Case Report

ANNALS OF MEDICINE & SURGERY

OPEN

Takayasu arteritis and interstitial lung disease: a case report and literature review

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Introduction and importance: Although pulmonary artery involvement is well recognized, the incidence of interstitial lung disease (ILD) with Takayasu arteritis is very rare. The pathophysiology of ILD in Takayasu is still incompletely understood, in contrast to several studies establishing the relationship between ANCA-associated vasculitis and ILD. The management of this patient involved a multidisciplinary approach with long-term follow-up.

Case presentation: The authors present a case of HRCT-proven interstitial lung disease in a patient with Takayasu arteritis and heart failure. The patient was on long-term corticosteroids on and off for several years and recently developed progressive dyspnea with a dry cough. After reviewing her history and physical examination, pulmonary function tests (PFTs) and high-resolution computed tomography (HRCT) were performed, and interstitial lung disease was diagnosed. The patient was managed by a team of pulmonologists, rheumatologists, and cardiologists and gradually improved after adjustment of medications, including corticosteroids and mycofenolate, and via long-term oxygen therapy.

Clinical discussion: Takayasu arteritis is a rare form of systemic vasculitis that can involve the pulmonary vasculature, such vasculitis with associated parenchymal involvement is rare. ILDs have been demonstrated with ANCA-associated vasculitis; however, whether the pathophysiology applies to Takayasu is unknown. Since Takayasu can be debilitating to the patient, the association of ILDs can have further prognostic implications. Given that no established guidelines exist to address this association, management is based on clinical expertise.

Conclusion: The authors report a case of Takayasu arteritis and associated ILD and its pharmacological management. Takayasu arteritis is a very uncommon type of vasculitis, and pulmonary parenchymal involvement further contributes to this case's rarity. As the management of Takayasu arteritis alone is cumbersome, the addition of another significant comorbidity, such as ILD, can pose several threats to the patient. Given the rarity of this association, no established guidelines exist, making clinical expertise crucial for managing such patients. Further research is needed to explore the underlying mechanisms and develop evidence-based treatment strategies for this rare combination.

Keywords: interstitial lung disease, Takayasu arteritis, vasculitis

Introduction

Takayasu arteritis (pulseless disease) is a systemic inflammatory condition that is characterized by damage to the large and medium arteries and their branches[1]. Takayasu arteritis (TAK) is a rare autoimmune disease that affects the vasculature and has an incidence rate of 0.4–1.5 per million[2]. Clinically, TAK has an early nonspecific phase with constitutional symptoms such as fever, malaise, and weight loss. It is not until the development of symptoms of arterial insufficiencies, hypertension in renovascular stenosis, or neurological manifestations with carotid artery stenosis that the patient starts seeking treatment[1]. In the context of the pulmonary system, pulmonary vascular involvement is one of the key hallmarks of several types of vasculitides and is associated with significant morbidity and mortality[3]. Although ANCA vasculitis commonly has pulmonary vascular involvement[4], patients with Takayasu arteritis rarely present with interstitial

HIGHLIGHTS

- Takayasu arteritis is a rare type of vasculitis that may have pulmonary manifestations involving lung vasculature, but parenchymal involvement is extremely rare.
- The pathophysiology of interstitial lung disease in Takayasu arteritis remains elusive.
- The patient exhibited overlapping symptoms, complicating the diagnosis and necessitating a multidisciplinary approach involving rheumatology, pulmonology, and cardiology teams.
- The case emphasizes the importance of early recognition and tailored management of such rare combinations to improve patient outcomes.

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Sponsorships or competing interests that may be relevant to content are disclosed at the end of this article.

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Annals of Medicine & Surgery (2024) 86:6303–6310

Received 16 July 2024; Accepted 26 August 2024

Published online 4 September 2024

http://dx.doi.org/10.1097/MS9.0000000000002544

lung disease (ILD). The presence of ILD in a patient already suffering from chronic vasculitis can pose several threats in terms of management and prognosis. Owing to the rarity of such concurrence, the literature related to management has been limited to a few case reports[5–8]. Nevertheless, the increase in the incidence of autoimmune ILDs in recent years necessitates a shift in focus to such autoimmune vasculitis-associated ILDs amidst the changing trends of disease patterns[9]. Given the complexities involved in managing two chronic and potentially debilitating conditions simultaneously, there is a pressing need for multidisciplinary approaches that incorporate the expertise of rheumatologists, pulmonologists, and cardiologists. By analyzing the clinical presentation, diagnostic workup, and treatment outcomes of patients with TAK-associated ILD, this study aims to contribute valuable insights to the existing body of knowledge. These insights could help reform future clinical practice, guiding healthcare providers in making evidence-based decisions that improve patient outcomes. Here, we describe a patient with Takayasu arteritis who was recently diagnosed with interstitial lung disease and its multidisciplinary management.

