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Case Report

A Case Reports On Bilateral Diffuse Cystic Lung Disease (Pulmonary Langerhans' Cell Histiocytosis)

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ABSTRACT

Diffuse Cystic Lung Disease (DCLD) is an uncommon and complex condition marked by the presence of numerous air-filled cysts scattered throughout the lung tissue. This case report discusses about a 67-year-old man who came to the hospital with symptoms including nausea, vomiting, fever, persistent cough, and chest discomfort. His medical background was notable for rheumatoid arthritis, Hypothyroidism, Cholelithiasis, and Grade I Fatty Liver Infiltration, along with a history of undergoing coronary artery angiography. Imaging studies revealed a collapse and consolidation in the lower lobe of the left lung, alongside multiple cysts in both lungs and areas of ground-glass opacity. Blood tests indicated signs of inflammation and abnormalities in hematological and liver function parameters. Based on the clinical and radiological findings, a final diagnosis of bilateral DCLD Pulmonary langerhans cell histiocytosis, was made. The patient was managed with bronchodilators, corticosteroids, NSAID's, analgesics, and antiemetics. This case underscores the diagnostic complexity and therapeutic challenges of DCLD, emphasizing the urgent need for more research to enhance early detection and improve treatment outcomes.

Keywords: Diffuse cystic lung disease, Lung cysts, Collapse consolidation, Diagnosis, Treatment challenges.

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INTRODUCTION

Diffuse Cystic Lung Disease (DCLDs) represent a diverse group of pulmonary disorders characterized by the presence of multiple spherical or irregular air filled spaces within the lung parenchyma. These cysts typically have well-defined borders, thin walls, and surrounded by otherwise normal lung tissue (Hansell *et al*., 2008). Determining the true prevalence of DCLDs in India remains difficult due to limited large scale epidemiological studies. Furthermore, given the country's high burden of tuberculosis, some cases of DCLD may be linked to post—tuberculous lung damage, where cystic changes can arise as a sequel of prior infection (Wang *et al*., 2023). In some parts of India, high smoking rates may be a contributing factor to the prevalence of smoking-related DCLD (Gupta *et al*., 2015). According to reports, between 7.4 and 46.2% of patients with Sjogren illness develop DCLD. If it is conservatively assumed that 5-10% of people with Sjogren disease develop DCLD, the average number of individuals with Sjogren-related cystic lung disease would be 21-43 per million (Franciosi *et al*., 2024). The term "Bilateral Diffuse Cystic Lung Disease" (B/L DCLD) describes a particular pattern of lung involvement marked by the existence of many cysts in both lungs. These cysts may differ in number and size and are usually dispersed in a diffuse manner (Kunisaki *et al*., 2019). Many factors, including congenital, acquired, viral, inflammatory, and neoplastic origins, can contribute to the etiology of DCLD. Some of the congenital causes are Birt-Hogg-Dube syndrome, Ehlers-Danlos syndrome, and Proteus' syndrome; neoplastic causes are non-Langerhans' cell histiocytosis, Erdheim Chester disease, Lymphangioleiomyomatosis (LAM), and pulmonary Langerhans' cell histiocytosis; and inflammatory causes are pneumocystis *jiroveci*, staphylococcal pneumonia, and recurrent respiratory papillomatosis. The precise processes by which cysts arise in DCLDs are not well understood and probably differ based on the underlying illness. Cyst development has generally been associated with three main processes: (1) air space dilatation due to one-way blockage in small airways, which causes air to enter but not exit air spaces; (2) ischemia, which results in small bronchiole cellular death; and (3) remodeling from matrix-degrading proteolytic enzymes (Colombat *et al*., 2008; Gupta *et al*., 2017; Kikuchi *et al*., 2006; Ohdama *et al*., 1996; Ryu and Swensen, 2003). Most cases having common signs and symptoms include dyspnea, chest pain, cough, pneumothorax, chylothorax, and hypoxia. In most cases, clinicians can use a noninvasive method

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to accurately diagnose DCLD by combining essential radiologic assessment of cyst morphological concepts and location with important demographic factors (e.g., sex, focused history of illness and physical evaluation, detection of extra pulmonary visualization defects, assessment for the existence of disease, and particular laboratory biomarkers). The first suggested diagnostic decision point is ruling out the potential existence of common cyst mimics (emphysema, cavitary disease, or cystic bronchiectasis). This is followed by differentiating between paucicystic (≤10 cysts) entities and the more severe diffuse, bilateral, and multilobed modifications typical of multicystic DCLDs to further refine the diagnostic process for making decisions. The diagnostic tests include High-Resolution Computed Tomography (HRCT) scan, pulmonary function tests, bronchoscopy, chest X-ray, sputum culture, blood tests, and lung biopsy. The goal of treating diffuse cystic lung disease is to control the underlying cause and reduce symptoms; depending on the individual, treatment options may involve medication, oxygen administration, or surgery (Gupta *et al.*, 2016; Lee *et al.*, 2019; Paciocco *et al.*, 2004; Park and Lee, 2017; Singla and Gupta, 2021).

