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When Wheezing is Not Asthma: A Case of Kartagener's Syndrome

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ABSTRACT

Kartagener's syndrome, a distinct form of primary ciliary dyskinesia, is characterized by the classic triad of chronic sinusitis, bronchiectasis, and situs inversus. We report the case of an 18-year-old male, followed since childhood for severe treatment-resistant asthma, admitted with acute respiratory distress. Investigations revealed bilateral infected bronchiectasis, complete situs inversus, and chronic sinusitis, confirming the diagnosis of Kartagener's syndrome. Management included oxygen therapy, empirical antibiotic therapy, respiratory physiotherapy, and updated vaccinations, with a favorable outcome. This case illustrates that wheezing does not always correspond to asthma, highlighting the importance of an accurate etiological diagnosis and appropriate management to prevent long-term respiratory and cardiovascular complications.

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KEYWORDS:
Kartagener's syndrome, Primary ciliary dyskinesia, Bronchiectasis, Situs inversus, Chronic sinusitis, Wheezing, Atypical asthma, Etiological diagnosis, Respiratory management, Cardiovascular complications

INTRODUCTION

Kartagener's syndrome is a particular form of primary ciliary dyskinesia (PCD). It is an autosomal recessive genetic disorder characterized by abnormalities in ciliary morphology and motility, leading to impaired mucociliary clearance [1]. Clinically, it is defined by a triad combining chronic sinusitis, bronchiectasis, and total or partial situs inversus. In our case, this syndrome was identified in the context of acute respiratory distress.

CASE REPORT

We report the case of an 18-year-old male admitted for acute respiratory distress, in whom the etiological workup led to the diagnosis of Kartagener's syndrome. He had been followed since early childhood for severe asthma refractory to standard therapies. His family history revealed paternal grandfather with asthma, with no reported consanguinity in the family and no other significant antecedents.

The patient was admitted to the pulmonology department for acute respiratory distress on a background of chronic symptoms. These included paroxysmal productive cough with greenish sputum sometimes streaked with blood, without diurnal predominance, associated with diffuse burning chest pain of moderate intensity (3/10 on VAS). He also complained of exertional and resting dyspnea, classified as stage III according to Sadoul, without orthopnea. In addition, he reported abdominal bloating, hypogastric pain without bowel movement or flatus arrest, and partial anosmia. The symptoms evolved in a febrile context (predominantly nocturnal) with deterioration of general condition.

Clinical examination revealed a patient in respiratory distress with tachypnea at 32 cycles per minute and oxygen saturation at 80% on room air. There was bilateral digital clubbing and bilateral lower-limb edema with pitting up to the knees. Thoracic inspection showed distension; pulmonary auscultation revealed diffuse bilateral crackles and wheezes. The abdomen was distended, without palpable organomegaly. ENT examination revealed congestive nasal mucosa, bilateral mucopurulent discharge, and tenderness over the maxillary sinuses. A right hydrocele with positive transillumination was also noted.

Contrast-enhanced cervicofacial and thoracoabdominal CT scan revealed several abnormalities. Facial and cervical slices showed almost complete mucopurulent filling of the maxillary sinuses with ethmoidal mucosal thickening. No cervical lymphadenopathy or vascular abnormalities were found. Thoracoabdominal images demonstrated bilateral cystic and cylindrical bronchiectasis predominantly in the lower lobes (Figures 1 and 2), with bronchial wall thickening and mucoid impactions (Figures 1 and 2). A complete situs inversus was evident, with dextrocardia (Figure 4), the liver

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located on the left, the spleen on the right, and inversion of other thoracoabdominal structures (Figure 3).

Echocardiography confirmed dextrocardia, showing right-sided positioning of all cardiac chambers, associated with dilation of the right cavities and pulmonary arterial hypertension estimated at 75 mmHg, without intracardiac thrombus.

