IJN
Iranian Journal of Neonatology
http://ijn.mums.ac.ir

Open Access

Case Report

Navigating the Challenges of Neonatal Type 2 CPAM: A Case Study of Successful Pneumonectomy and Intensive Care

Masayu Amanda Ledika[1*], Tetty Yuniaty[1], Aris Primadi[1], Fiva Aprilia Kadi[1], Hasrayati Agustina[2], Citra Yulia Sari[1]

1. Department of Pediatrics, Faculty of Medicine, Padjadjaran University, Dr. Hasan Sadikin General Hospital, Bandung, West Java 40161, Indonesia
2. Department of Pathology Anatomy, Padjadjaran University, Dr. Hasan Sadikin General Hospital, Bandung, West Java 40161, Indonesia

ABSTRACT

Background: Congenital Pulmonary Airway Malformation (CPAM) is a rare problem in the development of the fetal tracheobronchial tree. If it is not treated properly, it can cause serious illness and even death. This report documents and emphasizes the challenges associated with postnatal CPAM diagnosis, the risks of significant surgical intervention, and the importance of meticulous clinical management to achieve the best possible patient outcomes.

Case Report: This case report details the condition of a 14-day-old male neonate diagnosed with Type II CPAM, a collapsed area in his left lung, pneumonia, ASD, and VSD. The patient underwent surgery to remove his left lung on the 19th day after birth (5 days after admission). Chest tubes were placed after the operation. Although he experienced some complications after surgery, he gradually improved and was discharged 42 days after the surgery.

Conclusion: Early surgery for symptomatic CPAM can lead to good outcomes, even in the presence of serious complications. To achieve the best results, it is important to have a multidisciplinary team, robust infection control, and long-term follow-up, especially in places with limited resources.

Keywords: Congenital pulmonary airway malformation, CPAM, Neonate, Resection surgery, Respiratory distress

Introduction

Congenital Pulmonary Airway Malformation (CPAM), formerly known as Congenital Cystic Adenomatoid Malformation of the Lung (CCAM), is a congenital lung condition present at birth. This occurs due to improper lung development early in pregnancy, typically around the 7th week, impacting the small airways known as terminal bronchioles. This condition occurs in about 1 out of every 25,000 to 35,000 births and accounts for up to 25% of all congenital lung problems. The disease causes excessive cell growth and reduced cell death, leading to abnormal growth of small airways with connected cysts. Although these growths are linked to the airway, they do not have a normal bronchial system inside the lung (1).

CPAM is classified into five distinct types: Type 0 (bronchial type, formerly called acinar dysplasia) features bronchial-type airways separated by extensive mesenchymal tissue; Type 1 (bronchial/bronchiolar type) displays cysts larger than 2 cm in diameter; Type 2 (bronchiolar type) presents with multiple small cysts; Type 3 (bronchiolar/alveolar type) appears as a solid mass rather than cystic due to excessive bronchiolar structures separated by airspaces

* *Corresponding author*: Masayu Amanda Ledika, Department of Pediatrics, Faculty of Medicine, Padjadjaran University, Dr. Hasan Sadikin General Hospital, Bandung, West Java 40161, Indonesia. Email: masayu09001@mail.unpad.ac.id

Please cite this paper as:
Ledika MA, Yuniaty T, Primadi A, Kadi FA, Agustina H, Sari CY. Navigating the Challenges of Neonatal Type 2 CPAM: A Case Study of Successful Pneumonectomy and Intensive Care. Iranian Journal of Neonatology. 2026 Apr: 17(2). DOI: 10.22038/ijn.2026.87641.2687

resembling late fetal lung; and Type 4 (peripheral type) is characterized by thin-walled, often multiloculated peripheral cysts (2-4).

Small CPAM lesions often do not cause symptoms and are typically discovered later or incidentally. Newborn babies may experience difficulty breathing immediately after birth. Up to 25% of patients, typically those with Type II, may experience additional issues in the kidneys, intestines, bones, or heart (1). Current best practices support identifying CPAM before birth and early surgical resection of the affected lung tissue as the optimal treatment for symptomatic cases (5,6). This report discusses a newborn baby who had Type 2 CPAM, which was not diagnosed before birth. The baby showed symptoms, underwent left pneumonectomy, and then experienced some complications after the operation.

Case report

A 14-day-old male neonate was brought to our hospital because he had respiratory distress since birth. He was born naturally with assistance from a midwife, weighing 3150 g, and was immediately transferred to a local hospital. His mother, who was 19 years old and nulliparous, had no ultrasound checks during her normal full-term pregnancy. At 3 days of age, he had respiratory failure and needed endotracheal intubation and mechanical ventilation. Chest X-rays showed multiple cyst-like structures in the upper and lower parts of his left lung, and his heart was pushed to the right side (Figure 1). Doctors considered CPAM, but they also considered congenital diaphragmatic hernia. He was transferred to another hospital for a CT scan and further evaluation.

