OLGU SUNUMU / *CASE REPORT*

A Rare Presentation of Cystic Bronchiectasis with Acute Renal Failure and Electrolyte Imbalance

Kistik Bronşektazi ile Nadir Bir Birliktelik, Akut Renal Yetmezlik ve Elektrolit İmbalansı

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ABSTRACT

A 62-year-old female patient with a history of cystic bronchiectasis was brought to the emergency department with complaints of weakness, nausea, and shortness of breath. The patient was hospitalized at the internal medicine service for the accompanying acute renal failure, hypocalcemia, hypokalemia, and hypomagnesemia. The patient was started on oral active vitamin D therapy. Intravenous calcium treatment was given to the patient with a corrected calcium value of 6.4 mg/dl. In addition, intravenous potassium and intravenous magnesium replacement were performed. The patient's fluid intake and output were monitored to prevent fluid overload. The creatinine value of the patient decreased to the normal limits after five days of treatment. The patient, who had no electrolyte imbalance and whose complaints regressed, was discharged on the 8th day of hospitalization with recommendations. Clinicians should be careful about the potential risk of accompanying renal failure and electrolyte imbalance in patients with cystic bronchiectasis.

Keywords: *bronchiectasis; creatinine; acute renal failure; hypocalcemia; hypokalemia; hypomagnesemia*

ÖZET

Altmış iki yaşında kistik bronşektazi öyküsü olan kadın hasta halsizlik, bulantı ve nefes darlığı şikâyetleri ile acil servise getirildi. Hasta, eşlik eden akut böbrek yetmezliği, hipokalsemi, hipokalemi ve hipomagnezemi nedeniyle dâhiliye servisine yatırıldı. Hastaya oral aktif vitamin D tedavisi başlandı. Düzeltilmiş kalsiyum değeri 6,4 mg/dl olan hastaya intravenöz kalsiyum tedavisi başlandı. Hastaya ek olarak intravenöz potasyum ve intravenöz magnezyum replasmanı yapıldı. Hastanın volüm yüklenmesini önlemek için sıvı alımı ve idrar çıkışı izlendi. Tedaviden beş gün sonra hastanın kreatinin değeri normal sınırlara geriledi. Elektrolit imbalansı olmayan ve şikâyetleri gerileyen hasta yatışının 8. gününde önerilerle taburcu edildi. Klinisyenler, kistik bronşektazili hastalarda renal yetmezlik ve elektrolit imbalansı eşlik etme potansiyeline karşı dikkatli olmalıdır.

Anahtar kelimeler: *bronşektazi; kreatinin; akut renal yetmezlik; hipokalsemi; hipokalemi; hipomagnezemi*

Introduction

Bronchiectasis, first described by Laennec in 1819, is a long-term condition where the bronchi become permanently enlarged and thickened. This is accompanied by persistent coughing, the production of sputum, and repeated infections in the respiratory system. Bronchiectasis is divided into cylindrical, varicose, and cystic bronchiectasis according to the radiological and pathological images of the airways[1].

Case

A 62-year-old female patient with known cystic bronchiectasis, seasonal allergic rhinitis, Chronic obstructive pulmonary disease, Hypertension (HT), type 2 Diabetes Mellitus (DM), and coronary artery disease was admitted to the emergency department of our hospital on February 23rd of 2023 with complaints of weakness, nausea, shortness of breath, and weight loss. Physical examination of the patient: arterial blood pressure of 130/80 mmHg, a pulse of 102 beats per minute in sinus rhythm, and an oxygen saturation level of 97 percent. The patient was alert, oriented, and

