Journal of
Medical Case Reports

CASE REPORT

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Kartagener syndrome in a young Ethiopian boy: a case report

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Abstract

Introduction Kartagener syndrome is a subset of a larger group of ciliary motility disorders called primary ciliary dyskinesias. The syndrome includes the clinical triad of chronic sinusitis, bronchiectasis, and situs inversus. Patients usually present with recurrent respiratory tract infections due to ineffective mucociliary clearance. Females and males are equally affected. Kartagener syndrome occurs in about 1 in 32,000 to 40,000 births worldwide.

Case presentation This case involved a 17-year-old Black African Semitic male patient who presented to our hospital with a complaint of intermittent productive cough, which started when he was 5 years old. He had a history of repeated treatment for lower respiratory tract infection and chronic sinusitis with frequent exacerbation. On examination, he had coarse crackles over the left posterior lower lung field. Heart sounds were appreciated on the right side. During imaging investigations, his chest X-ray posterior-anterior view showed dextrocardia and right side gastric shadow with left paracardiac bronchiectatic changes. A high-resolution chest computed tomography scan was suggestive of complete situs inversus. There were left lower lobe bronchiectatic changes. An electrocardiogram showed features of dextrocardia. Routine laboratory tests were within normal range. He was treated with thoracic physiotherapy, azithromycin 500 mg three times per week, and mucolytics, with no apparent exacerbations in the last 6 months.

Conclusion The diagnosis of Kartagener syndrome is typically delayed because the clinical symptoms are easily mistaken for common infections. Since there is no specific treatment for Kartagener syndrome, early diagnosis and management of Kartagener syndrome are critical to prevent irreversible lung damage and chronic lifelong sequelae. A high index of suspicion is needed to make an early diagnosis so that timely treatment options may be offered to prevent problems associated with Kartagener syndrome.

Keywords Kartagener syndrome, Primary ciliary dyskinesias, Bronchiectasis, Situs inversus, Dextrocardia

Introduction

Kartagener syndrome (KS) is a subset of a larger group of ciliary motility disorders called primary ciliary dyskinesias (PCDs) [1]. It is a rare autosomal recessive genetic disorder that was first described by Siewert in 1904; however, Manes Kartagener recognized the clinical syndrome in 1933 [2]. The syndrome includes the clinical triad of chronic sinusitis, bronchiectasis, and situs inversus. The *dynein axonemal intermediate chain 1* (DNAI1) and *dynein axonemal heavy chain 5* (DNAH5) gene mutation in KS causes decreased ciliary motility, which increases the risk of infertility, left–right body alignment problems,

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and recurrent sinopulmonary infections. Normal ciliary function is crucial for sperm movement, respiratory system defense, and the proper positioning of internal organs during development [2]. In the embryonic phase, consistent ciliary movement dictates the placement of internal organs; however, in Kartagener syndrome (KS), the organs do not migrate to the left side owing to impaired ciliary movement, leading to situs inversus. Individuals with KS often experience repeated respiratory infections owing to ineffective mucociliary clearance. [3]. PCD does not have a gold-standard diagnostic test and requires multiple complementary tests. The European Respiratory Society guidelines suggest using nasal nitric oxide (nNO) measurement, high-speed video microscopy analysis (HSVA), transmission electron microscopy (TEM), and genetic testing. If TEM is not available, immunofluorescence labeling of ciliary proteins can suggest a likely diagnosis. Overall, ciliary ultrastructural and beat pattern correlate well with the genotype; however, 26% of documented PCD patients (via genetics, nNO, and TEM) do not have ultrastructural abnormalities [4]. Females and males are equally affected [5]. The incidence of PCD in the world varies between 1 in 20,000 to 1 in 40,000 births. KS occurs in about 1 in 32,000 births worldwide [6, 7]. The prevalence or incidence of Kartagener syndrome (KS) in Ethiopia is currently unknown, and only a limited number of case reports exist, highlighting the rarity of this condition. Factors such as being a low-middle-income country, which restricts access to diagnostic testing, may contribute to the infrequent recognition of the disease. In this report, we present a case involving a 17-year-old boy diagnosed with Kartagener syndrome. This case report aims to positively influence the scientific community by underscoring the importance of early diagnosis and intervention for this disease.

