Cureus
Part of SPRINGER NATURE

Open Access Case Report

Central Airway Compression Associated With Mediastinal Lymphoma Presenting as Acute Wheezing in a 94-Year-Old Woman: A Case Report

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Review began 11/09/2026
Review ended 11/20/2026
Published 11/25/2026

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DOI: 10.7759/cureus.120249

Abstract

New-onset wheezing in older adults is commonly attributed to asthma or heart failure; however, central airway compression caused by malignancy may present with similar symptoms. We report the case of a 94-year-old woman who developed acute wheezing and severe tachypnea despite preserved oxygenation. Although acute heart failure was initially suspected, chest computed tomography revealed extensive cervical and mediastinal lymphadenopathy associated with central airway narrowing. Respiratory symptoms recurred during hospitalization and did not meaningfully improve with diuretics or noninvasive ventilation. Intravenous methylprednisolone (40 mg/day) was initiated for suspected lymphoma-associated airway compression, followed by marked symptomatic improvement the next day. Subsequent lymph node biopsy demonstrated a proliferation of small atypical lymphoid cells expressing CD20, CD79a, and CD5, suggestive of B-cell lymphoma. This case highlights the importance of considering malignant central airway compression in older adults presenting with unexplained or treatment-refractory wheezing, even when oxygenation is preserved. It also illustrates the potential role of corticosteroids in relieving symptoms associated with suspected lymphoma-related airway compression. In patients requiring urgent treatment, clinicians must balance respiratory stabilization with preservation of diagnostic accuracy and individualized goals of care.

Categories: Family/General Practice, Geriatrics, Oncology
Keywords: b-cell lymphoma, central airway obstruction, general medicine, mediastinal lymphadenopathy, older adults, rural, wheezing

Introduction

Wheezing is a common respiratory symptom frequently associated with bronchial asthma, chronic obstructive pulmonary disease, and heart failure. However, wheezing may also result from central airway obstruction caused by malignant or nonmalignant conditions. Central airway obstruction can present with nonspecific respiratory symptoms, including dyspnea, cough, and wheezing, which may mimic more common cardiopulmonary disorders and delay accurate diagnosis [1,2]. Furthermore, conventional chest radiography may fail to identify clinically significant airway narrowing, whereas computed tomography (CT) provides more detailed information regarding the location, extent, and underlying cause of airway obstruction [1,2].

Malignant central airway obstruction may result from endoluminal tumor growth, extrinsic compression, or a combination of these mechanisms. In patients with mediastinal lymphoma, enlarged lymph nodes may exert external pressure on the trachea or main bronchi, narrowing the airway lumen and increasing resistance to airflow. The resulting turbulent airflow can produce wheezing that resembles peripheral airway disease. However, persistent or recurrent wheezing, positional dyspnea, stridor, or limited response to conventional bronchodilator treatment may raise suspicion of central airway obstruction rather than asthma or chronic obstructive pulmonary disease [1,2]. Importantly, substantial airway narrowing may cause increased work of breathing and severe tachypnea before significant hypoxemia develops, because alveolar ventilation and gas exchange may initially remain relatively preserved. Thus, normal oxygen saturation does not exclude clinically significant central airway obstruction. Management requires consideration of the underlying malignancy, severity of respiratory compromise, available therapeutic interventions, and the patient's overall clinical condition. Recent clinical practice guidelines emphasize individualized management, including therapeutic bronchoscopy when appropriate, alongside systemic or local treatment directed at the underlying disease [3].

In older patients, multimorbidity, physiological vulnerability, and the potential burdens of invasive diagnostic procedures may complicate the evaluation and management of acute respiratory symptoms. Nevertheless, chronological age alone should not preclude investigation of potentially treatable causes of respiratory distress. We report the case of a 94-year-old woman who presented with acute wheezing and

severe tachypnea despite preserved oxygenation. Chest CT revealed extensive mediastinal lymphadenopathy associated with central airway narrowing, and subsequent lymph node biopsy suggested a CD5-positive B-cell lymphoma. Her respiratory symptoms improved following corticosteroid administration, although a direct causal relationship could not be established. This case emphasizes that new-onset or recurrent wheezing accompanied by substantial respiratory distress should prompt reassessment for structural airway abnormalities, even when oxygen saturation remains normal. It also highlights the challenges of balancing urgent symptom management with diagnostic uncertainty and individualized goals of care [3,4].