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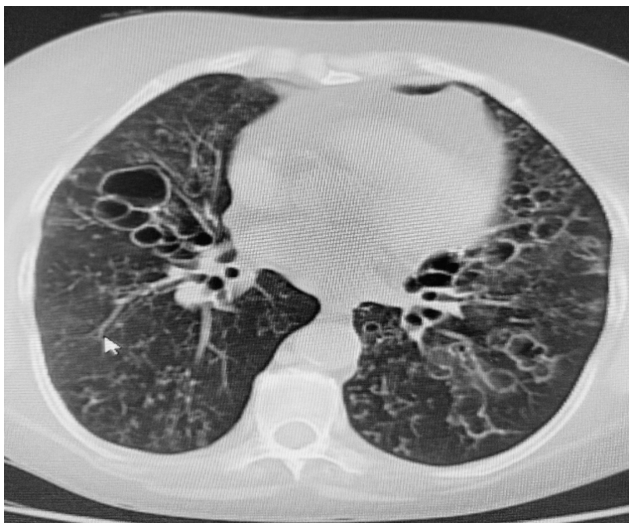

Figure 1. *Thorax tomography imaging.*

cooperative, but bilateral breath sounds were reduced during auscultation. Widespread rhonchi were heard in the bilateral lungs and rales in the basals. Although there was no tenderness, guarding, or rebounding during palpation of the abdomen, bilateral pretibial edema as grade one positive, umbilical hernia and widespread venous collaterals were detected.

Medicines used by the patient include ramipril 5 mg and hydrochlorothiazide 25 mg once a day, acetylsalicylic acid 100 mg once a day, levocetirizine and montelukast once a day, salmeterol 50 ug and fluticasone 500 ug twice a day, tiotropium 18 ug once a day, n-acetylcysteine 600 mg once a day, sitagliptin 50 mg and metformin 1000 mg twice a day, propylthiouracil 50 mg once a day. Laboratory tests in the emergency room are shown in Table 1.

Urinary system ultrasonography was performed in the emergency department to investigate the cause of acute renal failure, and no postrenal findings were observed. The cross-sectional image from the patient's thorax tomography is shown in Fig. 1. Thorax tomography was performed in the emergency department due to a complaint of shortness of breath. The report stated that the patient had calcific plaque buildup in their thoracic aorta, as well as widespread tubular cystic bronchiectasis (sequelae changes), which was more pronounced in the basal sections of both lungs; the patient also had a sliding hernia, but there was no indication of active infiltration or mass in the lung tissue (parenchyma areas); minimal fluid was present in the pleural area. Since the patient did not have typical chest pain symptoms and

Table 1. *Laboratory findings*

Parameter	Admission	Range
pH	7.35	7.35–7.45
pO_2	40	(83–108) mmHg
sO_2	%66	95–99
lactate	2.2	0.5–1.6 mmol /L
HCO_3	22.8	22–26 mmol/L
pCO_2	43	35–48 mmHg
WBC	15.520	4.000–11.000 mm^3
hemoglobin	10.2	12–16 g/dl
hematocrit	%32	36–46
MCV	78	80–96 fL
neutrophil	10.770	2.000–7.000 mm^3
lymphocyte	2760	800–4.000 mm^3
platelet	469.000	150.000–450.000 mm^3
glucose	78	74–10 mg/dl
creatinine	2.4	0.5–0.9 mg/dl
ALT	12	0–33 U/L
AST	18	0–32 U/L
total bilirubin	0.2	0–1.2 mg/dl
direct bilirubin	0.14	0–0.3 mg/dl
LDH	351	135–214 u/L
amylase	99	28–100 u/L
calcium	6.4	8.6–10.2 mmg/dl
sodium	135	136–145 mmol/L
potassium	3.2	3.5–5.1 mmol/L
CRP	93.2	0–5 mg/L
PT	9, 6	8.4–10.6 seconds
APTT	40.1	23.6–30.6 seconds
INR	1.1	0.8–1.2
albumin	4.1	3.5–5.2 gr/L
troponin T	0.03	0–0.014 microgram/L
pro BNP	705	0–125 pygogram/ml

pCO_2: Partial pressure of carbon dioxide; **MCV:** Mean corpuscular volume; **ALT:** Alanine aminotransferase; **AST:** Aspartate aminotransferase; **LDH:** Lactate dehydrogenase; **CRP:** C reactive protein; **PT:** Prothrombin time; **APTT:** Activated partial thromboplastin time; **INR:** International normalized ratio; **pro-BNP:** Pro-brain natriuretic peptides.

