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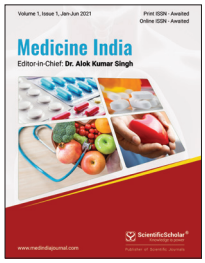

Case Report

Unusual presentation of rheumatoid arthritis as bilateral pleural effusion and asymmetrical arthropathy

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Received : 22 September 2022
Accepted : 27 December 2022
Published : 21 January 2023

DOI
10.25259/MEDINDIA_9_2022

Quick Response Code:

ABSTRACT

Rheumatoid arthritis (RA) is one of the most frequent chronic autoimmune diseases affecting the joints and the appendicular skeletal system. RA can present with multiple presentations and pulmonary involvement also can occur in RA. Inflammatory pleural effusions are uncommon in RA individuals, occurring in roughly 2–5% of cases. Here, we report a case where the patient came with pleural effusion on both sides and asymmetrical arthritis with a background of cervical radiculopathy due to an injury that occurred at school age. Further examination of the individual and the pleural fluid revealed that it was compatible with pleural effusion caused by RA. The joints on the weak side were devoid of all intense features of RA. Oral non-steroidal anti-inflammatory medications and disease-modifying anti-rheumatic medicines worked effectively for the patient.

Keywords: Asymmetrical, Radiculopathy, Rheumatoid arthritis, Pleural effusion

INTRODUCTION

Rheumatoid arthritis (RA) is an autoimmune illness that causes symmetrical chronic inflammatory polyarthritis. The disease's hallmark is joint inflammation. Advancement of this disorder causes erosion and destruction of bones and joints, resulting in the typical presentation of symmetrical deforming polyarthropathy. RA may present with different extra-articular manifestations and organ-specific involvement.[1] Pleuritis is the most common presentation among the pulmonary manifestations.[2,3] Inflammatory pleural effusions are uncommon complications that affect roughly 2–5% of RA individuals.[4,5] Here, we report a unique presentation of an RA individual with pleural effusion on both sides and asymmetrical RA changes with significant attenuation in severity over his right hand, which was also impacted by long-standing weakness due to cervical radiculopathy from a childhood injury.

CASE REPORT

A 60-year-old male individual came with the problem of prolonged left-hand pain and pleuritic chest pain for the past 10 days. His left-hand discomfort was sporadic and worst over the wrist and minor joints, with increased stiffness during the morning that lasted more than an hour and was accompanied by increasing local warmth and edema. He denied experiencing numbness or paresthesia in his extremities. On the other hand, his right hand was undisturbed by joint discomfort or swelling. Following childhood trauma, it has become noticeably weaker and less functioning. No eye discomfort, redness,

impaired vision, skin lesion, or burning micturition were reported by the patient, and he did not have any additional rheumatologic symptoms.

His vitals were normal on assessment. His blood pressure was 124/76 mmHg, PR was 78 beats/min, and RR was 20 cycles/min. Asymmetrical upper extremity polyarthropathy was discovered during a general physical examination. There was bony enlargement above his left hand's wrist, ulnar deviation, and Z thumb deformity [Figure 1]. There were edema and pain across his proximal interphalangeal joints, with his distal interphalangeal joints being spared. He could not bend or extend his left wrist, and his left hand could not even create a fist. Despite an ulnar deviation in his right hand, the wrist's range of motion was maintained at around 40° and 60°. With his right hand, he was still able to create a fist. Reduced air passage in the infra-scapular and infra-axillary areas on both sides and stony dullness were found on percussion, and fine crepitations were heard on auscultation.