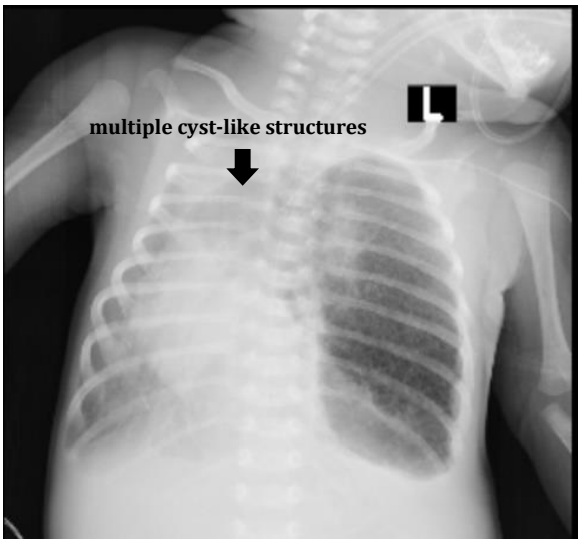

Figure 1. Chest X-rays revealed multiple cyst-like structures

The patient's vital signs were stable even though he was on a mechanical ventilator. He tolerated enteral feeds up to 90 mL per kilogram of body weight per day. He showed only small chest movements when breathing, had a grade 2 heart murmur near the breastbone, and his heartbeat was felt on the right side of his chest. A thoracic CT scan revealed that the anterior portion of the right lung's upper lobe had herniated into the left chest cavity (Figure 2). The mediastinal organs were displaced to the right. The scan also showed patchy infiltrates with air-filled airways in the top and front parts of the right lung's upper lobe. Both main bronchi were open and the same size. The CT scan showed that the baby had Type II Congenital Pulmonary Airway Malformation, with collapse of the left lower lobe and pneumonia in the right upper lobe. Pathological examination revealed that the left lung sample contained two small cystic cavities with connected spaces, lined with cuboidal and pseudostratified ciliated epithelium with normal nuclei (Figure 3). The histopathology confirmed that the left lung had type II congenital cystic adenomatoid malformation.

The patient was transferred to our main hospital for comprehensive management by a multidisciplinary team including neonatologists, pediatric cardiologists, thoracic surgeons, and anesthesiologists. An echocardiogram showed a ventricular septal defect (5–6 mm), dextroposition of the heart, and a small patent ductus arteriosus. The patient required mechanical ventilation, and his condition slightly deteriorated. Anemia was successfully treated with a packed red blood cell transfusion, and no neurological or gastrointestinal complications were observed.

After evaluation by the cardiothoracic surgery and anesthesiology departments, the patient was prepared for surgical intervention. The surgery took place on the fifth day after admission (postnatal day 19). During the operation, multiple cysts were found in the upper part of the left lung. The entire left lung was removed, and chest tubes were placed to drain fluid.

After surgery, the right side of the chest expanded more, and up to 80 mL of blood drained from the right chest tube in the first 48 hours. The patient's condition worsened with repeated episodes of hypoxia and bradycardia. He required increased oxygen, up to 45% FiO_2, to keep his oxygen saturation above 90%. The patient remained on conventional mechanical ventilation.

