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CASE REPORT
ΕΝΔΙΑΦΕΡΟΥΣΑ ΠΕΡΙΠΤΩΣΗ

Congenital lobar emphysema in the neonatal period
A case report of diagnostic and management challenges

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ΑΡΧΕΙΑ ΕΛΛΗΝΙΚΗΣ ΙΑΤΡΙΚΗΣ 2026, 43(Συμπλ 1):132–137

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Συγγενές λοβιακό εμφύσημα στη νεογνική περίοδο: Αναφορά περίπτωσης με διαγνωστικές και διαχειριστικές προκλήσεις

Περίληψη στο τέλος του άρθρου

We describe the diagnostic challenges, surgical management strategies, and postoperative complications in an infant with congenital lobar emphysema (CLE). A 3-month-old female infant was brought to the hospital with progressive tachypnea, chest retractions, cyanosis, and feeding difficulties that resulted in failure to thrive. Initially, the patient was diagnosed with pneumonia and received intravenous antibiotics. Subsequently, chest radiography was misinterpreted as pneumothorax, leading to chest tube placement without clinical improvement. Further evaluation with chest computed tomography (CT) revealed hyperinflation of the left upper lobe, mediastinal shift, and compression of the surrounding lung tissue, establishing the diagnosis of CLE. The patient underwent left upper lobectomy, which immediately improved oxygen saturation. Postoperatively, the patient developed ventilator-associated pneumonia, sepsis caused by multidrug-resistant *Acinetobacter baumannii*, and persistent malnutrition, which were managed with last-line antibiotics, high-calorie enteral nutrition, and chest physiotherapy. In conclusion, this case highlights the importance of clinical vigilance for CLE in neonates with respiratory distress unresponsive to conventional therapy. Lobectomy is effective as a definitive treatment; however, optimal recovery requires multidisciplinary management, particularly infection prevention and nutritional rehabilitation.

Key words

Congenital lobar emphysema
Infant newborn
Lobectomy
Malnutrition
Nosocomial infections
Respiratory distress syndrome newborn

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Respiratory distress in neonates is always a serious clinical issue as it can lead to hypoxia, respiratory failure and ultimately death.[1] There is a variety of potential causes of respiratory distress in neonates from pneumonia and pneumothorax to congenital pulmonary malformation.[2] Among congenital conditions, congenital lobar emphysema (CLE) is of particular interest because it is often misdiagnosed as another pulmonary process.[3,4] CLE is a rare entity at 1 in 20,000–30,000 live births.[5] CLE is characterized by partial bronchial obstruction with one-way valve properties.[5] Usually, the obstruction will allow air to enter in inspiration but will not allow expired air to leave, leading to abnormal hyperinflation of the involved lobe, compression of adjacent lung, and shifting of the mediastinum to the opposite side.[6,7] The left upper lobe is the most common location affected, followed by the right middle lobe and right upper lobe.[5,8]

Clinically, CLE can present with a broad range of features from asymptomatic neonates to significant respiratory distress.[9,10] The radiological appearance of CLE can resemble pneumothorax or pneumonia, which can result in situations when CLE is overlooked or diagnosed incorrectly, and some other procedure is performed, most commonly chest tube placement.[4,11,12] Chest computed tomography (CT) remains the most sensitive modality to definitively answer the question by showing hyperinflation of the lobe along with the subsequent compressive mechanisms.[5,13] Few cases can be monitored conservatively; however, lobectomy is the definitive treatment in the setting of significant clinical distress.[14–16] In general, lobectomy tends to be associated with improved respiratory function but the postoperative course is frequently complicated by hospital-acquired infection, dependency upon the vent, and complications due to nutritional issues.[17,18] Herein, we present a case report to highlight the clinical progression of an infant with CLE including the diagnostic challenges, surgical management and post-operative complications, while also highlighting

the value of a multidisciplinary approach to maximize long-term outcomes.[19,20]