CASE REPORT

A 67-year-old male patient was admitted to the tertiary care hospital presenting with nausea that had begun earlier that morning, accompanied by fever, cough, and chest pain that had persisted since 5 days. He has a history of rheumatoid arthritis since 1 month. He was on medication for Cholelithiasis, rheumatoid arthritis, Hypothyroidism, coronary artery angiography, and Grade I fatty liver infiltration. His past medication history includes the T. TORVAS (Atorvastatin) 10 mg taken at night (0-0-1), T. THYRONORM (Levothyroxine Sodium) 50 µg taken on an empty stomach in the morning (1-0-0), T. AB Flo SR (Acebrophylline) 200 mg taken in the morning (1-0-0), Neb. FORACORT (Budesonide + Formoterol) 0.5 mg by nebulizer taken twice daily (1-0-0-1), and T. ASPIBOL (Aspirin + Glycine) 75 + 37.5 mg taken in the morning (0-1-0-0). His personal history was normal including appetite, sleep, and bowel and bladder movements. His vitals were as Pulse rate: 108 beats per minute (N: 60-100 beats per minute), respiratory rate: 22 bpm, and blood pressure: 150/90 mm of mercury.

Upon examination, he was alert and febrile, with all systemic evaluations returning normal results. Hematological tests indicated a reduction in red blood cell and basophil counts, alongside an elevated white blood cell count. Liver function tests revealed increased levels of total bilirubin, Serum glutamate oxaloacetate transaminase, and Serum glutamate pyruvate transaminase. Furthermore, both C-reactive protein and HbA1c levels were elevated, while potassium levels were found to be low (Table 1). A HRCT scan of the chest identified multiple cysts in both lung fields, consolidation collapse in the left lower lobe, and areas of ground-glass opacity (Figure 1). The computed tomography chest pain reveals collapse consolidation in left lower lobe with areas of breakdown, multiple cysts of varying sizes noted in bilateral lung fields with ground-glass attenuation rest of the lung fields and the possibilities could be LCH (Langerhans' Cell Histiocytosis) and LAM. The department of radiology performed abdominal ultrasound which reveals coarse heterogenous hepatic echotexture- likely chronic liver disease, bilateral Grade I/II renal parenchymal changes and mild left pleural effusion was noted. Based on all the subjective and objective evidences patient was diagnosed with bilateral diffuse cystic lung disease (pulmonary Langerhans' cell histiocytosis).

Day-wise Report on Day 1, the patient was started on treatment for a severe lung infection. The therapy included Inj. Meropenem (MERNEM) 1 g IV every 8 hr, a broad-spectrum antibiotic to control infection, and Inj. Cefoperazone + Sulbactam (CEFOR-S) 1 g IV every 12 hr for additional antibacterial coverage. To protect liver function, Inj. Glutathione (GLUTAVERYL) 600 mg IV once daily was given, and Normal Saline (0.9%) with KCl 40 milliequivalents/L was infused at 10 mL/hr to maintain hydration and electrolyte balance

On Day 2, the patient continued with the same medications as prescribed on the previous day. As the patient experienced nausea and vomiting, Inj. Ondansetron (EMESET) 4 mg IV was administered as a stat dose to control nausea. All other medications were continued as per the previous schedule, and the patient remained in a stable condition.

On Day 3, there were no new medications added, and the patient was maintained on the same treatment regimen. The patient showed signs of mild clinical improvement, with reduced infection symptoms and better tolerance to therapy.

On Day 4, after further diagnostic evaluation, a definitive diagnosis of Bilateral Diffuse Cystic Lung Disease (B/L DCLD) was made. Based on this, new medications were introduced, including T. Theophylline (UNICONTINUE) 400 mg once daily as a bronchodilator, T. Acetylcysteine (LUMENAC) 600 mg once daily as a mucolytic agent, T. Diltiazem (ANGIZEM) 90 mg once daily for heart rate control, and T. Aspirin (ECOSPRIN) 75 mg once daily as an antiplatelet for supportive care. These additions aimed to improve breathing, reduce mucus buildup, and provide symptomatic relief.