Case presentation

A 17-year-old Black African Semitic male patient came to our hospital reporting an intermittent cough that produces thick, whitish mucus. The cough began when he was 5 years old, but he did not seek medical attention at that time. His first visit to a healthcare facility occurred 5 years ago, during which he reported symptoms of nasal congestion, sneezing, and difficulty with smell. For these issues, he consulted different hospitals and was evaluated by an ear, nose, and throat (ENT) specialist, who diagnosed him with chronic sinusitis. He was treated with saline rinses and augmentin 625 mg three times per day. Following this, he had improvement of symptoms for a few weeks. He did not go back to the ENT specialist for follow-up owing to financial crisis. He had a history of repeated treatment for lower respiratory tract infections at local health institutions. He used to have exacerbation of these symptoms every 2–4 weeks. At present, he has been facing a progressive worsening of his productive cough, which is aggravated by walking over 30 min and alleviated by taking breaks. Furthermore, he has a history of nasal congestion and recurrent sneezing; he visited a local health center, where he was referred to our hospital. He has no history of smoking or alcohol intake. His mother reported that he was delivered at home after a full-term pregnancy via spontaneous vaginal delivery. He has no significant history of developmental delays. He is the firstborn child and has one younger brother, with no similar issues reported among other family members.

On physical examination, he was fairly stable with normal vital signs. His blood pressure measured 120/80 mmHg, with a pulse rate of 88 beats per minute, a respiratory rate of 24 breaths per minute, an axillary temperature of 36.8 °C, and an oxygen saturation level of 94% on room air. His body mass index was 16 (underweight). There were no visible nasal polyps present. He showed no signs of chest deformity or nasal flaring. No subcostal or intercostal retractions were noted. Coarse crackles were heard in the left posterior lower lung field. There was no cyanosis or clubbing. Normal heart sounds detected were at the base of the heart on the right side, and the point of maximal impulse was located at the right fifth intercostal space. No murmurs or gallops were detected.

He was admitted to the medical ward for investigation. A chest X-ray in the posterior-anterior (PA) view revealed dextrocardia along with a gastric shadow on the right side and bronchiectatic changes adjacent to the left cardiac border (Fig. 1). A high-resolution chest computed tomography (CT) scan demonstrated a complete reversal of the heart and major vessels, indicating dextrocardia (Fig. 2). The abdominal organs visualized on the CT scan displayed the stomach and spleen positioned on the right and the liver on the left, indicating complete situs inversus (Fig. 3). Additionally, there were bronchiectatic changes in the left lower lobe (Fig. 4). When an electrocardiogram (ECG) was performed, it revealed a right axis deviation, an inverted P wave in lead I, and an avL that suggested dextrocardia (Fig. 5). A two-dimensional echocardiogram revealed dextrocardia, with all four chambers normal. A dextrocardia exhibiting situs inversus of the aorta and inferior vena cava was confirmed by a Doppler examination. The results of routine laboratory tests performed to rule out concomitant infectious processes, such as the complete blood count and the erythrocyte sedimentation rate (ESR), were normal. Liver and renal function tests and fasting blood sugar were within the normal range (Table 1).

He declined to undergo a semen analysis. We consulted an ENT specialist, who after careful evaluation, suggested conducting tests related to Kartagener syndrome,