Case summary

History

A 42-year-old female with a known history of Takayasu arteritis (Fig. 1) under immunosuppression with prednisolone tablets (on and off) for 13 years presented with persistent dry cough for 6 months and difficulty breathing for 6 months. The onset of her cough was insidious, progressive, and nonpurulent, without any aggravating or relieving factors, and there were no diurnal variations. She described her shortness of breath as progressive, initially presenting while climbing uphill only 1 year back (MMRC I), and over the past few months, she had been short of breath even while walking on level ground and had to walk slower than her peers (MMRC II). A detailed history of medication use and environmental exposure at home, work, and other places where the patient frequently visits was obtained. The patient had been using tablet (Tab) prednisolone inconsistently for several years and had been to several clinics for a workup of TAK. She has been followed up at our hospital for the past 9 months, and her medications have been adjusted in our center over the past 9 months. She was taking 20 mg of Tab prednisolone once daily and Tab Mycofenolate Mofetil 500 mg twice a day for Takayasu arteritis. A cardiovascular workup was also performed 9 months prior. Echocardiography revealed left ventricular (LV) systolic function [ejection fraction (EF): 60–65], and there were no regional wall motion abnormalities. There was intermediate LV diastolic dysfunction, with an E/e of – 4.12 LV filling pressure. The right ventricular dimensions were within the normal range, but there was inadequate systolic function (tricuspid annular plane systolic excursion, TAPSE - 14 mm). Mild tricuspid regurgitation was noted with estimated pulmonary arterial systolic pressure (PASP) (27 + 5 = 32 mmHg). The patient was then diagnosed with heart failure with preserved ejection fraction (HFpEF) by the New York Heart Association (NYHA II) and was treated with 25 mg of Tab metoprolol and 25 mg of tab spironolactone. Since then, the patient has been under routine follow-up with adjustments of the medications as needed. A CT aortogram was also conducted 2 months prior, which revealed marked stenosis of the subclavian artery and the distal infrarenal aorta (nonvisualized bilateral common iliac vessels) and multiple systemic collaterals arising from the inferior epigastric, superior epigastric, internal thoracic, and iliolumbar arteries. She had been asymptomatic since treatment until her recent presentation. Serial ESR also revealed a progressive reduction from 106 to 12, and the CRP concentration decreased from 118 mg/l to 23 mg/l. Her current assessment revealed a heart rate of 60 bpm; her blood pressure was unrecordable on all 4 limbs, her temperature was normal, her respiratory rate was 20 breaths/min, and her SPO_2 was 90% at 2 l O_2. On respiratory examination, she had bilateral coarse crepitations over the mammary, inframammary, and infrascapular areas, and her breath sounds were normal vesicular type.