On Day 5, the treatment plan was further expanded for respiratory stabilization and supportive care. The patient was started on Nebulization with Duolin (Levosalbutamol + Ipratropium) every 8 hr and Nebulization with Formanide (Formoterol + Budesonide) as needed to improve airflow and reduce inflammation. Inj. Multivitamin, 10 mL IV once daily was added for nutritional support, and T. Montair LC (Montelukast + Levocetirizine) once daily was given for antiallergic and bronchodilator support. Inj. Paracetamol (PCM) 1 g IV was administered as required for fever or pain, Syrup Bentonine (B-Complex + l-lysine) 10 mL

once daily was prescribed to boost appetite and recovery, and Inj. Ondansetron (EMESET) 4 mg IV was continued as needed for nausea. Overall, the patient's condition showed gradual improvement with this comprehensive treatment plan from Day 1 to Day 5 (Table 2).

DISCUSSION

DCLDs include a different type of pulmonary disorder characterized by the presence of multiple thin-walled cysts distributed throughout the tissues of the lung. A crucial part is diagnosing accurately, as varying treatment strategies are observed based on the particular underlying cause. Diagnosis for diffuse cystic lung disease is extensive, including various conditions such as lymphangioleiomyomatosis (LAM), Birt-Hogg-Dube syndrome, Pulmonary Langerhans' Cell Histiocytosis (PLCH), lymphocytic interstitial pneumonia, and certain metastatic cancers. Each of these disorders exhibits distinct clinical, radiological, and pathological features that facilitate diagnosis and management. For instance, LAM predominantly affects women of reproductive age and is characterized by extensive cystic alterations due to the proliferation of atypical smooth muscle-like cells. The average age at diagnosis is approximately 35 years, although rare cases have been reported in prepubescent girls and individuals in their eighties. Birt-Hogg-Dube syndrome is an autosomal dominant disorder caused by mutations in the folliculin gene, leading to pulmonary cysts, renal tumors, and skin manifestations. PLCH, often linked to smoking, typically presents with nodules and cysts, primarily in the upper lung lobes. A very rare lung condition known as PLCH typically, but not always, affects cigarette smokers. Lymphocytic interstitial pneumonia, associated with autoimmune conditions such as Sjögren's syndrome, is marked by widespread cysts and interstitial infiltrates. Additionally, metastatic cancers, including colorectal adenocarcinoma, may also present as diffuse cystic lung disease.

In this case, the definitive diagnosis was bilateral diffuse cystic lung disease (pulmonary Langerhans' cell histiocytosis). HRCT and computed tomography chest confirmed the diagnosis. Hence, a test like a biopsy is not recommended, and due to the high age, these invasive procedures are not advisable.

The main pathogenesis involved in the PLCH was A diverse group of antigen-presenting cells, dendritic cells are divided into several subsets based on their origin, location, surface phenotype, and functional characteristics. As a first line of defense, Langerhans' cells—a particular subpopulation of dendritic cells—are present in the epidermis and beneath the tracheobronchial tree's epithelium. They evaluate antigens that are deposited in the airway after inhalation. After coming into contact with danger signals, such as toll-like receptors expressed by infectious pathogens or substances secreted by nearby wounded or necrotic cells, these airway Langerhans' cells become activated. Numerous alterations brought about by activation facilitate antigen presentation and migration to local lymphoid tissues, which trigger adaptive immune responses. Additionally, Langerhans' cells are presumably crucial in limiting needless airway inflammation to harmless antigens deposited in the airway and in establishing tolerance toward benign inhaled antigens. Understanding the pathophysiology of PLCH requires deciphering the methods by which Langerhans' cells coordinate immune responses in the airways.

The differential diagnosis primarily includes cystic lung diseases like LCH and LAM. But LAM was less likely even though it shows the characteristics of multiple cysts, as LAM is mostly seen in and mostly impacts women who are of reproductive age and around 35 years old. But in this case, LAM was less likely due to the patient's gender and age, and LCH is more likely the definitive diagnosis. The presence of clinical manifestations, bilateral lung cysts, ground-glass attenuation, regions of breakdown and collapse consolidation in the left lower lobe, along with age and previous history of autoimmune disorders, are all factors that confirm PLCH. Thus, integrating the clinical presentation, radiological evidence, and laboratory findings, PLCH was confirmed as the most plausible diagnosis. Bronchodilators, corticosteroids, NSAIDs, analgesics, and antiemetics were used to treat the patient.