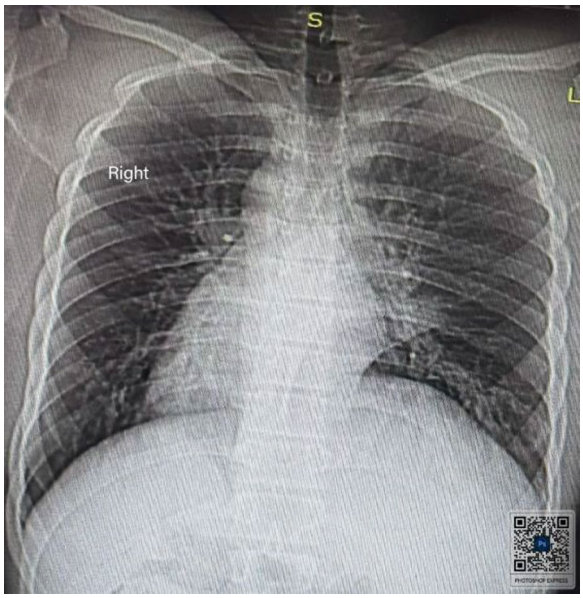

Fig. 1 Chest X-ray on posterio-anterior view showed dextrocardia with bronchiectatic changes in the left paracardiac lung parenchyma

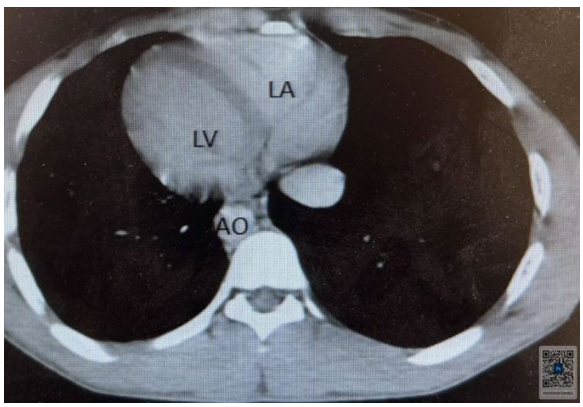

Fig. 2 High-resolution chest computed tomography scans showing complete reversal of heart and great vessels. *LV* left ventricle, *LA* left atrium, *AO* aorta

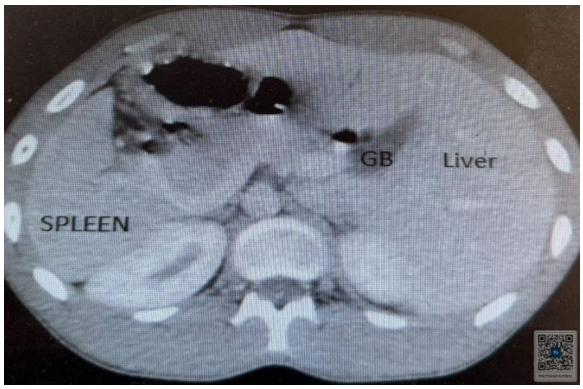

Fig. 3 High-resolution chest computed tomography scans showing abdominal organs, with stomach and spleen on right side and liver on left side, suggestive of complete situs inversus

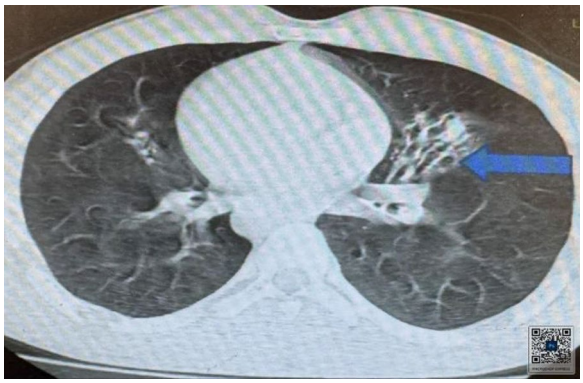

Fig. 4 High-resolution chest computed tomography scans showing lower lobe bronchiectatic changes (blue arrow)

including an audiometric test to evaluate hearing and ciliary motility studies to examine the function of cilia in the respiratory system. Regrettably, neither of these tests could be performed in the town because they were not available.

Following thorough evaluation and imaging tests, he was diagnosed as having Kartagener syndrome. He spent 3 days in the hospital. Education and counseling regarding his condition were provided. Upon discharge, he was recommended to undergo thoracic physiotherapy, take azithromycin 500 mg three times a week, and take guaifenesin syrup 200 mg three times daily. The focus of thoracic physiotherapy for Kartagener syndrome is on employing techniques such as postural drainage, percussion, and vibrations to aid in mucus clearance from the airways, since the condition causes compromised ciliary function, making it challenging for the body to expel secretions naturally. Azithromycin may assist in managing recurring respiratory infections by functioning as a preventive antibiotic owing to its antiinflammatory effects, especially during episodes of the illness; in essence, it is utilized to alleviate symptoms and avert complications linked to the syndrome by diminishing the bacterial load in the airways. Mucolytic agents are frequently used to thin mucus and facilitate its clearance.