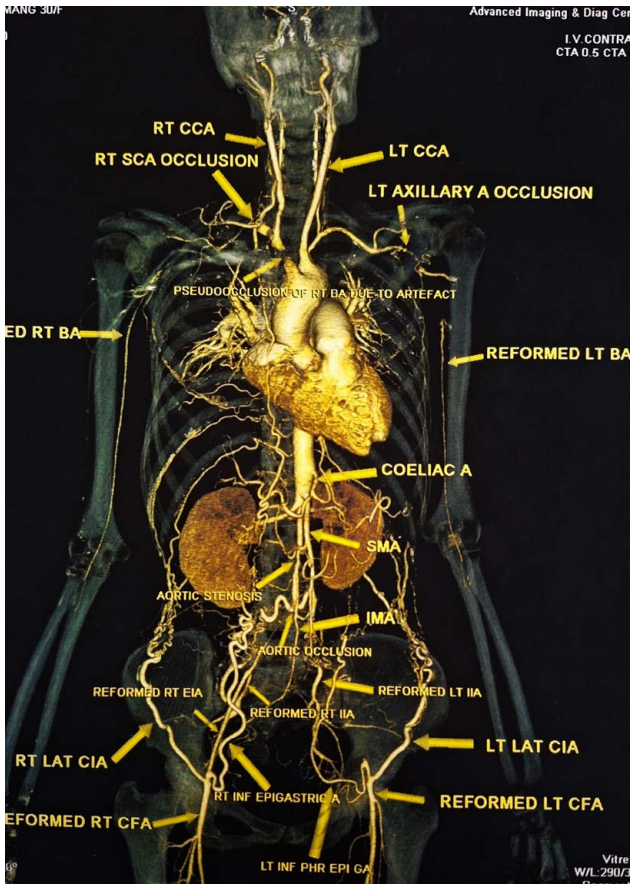

Figure 1. 3D CT Aortography conducted 9 months back with following findings: (1) Irregular long segment concentric wall thickening (max 2.5 mm) involving infrarenal portion of abdominal aorta causing multifocal areas of luminal narrowing (max 3 mm) ~4.2 cm distal to origin of renal arteries (about 80% area stenosis). (2) Alternating fusiform dilated segment measuring about 9.3 mm in length and 7.6 mm in maximum diameter noted in between the stenotic region. (3) No evidence of calcification, intraluminal thrombus, or dissection. Distal abdominal aorta about 9 cm from the origin of renal arteries shows no opacification with nonvisualized common iliac arteries. (4) Proximal right subclavian artery also shows diffuse wall thickening and marked narrowing with an irregular outline. Distal reformation of the axillary artery was noted via collaterals from the internal thoracic artery. Short segment diffuse concentric wall thickening (max 3 mm) noted involving the arch of the aorta and proximal brachiocephalic artery without significant luminal stenosis. Multiple small foci of transmural wall calcifications were noted. (5) Lumbar arteries are dilated with multiple collateral vessels arising from inferior epigastric, superior epigastric, and ilio-lumbar arteries.

Investigations

Baseline investigations, including complete blood count, blood and urine cultures, and a renal function test, were performed, which returned negative results. Sputum cultures for bacteria, tuberculosis, and common fungal infections, as well as for atypical, opportunistic, and atypical infections, also returned negative. C-reactive protein (CRP) and the erythrocyte sedimentation rate (ESR) were elevated at 87.6 mg/dl and 12 mm/h, respectively, but a repeat CT aortogram did not reveal any progression (Fig. 2). Owing to her past medical history of HFpEF, NT-pro-BNP, and echocardiography were advised. NT-pro-BNP levels were normal, and her echocardiographic features were similar to those of the last echo report, which was conducted 3 months prior. A pulmonary function test revealed features of restrictive lung disease.

HRCT of the chest without contrast agent revealed mosaic attenuation of the lung parenchyma. A heterogeneous distribution of fibrotic densities with reticulation and traction bronchiectasis was noted in the bilateral lungs, predominantly involving the bilateral lower lobes, right middle, and left lingula. There was subpleural sparing. Tiny centrilobular nodular densities were observed in bilateral lung basal segments with surrounding minimal ground glass opacities (L > R). Interlobular septations and thickening of the bronchovascular bundles were noted. Honeycombing was not present (Fig. 3). All these features suggested 'probable UIP'. An autoimmune workup was subsequently performed. The results for rheumatoid factor, anticitrulline peptide antibody, anti-dsDNA, anti-sm antibodies, ACE level, and anticentromere antibodies were negative. However, her ANA titer was more than 1:320, and she was speckled. The patient was advised to undergo bronchoalveolar lavage and/or lung biopsy, but they refused because of financial constraints. After a detailed clinical workup, laboratory investigations and a comprehensive medical chart review, a diagnosis of interstitial lung disease was made.