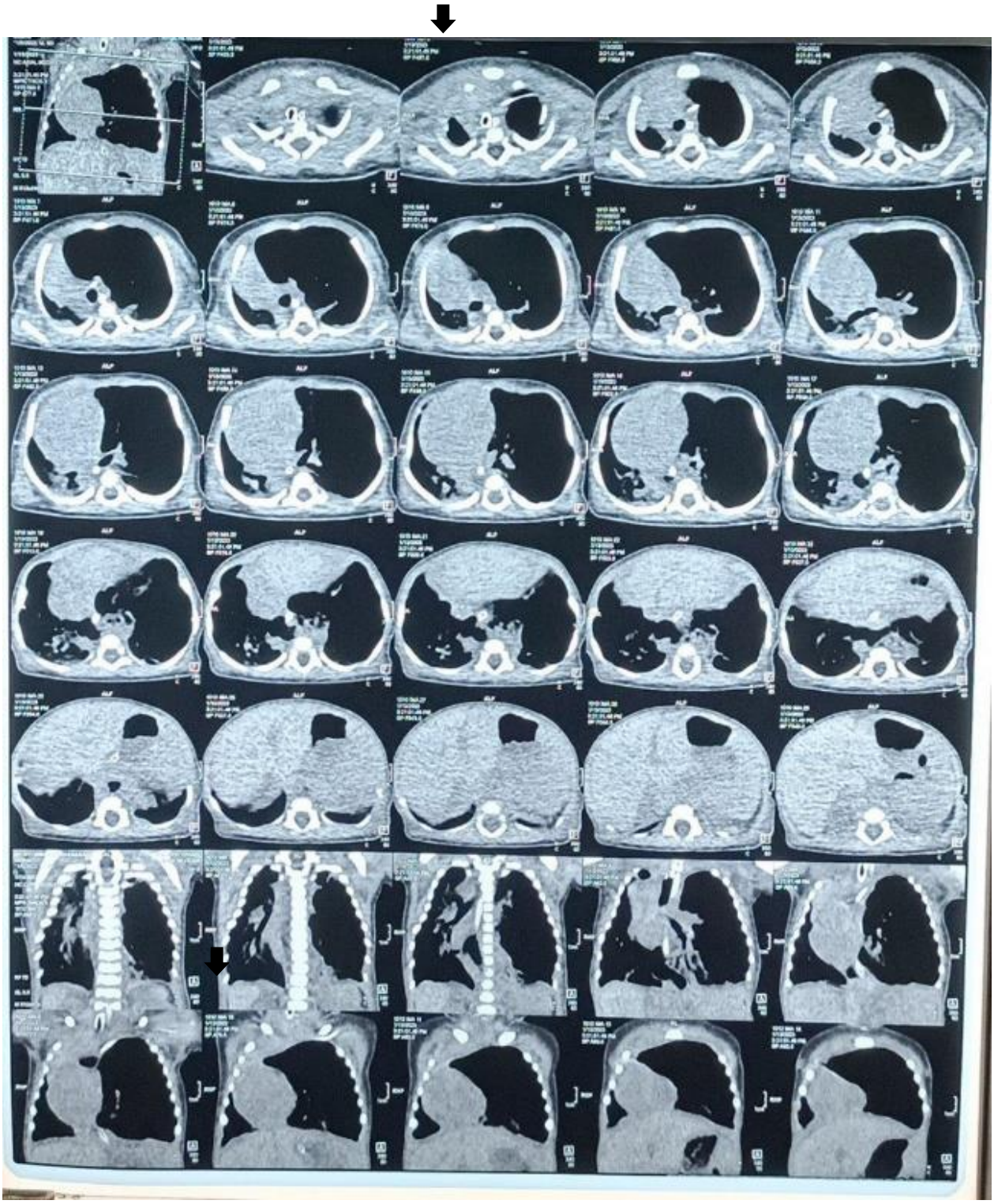

Figure 2. A thoracic CT scan lung's upper lobe had pushed into the left chest cavity

The endotracheal tube (size 3.5) was advanced to 10 cm at the lips, to avoid injury to the left bronchus after the left lung was removed. This helped ensure ventilation of the right lung.

Postoperatively, the patient received total parenteral nutrition and medications including fentanyl, midazolam, paracetamol, inotropes, two antibiotics (meropenem and amikacin), and an