indicated risk factors of acute coronary syndrome, the patient's electrocardiogram (ECG) was taken to the emergency department for further examination; however, it did not reveal any indications of acute coronary syndrome. The troponin value, studied in the emergency department with 2-hour intervals, was determined as 0.03 ug/L, consistent with the previous value.

The patient was hospitalized with the diagnoses of acute renal failure, hypokalemia, hypocalcemia, hypomagnesemia, and accompanying pre-existing cystic bronchiectasis, seasonal allergic rhinitis, COPD, HT, type 2 DM, coronary artery disease, and coronary artery disease. Upon investigation of the cause of acute renal failure, it was discovered that the patient did not have a history of nonsteroidal anti-inflammatory drug use or complaints of diarrhea or vomiting; however,

the patient's daily water intake was found to be low, which could be a contributing factor to the condition. The patient was observed to be on multiple medications and had been taking ramipril 5 mg and hydrochlorothiazide 25 mg once a day for approximately one year as an antihypertensive medication.

Parathormone, phosphorus, 25-OH Vitamin D3, magnesium tests, and other routine tests were requested from the patient due to accompanying hypocalcemia. The test results were as follows; tsh: 0.5 (0.2–4.2) mu/L, ft4:1.3 (0.9–1.7) ng/dl, uric acid: 11.9 (2.4–5.7)) mg/dl, alkaline phosphatase (ALP): 69 (30–120) U/L, gamma-glutamyl transferase (GGT): 12 (5–36) U/L, magnesium: 1.0 (1.6–2, 6) mg/dl, phosphorus: 4.2 (2.5–4.5) mg/dl, parathormone: 131 (15–65) ng/L, procalcitonin: 0.12 (0–2) ug/L, 25-OH Vitamin D3:2.47 (20–50) ng/ml, iron: 53 (37–145) ug/dl, iron-binding: 223 (135–392) ug/dl, ferritin: 150 (13–150) ug/L, folate: 9.9 (3.8–20) ug/L, b12:417 (197–771) nanogram/L

Acute renal failure, severe vitamin D deficiency, hypocalcemia, hypomagnesemia, and hypocalcemia were detected in the patient. The patient's treatment with sitagliptin+metformin and ramipril+hydrochlorothiazide was discontinued. Blood pressure was monitored, and 10 mg of amlodipine was ordered daily if necessary. The patient was started on oral active vitamin D therapy. Intravenous calcium treatment was given to the patient with a corrected calcium value of 6.4 mg/dl. In addition, intravenous potassium and intravenous magnesium replacement were performed. The patient's fluid intake and output were monitored to prevent fluid overload, and the fluid intake was followed up to be 500 cc more than the fluid output. Laboratory findings after treatment are shown in Table 2. The creatinine value of the patient was within normal limits. The patient, who had no electrolyte imbalance and whose complaints regressed, was discharged on the 8th day of hospitalization with recommendations.

Table 2. *Laboratory findings after treatment*

Parameter	Level	Range
magnesium	1.9	1.6–2.6 mg/dl
glucose	84	74–10 mg/dl
creatinine	0.8	0.5–0.9 mg/dl
ALT	10	0–33 U/L
AST	14	0–32 U/L
total bilirubin	0.2	0–1.2 mg/dl
direct bilirubin	0.	0–0.3 mg/dl
LDH	208	135–214 u/L
calcium	8.5	8–6–10.2 mg/dl
sodium	135	136–145 mmol/L
potassium	4.5	3.5–5.1 mmol/L
CRP	4	0–5 mg/L

ALT: Alanine aminotransferase; **AST:** Aspartate aminotransferase; **LDH:** Lactate dehydrogenase; **CRP:** C-reactive protein.