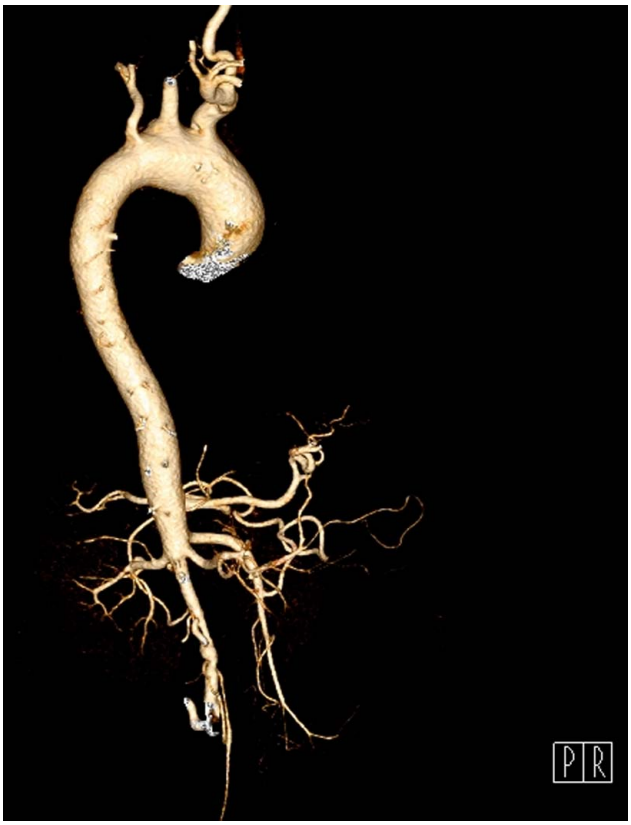

Figure 2. Repeat 3D CT aortogram conducted again during the case management. Findings revealed marked stenosis of the subclavian artery and the distal infrarenal aorta (nonvisualized bilateral common iliac vessels) and multiple systemic collaterals arising from the inferior epigastric, superior epigastric, internal thoracic, and iliolumbar arteries.

Management

A multidisciplinary approach with consultations from rheumatology, cardiology, and pulmonary medicine was used, and the patient was managed with pulmonary rehabilitation, long-term domiciliary oxygen therapy, long-term prednisolone therapy (7.5 mg) once daily, and mycophenolate at 500 mg twice daily. The patient was advised to continue her heart failure medications as well: Tab metoprolol 25 mg once daily and tab furosemide 10 mg once daily. Gradually, the patient's shortness of breath improved, PFT also improved, and she was advised to perform routine follow-ups to adjust her medications every month. To monitor her cardiovascular profile and PFT, the patient was advised to undergo echocardiography and PFT after 3 months.

Discussion

This case report highlights the unusual presentation of ILD in a 42-year-old female patient with a long-standing history of TAK who was recently diagnosed with HFpEF. The patient, who had been receiving immunosuppressive therapy for more than a decade, presented with progressive respiratory symptoms, including persistent dry cough and shortness of breath, eventually leading to a diagnosis of ILD. HRCT of the chest revealed features consistent with probable UIP, a pattern rarely associated with Takayasu arteritis. The novelty of this case lies in the rare co-occurrence of ILD with Takayasu arteritis, a combination scarcely reported in the medical literature. Pulmonary rehabilitation and long-term domiciliary oxygen therapy alongside continued immunosuppressive and heart failure treatment resulted in clinical improvement. This case emphasizes the need for individualized patient care with a multidisciplinary approach in such complex cases.

Takayasu arteritis (TAK) is a rare type of vasculitis that primarily affects large arteries through inflammation and obliteration. Pulmonary involvement is a common manifestation of TAK, but it mostly affects the pulmonary vasculature, and parenchymal involvement is extremely rare[5]. Although ANCA-associated small vessel vasculitis is more likely to cause permanent lung parenchymal damage, it is rare for patients with large vessel disease, such as TAK, to exhibit pulmonary parenchymal manifestations, and knowledge related to this topic is limited to a few case reports[6–8]. Mehta *et al*.[6] reported a case of biopsy-proven ILD associated with Takayasu arteritis that was successfully managed with tapering prednisolone therapy, mycophenolate mofetil and abatacept. Greene *et al*.[7] presented a patient with TAK who was shown to have interstitial pulmonary fibrosis and mesangial glomerulonephritis, which suggested that a common antigenic stimulus resulted in tissue injury. Similarly, Dziadzio