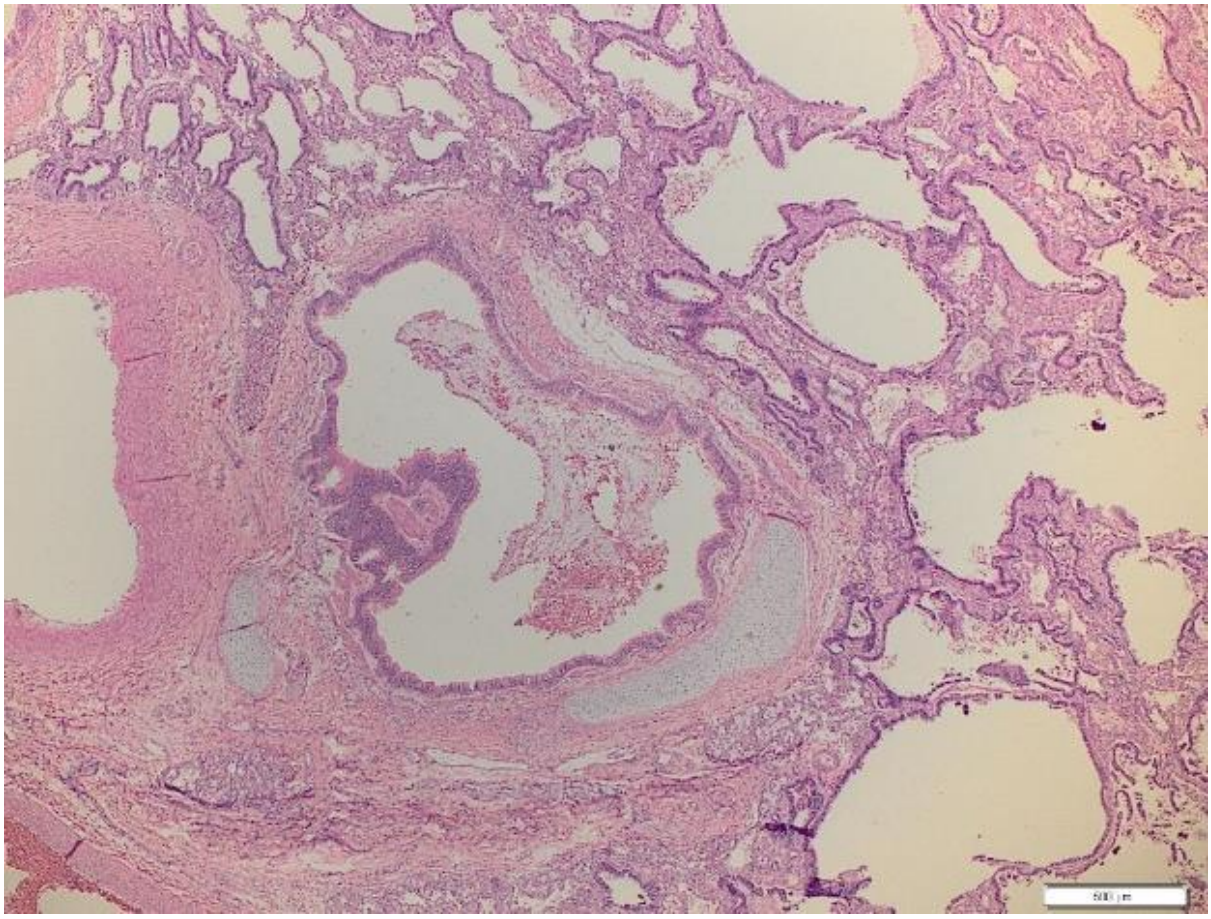

Figure 3. The pathological examination showed Type II congenital cystic adenomatoid malformation

antifungal (fluconazole). The patient also received albumin transfusions and regular nebulizer treatments. The chest X-ray showed post-pneumonectomy changes on the left side. It also showed pneumonia and fluid around the right lung, and partial collapse of the right upper lobe (Figure 4). A pus culture from the surgical wound identified multidrug-resistant, carbapenem-resistant *Acinetobacter baumannii*. Vancomycin was added to the treatment regimen for 7 days. Although the patient's condition stabilized, he remained in critical condition.

Blood cultures obtained on postoperative day 3 (reported on day 7) also tested positive for multidrug-resistant (carbapenem-resistant) *Acinetobacter baumannii*. Laboratory findings indicated deterioration, with hemoglobin of 9.6 g/dL and severe thrombocytopenia (6,000/mm^3), although C-reactive protein improved from 12.71 g/L post-surgery to 5.58 g/dL. After consultation with the pediatric infectious diseases division, the antibiotic regimen was adjusted to ampicillin-sulbactam (50 mg/kg/dose every 6 hours) for 7 days, followed by ceftazidime. The antifungal was changed to micafungin (10 mg/kg every 24 hours) for 7 days. The patient slowly improved, received blood and albumin transfusions, started gastric feeds, required less analgesia, was weaned off inotropes, and mechanical ventilatory support was gradually reduced.

On postoperative day 16, the patient's blood culture revealed another multidrug-resistant organism, *Achromobacter denitrificans*. The antibiotic cefepime, to which the organism was moderately susceptible, was given at 50 mg/kg every 12 hours. The patient showed improvement in physical exams and lab tests. The endotracheal tube was removed on postoperative day 17 (when the patient was 43 days old), and treatment for bronchopulmonary dysplasia was given following DART guidelines. The patient still required supplemental oxygen and was given low-flow oxygen at 0.1 liters per minute with 100% FiO_2 to keep oxygen saturation above 92%. The patient