After a month, he returned to our hospital for his scheduled appointment. He reported that his cough had improved and mentioned experiencing one exacerbation 3 weeks after being discharged, which involved a worsening of cough accompanied by the production of yellowish sputum and difficulty breathing. He sought help at the local health center and was treated with cephalexin 500 mg two times daily for 1 week, resulting in significant

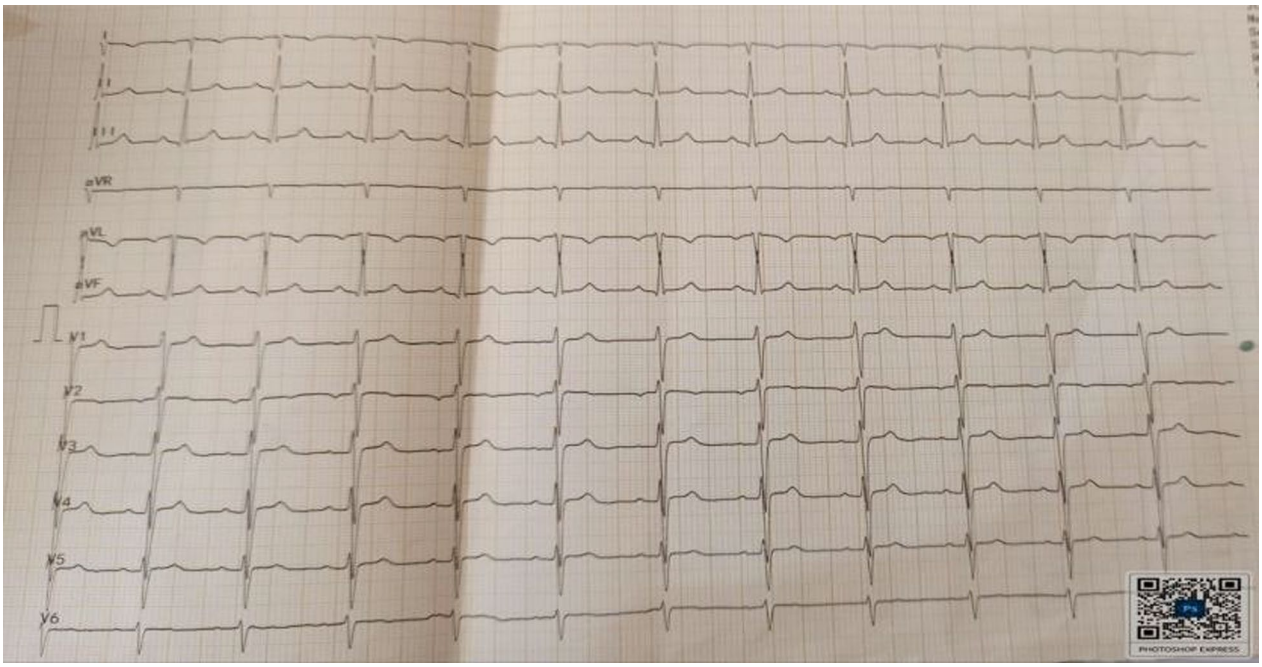

Fig. 5 Electrocardiogram showing right axis deviation, inverted P wave in lead I, and avL suggestive of dextrocardia

Table 1 Laboratory values from admission time and his follow up visits with reference ranges

Lab results	At admission	After 1 month	After 6 months	Reference
Hgb (g/dL)	13.8	13.1	13.6	12.3–15.3
WBC (10^3/ul)	6.6	7.8	6.8	4.00–11.00
Neutrophils (%)	56	60	64	40–60
Lymphocytes (%)	25	22	18	20–40
FBS (mg/dL)	98	84	NA	70–100
Creatinine (mg/dL)	0.9	NA	0.8	0.3–1.3
Urea nitrogen (mg/dL)	20	NA	20	6–22
ESR	11	NA	NA	0–20
Gene-Xpert MTB/RIF assay from sputum	Negative	NA	NA	Negative