Bilateral blunting of costophrenic angle was seen on chest X-ray [Figure 2]. CECT of the chest revealed bilateral pleural effusion. Fluid overload, anasarca, and systemic illnesses such as heart failure, chronic liver disease, or nephrotic syndrome were not seen. RA factor, ANA by hep2, and anti-ccp were sent together with normal investigations because of joint discomfort and pleural effusion. His Hb was 13.6 g/dL, TLC was 5680/mm^3, platelet count was 208000/mm^3, ESR was 64 mm in 1st h, RF (65 U/mL), anti-ccp (14 U/mL), and Quantitative CRP were high (80.6 mg/L). An X-ray of the hands revealed extensive erosion of the left wrist, with carpal bone collapse and juxta-articular osteopenia on both sides [Figure 3]. NCS and EMG studies showed right lower cervical radiculopathy. Serum ANA by the hep2 method was negative. A diagnostic pleural tap was performed using ultrasonography. Exudative neutrophil predominates effusion was detected in the pleural fluid investigation. ADA was 20. Diagnosis of RA was made based on EULAR/ACR criteria.

The patient was given naproxen for 1 week and then began on methotrexate 7.5 mg once a week, along with folic acid and sulphasalazine 500 mg twice daily, with regular follow-up. Over the next month, the patient's symptoms improved drastically, and a subsequent chest X-ray revealed no remaining pleural effusion. Moreover, he had his hand movements optimized through rehabilitation.

DISCUSSION

In connective tissue diseases, pulmonary manifestations are prevalent, and all parts of pulmonary tissue can be affected. Pleural abnormalities and ILD are RA's most pervasive pulmonary symptoms, while interstitial fibrosis and pulmonary hypertension are the frequent respiratory features in scleroderma. In our case, the patient came to the hospital with pleural effusion on both sides and pleuritic chest pain.

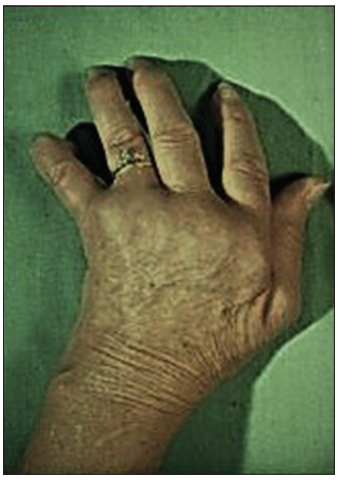

Figure 1: Left hand showing ulnar deviation of wrist and Z thumb deformity.

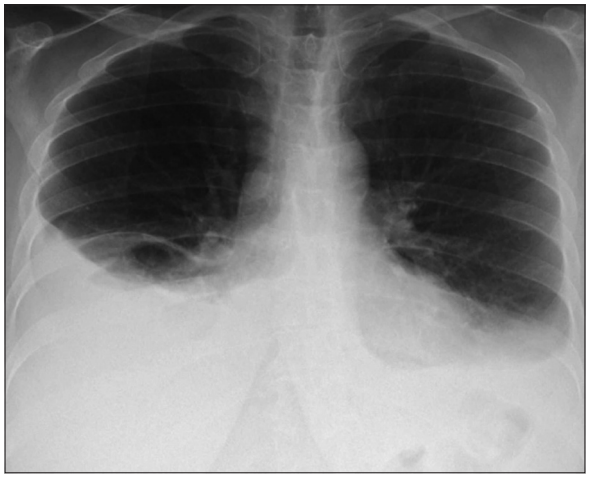

Figure 2: Chest X-ray showing bilateral blunting of costophrenic angle.

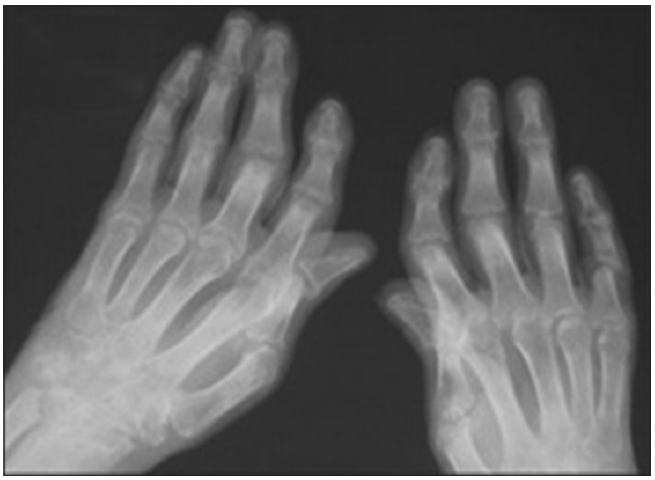

Figure 3: X-ray of hands showing advanced degeneration of the left wrist with the collapse of the carpal bones.