Case Presentation

A 94-year-old woman with a history of hypertension was brought to the emergency department after suddenly developing severe coughing and tachypnea while participating in a physical exercise program at a community day-care facility. Before admission, she required partial assistance with activities of daily living and was considered clinically frail. However, her cognitive function was relatively preserved, without apparent cognitive impairment affecting communication or participation in clinical decision-making. She had no history of bronchial asthma or smoking and no known drug allergies. Her regular medications included amlodipine (10 mg/day) and loxoprofen (120 mg/day).

On arrival, her blood pressure was 149/85 mmHg, heart rate 55 beats/minute, respiratory rate 52 breaths/minute, body temperature 36.9°C, and oxygen saturation 99% on room air. Despite preserved oxygenation, she exhibited marked tachypnea and orthopnea. Bilateral wheezing was audible on lung auscultation, although no stridor was detected. Cardiac auscultation revealed a regular rhythm without murmurs. No jugular venous distention was noted, but bilateral pitting edema was present in the lower extremities. Her abdomen was soft and nontender, and her extremities were not cold.

Initial laboratory investigations revealed mild normocytic anemia (hemoglobin, 10.6 g/dL; mean corpuscular volume, 85.7 fL) and a slightly elevated C-reactive protein level (0.48 mg/dL). Cardiac biomarkers were not elevated, with troponin I <0.010 ng/mL and brain natriuretic peptide 38.9 pg/mL. Arterial blood gas analysis on room air demonstrated respiratory alkalosis (pH, 7.552; $PaCO_2$, 27.5 mmHg; PaO_2, 102.0 mmHg; and HCO_3^-, 24.1 mmol/L), consistent with marked hyperventilation without hypoxemia. The initial laboratory findings are summarized in Table *1*.

Parameter	Result	Reference range
Hematology		
White blood cell count (×10³/μL)	5.30	3.5–9.1
Neutrophils (%)	73.8	44.0–72.0
Lymphocytes (%)	14.4	18.0–59.0
Hemoglobin (g/dL)	10.6	11.3–15.2
Hematocrit (%)	30.6	33.4–44.9
Mean corpuscular volume (fL)	85.7	79.0–100.0
Platelet count (×10⁴/μL)	19.3	13.0–36.9
Biochemistry		
Total protein (g/dL)	6.5	6.5–8.3
Albumin (g/dL)	3.3	3.8–5.3
Total bilirubin (mg/dL)	0.8	0.2–1.2
Aspartate aminotransferase (U/L)	17	8–38
Alanine aminotransferase (U/L)	8	4–43
Lactate dehydrogenase (U/L)	192	121–245
Blood urea nitrogen (mg/dL)	16.4	8–20
Creatinine (mg/dL)	0.84	0.40–1.10
Sodium (mEq/L)	141	135–150
Potassium (mEq/L)	3.8	3.5–5.3

Chloride (mEq/L)	107	98–110
Ferritin (ng/mL)	208.1	14.4–303.7
C-reactive protein (mg/dL)	0.48	<0.30
Immunology		
Immunoglobulin G (mg/dL)	1,261	870–1,700
Immunoglobulin M (mg/dL)	71	35–220
Immunoglobulin A (mg/dL)	237	110–410
Cardiac biomarkers		
Troponin I (ng/mL)	<0.010	<0.010
BNP (pg/mL)	38.9	<40
Arterial blood gas (room air)		
pH	7.552	—
$PaCO_2$ (mmHg)	27.5	—
PaO_2 (mmHg)	102.0	—
HCO_3^- (mmol/L)	23.4	—

TABLE 1: Laboratory findings on admission.

Laboratory investigations demonstrated mild normocytic anemia, a slight elevation in C-reactive protein, and respiratory alkalosis characterized by reduced $PaCO_2$ and elevated pH despite preserved arterial oxygenation. The relatively low BNP level (38.9 pg/mL) provided limited support for acute cardiac decompensation as the predominant cause of respiratory distress, although heart failure with preserved ejection fraction could not be completely excluded. The preserved PaO_2 (102.0 mmHg on room air) highlighted the discrepancy between severe tachypnea and maintained oxygenation. Arterial blood gas analysis was performed while the patient was breathing room air.

