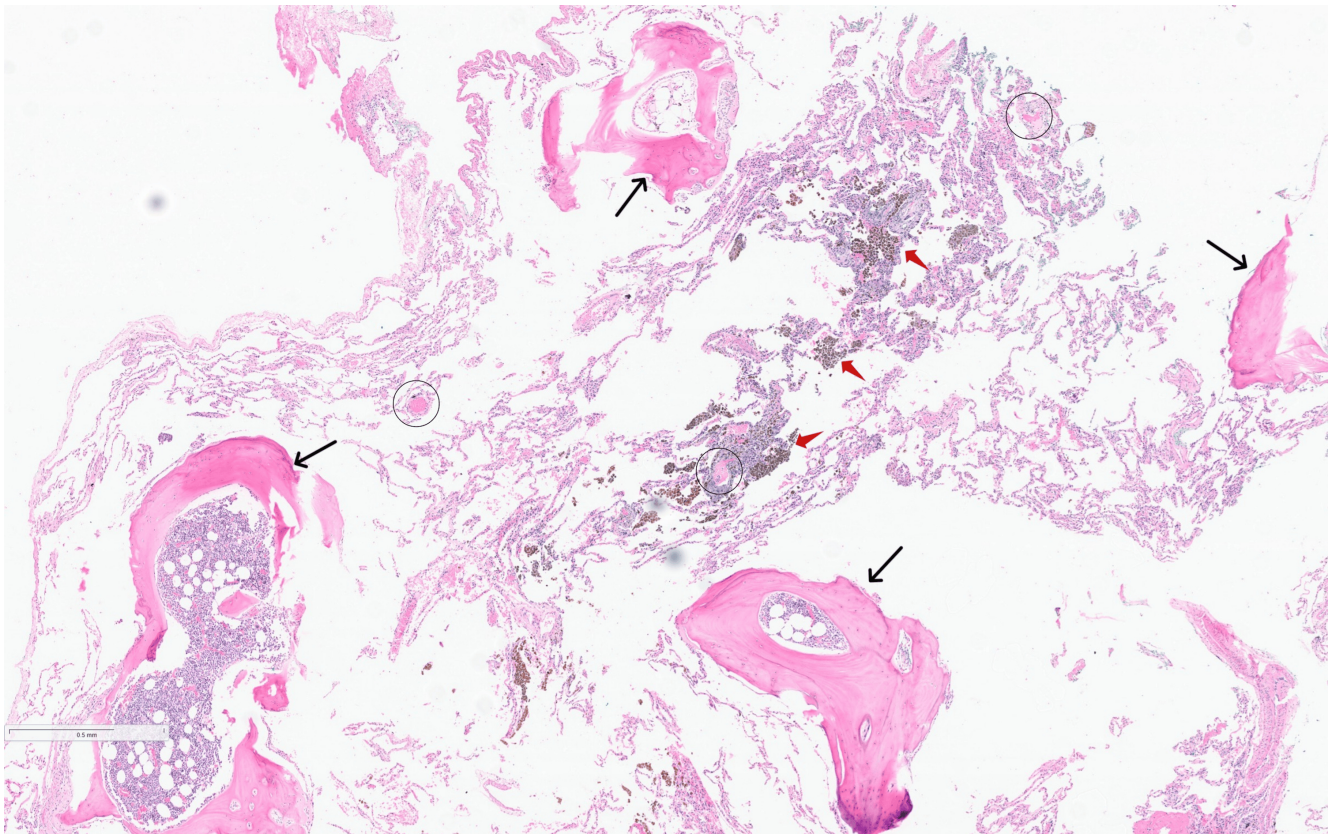

Figure 4: Histopathology slide showing multiple pulmonary ossifications. Notice the presence of lamellar bone containing fat and hematopoietic elements (black arrows), as well as very focal anthracosis at the periphery (red arrows) and the presence of fresh intra-alveolar hemorrhages (black circles). (hematoxylin and eosin staining; low-power magnification; 40× objective).