Hgb hemoglobin, *WBC* white blood cells, *ALT* alanine aminotransferase, *AST* aspartate aminotransferase, *ALP* alkaline phosphatase, *ESR* erythrocyte sedimentation rate, *MTB/RIF* mycobacterium tuberculosis/rifampicin

improvement after 4 days of treatment. Upon examination, his vital signs were all within normal limits. A notable finding during the lung exam was the presence of coarse crackles in the left posterior lower lung field. His complete blood count was within the normal range. He returned 3 months after the initial diagnosis, stating that he had not experienced any exacerbations since the last follow-up. He continued his regimen of azithromycin and chest physiotherapy.

During his follow-up appointment 6 months later, he reported no exacerbations and noted an increase in his exercise tolerance. He stated that he could walk for up to 60 min without experiencing any symptoms when visiting his uncle. There were no significant findings during the physical examination. It was recommended that he continue with chest physiotherapy and take azithromycin 500 mg three times weekly. He also received vaccinations for influenza and pneumococcal disease. This was his latest follow-up appointment, and he was scheduled to return after 3 months.

On this visit, he came with his younger brother, who was 12 years old. We inquired about any respiratory issues, such as cough, chest pain, shortness of breath, runny nose, or frequent sneezing. He indicated that he

had experienced none of these symptoms. His vital signs were within the normal limits, showing a blood pressure of 110/70 mm Hg, a pulse rate of 68 beats per minute, a respiratory rate of 14 breaths per minute, and an oxygen saturation of 94% in room air. The examination of his respiratory system showed normal air entry in both lungs. There was no clubbing of fingers. We performed a chest X-ray, abdominal ultrasound, electrocardiogram, and echocardiogram to look for any abnormalities on the lung parenchyma, including bronchiectasis, and to check for the normal positions of his organs including the heart, liver, and spleen. All tests were found to be normal.

Discussion

Ciliary motility disorders can be either congenital or acquired. Congenital disorders are referred to as primary ciliary dyskinesia (PCD). Nearly half of patients with PCD exhibit situs inversus. Cases of PCD accompanied by situs inversus are termed Kartagener syndrome [1]. Primary ciliary dyskinesia (PCD) is a genetically diverse condition stemming from defects in motile cilia. Although the exact prevalence is not known, the estimated incidence is around 1 in 20,000 to 40,000 live births [2, 6]. Since Siewert's initial description of the condition in 1904, additional cases of Kartagener syndrome have been documented, including in African countries [7–11].

Our patient in this report presented with situs inversus totalis, characterized by the heart and abdominal organs being arranged in a mirror image to the normal anatomy. Most mutations associated with this condition are believed to involve two specific genes, DNAH5 and DNAI1. The full syndrome exhibits significant familial patterns, often manifesting within a single generation and among multiple siblings. These characteristics, along with the high prevalence of consanguinity among apparently unaffected parents of the afflicted children, support the idea that the genetic anomaly is inherited as an autosomal recessive trait [1]. Since we do not have access to genetic testing, saccharin testing, radioaerosol testing, or nasal nitric oxide (NO) testing at our facility, we conducted a thorough evaluation of his parents and brother, which resulted in normal findings. None of them experienced any respiratory problems, and physical assessments of all systems were within normal parameters. Imaging studies of his brother demonstrated normal lung tissue and correct placement of all internal organs.

