Case Report

Bronchiectasis as rare initial manifestation of Rheumatoid arthritis- A case report

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Abstract

Bronchiectasis is defined as an irreversible abnormal dilatation of the bronchial tree. It has a variety of underlying causes, with a common etiology of chronic inflammation. Bronchiectasis is one of the most prevalent respiratory signs of rheumatoid arthritis (RA). Although a hereditary predisposition to RA-related bronchitis has been shown, the notion of chronic bacterial infection-induced autoimmunity causing permanent bronchial damage is becoming increasingly popular among researchers. Patients with RA who have a persistent cough, excessive salivation, or recurrent respiratory infections should be suspected of having bronchiectasis; high-resolution thoracic computer tomography is required to establish the diagnosis. A multimodal treatment strategy is used to treat patients with bronchiectasis linked to RA

Introduction

Bronchiectasis is a common extra articular manifestation of Rheumatoid Arthritis with prevalence ~18% [2]. However prevalence of bronchiectasis as initial manifestation of RA is rare and not found in literature. RA isan autoimmune illness that affects the multiple systems in the body and is often characterized by extra-articular manifestations during initial presentation. RA is a chronic inflammatory disease that mostly affects the synovial joints and is frequently brought on by the interplay of genes and environmental factors.[1] Bronchiectasis is defined as an irreversible abnormal dilatation of the bronchial tree. It has a variety of underlying causes, with a common pathology of chronic inflammation.

Case report

A 40-year-old male patient was admitted to the Respiratory Medicine Department of NRSMCH with chief complaints of multiple episodes of respiratory tract infection with profuse sputum for 5 years and joint pain for 7 months. Joint pain involvedsmall joints of hands and foot was present, which was insidious in onset, gradually progressive, started 7 months back, and was maximum after waking up from sleep and relieved gradually with activity of an hour; suggesting inflammatory joint pain. No significant history of hospital admission or chronic medication intake was present.

General examination was unremarkable except for mild pallor.The examination of the respiratory system revealedbiphasic coarse rhonchi and crepitation over bilateral infrascapular areas. On joint examination, bilateral symmetrical swollen and tender small joints involvement of hands including metacarpophalangeal (MCP), proximal interphalangeal(PIP), and wrist joint was present with sparing of the distal interphalangeal(DIP) joints. Also in the foot metatarsophalangeal (MTP) joint along with PIP was involved. No pain or tenderness of the axial skeleton was noted. Nojoint deformity was noted.

Routine blood investigation showed mild anemia(10.9g/dl) with normal Total Leukocyte count (7500/ul), and platelets. Renal and Liver function tests were unremarkable. Serology for routine viral markers was negative. Sputum for acid-fast bacilli was negative.A Chest X-ray revealed bilateral lower-zone fibro cystic opacities(**Figure 1**). Following this, a High-Resolution Computed Tomography(HRCT) scan of the thorax was done which revealed cylindrical bronchiectasis in bilateral lower lobe(**Figure 2**).Bronchoscopy was done,

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bronchoalveolarlavage fluid gram stain, fungal stain and culture shows no growth. BAL AFB, CBNAAT was negative. BACTEC culture for M.TB and NTM was negative.Total serum IGE was 78 IU/ml. Ruling out infective and allergic conditions. Autoimmune workup showed RA factor 312, IU/ml, CRP 15.2 mg/L, Anti-CCP 1146 EU/ml (highly raised), and other autoimmune antibody profiles came back negative. Putting the clinical and lab findings in ACR-EULAR RA diagnostic criteria gives a score of 10/10(definite RA). A final diagnosis of Rheumatoid arthritis(RA) with exacerbation of bronchiectasis was made. Appropriate antibiotics, analgesics, and bronchodilators were given while admitted. The patient was discharged after 14 days with advice for airway clearance and a plan for starting management of RA after consultation with the Rheumatology department.The patient is now under regular follow-up under the Department of Respiratory Medicine and 6 months into therapy for RA is doing well with the resolution of his symptoms

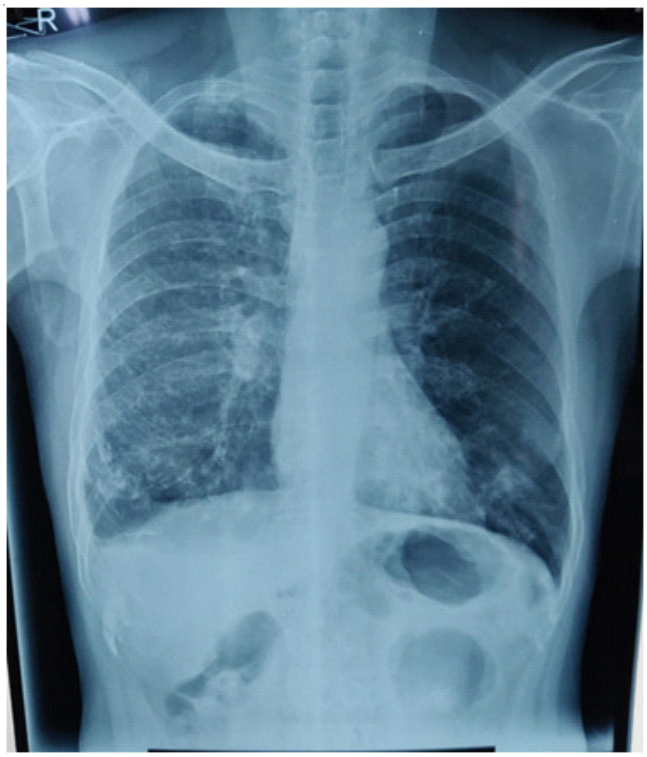

Figure 1 – Chest radiography in postero anterior view showing both lower zone predominant fibro cystic opacities.