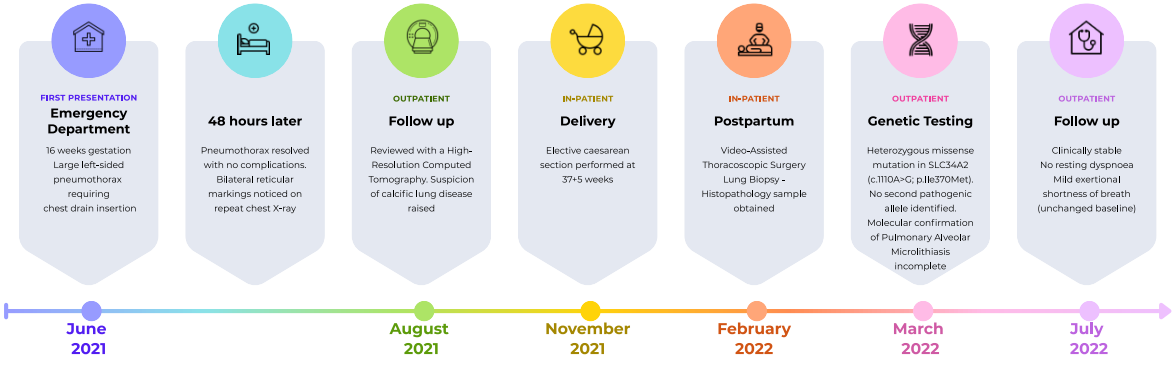

Figure 5: A timeline showing the sequence of how the events unfolded.

Table 1: Summary of reported cases and their outcomes.

Author	Age (years)	Diagnosis Timing	Pulmonary Function	Symptom Onset During Pregnancy	Delivery	Outcome
Abba [17]	37	Known PAM, family history	FEV_1 3.6 L (90% predicted), FVC 3.8 L, DLCO 82% predicted	Asymptomatic during pregnancy	Cesarean section at approximately 8 months due to placenta previa	Favorable maternal outcome
Rodríguez et al. [22]	36	Diagnosed during unrecognized pregnancy	Not reported	Became symptomatic at 34 weeks	Cesarean section at 38 weeks	Maternal death 4 months postpartum due to progressive respiratory failure
Souza Filho et al. [18]	26	Diagnosed 10 months before pregnancy	Severe restriction (FVC 25.9% predicted, FEV_1 13.9% predicted)	Worsening at 28 weeks	Cesarean section at 32 weeks	Maternal survival
Erdem et al. [19]	36	Diagnosed 4 years before pregnancy	Not reported	Worsening during second–third trimester	Cesarean section at 31 weeks due to preterm labor	Maternal survival
Sethy et al. [20]	27	Diagnosed 1 week postpartum	Mild restriction, DLCO 48% predicted	Dyspnoea during second trimester	Vaginal delivery at term	Favorable maternal outcome
Aktürk et al. [16]	37	Diagnosed 5 years before pregnancy	Not reported	Symptomatic at approximately 35 weeks	Preterm cesarean section	Maternal survival
Özel et al. [3]	21	Known PAM before pregnancy	FEV_1 2.98 L (predicted 4.12 L), FVC 3.42 L	Symptoms after 35 weeks	Cesarean section at 38 weeks after failed induction	Favorable maternal and neonatal outcome
Figueiredo et al. [21]	32	Known PAM before pregnancy	Not reported	Asymptomatic during pregnancy	Vaginal delivery at term	Favorable maternal and neonatal outcome
Borg Axisa et al. (Our case)	26	Suspected during pregnancy and further evaluated postpartum	FVC 1.64 L (43%)	Symptoms at 16 weeks	Cesarean section at 37+5 weeks	Favorable maternal and neonatal outcome

Abbreviations: DLCO, diffusing capacity of the lung for carbon monoxide; FEV_1, forced expiratory volume in 1 second; FVC, forced vital capacity; PAM, pulmonary alveolar microlithiasis.

4. Conclusion

This case describes a clinical and radiological PAM-like phenotype presenting with a spontaneous pneumothorax in pregnancy. Although imaging features were highly suggestive, atypical histopathology and incomplete molecular confirmation necessitate diagnostic caution. The patient remained clinically stable throughout pregnancy, with preserved oxygenation on follow-up. This case report underscores the importance of multidisciplinary evaluation, cautious interpretation of genetic findings, and longitudinal follow-up when rare lung diseases are encountered during pregnancy.

Conflicts of Interest

The authors declare no competing interests that could have influenced the objectivity or outcome of this research.

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None.

Ethics Approval

Formal ethics committee or institutional review board review was waived because this manuscript describes a single case report and does not constitute research involving human subjects.

Informed Consent

Written informed consent was obtained from the patient for publication of this case report, including accompanying clinical information and radiologic and histopathologic images.

Large Language Model

None.

Author Contributions

The original draft was written by ABA and JZ. Review and editing were performed by LC. Supervision was provided by CZ.

Data Availability

The data supporting the findings of this case report are available from the corresponding author upon reasonable request. The data are not publicly available because they contain information that could compromise patient privacy and confidentiality.

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