Table 1: Laboratory investigations report.

Test	Observed value	Reference value
Hematology		
WBC	14,880 cells/cumm	4,000 – 11, 000 cells/cumm
RBC	4.37 million cells/cumm	4.5-5.5 million cells/cumm
Basophils	0%	1-2%
CRP	55.7 mg/100 mL	<6 mg/100 mL
Electrolytes		
Potassium	2.9 g/100 mL	3.5-5.0 g/100 mL
Biochemistry		
HbA1c	7.1%	<5.7%
Liver function tests		
Total bilirubin	1.6 mg/100 mL	0.2-1.3 mg/100 mL
SGOT	55 U/L	<35 U/L
SGPT	52 U/L	<45 U/L

Abbreviations: CRP: C-reactive protein; RBC: Red blood cell; SGOT: Serum glutamate oxaloacetate transaminase; SGPT: Serum glutamate pyruvate transaminase; WBC: White blood cell.

Table 2: **Pharmacological treatment.**

Sl. No	Brand name	Generic name	Dose	ROA	Frequency	Duration
1.	Inj. MVI	Multivitamins	1injection	IV	OD (0-1-0)	5 days
2.	Inj. NEOMOL	Paracetamol	1 g	IV	TID (1-1-1)	4 days
3.	Inj. GLUTAVERGE	Glutathione	600 mg	IV	OD (1-0-1)	5 days
4.	Inj. EMESET	Ondansetron	4 mg	IV	SOS	5 days
5.	Inj. MERNEM	Meropenem	1 g	IV	TID (1-1-1)	5 days
6.	Inj. PAN	Pantoprazole	40 mg	IV	OD (1-0-0)	5 days
7.	T. ANGIZEM	Diltiazem	90 mg	PO	OD (1-0-0)	5 days
8.	T. MONTAIR-LC	Montelukast+ Levocetirizine	5 + 10 mg	PO	OD (0-0-1)	3 days
9.	T. LUMENAC	Acetylcysteine	600 mg	PO	BD (1-0-1)	5 days
10.	T. ASPIRIN	Aspirin	75 mg	PO	OD (0-1-0)	1 day
11.	T. UNICONTINE	Theophylline	400 mg	PO	OD (0-0-1)	5 days
12.	Neb. Formanide	Formoterol+ Budesonide	1respule	PN	BD (1-0-1)	5 days
13.	Neb. DUOLIN	Levosalbutamol + ipratropium	500 µg + 1.25 mg	PN	TID (1-1-1)	5 days
14.	Syp. BETONIN	B-complex vitamins	10 mL	PO	OD (0-0-1)	1 day

Abbreviations: MVI: Multivitamin.

Table 3: **Discharge medications.**

Sl. No.	Brand name	Generic name	Dose	ROA	Frequency	Duration
1.	T. ANXOTIC ALA	Multivitamins	1 tab	PO	OD (0-1-0)	5 days
2.	T. METRONIDAZOLE	Metronidazole	400 mg	PO	BD (1-0-1)	5 days
3.	T. DOLO	Paracetamol	650 mg	PO	TID (1-1-1)	4 days
4.	T. ZOFER	Ondansetron	4 mg	PO	SOS	5 days
5.	T. PAN	Pantoprazole	40 mg	PO	OD (1-0-0)	5 days
6.	T. ANGIZEM	Diltiazem	90 mg	PO	OD (1-0-0)	5 days
7.	T. MONTAIR-LC	Montelukast+ levocetirizine	5 + 10 mg	PO	OD (0-0-1)	3 days
8.	T. LUMENAC	Acetylcysteine	600 mg	PO	BD (1-0-1)	5 days
9.	T. ASPIRIN	Aspirin	75 mg	PO	OD (0-1-0)	5 days
10.	T. UNICONTINE	Theoph ylline	400 mg	PO	OD (0-0-1)	5 days
11.	Neb. Formanide	Formoterol+ budesonide	1respule	PN	BD (1-0-1)	5 days
12.	Neb. DUOLIN	Levosalbutamol + ipratropium	500 µg + 1.25 mg	PN	TID (1-1-1)	5 days
13.	Syp. BETONIN	B-complex vitamins	10 mL	PO	OD (0-0-1)	5 days

Both invasive and preventive techniques are used in management of PLCH. Bronchodilators, corticosteroids, NSAIDs, analgesics, and antiemetics were used to treat the patient in our case. At the time of release, the patient's condition was recovered and healthy, and their vital signs returned to normal (Table 3).