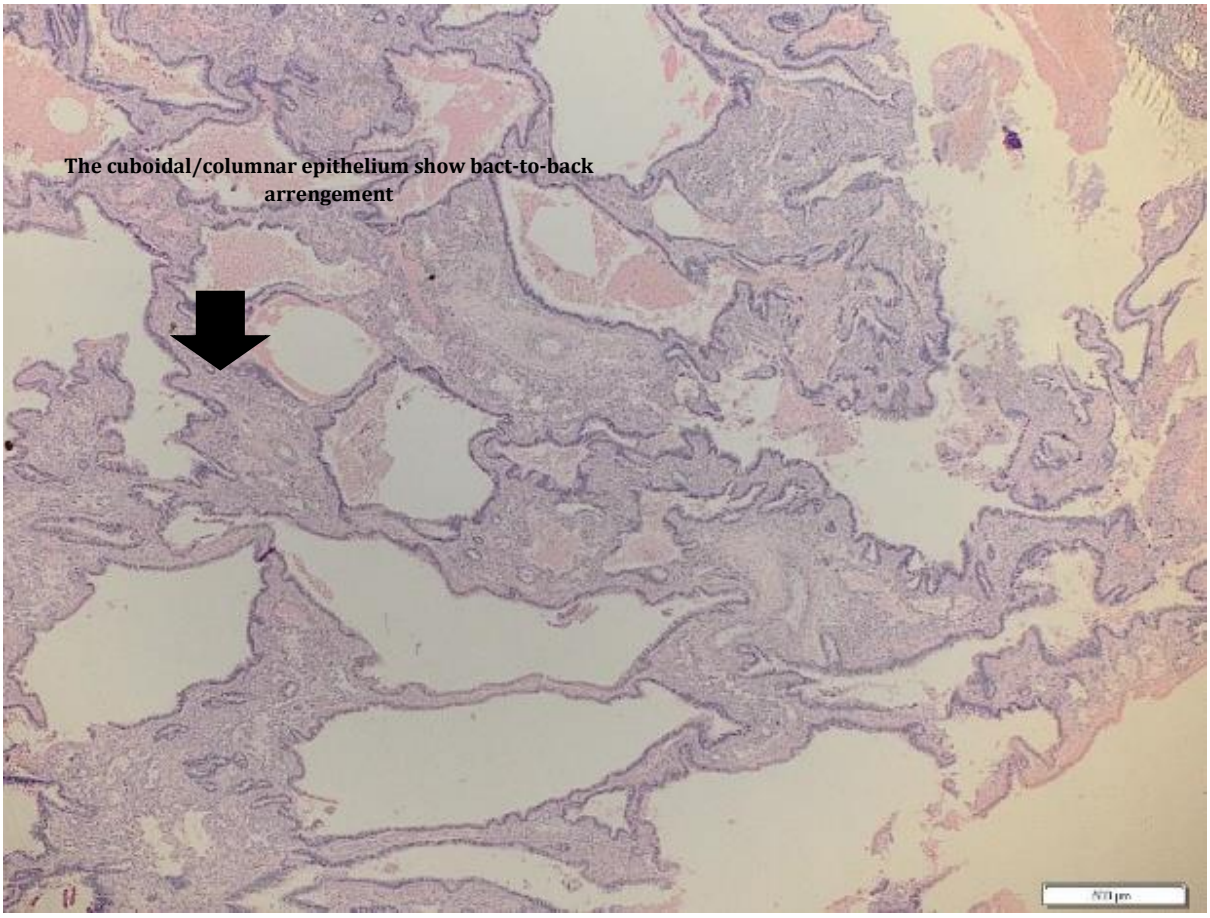

Figure 4. Histopathological examination of the left lung demonstrating Type II CPAM

developed cholestatic jaundice, and tests on day 23 after surgery showed positive anti-cytomegalovirus antibodies (IgM 3.7 and IgG 57.8). Because of this, the patient was treated with oral valganciclovir.

The patient's condition continued to improve, and he was discharged 42 days after surgery, with post-discharge status documented on the chest X-ray (Figure 5). Upon discharge, the patient was receiving enteral nutrition through an orogastric tube, with a continuous 0.1 liter/minute low-flow oxygen supply at 100% FiO_2, and was under valganciclovir therapy. After being discharged, the patient encountered a single episode of pneumonia that was successfully resolved with antibiotic treatment.

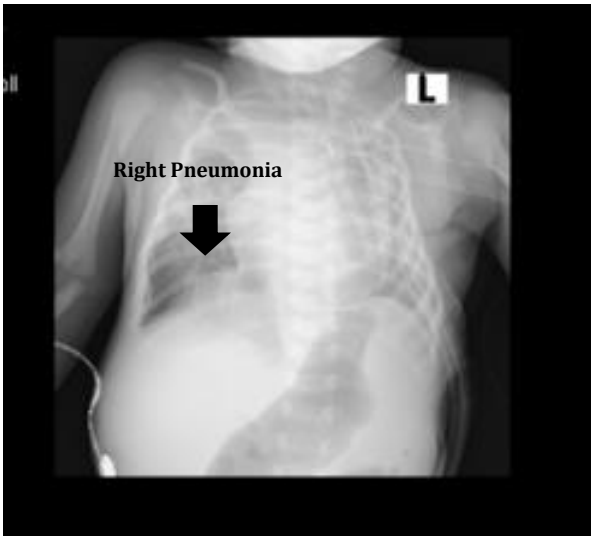

Figure 5. Chest X-ray showed pneumonia

Ethical Approval

This case report was conducted in accordance with the ethical principles of the Declaration of Helsinki. Written informed consent was obtained from the patient's parents/legal guardians for the publication of this case report and any accompanying images. No ethical committee approval was required for this case report as it involves routine clinical care data.

Discussion

Our patient presented with symptoms of CPAM soon after birth but was successfully treated with open chest surgery and removal of the whole left lung. CPAM is a rare cause of breathing problems in newborns. It arises from abnormal airway development due to an issue during the 35th day of pregnancy (3, 7). The exact cause is still unknown, but studies in rats show that excessive growth factors involved in lung development may play a role (8, 9). Recent research found changes in genes linked to lung cancer, which might increase the chance of malignancy developing (10). The condition was first documented in 1949 by Chin and Tang (11), with Kwittken later providing a more detailed microscopic description in 1962 (12).