BNP: brain natriuretic peptide; $PaCO_2$: partial pressure of arterial carbon dioxide; PaO_2: partial pressure of arterial oxygen; HCO_3^-: bicarbonate

Electrocardiography demonstrated sinus rhythm at 59 beats/minute. Chest radiography showed bilateral hilar opacities. Transthoracic echocardiography revealed preserved left ventricular systolic function, with an ejection fraction greater than 55%, and no pericardial effusion or interventricular septal flattening. Initial chest radiography demonstrated bilateral hilar prominence (Figure *1*, Panel A).

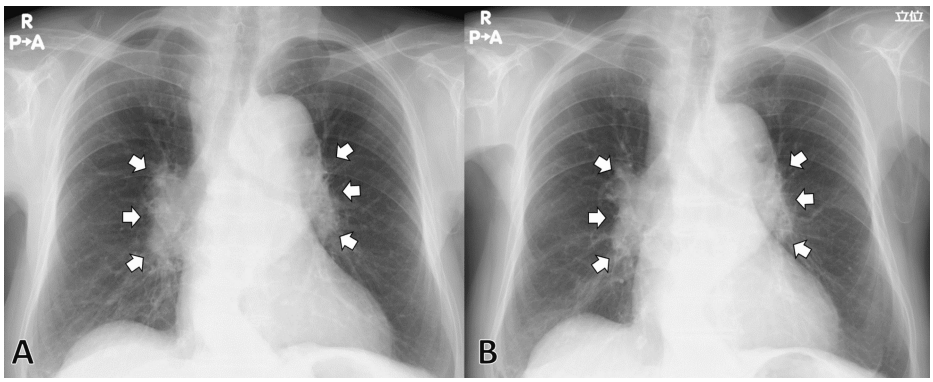


FIGURE 1: Chest radiographic findings before and after treatment.

(A) Pretreatment chest radiograph demonstrating bilateral hilar opacities/prominence (arrows). (B) Post-treatment chest radiograph demonstrating improvement of the bilateral hilar opacities/prominence (arrows).

Subsequently, chest CT revealed extensive mediastinal and hilar lymphadenopathy associated with narrowing of the central airway (Figure *2*, Panel A).

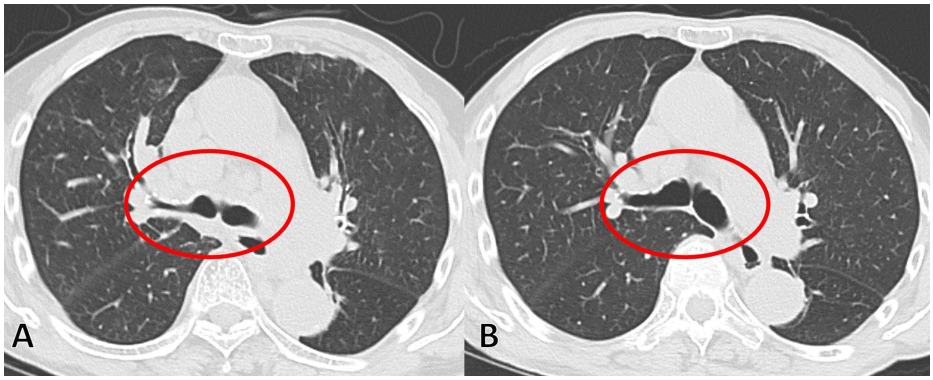


FIGURE 2: Chest computed tomography findings before and after corticosteroid initiation.

(A) Pretreatment axial chest computed tomography demonstrating extensive mediastinal and hilar lymphadenopathy associated with central airway narrowing (red circle). (B) Follow-up chest computed tomography obtained seven days after initiation of intravenous methylprednisolone, demonstrating reduced mediastinal and hilar lymphadenopathy and improvement in central airway narrowing (red circle). The radiological changes were assessed qualitatively because quantitative measurements of airway diameter were unavailable.

Bronchoscopic assessment was not performed; therefore, direct endoscopic evaluation of the airway narrowing was unavailable.

