A case of rheumatoid arthritis with valvular and vocal cord involvement

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ABSTRACT

Chronic inflammatory polyarticular joint pain is the most common presenting feature of rheumatoid arthritis (RA). Apart from it, other system involvement symptoms and signs are frequently associated. We are reporting a case of RA that presented as a respiratory case, was diagnosed to have valvular heart disease and was eventually diagnosed as RA with cardiovascular involvement along with the unique and rarely reported laryngeal involvement causing loss of voice temporarily.

Key words: *Rheumatoid arthritis, Valvular heart disease, Vocal cord palsy*

Rheumatoid arthritis (RA) is a chronic inflammatory disorder characterized by synovial inflammation of joints causing typical symptoms of small joints inflammatory polyarthritis. It is the most common form of chronic inflammatory arthritis. It involves joints of the hands and larger joints as well. It also presents with extra-articular manifestations in the form of fatigue, subcutaneous nodules, lung involvement, pericarditis, peripheral neuropathy, vasculitis, and hematological abnormalities such as anemia of chronic disease, neutropenia, splenomegaly, Felty syndrome, leukemia, and lymphomas [1].

Among the patients of RA, the incidence of valvular heart disease (VHD) as a result of inflammation caused by RA has been reported and evaluated [2]. The involvement was mainly in the form of the left side valve thickening, nodules, and regurgitation. RA is also known to alter the risk of atherosclerosis and heart failure [3]. We are reporting a case who had VHD as a result of his RA along with laryngeal involvement of RA which is an atypical clinical feature of the disease [4,5].

CASE REPORT

A 38-year-old patient presented to our hospital after having been referred from Solapur Medical College for the evaluation and further management of consolidation of lungs causing persistent cough and bloody expectoration. The patient had developed the complaints 1 month ago. He had associated breathlessness. The complaints were subacute onset and progressive over the past 1 month. There was no history of such complaints in the past. There were no associated complaints such as chest pain, swelling of body, or palpitations. The cough was frequent and associated with bloody expectoration. There was a history of associated fever. A history of exposure to hazardous substances, gases, and smoking was not present. There was no history of any heart diseases.

On general examination, tachypnea, tachycardia, and pallor were present. Pulse was high volume, 110/min and regular in nature. Blood pressure was 140/80 mmHg. O_2 saturation was 92% on room air. On systemic examination, the chest showed crepitations over the right infraclavicular area anteriorly with bronchial breath sounds, a few crepitations on the left side anteriorly, and normal vesicular breath sounds on all other regions. Cardiovascular (CV) examination showed an outward and downward apex and normal S1 and S2. No murmurs were heard. Other systems examination was within normal limits.

Our differential diagnoses at this stage were the consolidation of right upper lobe infective, pulmonary tuberculosis, and underlying cardiac pathology in view of a high-volume pulse and an outward and downward displaced apex.

The initial X-ray that was obtained showed heterogeneous opacities in the right upper zone. Electrocardiogram was suggestive of the left ventricular hypertrophy. Total leukocyte count was increased (27,000/mm^3) with neutrophils on the higher side. Sputum studies for Gram staining, culture, and *Mycobacterium tuberculosis* were awaited.

The patient was started on amoxicillin-clavulanic acid and azithromycin considering it to be a case of pneumonia. The patient, however, continued to be breathless and the saturation hovered around 94–96% even on oxygen supplementation. Computed tomography (CT) scan of the chest was planned to look at the lesion in a more detailed manner. CT pulmonary angiography was also done. It was suggestive of multiple nodular opacities in bilateral lungs with "tree in bud" appearance suggestive of an infective etiology. There was evidence of pulmonary embolism in subsegmental branches on both sides. The high-resolution computed tomography done at the previous hospital had only shown bilateral consolidation and ground-glass opacities.

We started evaluating him for the cause of his pulmonary artery thrombus. Transthoracic 2D echocardiogram was done which showed moderate mitral regurgitation and moderate aortic regurgitation along with mild pulmonary artery hypertension. The thrombophilia profile was sent. Sputum studies came by now which showed tuberculosis bacterium to be absent but the presence of methicillin-resistant coagulase-negative *Staphylococcus aureus*. The antibiotics were tailored accordingly.

Hence, now, we had a case of the infective etiology pneumonia with pulmonary embolism, the etiology of which was under consideration, and rheumatic-like heart condition. There were a few possible scenarios in front of us. The first possibility could be that the patient had pneumonia, pulmonary embolism due to whatever reason, and rheumatic heart disease. This, however, seemed less likely as the patient had had no past complaints suggestive of rheumatic heart condition. The second scenario was that pneumonia had resulted in infracted parts of the lungs due to pulmonary embolism which, in turn, could have happened due to a primary thrombotic or a secondary thrombotic condition or a heart disease. It was to look for a thrombotic condition that we had sent a thrombophilia profile and also began to look for a secondary cause like a connective tissue disorder which could also explain the heart condition.