CASE PRESENTATION

A female infant was brought to the hospital with progressive tachypnea, chest retractions, cyanosis, and feeding difficulties leading to failure to thrive. She was born at term via cesarean section due to prolonged second stage of labor, with a birth weight of 2,800 g. The neonatal adaptation was uneventful, with no signs of respiratory distress or cyanosis. The pregnancy history was normal, without intrauterine infection or congenital abnormalities detected during antenatal examinations. The infant was formula-fed from birth due to feeding difficulties, although initial growth was adequate. At the age of two months, the patient began to exhibit persistent tachypnea, intermittent cyanosis, chest retractions, and feeding intolerance. These symptoms progressed to impaired growth, as her weight failed to increase in accordance with the growth curve. She was subsequently taken to a secondary hospital, where she was diagnosed with pneumonia or pneumothorax. Chest radiography demonstrated hyperlucency of the left hemithorax, which was misinterpreted as pneumothorax, leading to chest tube insertion. However, the patient's condition did not improve, and respiratory distress persisted.

After discharge, the patient experienced deterioration and required rehospitalization with intravenous antibiotics and oxygen therapy, but without significant improvement. Due to the suspicion of a congenital pulmonary anomaly, the patient was eventually referred to our hospital. Upon arrival, she was in severe respiratory distress with an oxygen saturation of only 30% on room air, necessitating immediate intubation and mechanical ventilation. Physical examination revealed decreased breath sounds on the left hemithorax and central cyanosis. Chest radiography demonstrated hyperinflation of the left upper lobe with mediastinal shift to the right (fig. 1A), and thoracic CT scan subsequently confirmed the diagnosis of CLE in the left upper lobe with compression of the adjacent lung tissue (tab. 1).

Additional investigations revealed the following results: Arterial blood gas analysis showed a pCO_2 of 56 mmHg and a pH of 7.37, indicating compensated respiratory acidosis; hemoglobin level was 10.1 g/dL with neutrophilic leukocytosis; and blood culture grew multidrug-resistant *Acinetobacter baumannii*. Due to persistent hypoxia despite maximal ventilatory support, progressive mediastinal shift, and failure of conservative therapy, the medical team decided to perform a left upper lobectomy.

Following the lobectomy, the patient demonstrated marked improvement in oxygenation, with oxygen saturation increasing to 95–98% despite continued mechanical ventilation at lower pressure settings. The mediastinal shift was reduced after surgery, and serial chest radiographs showed gradual re-expansion of the contralateral lung, confirming the effectiveness of the intervention. Nevertheless, the postoperative course was complicated by persistent ventilatory dependence, recurrent pneumonia, and nosocomial infections, which prolonged hospitalization and required comprehensive multidisciplinary management. A comparison of the patient's preoperative, postoperative, and follow-up clinical conditions is summarized in table 2, demonstrating significant improvements in oxygenation and ventilatory parameters, while also highlighting the ongoing challenges related to recurrent infections and nutritional deficiencies.

Despite improvement in oxygen saturation, the patient continued to experience oxygen dependency during the follow-up phase, requiring high flow nasal cannula (HFNC) and a tracheal mask due to recurrent pneumonia. Follow-up radiological examinations demonstrated improvement in mediastinal deviation and re-expansion of the contralateral lung, although residual pulmonary abnormalities were still evident (fig. 1B). The patient also underwent regular chest physiotherapy to support compensatory lung growth, improve respiratory function, and reduce the risk of recurrent infections.

COMMENTS

This case report describes a 3-month-old female neonate with CLE who was initially misdiagnosed as having pneumothorax, leading to an inappropriate chest tube insertion before the correct diagnosis was established through CT scan and subsequently managed with lobectomy.[3,4] The main findings of this case highlight the importance of early imaging-based diagnosis and the fact that the postoperative course is not always straightforward, as the patient faced challenges of multidrug-resistant nosocomial infection and malnutrition.[8,17,21] Previous studies have also emphasized that misdiagnosis of CLE as pneumothorax is a common issue that can worsen patient outcomes.[4,9] Furthermore, other research has reported that lobectomy remains the definitive therapy with a high success rate, although postoperative challenges are often related to infectious complications and nutritional recovery.[15,16] The direct reason for these findings is the limited clinical experience in recognizing this rare condition, whereas the indirect reason lies in the limited radiology facilities available in secondary hospitals.[20] Therefore, this case underscores that while lobectomy is effective in improving ventilation in CLE, long-term recovery requires a multidisciplinary approach.[18,19]