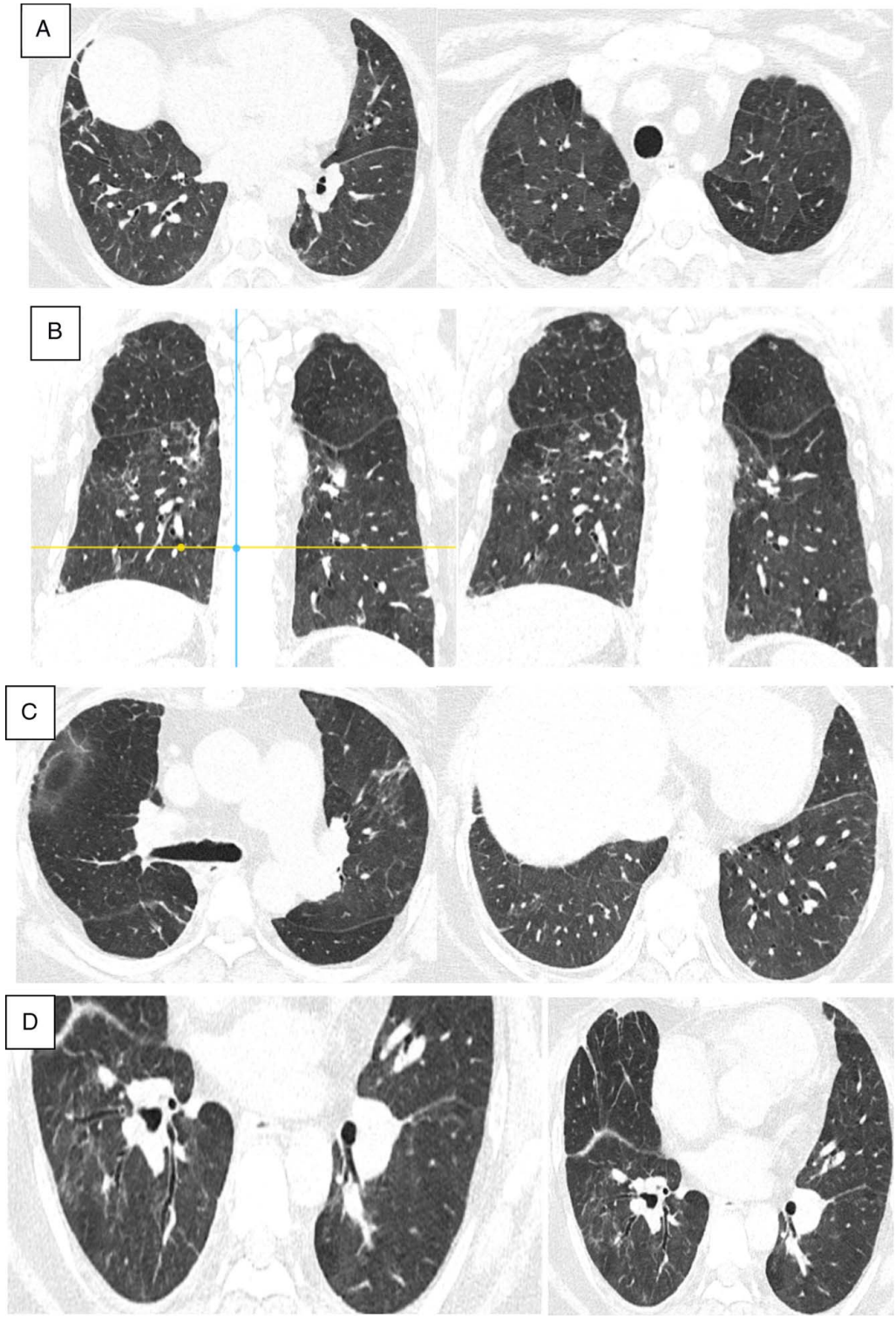

Figure 3. HRCT chest of the patient showing. A: Mosaic attenuation of the lung parenchyma (left and right images) with interlobular septation thickening. B: Lower lobes predominance of traction bronchiectasis and reticulations with subtle ground glass opacities. (Left and right images). C: Reticulations in the right middle lobe and Lingula (left image) and fine reticulations in lower lobes (right image) with relative subpleural sparing D: Traction Bronchiectasis and subtle ground glass opacities in lower lobes (left and right images). Thickening of bronchovascular bundles can also be noted. All of these findings are consistent with Interstitial lung disease (probable UIP pattern).

et al.[8] described a case of TAK with parenchymal pulmonary involvement associated with spondylarthropathy.