CPAM is classified into five types (0–4) based on clinical and pathological features. Type 0 affects all lung lobes and is not compatible with survival. Type 1, representing 60–65% of cases, consists of single or multiple large cysts lined with ciliated, pseudostratified, or columnar epithelium. Type 2, which makes up 20% of cases, shows many small cysts lined with cuboidal or columnar cells without mucus or cartilage. This type is associated with other birth defects in 60% of cases. Type 3 appears as a large, gland-like mass without cysts, usually in one lung lobe, which can grow large enough to cause poor development of the other lung (2, 3, 13). In our case, histopathology showed the left lung had two small cysts with connected spaces. These were lined with cuboidal and pseudostratified ciliated cells with normal nuclei. This ruled out other congenital lung problems like pulmonary sequestration and bronchogenic cysts.

After birth, CPAM can be symptomatic or asymptomatic. Respiratory distress is a common sign of CPAM in newborns, as seen in our patient. Other common causes of breathing problems in newborns, such as congenital pneumonia, heart disease, or diaphragmatic hernia, were ruled out by chest X-rays. However, in most cases, a chest CT scan provides the definitive diagnosis of CPAM.

In our patient's case, the CT scan findings strongly suggested type II CPAM in the upper lobe of the left lung, with collapse of the left lower lobe and pneumonia in the right lung. The presence of an intact diaphragm eliminated congenital diaphragmatic hernia as a possibility. Older studies showed worse results for types 2 and 3, but recent data show better outcomes for most types of CPAM. The only exception is type 4, which has large, thin-walled cysts that may transform into a cancer called type 1 pleuropulmonary blastoma (6, 13).

Type 2 CPAM originates in the bronchiolar regions and is the second most common variant. It usually presents as multiple small cysts without a mass effect. The prognosis is favorable, and this type does not have malignant potential (6). In our patient's case, there was also a moderate-sized VSD and rightward displacement of the heart. The combination of CPAM and congenital heart disease is rare, occurring in only 15–20% of cases, but can be successfully managed with careful planning and a multidisciplinary team approach, as demonstrated in our case (14). Cardiac defects are not considered poor prognostic factors in CPAM (1).

Surgery was the main treatment for our patient because of severe respiratory distress and large cysts in the left lung that pushed the mediastinum and heart to the right. Symptomatic CPAM usually requires urgent surgery to remove the affected part of the lung, using either open chest surgery (thoracotomy) or keyhole surgery (thoracoscopic) (15,16). At our hospital, the surgery was performed using the thoracotomy method. Because of the size and number of cysts found during surgery, the entire left lung was removed. After surgery, a single-lung ventilation strategy was used, similar to a case report by Giwangkencana et al. This involved inserting the endotracheal tube deeper (11 cm from the lips) to ensure the right lung received adequate ventilation and to protect the left bronchus until the tube was removed (17). In our case, the placement of the tube in the right main bronchus may have contributed to atelectasis in the upper right lobe. Excessively deep positioning can exclude the upper lobe bronchus, resulting in right upper lobe collapse and hypoxia (18). The atelectasis in the right upper lobe was treated with continuous antibiotic therapy, adjusting the endotracheal tube position to 10 cm from the lips, and proper patient positioning.

The management of asymptomatic CPAM after birth remains contested, with differing views on elective surgery versus conservative management (19). Supporters of planned surgery after birth focus on concerns about recurrent pneumonia, poor lung growth caused by the mass effect, and the potential for malignant transformation (19, 20). The opposing viewpoint argues that surgical risks outweigh these potential complications (21, 22). Postoperative complications such as

pneumonia, pneumothorax, and pectus excavatum occur about four times more often in children with symptomatic CPAM than in those with asymptomatic lesions (19). Pneumonia and sepsis after surgery were treated with antibiotics and intensive care. Our patient had multiple risk factors that predisposed him to sepsis, such as mechanical ventilation, a peripherally inserted central catheter, and major surgery. The sepsis was caused by *Acinetobacter baumannii*, a Gram-negative coccobacillus that has emerged as a significant hospital-acquired pathogen globally (23). Ampicillin-sulbactam was chosen as the antibiotic treatment upon the availability of blood culture results. Ampicillin-sulbactam is unique because the sulbactam component (a beta-lactamase inhibitor) shows excellent standalone bactericidal activity against *Acinetobacter* even in the absence of ampicillin. This mechanism enables sulbactam to retain effectiveness in the presence of resistance to other beta-lactam antibiotics and carbapenems. In the treatment of highly resistant *Acinetobacter* infections, some published reports have suggested higher doses of ampicillin-sulbactam than those usually recommended by manufacturers. Small retrospective studies compared ampicillin-sulbactam to other treatments, used alone or with other drugs, for *Acinetobacter* infections. These studies show that ampicillin-sulbactam works at least as well as other treatments. The success rates for ampicillin-sulbactam in these studies were between 83 and 93 percent (23, 24).