In this case, the patient presented with persistent respiratory distress, severe hypoxia, and ventilator dependence that did not improve despite maximal supportive therapy. Radiological evaluation revealed extensive hyperinflation of the left upper lobe with significant compression of the contralateral lung and mediastinal structures, posing a serious risk of ventilatory failure, prolonged hypoxia, and hemodynamic instability.[6,10] This progressive condition

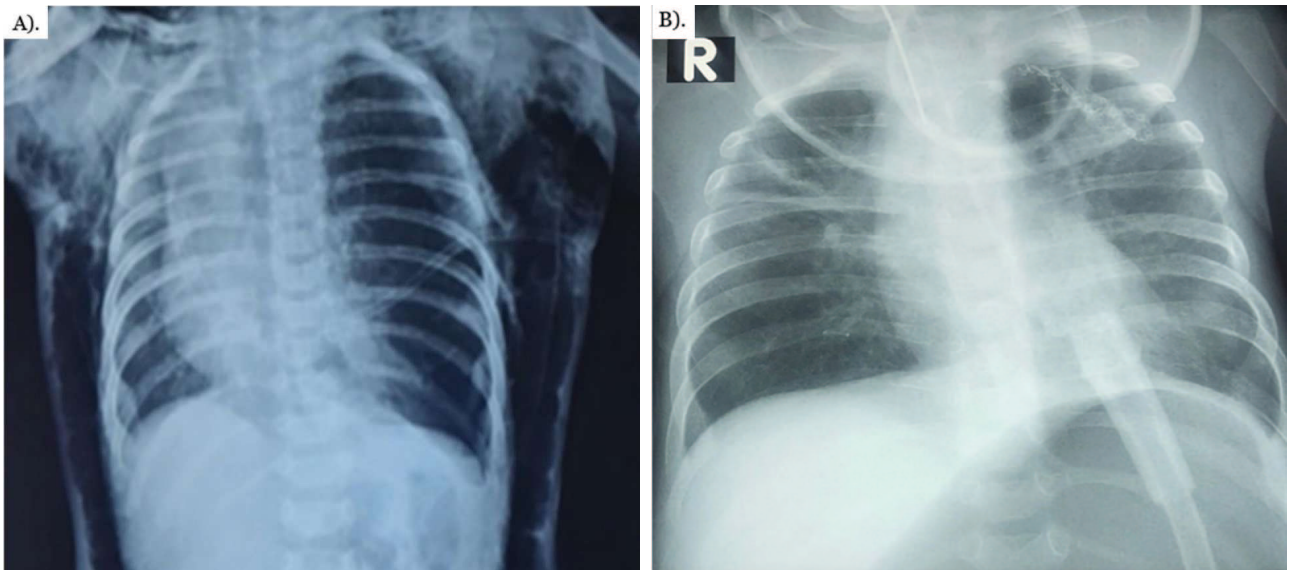

Figure 1. Radiological chest X-ray findings in a neonate with congenital lobar emphysema (CLE). **(A)** Preoperative chest X-ray showing hyperinflation of the left upper lobe with mediastinal shift to the right, consistent with typical CLE findings. **(B)** Postoperative chest X-ray following left upper lobectomy, demonstrating improved expansion of the contralateral lung and partial correction of mediastinal shift.

Table 1. Summary of diagnostic workup.

Investigation	Findings
Chest X-ray	Hyperinflation of the left upper lobe with contralateral mediastinal shift; suggestive of pneumonia
CT scan (thorax)	Confirmation of congenital lobar emphysema (CLE) in the left upper lobe; mediastinal shift; compression of adjacent structures
Blood culture	Growth of pandrug-resistant *Acinetobacter baumannii*, resistant to all tested antibiotics except tigecycline
Arterial blood gas (ABG)	pH: 7.37 (normal), pCO_2: 56 mmHg (elevated), HCO_3^-: 32.7 mmoL/L (compensated respiratory acidosis)
Complete blood count (CBC)	Hemoglobin: 10.1 g/dL (mild anemia), Neutrophils: 89.9% (neutrophilic leukocytosis)

CT: Computed tomography

Table 2. Postoperative and follow-up outcomes.