DCLD encompasses a group of rare pulmonary disorders characterized by the presence of multiple air-filled cysts of varying sizes and distributions throughout the lung parenchyma. These cysts often lead to respiratory complications such as breathlessness, persistent coughing, and recurrent pulmonary infections. Diagnosis typically involves HRCT and, in some cases, histopathological examination through lung biopsies.

Management of DCLD is largely dependent on the underlying cause, severity of symptoms, and disease progression.

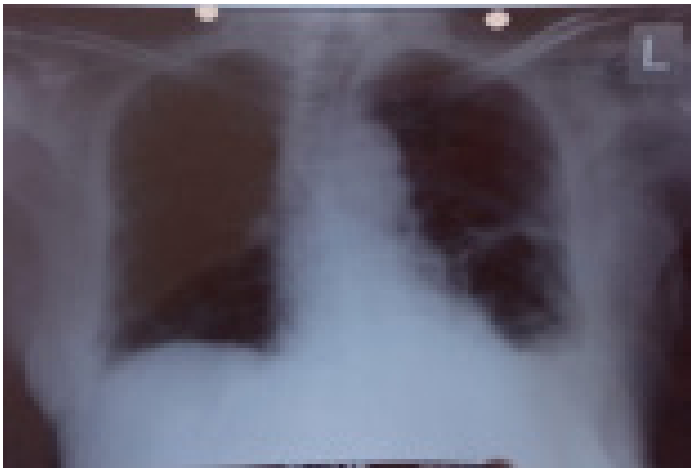

Figure 1: Chest Radiograph (Anteroposterior view).

Treatment strategies may include pharmacological therapy, regular monitoring, and in advanced cases, consideration for lung transplantation. Medications such as bronchodilators, corticosteroids, and nonsteroidal anti—inflammatory drugs are commonly employed to manage symptoms and reduce inflammation.

CONCLUSION

This case underscores the importance of a multidisciplinary approach involving pulmonologists, radiologists, and thoracic surgeons to provide comprehensive care. Regular follow up and individualized treatment adjustments are essential for optimizing outcomes and improving the quality of life in patients diagnosed with DCLD.

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ABBREVIATIONS

DCLD: Diffuse cystic lung disease; **NSAIDs:** Non-steroidal anti-inflammatory drug; **B/L DCLD:** Bilateral diffuse cystic lung disease; **LAM:** Lymphangioleiomyomatosis; **HRCT:** High resolution computed tomography; **HbA1c:** Glycated hemoglobin; **B/L VBS:** Bilateral vesicular breath sounds; **IV:** Intravenous; **mEq/L:** Milliequivalent per liter; **SpO^2:** Oxygen saturation; **PLCH:** Pulmonary langerhans cell histiocytosis.

CONFLICT OF INTEREST

The authors declare that there is no conflict of interest.

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ETHICAL STATEMENTS

All necessary measures have been taken to safeguard patient confidentiality.

REFERENCES

Baldi, B. G., Carvalho, C. R., Dias, O. M., Marchiori, E., & Hochhegger, B. (2017). Diffuse cystic lung diseases: Differential diagnosis. *Jornal Brasileiro de Pneumologia: Publicacao Oficial da Sociedade Brasileira de Pneumologia e Tisilogia*, *43*(2), 140-149. doi: https://doi.org/10.1590/s1806-37562016000000341101590/s1806-37562016000000341, PubMed: https://www.ncbi.nlm.nih.gov/pubmed/28538782.

Colombat, M., Caudroy, S., Lagonotte, E., Mal, H., Danel, C., Stern, M., (2008). Pathomechanisms of cyst formation in pulmonary light chain deposition disease. *European Respiratory Journal*, *32*(5), 1399-1403. doi: 10.1183/09031936.00132007, PubMed: 18978139.

Franciosi, A. N., Gupta, N., Murphy, D. J., Wikenheiser-Brokamp, K. A., & McCarthy, C. (2024). Diffuse cystic lung disease: A clinical guide to recognition and management. *Chest*, *167*(2), 529-547. doi: 10.1016/j.chest.2024.08.008, PubMed: 39168181.