Based on these findings, the diagnosis of Kartagener's syndrome with superinfected bronchiectasis was established. The patient was hospitalized and placed on low-flow oxygen therapy (3 L/min). Empirical intravenous antibiotic therapy with amoxicillin– clavulanic acid (1 g/125 mg every 8 hours for 10 days) was initiated to treat bronchiectasis superinfection. Active and passive respiratory physiotherapy was also undertaken. Influenza, pneumococcal, and SARS-CoV-2 vaccinations were scheduled. Referral to urology was arranged for hydrocele management.

The clinical course was rapidly favorable, marked by improvement in oxygen saturation allowing withdrawal of oxygen therapy with maintenance of satisfactory SpO_2 on room air, reduction in tachypnea, resolution of fever, and regression of chest pain. Sputum production became less abundant and less purulent, and the patient's general condition improved significantly.

At discharge, continuation of oral antibiotic therapy, respiratory physiotherapy, regular pulmonology follow-up, as well as ENT and urology evaluations were recommended.

DISCUSSION

Kartagener's syndrome was first described in 1935 by Manes Kartagener. It is classically defined by the triad of bronchiectasis, chronic sinusitis, and situs inversus [2]. It accounts for approximately 50% of cases of primary ciliary dyskinesia (PCD). This rare genetic disorder is most commonly transmitted in an autosomal recessive pattern, although dominant and Xlinked forms have also been reported [2].

The pathogenesis is based on mutations in the genes encoding dynein, located on chromosomes 5, 9, and 7, leading to morphological and/or functional abnormalities of the cilia [2]. The age at diagnosis varies across studies, with clinical manifestations often appearing early in childhood [2,3,4,5]. In a study by Elguedri, the mean age at diagnosis was 17 years, ranging from 6 to 27 years [6]. Our patient, diagnosed at 18 years of age, therefore falls within this range, in line with literature data.

In rare cases, PCD may be associated with additional malformations, particularly cardiac, renal, or sensory anomalies, resulting in complex phenotypes. However, clinical manifestations primarily involve the respiratory system and the genital tract [7,8].

Respiratory symptoms appear early, with signs of respiratory distress reported in 50–70% of cases in different series [9]. Other symptoms, although non-specific, are consistently chronic in nature: chronic productive cough, daily bronchorrhea, and exacerbations with mucopurulent sputum sometimes streaked with blood. These episodes may be accompanied by fever and wheezing, mimicking atypical asthma, which often fails to respond to conventional therapy [9], as observed in our patient.

Bronchiectasis is a constant finding in adults, with a predominance in dependent lung regions such as the middle lobe, lingula, and lower lobes [10]. In our case, bronchiectasis predominated in the lower lobes, consistent with the usual topographic distribution described in the literature.

Cardiac malformations are found in approximately 3% of patients with Kartagener's syndrome [11,12]. In addition, several authors have reported cases of pulmonary arterial hypertension (PAH) [13]. Our patient presented with complete situs inversus without associated cardiac malformations, but with significant PAH.

Rhinosinusal involvement in PCD is less extensively documented in the literature. Nasal cavity examination, ideally performed under endoscopy, usually reveals diffuse mucosal inflammation with thick, adherent secretions. Sinus opacifications are most often partial. In older children and adults, hypoplasia or even agenesis of the frontal sinuses may be observed, along with opacification of the maxillary sinuses [14]. Our patient presented with mucopurulent filling of the maxillary sinuses, in perfect agreement with these reported findings.

To date, no specific etiological treatment exists. Management is mainly based on regular respiratory physiotherapy to ensure bronchial drainage, combined with antibiotic therapy in cases of superinfection. It also includes appropriate vaccination coverage (influenza, pneumococcal, and COVID19) and close cardiopulmonary monitoring [14,11,12,15]. Thoracic or ENT surgery remains exceptional, reserved for cases of medical treatment failure.

CONCLUSION

This case illustrates that wheezing does not always correspond to asthma, even when the patient is symptomatic. Hence the importance of establishing a precise etiological diagnosis. This can contribute to the early identification of the cause of wheezing and to adapted management, including surgical intervention when necessary, in order to limit long-term respiratory and cardiovascular complications that may threaten both vital and functional prognosis.