Based on the initial findings of orthopnea, bilateral lower-extremity edema, and hilar opacities on chest radiography, acute congestive heart failure was initially considered. However, the relatively low brain natriuretic peptide (BNP) level (38.9 pg/mL), preserved left ventricular systolic function (ejection fraction >55%), and absence of other clear clinical findings supporting acute cardiac decompensation made acute heart failure a less likely primary explanation for the respiratory symptoms, although heart failure with preserved ejection fraction could not be completely excluded. During the emergency department evaluation, her tachypnea and wheezing resolved spontaneously within approximately one hour, and her respiratory rate decreased to 20 breaths/minute. Given the sudden onset of symptoms during physical activity and their transient resolution, aspiration-related bronchospasm or transient airway irritation was also considered a possible contributor to the initial episode. She was admitted to the general medicine ward for observation and further evaluation.

On the first day of hospitalization, episodes of tachypnea and wheezing recurred. Noninvasive positive-pressure ventilation and intravenous furosemide were administered, but neither intervention resulted in meaningful clinical improvement. Given the extensive mediastinal and hilar lymphadenopathy and associated central airway narrowing demonstrated on chest CT, extrinsic airway compression was considered a possible contributor to the recurrent respiratory distress. On the third day of hospitalization, treatment directed at pulmonary congestion was discontinued, and intravenous methylprednisolone (40 mg/day) was initiated because of concern about possible progression of airway compromise. The respiratory rate immediately before corticosteroid initiation was approximately 40-49 breaths/minute. On the following day, the respiratory rate decreased to approximately 20-29 breaths/minute, accompanied by marked improvement in wheezing and respiratory distress. The corticosteroid dose was subsequently tapered, and treatment was continued for approximately one month.

Her respiratory symptoms improved markedly on the following day. Follow-up chest radiography showed less prominent hilar opacities. On the fourth day of hospitalization, we performed an excisional biopsy of the right cervical lymph node. Histopathological examination revealed diffuse proliferation of small atypical lymphoid cells. Immunohistochemical staining demonstrated expression of CD20, CD79a, and CD5 in the atypical lymphoid cells, whereas the neoplastic cells were negative for CD3 and CD30. Scattered background T lymphocytes were positive for CD3. Additional immunohistochemical investigations, including cyclin D1, SOX11, CD23, LEF1, and Ki-67, were not performed in the regional hospital setting. The available histopathological and immunohistochemical findings were suggestive of a CD5-positive B-cell lymphoma; however, differentiation among mantle cell lymphoma (MCL), chronic lymphocytic leukemia (CLL)/small lymphocytic lymphoma (SLL), and other CD5-positive B-cell lymphomas was not possible. Therefore, a definitive histological subtype could not be established (Figure *3*).

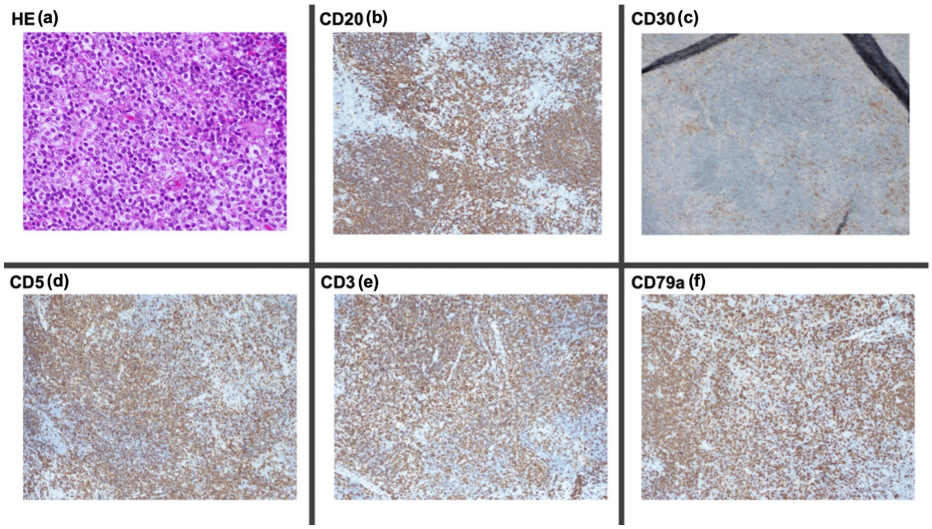


FIGURE 3: Histopathological and immunohistochemical findings of the excised cervical lymph node.