Respiratory system involvement is a common and important feature of ANCA-associated systemic vasculitis, unlike other SV[10]. The association of interstitial lung disease with ANCA-associated vasculitis is well established, with prevalence rates of up to 23% in patients with granulomatosis with cholangitis and 45% in patients with microscopic cholangitis[11]. However, no studies have reported the prevalence of ILDs in patients with large vessel vasculitis, specifically Takayasu arteritis, as this unusual association has been demonstrated in only a few case reports[6–8].

There is a large difference between the pathophysiologies of ILD and Takayasu arteritis. Studies related to the pathophysiology behind vasculitis-associated ILD are mostly related to ANCA-associated SV (AASV), and whether this ANCA-related association can be extrapolated to other systemic vasculitis, such as TAK, remains elusive[12–14]. Among the several mediators postulated to contribute to the development of ILD in AASV, repeated episodes of intra-alveolar hemorrhage seem to be key predecessors of pulmonary fibrosis. In their review of open lung biopsy samples from patients with AASV, Travis *et al*.[15] reported that ~49% of patients had alveolar hemorrhage, and Schnabel *et al*.[16] reported that hemosiderin-laden alveolar macrophages are common in the bronchoalveolar lavage fluid of patients with primary systemic vasculitis but are uncommon in those with collagen vascular diseases and rheumatoid arthritis (RA). Furthermore, some of the initial CT images of consolidation consistent with alveolar bleeding evolved into areas of honeycombing during the long-term follow-up of ANCA patients[17]. On the other hand, the pulmonary vascular manifestations of TAK mostly involve various degrees of irregularity, stenosis, and obstruction, and a study by Vanoli *et al*.[18] demonstrated 93% stenosis, occlusion (57%), dilatation (16%), and aneurysm (7%). Pulmonary hemorrhage in TAK is unusual, and cases have been limited to a few case reports[19,20]. Similarly, reactive oxygen species such as hypochlorous acid and proteolytic enzymes, which are released by anti-MPO ANCAs, can trigger fibroblast proliferation and induce pulmonary fibrosis[13]. ANCA activity is not present in TAK, and instead, its pathophysiology largely depends upon CD4+- and CD8+-dependent cell-mediated immunity, resulting in granuloma formation and the release of proteases such as matrix metalloproteases (MMPs), ultimately leading to chronic inflammation and fibrosis[1]. However, no research has clarified the role of this pathophysiological process in interstitial lung disease, and knowledge of its role in promoting pulmonary fibrosis is limited.

The diagnosis of interstitial lung disease consists of evaluating a detailed patient history and physical exam coupled with laboratory testing, imaging, physiologic testing, and possibly a biopsy. High-resolution CT (HRCT) is of paramount importance in diagnosing ILD, especially in resource-limited countries where biopsy may not be financially or technically feasible. HRCT describes ILDs in terms of (a) upper lobe-versus lower lobe-predominance; (b) peripheral versus central (central is also termed peribronchiolar); and (c) whether the opacities spare the extreme periphery of the lung[21]. The presence of pulmonary fibrosis is indicated by a) reticular opacities, (b) traction bronchiectasis, (c) honeycomb changes, and (d) volume loss[21]. Consolidations and ground glass opacities should also be considered. As with histopathology for ILD, CT imaging patterns may be described as representing a) typical interstitial pneumonia (UIP), b) nonspecific interstitial pneumonia (NSIP), or c) organizing pneumonia (OP). NSIP is usually homogeneous, whereas UIP is typically heterogeneous, patchy, and irregular[22]. The extent of honeycombing and traction bronchiectasis is greater in UIP than the extent of ground glass opacity or micronodules, which are more commonly associated with an NSIP pattern[22]. Honeycombing must be present for the diagnosis of UIP; however, a subpleural and basal predominant heterogeneous distribution of reticular distribution with peripheral traction bronchiectasis in the absence of honeycombing can be classified as 'probable UIP'[23]. HRCT in our patient revealed an apicobasal gradient with a lower lobe predominance of traction bronchiectasis and reticulations with minimal ground glass opacities. There were relative subpleural sparing and reticular changes with interlobular septation and thickening of the bronchovascular bundle. Thus, a probable UIP pattern of ILD was diagnosed. The patient refused bronchoalveolar lavage or lung biopsy because of financial constraints.