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Figures

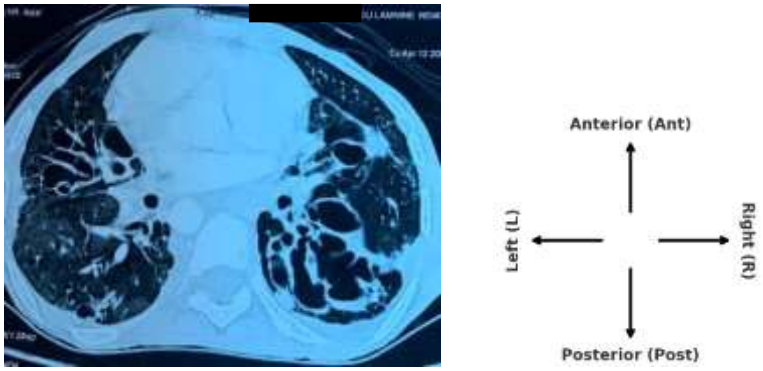

Figure 1

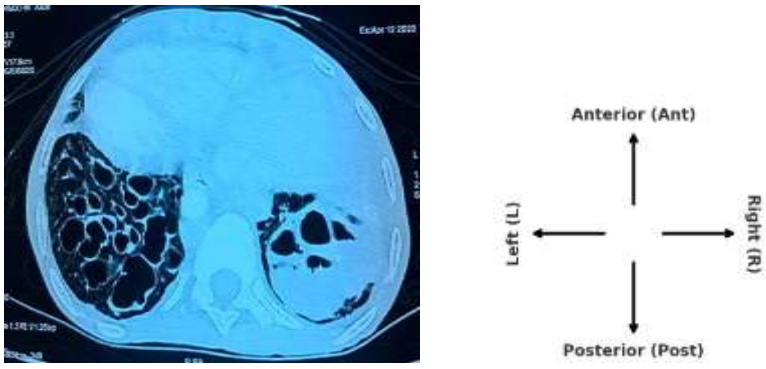

Figure 2

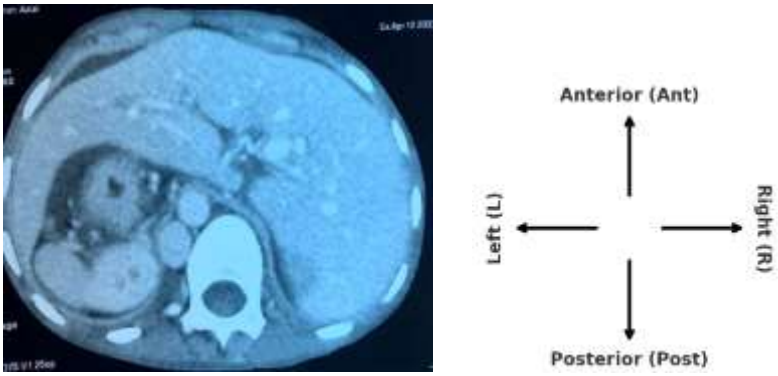

Figure 3

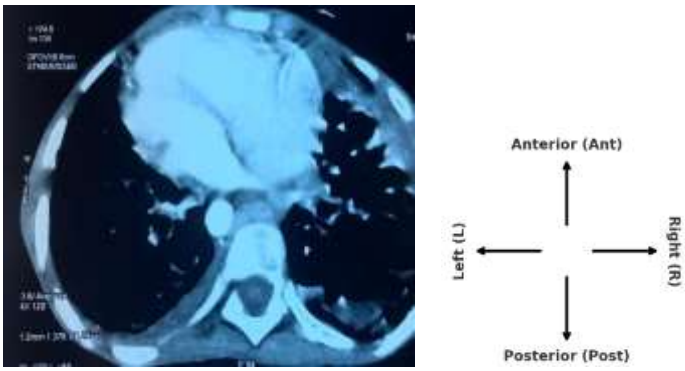

Figure 4