(a) Hematoxylin and eosin (H&E) staining demonstrating diffuse proliferation of small atypical lymphoid cells. (b) CD20 staining showing positive expression in the atypical lymphoid cells. (c) CD30 staining showing negative expression in the neoplastic cells. (d) CD5 staining demonstrating positive expression in the atypical lymphoid cells. (e) CD3 staining highlighting scattered background T lymphocytes, whereas the neoplastic cells were negative. (f) CD79a staining demonstrating positive expression in the atypical lymphoid cells. These findings were suggestive of a CD5-positive B-cell lymphoma but were insufficient to establish a definitive histological subtype.

The H&E image (a) was reported at ×400 magnification, whereas the immunohistochemical images (b–f) were reported at ×40 magnification.

Following initiation of intravenous methylprednisolone, the patient's wheezing and tachypnea improved markedly, with the respiratory rate decreasing from the 40s breaths/minute before treatment to the 20s breaths/minute on the following day. Follow-up chest radiography demonstrated improvement in bilateral hilar prominence (Figure *1*, Panel B), and chest CT performed seven days after corticosteroid initiation showed improvement in mediastinal and hilar lymphadenopathy and associated central airway narrowing (Figure *2*, Panel B). Methylprednisolone was subsequently tapered over approximately one month.

Given the patient's advanced age, pre-existing frailty, and partial dependence in activities of daily living, further diagnostic investigations and lymphoma-directed treatment were considered in the context of her overall condition and care preferences. Her cognitive function was relatively preserved, and she expressed a preference for symptom-focused supportive management rather than aggressive lymphoma-directed treatment. Accordingly, management prioritized relief of respiratory symptoms and maintenance of daily functioning. After clinical improvement, she remained stable without recurrent wheezing or tachypnea during the reported hospitalization and was discharged home to resume her previous level of daily activities.

Discussion

This case highlights three clinically important considerations: the recognition of central airway compression as a cause of acute wheezing despite preserved oxygenation, the potential role of corticosteroids in the initial management of suspected lymphoma-associated airway compromise, and the importance of individualized diagnostic and therapeutic decisions in very elderly patients. The distinctive feature of this case was the discrepancy between severe respiratory distress, characterized by a respiratory rate of 52 breaths/minute at initial presentation, and preserved oxygenation. Acute heart failure was initially considered because of orthopnea, bilateral lower-extremity edema, and hilar opacities on chest radiography. However, the relatively low BNP level, preserved left ventricular systolic function, and lack of meaningful improvement following diuretic therapy made acute heart failure less likely as the predominant cause of respiratory distress. Nevertheless, preserved systolic function and a relatively low BNP level do not completely exclude heart failure, particularly heart failure with preserved ejection fraction. The initial spontaneous resolution of wheezing also suggested that transient bronchospasm, potentially related to aspiration or airway irritation, might have contributed to the presenting episode. Although these alternative mechanisms could not be definitively excluded, the recurrence of respiratory symptoms, limited response to conventional treatment, and CT findings of extensive mediastinal lymphadenopathy with central airway narrowing supported extrinsic airway compression as an important contributor to the clinical presentation.

The clinical manifestations of malignant central airway obstruction depend on the anatomical location,

degree of narrowing, and underlying mechanism. Wheezing may occur when airflow becomes turbulent through a narrowed central airway, potentially resembling bronchial asthma. Importantly, preserved oxygen saturation does not exclude clinically significant airway obstruction, because respiratory mechanics and oxygenation may be affected differently during the early stages of airway compromise [5]. In this case, severe tachypnea and respiratory alkalosis preceded recognition of the structural airway abnormality. The initial spontaneous improvement and subsequent symptom recurrence further complicated clinical interpretation. These findings emphasize that persistent or recurrent wheezing, particularly when the clinical course does not fit common cardiopulmonary disorders, should prompt reassessment and consideration of structural airway disease.