Clinical manifestations of PCD can differ; some neonates may experience respiratory distress at birth, while others might later develop a chronic cough that produces mucus, often caused by bronchiectasis, which does not respond to treatment. They may also experience atypical asthma as well as chronic rhinosinusitis and otitis. In women, this condition can lead to ectopic pregnancies and subfertility, while men may face infertility [12]. The condition is congenital, with symptoms typically appearing at a young age, highlighting the necessity for pediatricians to recognize this disorder as a significant, although rare, differential diagnosis in children who exhibit recurrent symptoms in both the upper and lower respiratory systems [13]. As the condition advances, clinical and radiographic signs of bronchiectasis become evident; preschool-aged children may show signs of bronchiectasis and obstructive impairment [14]. Our patient showed signs of a long-standing productive cough along with a history of multiple pneumonia treatments. He also experiences ongoing issues related to rhinosinusitis. Chronic complications that may stem from KS include infertility, bronchiectasis, chronic bronchitis, chronic sinusitis, frequent respiratory infections, and chronic pulmonary hypertension. Routine vaccinations for influenza and pneumococcal disease, along with avoiding polluted environments, can help prevent recurrent infections [2, 15]. Our patient had manifested bronchiectasis in the left lower lobe, chronic sinusitis, and experienced repeated respiratory tract infections during his admission to our hospital.

Screening methods consist of saccharin testing, radioaerosol testing, and nasal nitric oxide (NO) assessment. Children who exhibit clinical signs of PCD, have radiographic results indicating bronchiectasis or bronchial wall thickening, or present with heterotaxy should undergo screening, while ensuring the exclusion of diseases that may present similarly, such as cystic fibrosis, immunodeficiency, and chronic aspiration. Any positive results from screening require follow-up with confirmatory diagnostic tests. The diagnosis of Kartagener syndrome is primarily based on clinical symptoms, supported by various diagnostic evaluations including radiological examinations (chest x-ray and computed tomography), ciliary motility assessments (nasal biopsy and electron microscopy, high-speed video microscopy, and semen analysis) and genetic analysis [16]. The suggested diagnostic criteria for this syndrome include a history of chronic bronchial infections and rhinitis dating back to early childhood, along with one or more of the following characteristics: (1) situs inversus or dextrocardia in the patient or a sibling, (2) spermatozoa that are present but lack motility, (3) absent or compromised tracheobronchial clearance, and (4) cilia exhibiting specific ultrastructural defects, as seen through electron microscopy [2].

Diagnosis can be established through tests that demonstrate dysfunctional cilia, biopsies, and genetic examinations. Analysis of semen in males after puberty may indicate either abnormal sperm movement or aspermia.

The Saccharin test serves as a preliminary assessment to identify dysfunctional mucociliary clearance. This assessment evaluates the duration required for a saccharin pellet placed on the inferior turbinate to be tasted, with 30 min being the threshold that differentiates healthy individuals from those with impaired nasal mucociliary clearance. The measurement of exhaled nasal nitric oxide involves evaluating the expired NO from one nostril. Although there are no standardized cutoff values, nasal nitric oxide levels in individuals with PCD typically range from only 10% to 20% of the average normal levels [1]. Our patient declined to undergo a semen analysis, which prevented us from verifying abnormal sperm motility. We did not perform the other necessary tests owing to their unavailability at our hospital. The limited access to various diagnostic options posed a considerable challenge for diagnosis. Our patient encountered several instances of sinopulmonary infections. Imaging results revealed signs of bronchiectasis, dextrocardia, and situs inversus, meeting the diagnostic criteria for KS. Laboratory screening assessments and confirmatory tests, which required a better clinical setting, were not carried out.

When a diagnosis of Kartagener syndrome is considered, other differential diagnosis should also be considered and ruled out, such as allergic bronchopulmonary aspergillosis, alpha1-antitrypsin (AAT) deficiency, bronchial obstruction, asthma, chronic aspiration, chronic obstructive pulmonary disease (COPD), congenital cartilage deficiency, cystic fibrosis, foreign body aspiration, and idiopathic interstitial pneumonia [17]. However, in our case, the patient's medical history revealed chronic rhinosinusitis with recurrent exacerbations of respiratory tract symptoms. Additionally, the presence of situs inversus totalis provided a significant clue supporting the inclusion of KS as the primary consideration among the differential diagnoses.