Gupta, N., Langenderfer, D., McCormack, F. X., Schauer, D. P., & Eckman, M. H. (2017). Chest computed tomographic image screening for cystic lung diseases in patients with spontaneous pneumothorax is cost effective. *Annals of the American Thoracic Society*, *14*(1), 17-25. doi: 10.1513/AnnalsATS.201606-459OC, PubMed: 27737563.

Gupta, N., Vassallo, R., Wikenheiser-Brokamp, K. A., & McCormack, F. X. (2015). Diffuse cystic lung disease. Part I. *American Journal of Respiratory and Critical Care Medicine*, *191*(12), 1354-1366. doi: 10.1164/rccm.201411-2094CI, PubMed: 25906089.

Gupta, N., Wikenheiser-Brokamp, K. A., Fischer, A., & McCormack, F. X. (2016). Diffuse cystic lung disease as the presenting manifestation of Sjögren syndrome. *Annals of the American Thoracic Society*, *13*(3), 371-375. doi: 10.1513/AnnalsATS.201511-759BC , PubMed: 26741500.

Hansell, D. M., Bankier, A. A., MacMahon, H., McLoud, T. C., Müller, N. L., & Remy, J. (2008). Fleischner Society: Glossary of terms for thoracic imaging. *Radiology*, *246*(3), 697-722. doi: 10.1148/radiol.2462070712, PubMed: 18195376.

Kikuchi, E., Kinoshita, I., Yamazaki, K., Itoh, T., Shimizu, T., Shimizu, H., & Nishimura, M. (2006). Epithelioid sarcoma presenting as pulmonary cysts with cancer antigen 125 expression. *Respirology*, *11*(6), 826-829. doi: 10.1111/j.1440-1843.2006.00925.x, PubMed: 17052316.

Kunisaki, S. M., Saito, J. M., Fallat, M. E., St Peter, S. D., Kim, A. G., Johnson, K. N. (2019). Current operative management of congenital lobar emphysema in children: A report from the Midwest Pediatric Surgery Consortium. *Journal of Pediatric Surgery*, *54*(6), 1138-1142. doi: 10.1016/j.jpedsurg.2019.02.043, PubMed: 30898401.

Lee, K. C., Kang, E. Y., Yong, H. S., Kim, C., Lee, K. Y., Hwang, S. H., & Oh, Y. W. (2019). A stepwise diagnostic approach to cystic lung diseases for radiologists. *Korean Journal of Radiology*, *20*(9), 1368-1380. doi: 10.3348/kjr.2019.0057, PubMed: 31464115.

Ohdama, S., Akagawa, S., Matsubara, O., & Yoshizawa, Y. (1996). Primary diffuse alveolar septal amyloidosis with multiple cysts and calcification. *European Respiratory Journal*, *9*(7), 1569-1571. doi: https://doi.org/10.1183/09031936.96.0907156910.1183/09031936.96.09071569, PubMed: https://www.ncbi.nlm.nih.gov/pubmed/8836676883676.

Paciocco, G., Uslenghi, E., Bianchi, A., Mazzarella, G., Roviaro, G. C., Vecchi, G., & Harari, S. (2004). Diffuse cystic lung diseases: Correlation between radiologic and functional status. *Chest*, *125*(1), 135-142. doi: 10.1378/chest.125.1.135, PubMed: 14718432.

Park, S., & Lee, E. J. (2017). Diagnosis and treatment of cystic lung disease. *Korean Journal of Internal Medicine*, *32*(2), 229-238. doi: 10.3904/kjim.2016.242, PubMed: 28264540.

Ryu, J. H., & Swensen, S. J. (2003). Cystic and cavitary lung diseases: Focal and diffuse. *Mayo Clinic Proceedings*. Amsterdam: Elsevier, *78*(6), 744-752). doi: 10.4065/78.6.744 , PubMed: 12934786.

Singla, A., & Gupta, N. (2021). Approach to the diagnosis and management of diffuse cystic lung diseases. In N. Gupta, K. A. Wikenheiser-Brokamp & F. X. McCormack (Eds.),*Diffuse cystic lung diseases* (pp. 339-370). Cham: Springer International Publishing. doi: 10.1007/978-3-030-63365-3_18.

Wang, L., Liu, J., Yang, H., & Peng, L. (2023). Diffuse cystic lung disease caused by tuberculosis infection: Case series. *Journal of Infection and Public Health*, *16*(4), 526-530. doi: 10.1016/j.jiph.2023.02.013, PubMed: 36801632.

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