Parameter	Pre-lobectomy	Post-lobectomy	Follow-up period
Oxygen saturation (SpO_2)	30% on room air; 100% with mechanical ventilation (PEEP: 8 cmH_2O, FiO_2: 100%)	95–98% on mechanical ventilation (PEEP: 5 cmH_2O, FiO_2: 40%)	Required supplemental O_2, fluctuating SpO_2 due to recurrent pneumonia
Ventilatory settings	High-pressure support; frequent desaturations	Lower-pressure support; stable oxygenation	Escalation to HFNC and tracheal mask with fluctuating FiO_2 requirements
Nutritional status	Severe malnutrition; limited intake via orogastric tube	Improved intake with high-calorie formula; weight gain observed	Persistent malnutrition despite high-calorie intake
Infection markers (CRP)	Elevated, indicating active infection	Normalized, indicating resolution of infection	Recurrent infection episodes requiring prolonged antimicrobial therapy

PEEP: Positive end-expiratory pressure, HFNC: High-flow nasal cannula

reinforced the indication for early lobectomy, particularly since conservative management had failed, as evidenced by recurrent hospitalizations, increased work of breathing, persistent oxygen requirement, as well as feeding intolerance and failure to thrive due to high respiratory burden and inadequate caloric intake.[5,14,15] Several previous reports have emphasized that early surgical intervention in CLE is associated with favorable long-term outcomes, given the

remarkable compensatory capacity and functional adaptation of neonatal lungs following lobectomy.[16–18] Resection of the hyperinflated lobe allows re-expansion of healthy lung tissue, thereby improving ventilatory efficiency and reducing airway obstruction.[13,22] Conversely, delaying surgery may result in irreversible lung damage, recurrent infections, and chronic pulmonary dysfunction, ultimately worsening the prognosis.[23] Therefore, lobectomy remains the treatment of choice in symptomatic neonates with CLE, particularly in the presence of significant respiratory compromise.[14,16]

Following lobectomy, the patient developed several serious complications that prolonged the recovery process. The major complications were nosocomial infection caused by multidrug-resistant *Acinetobacter baumannii* and persistent malnutrition due to difficulties with enteral intake.[8,17] These findings indicate that although lobectomy can improve oxygenation status, long-term success is highly influenced by secondary postoperative conditions.[18] Similar observations have been reported in previous studies, where CLE patients with nosocomial infections experienced longer hospital stays and higher morbidity risk.[15,17] Other reports have also emphasized that poor nutritional status can delay postoperative pulmonary recovery and increase the risk of recurrent infections.[18,19] The direct contributing factors to these complications were prolonged ventilator use and preoperative malnutrition, whereas the indirect causes included limited infection control and challenges in fulfilling oral nutritional requirements.[20] Therefore, it can be tentatively concluded that the prognosis of CLE patients after lobectomy largely depends on nutritional management and the prevention of nosocomial infections.[16,18,24]

Theoretically, CLE occurs as a result of partial bronchial obstruction that produces a "one-way valve" mechanism, allowing air to enter during inspiration but trapping it during expiration.[5,7] This obstruction may be intrinsic, such as bronchial cartilage dysplasia, bronchomalacia, or other structural abnormalities; or extrinsic, such as compression by anomalous blood vessels or a mass.[8,10] Consequently, the affected pulmonary lobe undergoes progressive hyperinflation, compressing adjacent lung tissue and shifting the mediastinum.[6,7] Histopathologically, the lobe demonstrates alveolar overdistension with reduced elastin, thereby decreasing elastic recoil and worsening air trapping.[15] Clinically, this condition is often misidentified as pneumothorax, since both present as hyperlucency on radiographs; however, CLE still preserves intact bronchovascular markings.[4,9] In infants, the highly compliant chest wall accelerates the progression of hyperinflation, leading to respiratory distress and respiratory failure.[5] Therefore, this pathophysiological theory underscores why conservative management is often ineffective in severely symptomatic cases, and lobectomy remains the curative treatment of choice.[14,16,18]

This study carries several benefits and clinical implications. First, it raises clinician awareness of the possibility of CLE in neonates with respiratory distress unresponsive to conventional therapy. Second, it emphasizes the importance of chest CT scan as the gold standard in the differential diagnosis of pulmonary hyperlucency in neonates. Third, it highlights that lobectomy remains the primary choice for symptomatic CLE, with favorable long-term outcomes when performed in a timely manner. Fourth, it underscores that postoperative management must encompass both nosocomial infection prevention and nutritional rehabilitation. Fifth, this case provides a forward-looking perspective that the development of a multidisciplinary approach –encompassing pulmonology, surgery, nutrition, infectious disease, and physiotherapy– represents the most effective strategy to improve outcomes in CLE patients.