Discussion

Rheumatoid arthritis is a chronic inflammatory disease ofunknown etiology characterized by symmetric polyarthritis

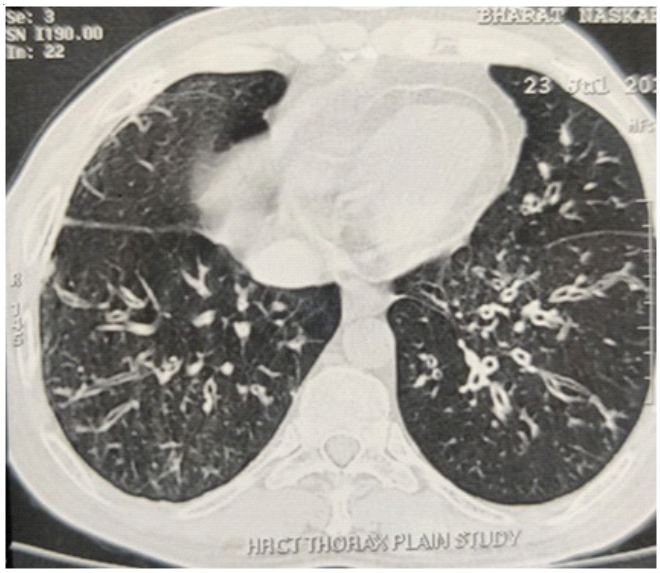

Figure 2 – HRCT chest showing bilateral lower lobebronchiectasis(shown by arrow).

and isthe most common form of chronic inflammatory arthritis.[1]

RA can lead to various extraarticular manifestations; and involvement of all major organ- systems including cardiac, pulmonary, central nervous system, renal, skin,oral, ocular, endocrine, and Musculo skeletal system. RA may be complicated by pulmonary disease with the involvement of any compartment of the lungs(airways, interstitium, Pleura, pulmonary vascular), which can manifest before, synchronously, or after the development of articular disease.[1,3,4].

Regarding impact of bronchiectasis in RA; bronchiectasissevere enough to produce clinical symptoms, may complicate the use of immunosuppressive medications, particularly anti-TNF agents as both bronchiectasis and anti-TNF therapy increase the risk of certain pulmonary infections. Among patients with rheumatoid arthritis and bronchiectasis, mortality rates are higher than for either condition alone [12]. There are no specific guidelines for the management of rheumatoid arthritis with bronchiectasis, and therapy is the same as for either condition alone, with bronchodilators, antibiotics and bronchial hygiene used to treat bronchiectasis.

Increasing evidence suggests, that the mucosa of the lung plays a crucial role in the very beginning of pathogenesis of RA. According to it, various environmental factors lead to post-translational protein modification(such as citrullination) in the airways.In the context of specific genes, autoimmunity against those proteins develops locally initially(IgA and IgG ACPA).

Usual interstitial Pneumonia	Non specific interstitial Pneumonia	Organizing pneumonia	Diffuse alveolar damage	Follicular bronchiolitis	Constrictive bronchiolitis	Bronchiectasis	Pleural disease	Alveolar hemorrhage
++	++	+	±	±	+	++	+++	±

Also, antibodies against the Fc domain of self-Ig proteins are formed, called RF. Later these antibodies find their way into systemic circulation due to loss of mucosal barrier integrity. Analysis of induced sputum has revealed the presence of these antibodies years before in RA patients when serum Anti ccp and RF are absent.[4,5,6]

Common manifestations of lung involvement in RA are summarized in Table no 1[7,8]

Commonly seen patterns include usual interstitial pneumonia (UIP)-like pattern with bilateral subpleural reticulation with or without honeycombing - considered to be a hallmark of worse prognosis. Non-specific interstitial pneumonia (NSIP)-like pattern with predominant ground-glass opacities.Inflammatory airway disease pattern with centrilobular branching lines with or without bronchial dilatation. Organizing pneumonia-like pattern with patchy areas of consolidation. Caplan Syndrome is a condition of Rheumatoid Arthritis associated with Upper Lobe Predominant Lung Nodules. These nodules can cavitate, and there may also be a pleural effusion. The common term RA-associated Interstitial lung disease (RA-ILD) is used to describe the radiological findings of RA. It is a source of substantial morbidity and mortality for affected patients. [9,10]

Conclusion

CTDs are a heterogeneous group of disorders with a proposed autoimmune etiology. Multiple CTDs have variable degrees of lung involvement which is seen on HRCT thorax. Lung involvement in RA is common however bronchiectasis as an initial presenting manifestation of RA is rare .Workup for bronchiectasis should always include an autoimmune profile especially where obstructive and infective causes have been ruled out. Treating physicians must be aware of the combination of typical joint findings on clinical examination along with recurrent chest infections which can give a clue towards diagnosis.

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