Previous case reports have described mediastinal lymphoma presenting with airway compromise and rapid symptomatic improvement following treatment. Athish et al. reported a case of mediastinal T-lymphoblastic lymphoma in which corticosteroid administration during diagnostic evaluation was associated with radiological tumor regression [6]. Similarly, Anai et al. described an 85-year-old man with adult T-cell leukemia/lymphoma who developed tracheal stenosis and superior vena cava syndrome due to a large mediastinal mass. Combined corticosteroid therapy and palliative radiotherapy resulted in marked clinical and radiological improvement [6]. Unlike these reports, our patient was 94 years old, initially presented with wheezing despite preserved oxygenation, and showed symptomatic improvement following corticosteroid administration without documented concomitant lymphoma-directed radiotherapy or chemotherapy during the reported acute episode. Specifically, the respiratory rate decreased from the 40s breaths/minute immediately before corticosteroid initiation to the 20s breaths/minute on the following day, accompanied by marked improvement in wheezing and respiratory distress. Follow-up chest CT performed seven days after corticosteroid initiation demonstrated improvement in mediastinal and hilar lymphadenopathy and associated central airway narrowing. Although these findings were temporally associated with corticosteroid treatment, they do not establish a direct causal relationship. The absence of quantitative airway measurements before and after treatment further limits assessment of the magnitude of anatomical improvement.

Initiating systemic corticosteroids before histopathological confirmation requires careful consideration of both immediate clinical needs and potential diagnostic consequences. In the present case, corticosteroid treatment was initiated because of recurrent severe respiratory distress, limited improvement with initial interventions, and CT evidence of central airway narrowing associated with extensive mediastinal lymphadenopathy. Although the patient did not have documented hypoxemic respiratory failure at presentation, the recurrent symptoms and structural airway narrowing raised concern about possible progression of airway compromise. Therefore, symptomatic stabilization was prioritized over delaying treatment until histopathological confirmation. Intravenous methylprednisolone was initiated at 40 mg/day and subsequently tapered over approximately one month.

Corticosteroids may reduce lymphoma-associated inflammation and induce apoptosis of lymphoma cells, potentially providing rapid symptomatic relief but also altering tissue morphology and complicating pathological interpretation. Previous studies have raised concerns that corticosteroid administration before biopsy may compromise diagnostic accuracy in some lymphoma subtypes. However, a retrospective study of 320 patients with diffuse large B-cell lymphoma found no significant difference in initial biopsy diagnostic yield between patients who received corticosteroids before biopsy and those who did not (83% versus 81%) [7]. These findings should be interpreted cautiously because they may not be generalizable to other lymphoma subtypes. In our patient, an excisional lymph node biopsy was performed on the day following initiation of corticosteroid treatment, and the available histopathological and immunohistochemical findings were suggestive of a CD5-positive B-cell lymphoma. Nevertheless, the potential influence of prebiopsy corticosteroid exposure on tissue morphology and diagnostic interpretation cannot be excluded, particularly because definitive histological subclassification was not achieved [8]. This case illustrates the importance of balancing timely symptomatic treatment with prompt tissue sampling and recognition of the limitations of pathological interpretation following corticosteroid exposure.

Another important consideration is the diagnostic uncertainty associated with the histopathological findings. Although the expression of CD20, CD79a, and CD5 supported the presence of a CD5-positive B-cell lymphoma, these findings alone were insufficient to establish a definitive histological subtype. The differential diagnosis included MCL, CLL/SLL, and other CD5-positive B-cell lymphoid neoplasms. Additional immunohistochemical markers could have helped distinguish these entities. MCL typically expresses cyclin D1, reflecting the characteristic t(11;14)(q13;q32) translocation involving CCND1 and IGH, while SOX11 is particularly useful in identifying cyclin D1-negative MCL. In contrast, CLL/SLL generally expresses CD23 and LEF1, with CD200 providing additional discriminatory value, although overlapping immunophenotypic patterns may occur. Additional markers, including CD10 and BCL6, could have assisted in evaluating other B-cell lymphoma subtypes, while Ki-67 would have provided information regarding proliferative activity rather than definitive histological classification. Molecular investigations, such as fluorescence in situ hybridization for the CCND1::IGH rearrangement, could also have supported the diagnosis of MCL [9]. However, these additional immunohistochemical investigations were not performed in the regional hospital setting, and the available pathological findings were insufficient for definitive histological subclassification. Furthermore, because corticosteroid treatment had been initiated before biopsy, its potential influence on tissue morphology and pathological interpretation could not be entirely

excluded. Therefore, the final pathological assessment remained suggestive of a CD5-positive B-cell lymphoma rather than a definitively classified lymphoma subtype. This diagnostic limitation should be considered when interpreting the clinical course and response to treatment.