Management of ILD with Takayasu arteritis can be difficult because there are no established guidelines, and treatment includes immunosuppression guided by symptoms, clinical conditions, and supportive imaging. A multidisciplinary approach and strong clinical expertise might help slow the progression of the disease. The management of this disease may closely resemble that of ILDs associated with AASV, where the strategy is to induce remission via the use of potent immunosuppressants followed by a maintenance phase to prevent relapse[13]. Mycophenolate mofetil and rituximab could be potential options for these patients on the basis of the experience obtained for the treatment of ILD associated with other autoimmune disorders, such as systemic sclerosis (SSc)[24]. Novel antifibrotic therapies (pirfenidone and nintedanib) designed for idiopathic pulmonary fibrosis might be tested for TAK-associated ILD in the future. In severe cases, lung transplantation may also be considered. The use of steroids should be carefully monitored, as a study by Karabacak *et al*.[25] reported a greater incidence of glucocorticoid-related side effects in adults with Takayasu arteritis. Our patient was managed with pulmonary rehabilitation, long-term domiciliary oxygen therapy, long-term corticosteroid therapy at a tapering dose, and mycophenolate mofetil. Heart failure medications were also prescribed to manage HFpEF. The patient gradually improved and visited the OPD for routine follow-up, with symptomatic improvement along with improvement in her pulmonary function test. A summary of the case from history to management can be found in Figure 4.

This case report has several limitations. First, the diagnosis of interstitial lung disease (ILD) in this patient was made without histopathological confirmation, as the patient declined bronchoalveolar lavage or lung biopsy because of financial constraints. While high-resolution CT (HRCT) provides strong evidence for a probable UIP pattern, histological confirmation could have strengthened the diagnosis. Second, the management of ILD in the context of Takayasu arteritis (TAK) is largely empirical, as there are no established guidelines for treating this rare combination. The treatment decisions were based on clinical experience and extrapolation from the management of ANCA-associated ILDs, which may not be fully applicable to TAK-associated ILD. Third, the patient's inconsistent use of corticosteroids and immunosuppressants over the years may have influenced the disease course and response to treatment, complicating the interpretation of