Discussion and Conclusion

Recent scientific research in Türkiye indicates that the etiological factors that cause the development of bronchiectasis can be revealed in more than half of the patients. While some identifiable causes of bronchiectasis are localized to the lung, others are a component of systemic diseases. Pneumonia and other lower respiratory tract infections rank first among the causes of bronchiectasis[2]. Investigation of the underlying etiology is recommended in all patients with bronchiectasis. Childhood viral and bacterial respiratory tract infections are thought to play a role in developing bronchiectasis[1]. Although complications such as lung abscess, empyema, metastatic brain abscess, and amyloidosis have been reported due to bronchiectasis, these conditions are rarely observed with the use of selected antibiotics today. Patients diagnosed with concomitant cystic fibrosis during pediatric age have a short life expectancy, and around 50% of them may live beyond the age of 30. During bronchiectasis, patients commonly experience acute exacerbations of pneumonic infections, hemoptysis (coughing up blood), COPD, and cor pulmonale (enlargement and dysfunction of the right side of the heart due to lung disease)[3]. Bronchiectasis not caused by cystic fibrosis can be treated with several options, including medical treatment such as antibiotics and bronchodilators, physiotherapy, and surgical treatment[3].

According to a report on comorbid diseases in patients with bronchiectasis, at least one additional comorbid disease was observed in 44% of 138 patients followed in the departments of Chest Diseases and Thoracic Surgery at Atatürk University Training and Research Hospital. These were respectively COPD (23.1%), sinusitis (15.2%), hypertension (13.7%), peptic ulcer (10.8%), heart failure (10.1%), hepatitis (3.6%) and other (7.0%)[4].

In a multicenter study investigating the etiology in 287 amyloidosis patients in Türkiye, familial Mediterranean fever (FMF) was reported as the cause in 64%, tuberculosis in 10%, bronchiectasis, and COPD in 6%, rheumatoid arthritis in 4%, spondyloarthropathy in 3%,

chronic osteomyelitis in 2%, other causes in 4%, and unknown causes in 7%. Bronchiectasis and COPD were ranked third among the causes. Bronchiectasis can play a role in the etiology of chronic kidney failure secondary to amyloidosis[5].

Upon reviewing the literature, no direct study investigated the association between bronchiectasis and acute renal failure-electrolyte imbalance. Studies have shown that inflammatory markers are elevated in patients with concomitant bronchiectasis, especially in the geriatric population. The association between bronchiectasis and an inflammatory profile is thought to contribute to the development of acute renal failure. Electrolyte imbalance can occur in patients with diseases secondary to acute renal failure. Patients with bronchiectasis, who are treated with antihypertensive medications such as ACE inhibitors or angiotensin II receptor blockers, as well as diuretics (loop, thiazide, thiazide-like), should be monitored for electrolyte imbalance, as in our case.

This case gains importance due to being the first case reported in the literature where both electrolyte imbalance and acute kidney failure accompany cystic bronchiectasis. Although acute kidney failure is a common cause of most electrolyte imbalances (such as hyperkalemia and hyponatremia), the coexistence of hypokalemia and hypocalcemia is a rare condition. Chronic granulomatous diseases, among the causes of non-cystic bronchiectasis, are also associated with hypercalcemia. This shows us that, contrary to what we expect in our case, bronchiectasis patients may present with many electrolyte imbalances[6].

Bronchiectasis is significant in pediatric and respiratory disease practice and requires attention in internal medicine clinics and accompanying internal pathologies. Early detection and management of these accompanying internal pathologies can improve bronchiectasis patients' overall prognosis and quality of life. Further research is needed to understand better the mechanisms linking bronchiectasis and these comorbidities and develop more effective treatment strategies for this complex disease.

Financial Support and Interest Relationship

No person/organization financially supports the study, and the authors do not have any conflicts of interest.

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