The temporal association between corticosteroid administration and subsequent clinical and radiological improvement warrants cautious interpretation. In the present case, the respiratory rate decreased from the 40s breaths/minute immediately before corticosteroid initiation to the 20s breaths/minute on the following day, accompanied by improvement in wheezing and respiratory distress. Follow-up chest CT performed seven days after treatment initiation demonstrated reduced mediastinal and hilar lymphadenopathy and improvement in central airway narrowing. Although these findings were temporally associated with corticosteroid administration, they do not establish a direct causal relationship. Several alternative explanations should be considered. First, the spontaneous resolution of the initial respiratory episode before corticosteroid administration suggests that respiratory symptoms may have fluctuated independently of treatment. Second, transient bronchospasm or airway irritation, potentially associated with aspiration, may have contributed to the initial presentation and subsequently resolved. Third, supportive management and other concurrent physiological changes may have influenced the clinical course. Furthermore, quantitative measurements of airway diameter and lymph node size were unavailable, limiting objective assessment of the magnitude of radiological improvement. The absence of bronchoscopic assessment, if not performed, would also limit direct evaluation of the airway abnormality. Therefore, although corticosteroid treatment may have contributed to the observed improvement, its independent therapeutic effect on airway obstruction cannot be established from this single case. These findings should be interpreted as observations of temporal association rather than definitive evidence of corticosteroid efficacy.

Finally, this case raises important considerations for managing suspected malignancy in very elderly patients. Chronological age alone does not adequately reflect physiological reserve, functional independence, cognitive status, treatment tolerance, or individual care priorities. Hormigo-Sanchez et al. demonstrated that comprehensive geriatric assessment could identify heterogeneous levels of frailty among older patients with lymphoma, supporting individualized treatment planning [10]. In the present case, the patient required partial assistance with activities of daily living before admission and was considered clinically frail, although her cognitive function was relatively preserved. Importantly, she preferred symptom-focused supportive management rather than aggressive lymphoma-directed treatment. These clinical characteristics and care preferences informed an individualized approach that prioritized relief of respiratory distress and preservation of daily functioning rather than escalation of diagnostic procedures or initiation of intensive lymphoma-directed therapy. Although a formal comprehensive geriatric assessment was not documented, the patient's functional limitations, clinical frailty, cognitive status, and expressed preferences were relevant considerations in clinical decision-making.

Several limitations should be considered when interpreting this case. The precise anatomical severity of central airway narrowing was not quantitatively assessed, and definitive histological subclassification of the suspected CD5-positive B-cell lymphoma was not achieved. Although respiratory symptoms improved following corticosteroid initiation and subsequent CT demonstrated reduced mediastinal lymphadenopathy and airway narrowing, these observations do not establish a direct causal relationship. The spontaneous resolution of the initial respiratory episode and the possible contribution of transient bronchospasm or other physiological mechanisms further limit causal interpretation. In addition, the available follow-up information was insufficient to evaluate long-term clinical outcomes. Therefore, the findings should be interpreted as a description of the clinical course rather than evidence of corticosteroid efficacy or the superiority of conservative management.

Conclusions

Malignant central airway compression should be considered in older adults presenting with unexplained or recurrent wheezing, even when oxygenation is preserved. This case illustrates the importance of reassessing the underlying cause of respiratory distress when conventional treatment fails to produce meaningful improvement. Chest CT can help identify structural airway abnormalities and guide subsequent clinical evaluation. Although clinical and radiological improvement was observed following corticosteroid administration, a direct causal relationship between treatment and improvement in airway obstruction could not be established. Furthermore, this single case does not establish the efficacy of a particular corticosteroid regimen or provide evidence regarding the long-term prognosis of the suspected lymphoma. In very elderly patients, decisions regarding further diagnostic investigations and treatment should consider functional status, frailty, cognitive function, and individual goals of care rather than chronological age alone.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

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Critical review of the manuscript for important intellectual content: Ryuichi Ohta, Akiyo Horiguchi, Taichi Fujimori, Chiaki Sano

Supervision: Ryuichi Ohta, Chiaki Sano

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

Acknowledgements

The writing of the manuscript was assisted by ChatGPT and Grammarly for language support.

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