The second microorganism identified in the blood culture was *Achromobacter denitrificans*. This Gram-negative bacterium is motile and aerobic. It is non-fermentative and tests positive for catalase and oxidase. These bacteria are usually found in soil and water and rarely cause infections in humans. This organism has been associated with contaminated intravenous solutions or the use of humidifiers and incubators. In our patient's case, the humidified oxygen for mechanical ventilation and the intravenous fluids might have been the source of infection. Known risk factors for *Achromobacter* infection include immune compromise, HIV infection, cancer, cystic fibrosis, and hospitalization (25). For our patient, the immature neonatal immune system and extended hospitalization likely constituted the primary risk factors.

Treatment for *Achromobacter* pneumonia depends on susceptibility tests. Most *Achromobacter* strains are resistant to first- and second-generation cephalosporins, aminoglycosides, and narrow-spectrum penicillins. They are usually sensitive to sulfonamides, carbapenems, broad-spectrum penicillins, and third-generation cephalosporins. Their response to fluoroquinolones can vary (26). In our patient's case, tests showed the bacteria were resistant to ampicillin, cefazolin, ceftazidime, ceftriaxone, ciprofloxacin, and gentamicin. The patient was given cefepime, to which the organism was moderately susceptible. Antibiotics that were highly effective were imipenem and piperacillin-tazobactam, but these were not available at our hospital. Reports of *Achromobacter* infections in newborn babies are rare. However, these infections are important because they do not respond to common antibiotics like cephalosporins and aminoglycosides, which are often used to treat neonatal sepsis (27). It is very important to always follow strict infection control measures when caring for newborns in intensive care units. This helps prevent serious infections such as sepsis.

The number of reported Cytomegalovirus (CMV) infections in the Neonatal Intensive Care Unit was lower than the number found in studies that screened all babies for CMV infection (28). Diagnosing congenital CMV infection requires PCR testing of urine, saliva, or blood collected within 2–3 weeks of birth. Postnatal CMV infection, although usually asymptomatic, can cause severe disease. In our case, CMV infection was diagnosed with a repeated antibody test that showed seroconversion of the IgM antibody to positive, with elevated liver enzymes and cholestatic jaundice. CMV infection in our patient was treated with oral valganciclovir, resulting in improvement in both clinical and laboratory conditions, but continuous monitoring and future examinations for brain and hearing functions are warranted.

Conclusion

Congenital Pulmonary Airway Malformation (CPAM) is a rare cause of respiratory distress in newborn babies. A chest X-ray can help identify the problem initially. However, a CT scan of the chest is superior for visualizing congenital lung lesions. It is important to refer patients promptly to hospitals that have thoracic surgery and intensive care units to ensure they receive necessary surgical care. One major challenge in lower-middle-income countries is providing good postoperative care and preventing infections. After patients recover and leave the hospital, they still need long-term follow-up. This includes tests for brain and hearing function, vaccinations,

physiotherapy, and infection prevention. There is still a chance of pneumonia recurring after surgery. Therefore, it is important to ensure adequate vaccinations, prompt antibiotic treatment, and monitoring of lung function for ongoing care. The long-term consequences following surgery are not well understood; however, a small group of children with CPAM exhibited normal lung function between the ages of 4 and 9 years. We advocate for improved antenatal ultrasound screenings to enable early diagnosis and referral to tertiary centers for prompt treatment, resulting in better outcomes in managing CPAM.

Acknowledgments

The authors gratefully acknowledge the Department of Pediatrics, Faculty of Medicine, University of Padjadjaran/Dr. Hasan Sadikin General Hospital.

Conflicts of interest

The authors declare no conflicts of interest.