Pleural effusions in RA usually have mixed cell counts with high protein concentrations and are generally exudative.[6]

Rheumatoid factor levels in pleural fluid resemble serum levels. The pleural fluid's cytological and biochemical profile was comparable with pleural effusion in RA. The pleural effusion also had an increased cholesterol level, which is typical with RA.

The rheumatoid hands are classically present as symmetrical deforming polyarthropathy. Glick[7] proposed that relative disuse was associated with asymmetry of decreasing degree of RA joint alterations. At this time, the mechanism for disuse guards against RA is unknown.

There have been other alternative possibilities since then. One crucial concept proposed is the role of the neurological system in inflammation. JAM-B stands for Junctional Adhesion Molecule B (which has been proven to play a function in developing arthritis in mouse models earlier).[8] JAM-C, a human homolog of JAM-B, is known to be increased in synovial endothelium cells afflicted by RA. It promotes leukocyte adherence and transmigration to joint spaces, which aids attachment to synovial fibroblasts. In addition, the JAM-C cleaved molecule acts as a pro-angiogenic mediator.[9] Antibiotics that target JAM-C have been proven to postpone the onset of arthritis and reduce inflammation in animal models.

Overall, CNS innervation plays a role in controlling the microvascular setting of inflammatory arthritis by providing a signal necessary to keep up concentrations of effector molecules like JAM-C, enhancing the microenvironment's vascular permeability. When this signal is vanished due to denervation, the gene expression profile of endothelial cells in the synovium is altered, ensuing in lower vascular permeability, which results in less admittance to arthritogenic cells and chemicals, occurring in a more deficient inflammatory phenotype.[10]

CONCLUSION

Inflammatory polyarthritis and bilateral pleural effusion should raise the high index of suspicion of connective tissue illness. The rheumatoid factor and a reduced complement concentration in pleural fluid highly suggest RA-related pleural effusion. In paresis, the intensity of RA clinical symptoms is reduced. While it was previously considered to be due to forced immobility, mounting evidence suggests denervation causes the knockdown of genes involved in vascular permeability, imparting resistance to arthritis initiation. Important concluding points from this case reporting are when there is a coexisting CNS dysfunction in the ipsilateral limb; there is a need to address the likelihood of an uncommon occurrence of an asymmetrical RA with a reduction of its intensity.

AUTHOR'S CONTRIBUTIONS

1. Dr. Jonnalagadda Vihari: Concepts, Design, Data analysis, Manuscript editing and review, Manuscript preparation, 2. Dr. Neerukonda Sriteja: Data acquisition, 3. Dr. Samir Sahu: Definition of intellectual content, 4. Dr. Chandan Das: Definition of intellectual content, 5. Dr. Brijeshraj Swain: Data analysis, Statistical analysis, 6. Dr. Meghanad Meher: Data analysis, Statistical analysis, 7. Dr. Annamdevula Vamsi Krishna: Experimental studies, Clinical studies, 8. Dr. Adurty Aditya: Experimental studies, Clinical studies, 9. Dr. Tirumalaraju Veneeth Varma: Experimental studies, Clinical studies.

Declaration of patient consent

Patient's consent not required as patients identity is not disclosed or compromised.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

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How to cite this article: Vihari J, Sriteja N, Sahu S, Das C, Swain B, Meher M, *et al*. Unusual presentation of rheumatoid arthritis as bilateral pleural effusion and asymmetrical arthropathy. Med India 2023;2:4.