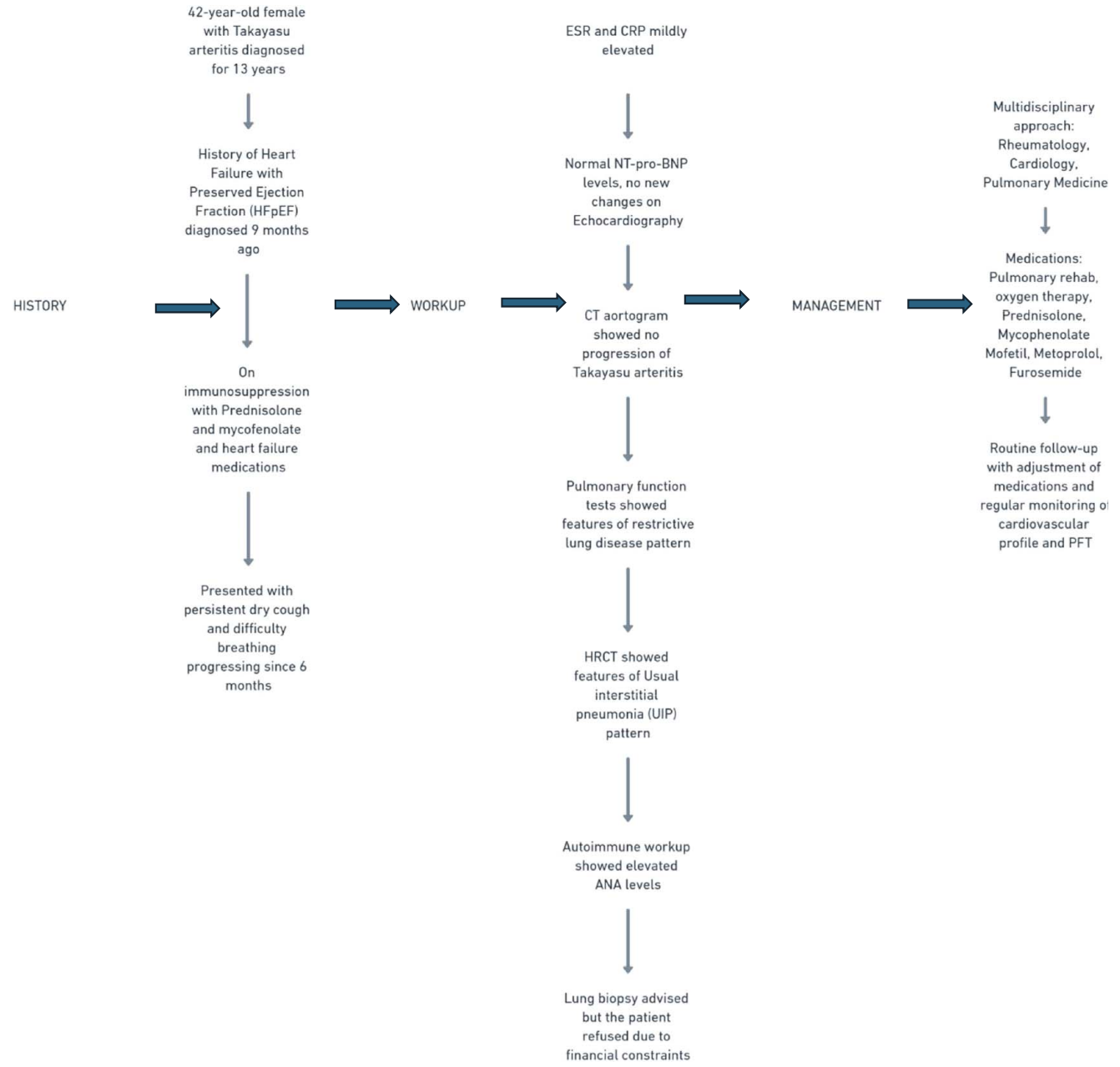

Figure 4. A summary of the case from history to management.

outcomes. Finally, this case report is limited by its single-patient design, making it difficult to generalize the findings to other patients with similar conditions.

Conclusion

This case report highlights the rare and challenging co-occurrence of Takayasu arteritis and interstitial lung disease in a 42-year-old female, demonstrating the complexities involved in managing overlapping autoimmune and respiratory conditions. The patient, who had a longstanding history of TAK and was recently diagnosed with HFpEF, presented with respiratory symptoms that ultimately led to the diagnosis of ILD with a probable UIP pattern. Management requires a multidisciplinary approach, including immunosuppressive therapy, pulmonary rehabilitation, and careful cardiovascular monitoring, which results in clinical improvement. The literature has established the association of ILDs with AASVs; however, research has failed to address the relationship between Takayasu arteritis and ILDs. This case highlights the importance of considering ILD as a potential complication in patients with chronic vasculitis, particularly when they present with unexplained respiratory symptoms. Because TAK is a rare type of vasculitis, research related to its role in ILDs has been limited to a few case reports. As such, the management of such patients can be confusing and tedious. This

case contributes to the limited literature on TAK-associated ILD and highlights the importance of individualized, multidisciplinary care in managing such complex cases. Further research is necessary to explore the pathophysiological mechanisms and optimal treatment strategies for this rare association. Research agendas for TAK-associated ILDs must focus more on their pathophysiological relationship to guide novel therapeutic agents and make management slightly less cumbersome.

Ethics approval

It is exempted at my institution. We do not need to get approval from the ethical committee for the case report.

Consent

Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

Source of funding

None.

Author contribution

K.B.: conceptualization, design and coordination of the study, visualization, data collection, wrote original draft and manuscript, and review of the manuscript; S.G., A.S., R.K.Y., and S.S.: wrote original draft and manuscript, and review of the manuscript.

Conflicts of interest disclosure

The authors declare that they have no financial conflict of interest with regard to the content of this report.

Research registration unique identifying number (UIN)

Not a first in man study.

Guarantor

Kritick Bhandari.

Data availability statement

All data generated or analyzed during this study are included in this published article.

Provenance and peer review

Not commissioned, externally peer-reviewed.

Statement

This article has been written in line with the SCARE guidelines[26].

Acknowledgement

The authors would like to express their sincere gratitude to the Department of Rheumatology, Cardiology, and Pulmonology of KIST Medical College for their invaluable contributions to the management of this patient. Their expertise and collaborative efforts were crucial in the diagnosis and treatment process, and their insights greatly enhanced the quality of care provided.

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