References

1. Dias R. Congenital pulmonary airway malformation. Anästh Intensivmed 2022;63:S15–S22.
2. Stocker JT, Madewell JE, Drake RM. Congenital cystic adenomatoid malformation of the lung. Classification and morphologic spectrum. Hum Pathol. 1977;8(2):155-171.
3. Bush A, Harcout J, Hewitt RJ, Nicholson AG, Wilmott RW, Deterding R, et al. Kendig's disorders of the respiratory tract in children. Congenital lung malformations. 2019:2793-3011.
4. Chowdhury MM, Chakraborty S. Imaging of congenital lung malformations. Semin Pediatr Surg 2015;24(4):168–175.
5. Vohra WI, Shakir M, Dhillo RA, Irfan O, Mirza A.. Multi-disciplinary management of a rare case of a complete lung congenital pulmonary airway malformation in a newborn from a developing country. Int J Pulm Res Sci 2022;6:555683.
6. Mehta PA, Sharma G. Congenital pulmonary airway malformation. [Updated 2023 Aug 7]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2023. Available from: https://www.ncbi.nlm.nih.gov/books/NBK551664/
7. Finder JD, Michelson P. Congenital Disorders of the Lung. In: Kliegman RM, Stanton BF, St Geme JW, Schor NF, editors. Nelson Textbook of Pediatrics. 20th ed. Philadelphia: Elsevier Saunders; 2016;2057–2059.
8. Lezmi G, Verkarre V, Khen-Dunlop N, Vibhushan S, Hadchouel A, Rambaud C, et al. FGF10 Signaling differences between type I pleuropulmonary blastoma and congenital cystic adenomatoid malformation. Orphanet J Rare Dis. 2013;8:130.
9. David M, Lamas-Pinheiro R, Henriques-Coelho T. Prenatal and postnatal management of congenital pulmonary airway malformation. Neonatology. 2016;110(2):101-115.
10. Hsu JS, Zhang R, Yeung F, Tang CSM, Wong JKL, So MT, et al. Cancer gene mutations in congenital pulmonary airway malformation patients. ERJ Open Res. 2019;5(1):00196-2018.
11. Ch'in KY, Tang MY. Congenital adenomatoid malformation of one lobe of a lung with general anasarca. Arch Pathol (Chic). 1949;48(3):221-229.
12. Ribet M, Pruvot FR, Dubos JP, Remy J, Sault MC, Gosselin B, et al. Congenital cystic adenomatoid malformation of the lung. Eur J Cardiothorac Surg. 1990;4(8):403-405.
13. Stocker JT. Congenital and developmental diseases. In: Dail DH, Hammar SP, eds. Pulmonary Pathology, 2nd ed. New York: Springer-Verlag; 1994:174–180.
14. Chiluveru SA, Dave NM, Dias RJ, Garasia MB. Congenital pulmonary airway malformation with atrial septal defect and pulmonary hypertension for lobectomy-anesthetic considerations. Ann Card Anaesth. 2016;19(2):372-374.
15. Delestrain C, Khen-Dunlop N, Hadchouel A, Cros P, Ducoin H, Fayon M, et al. Respiratory morbidity in infants born with a congenital lung malformation. Pediatrics. 2017;139(3):e20162988.
16. Lima M, D'Antonio S, Di Salvo N, Maffi M, Libri M, Gargano T, et al. Is thoracoscopic lung-sparing surgery in treatment of congenital pulmonary airway malformation feasible? J Ped Endos Surg. 2019;1(1):7-14.
17. Giwangkencana G, Andriyanto L, Zulfariansyah A, Lestari AN. One-lung ventilation techniques in infants and small children. J Ped Surg Case Rep 2022;82:102314.
18. Tognon C, Meneghini L, Fascetti LF, Gamba PG. The different approaches of single-lung ventilation in infants with pulmonary malformation. Int J Pediatr Res 2018;4(1): 1–6.
19. Kapralik J, Wayne C, Chan E, Nasr A. Surgical versus conservative management of congenital pulmonary airway malformation in children: A systematic review and meta-analysis. J Pediatr Surg. 2016;51(3):508-512.
20. Morini F, Zani A, Conforti A, van Heurn E, Eaton S, Puri P, et al. Current management of congenital pulmonary airway malformations: A "European Pediatric Surgeons' Association" Survey. Eur J Pediatr Surg. 2018;28(1):1-5. Erratum in: Eur J Pediatr Surg. 2018;28(1):e1.
21. Cook J, Chitty LS, De Coppi P, Ashworth M, Wallis C. The natural history of prenatally diagnosed congenital cystic lung lesions: long-term follow-up of 119 cases. Arch Dis Child. 2017;102(9):798-803.
22. Robinson A, Romao R, Mills J, Davies DA. Decision-making criteria for observational management of congenital pulmonary airway malformations (CPAMs). J Pediatr Surg. 2018;53(5):1006-1009.
23. Kanafani ZA, Kanj SS. Acinetobacter infection: Treatment and prevention. UpToDate Dec 2023.

Available from: https://www.uptodate.com/contents/acinetobacter-infection-treatment-and-prevention.
24. Qasemi A, Bayat Z, Akbari N, Babazadeh D. Bacterial resistance of acinetobacter baumannii: A global concern. Res Biotechnol Environ Sci. 2022;1(2): 36-42.
25. Rolston KV, Messer M. The in-vitro susceptibility of Alcaligenes denitrificans subsp. xylosoxidans to 40 antimicrobial agents. J Antimicrob Chemother. 1990;26(6):857-860.
26. Dar SA, Sameen D, Nazir M, Wani WA, Charoo BA. Rare case of neonatal sepsis by Achromonas denitrificans. J Clin Case Rep. 2018;8(1198):2.
27. Tran C, Bennett MV, Gould JB, Lee HC, Lanzieri TM. Cytomegalovirus infection among infants in neonatal intensive care units, California, 2005–2016. Am J Perinatol 2020;37(2):146–150.
28. Paes B, Kim D, Saleem M, Wong S, Mitchell I, Lanctot KL; and the CARESS investigators. Respiratory syncytial virus prophylaxis in infants with congenital airway anomalies compared to standard indications and complex medical disorders. Eur J Pediatr. 2019;178(3):377–385.