On specific questioning, the patient gave a history of joint pains which was inflammatory in nature and involved the small joints for the past 8–10 years. He also gave a history of taking steroids daily which he had been prescribed by a local doctor for his pains. On investigations, the patient's C-reactive protein, erythrocyte sedimentation rate, RA factor, and anti-cyclic citrullinated peptide levels were all strongly positive.

Hence, now, we had a diagnosis of a case of RA with CV involvement and pulmonary embolism with coexistent pneumonia. The patient was continued on antibiotics. He was also put on anticoagulants and angiotensin-converting enzyme inhibitor with diuretics. Disease-modifying antirheumatic drugs were planned for him with tapering of the steroids that he was taking.

The patient was doing well for the next few days in the ward when he developed a new symptom in the form of a sudden loss of voice. A central nervous system examination revealed nothing. We thought it could be a local laryngeal cause for which an ENT opinion was taken. There on laryngoscopic examination, the patient's left vocal cord was found to be paralyzed and the right-sided one was moving less. We ruled out local causes for recurrent laryngeal nerve palsy by looking at the CT imaging of the neck. After ruling out other possible causes for vocal cord palsy, we concluded that the palsy was due to the rheumatoid process involving the cricoarytenoid joints of the vocal cords which is very rare but reported. The patient was started on methotrexate and his symptoms began to resolve in about 2 weeks.

The final diagnosis that we made for this patient was a case of RA with valvular involvement in the form of aortic incompetence and mitral incompetence along with pulmonary embolism, most likely resulting from steroid misuse or the inflammatory disease process, complicated by pneumonia and vocal cord palsy. It turned out to be a rare case of RA presenting with vocal cord involvement along with several complications in the same patient.

DISCUSSION

RA is known to affect the heart in various ways. Roldan *et al*. studied 34 volunteers with RA with a mean age of 50±10 years underwent clinical evaluation and transesophageal echocardiography. Findings on transesophageal echocardiography were compared with those of 34 gender-matched healthy volunteers with a mean age of 42±6 years. Twenty patients (59%) had mainly (97%) left-sided VHD, valve nodules in 11 (32%), valve thickening in 18 (53%), valve regurgitation in 7 (21%), and valve stenosis in 1 (3%) compared with 5 controls (15%; nodules in 1, 3%; thickening in 4, 12%; and regurgitation in 1, 3%; $p \leq 0.05$ for all vs. patients). Valve regurgitation manifested as mild aortic regurgitation in four patients, moderate mitral regurgitation in four patients, and moderate tricuspid regurgitation in one patient. Mitral and aortic valve stenoses occurred in 1 patient (3%). No correlation was found between VHD and duration, activity, severity, pattern of onset and course, extra-articular disease, serology, or therapy of RA [2].

Peter *et al*. showed that patients with RA have a 1.5–2.0-fold increased risk of developing coronary artery disease (CAD) compared with the general population [6] similar in magnitude to the risk imparted by diabetes mellitus [7]. This increased CAD risk is evident even before the clinical recognition of RA: At diagnosis, individuals with RA were over 3 times as likely to have had a prior myocardial infarction than subjects without RA.

An expert committee of the European League Against Rheumatism has recommended that CV risk scores (e.g., Framingham) be multiplied by 1.5 in some patients with RA to reflect their increased risk of heart disease [8]. Patients with RA also have twice the risk of developing heart failure [9]. This risk is more pronounced in the RA patients who are rheumatoid factor positive than among seronegative patients. Patients with RA are less likely to have typical signs and symptoms of heart failure, tend to be managed less aggressively, and have poorer outcomes [10].

Mikkelsen *et al*. have reported three cases of laryngeal involvement in RA [4]. Grossman *et al.* found that half of the patients with RA had laryngeal symptoms [11]. The clinical presentation may vary from being asymptomatic to a constellation of upper aerodigestive symptoms. The array of symptoms includes odynophagia, foreign body sensation, dysphagia, sore throat, lump sensation in the throat, change in voice quality, referred otalgia, and respiratory symptoms [12].

In a study by Amernik on 77 patients with recognized RA with average disease duration of 9.4 years, the most frequent complaints were foreign body sensation in 51%, hoarseness in 47%, and voice weakness in 29% of the cases. In acute phases, patients may complain of burning, foreign body sensation in the throat, and difficulty in swallowing. In chronic cases, the cricoarytenoid joint is usually affected with resultant fixation, and airway symptoms may arise often necessitating an emergency tracheotomy [13].

The medical treatment consists of administering steroids or nonsteroidal anti-inflammatory drugs to avoid the formation of nodules and fibrosis. The effect of steroid treatment is less pronounced in cases of laryngeal nodules, probably due to the late diagnosis and the subtle clinical course of these lesions. The steroids may be given systemically or locally into the joint as reported by Habib [14].

CONCLUSION

RA being a chronic inflammatory disorder involves a variety of organ systems in the body and the presentations are also varied. This case is being presented to stress on the fact that unusual presentations of the disease should be kept in mind while diagnosing and treating the disease.

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