As there are no curative options yet, PCD treatment is directed at preventing and managing disease complications. Treatment guidelines are mainly based on expert opinion and extrapolations from cystic fibrosis and non-cystic fibrosis bronchiectasis guidelines, despite having distinct pathophysiologies. As a result of the paucity of evidence-based therapeutic options, treatment approaches have varied widely between different countries. An unequivocal beneficial effect was established on azithromycin maintenance therapy and, as such, this is the only evidence-based treatment found so far [18]. Our patient was treated with azithromycin three times per week, chest physiotherapy, and mucolytics. He showed significant symptomatic improvement. He has been to regular appointment visits for the last 6 months.

A retrospective study conducted in China included PCD patients from 2009 to 2021 to evaluate differences in clinical outcomes, pulmonary function, and chest CT findings between those who received azithromycin and those who did not. Patients treated with azithromycin experienced fewer respiratory exacerbations and demonstrated improved exercise tolerance compared with the group that did not receive the medication [19]. The primary advantage of azithromycin in Kartagener syndrome lies in its antiinflammatory properties, which can aid in diminishing airway inflammation and mucus production, typical symptoms of the disorder. In certain instances, a prolonged regimen of low-dose azithromycin might be contemplated to avert recurrent infections and address ongoing symptoms [2, 19].

Bronchiectasis and pneumonia can be managed through the use of inhaled bronchodilators, mucolytics, oral corticosteroids, and chest physiotherapy. Physiotherapy plays a crucial role in enhancing the clearance of the respiratory tract to help delay the onset and advancement of obstructive respiratory conditions. Engaging in physical exercise may assist in sputum clearance and has been shown to be more effective as a bronchodilator than bronchodilator medications themselves in individuals with PCD [13, 20]. It is essential to administer influenza and pneumococcus vaccines to reduce the risk of recurrent infections. Effective respiratory care for patients with PCD is crucial in avoiding permanent lung injury and may help prevent or slow the advancement of lung damage once a diagnosis is confirmed [19].

Alternative treatments for KS that have been utilized include recombinant human deoxyribonuclease (rhDNase), hypertonic saline, and acetylcysteine. While their effectiveness has not been established, they are still sometimes employed in efforts to provide symptomatic relief [1, 21]. In certain cases of advanced KS, a double lung transplant might be beneficial. There are reports of effective pulmonary procedures in a 23-year-old male, which included a left middle lobectomy, as well as successful lung transplantation in a 49-year-old female [22, 23]. An important part of the clinical visits at regular intervals should be monitoring the progression of the lung disease. The prognosis is generally considered favorable, and life expectancy is usually normal [13]. We aim to maintain this patient's treatment plans while conducting evaluations every 3–6 months. Given the lack of a standard treatment protocol globally, it is essential to develop interventions on the basis of evidence. Consequently, further trials are required to assess the efficacy of various mucolytics, maintenance antibiotic treatments, gene therapies, physical activity, and chest physiotherapy.

Conclusion

The diagnosis of KS is typically delayed because the clinical symptoms are easily mistaken for common infections. Since there is no specific treatment for KS, early diagnosis and management of KS are critical to prevent irreversible lung damage and chronic lifelong sequelae. Therefore, when a patient shows signs of recurrent respiratory infections and structural lung issues, KS should be included in the differential diagnosis. Accurately identifying KS during early life is vital for the overall prognosis of the syndrome, as many long-term complications can be mitigated with prompt management. An elevated level of suspicion and a collaborative approach involving a multidisciplinary team are necessary for making an early diagnosis, which allows for timely treatment options to prevent complications related to KS.

Acknowledgements
Not applicable.

Declaration of figure authenticity
The figures are created by the authors, who confirmed that the images are original with no duplication and have not been previously published in whole or in part.

Author contributions
HM and AL prepared the manuscript. SA conceptualized and prepared the images. All authors reviewed the manuscript.

Funding
None.

Availability of data and materials
No datasets were generated or analyzed during the current study.

Declarations

Ethics approval and consent to participate
Ethical clearance was obtained from the ethical committee of Woldia Comprehensive Specialized Hospital, and consent was obtained from the parents of our patient to prepare the case report.

Consent for publication
Written informed consent was obtained from the patient's legal guardian for publication of this case report and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

Competing interests
None.

Received: 4 February 2025 Accepted: 4 April 2025
Published online: 17 May 2025

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