This study has several limitations. First, the design is a single case report, which limits the generalizability of the findings. Second, the presence of confounding factors such as multidrug-resistant nosocomial infection influenced the clinical course, making it difficult to assess the pure outcome of lobectomy. Third, the limited facilities at the initial referral hospital contributed to delayed diagnosis, which may have introduced bias in the results. Fourth, this report only describes a clinical experience without comparison to a control group. Therefore, further research with a prospective design and a larger sample size is required to validate these findings.

In conclusion, this case highlights that CLE can be misdiagnosed as pneumothorax, thus requiring clinical vigilance and the use of CT scan to establish an accurate diagnosis. Lobectomy has been shown to be effective in improving ventilation and oxygenation, but the outcome is strongly influenced by the management of secondary complications, particularly nosocomial infection and nutritional status. It is expected that these findings may increase clinician awareness, accelerate early diagnosis, and strengthen the multidisciplinary approach in the management of CLE, thereby providing better long-term outcomes for patients.

ΠΕΡΙΛΗΨΗ

Συγγενές λοβιακό εμφύσημα στη νεογνική περίοδο: Αναφορά περίπτωσης με διαγνωστικές και διαχειριστικές προκλήσεις

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Περιγράφουμε τις διαγνωστικές προκλήσεις, τις στρατηγικές χειρουργικής διαχείρισης και τις μετεγχειρητικές επιπλοκές σε ένα βρέφος με συγγενές λοβιακό εμφύσημα (CLE). Θήλυ βρέφος 3 μηνών μεταφέρθηκε στο νοσοκομείο με προοδευτική ταχύπνοια, εισολκές του θώρακα, κυάνωση και δυσκολίες σίτισης που οδήγησαν σε αποτυχία ανάπτυξης. Αρχικά, η ασθενής διαγνώστηκε με πνευμονία και έλαβε ενδοφλέβια αντιβιοτικά. Στη συνέχεια, η ακτινογραφία θώρακα ερμηνεύτηκε λανθασμένα ως πνευμοθώρακας, οδηγώντας σε τοποθέτηση θωρακικού σωλήνα χωρίς κλινική βελτίωση. Περαιτέρω αξιολόγηση με αξονική τομογραφία θώρακα αποκάλυψε υπερδιάταση του αριστερού άνω λοβού, μετατόπιση του μεσοθωρακίου και συμπίεση του περιβάλλοντος πνευμονικού ιστού, επιβεβαιώνοντας τη διάγνωση του CLE. Η ασθενής υποβλήθηκε σε αριστερή άνω λοβεκτομή, η οποία βελτίωσε αμέσως τον κορεσμό οξυγόνου. Μετεγχειρητικά, η ασθενής εμφάνισε πνευμονία σχετιζόμενη με αναπνευστήρα, σήψη που προκλήθηκε από πολυανθεκτικό *Acinetobacter baumannii* και επίμονο υποσιτισμό, τα οποία αντιμετωπίστηκαν με αντιβιοτικά τελευταίας γραμμής, εντερική διατροφή υψηλής θερμιδικής αξίας και φυσικοθεραπεία θώρακα. Συμπερασματικά, αυτή η περίπτωση υπογραμμίζει τη σημασία της κλινικής επαγρύπνησης για το CLE σε νεογνά με αναπνευστική δυσχέρεια που δεν ανταποκρίνονται στη συμβατική θεραπεία. Η λοβεκτομή είναι αποτελεσματική ως οριστική θεραπεία. Ωστόσο, η βέλτιστη ανάρρωση απαιτεί διεπιστημονική διαχείριση, ιδιαίτερα πρόληψη λοιμώξεων και διατροφική αποκατάσταση.

Λέξεις ευρετηρίου: Λοβεκτομή, Νεογέννητο βρέφος, Νοσοκομειακές λοιμώξεις, Συγγενές λοβιακό εμφύσημα, Σύνδρομο αναπνευστικής δυσχέρειας νεογνού, Υποσιτισμός

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