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Can an Infection with SARS COV-2 cause or Exacerbate Rheumatoid Arthritis? Description of A Clinical Case

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ABSTRACT

Infectious disease can activate the immune system favoring a highly inflammatory response.
Rheumatoid Arthritis is a long term inflammatory disease characterized by an autoimmune trigger.

What happens when SARS COV 2 infection occurs in a patient suffering from early stage of rheumatoid arthritis?

In this clinical case we want to highlight that the infection by SARS COV 2 could esacerbate or precipitate a clinical picture of Rheumatoid Arthritis.

This condition should make you reflect on the probable therapeutic implications.

Keywords

SARS COV-2, Rheumatoid Arthritis, Inflammatory disease, Cytokine storm, Lung involvement.

Introduction

The relationship between rheumatoid arthritis and infectious diseases is really complex as well as the cause-effect link has to be established [1].

Whether, on the one hand, infectious processes are decisive in the induction of autoimmune diseases because they activate the immune system, on the other hand, pathogens behave like real and true adjuvants of the immune response favoring a highly inflammatory environment.

The infecting agent can cause activation of both innate and acquired immunity by various pathways [2].

We describe a cse of a 50 year old woman who has contracted SARS COV 2 infection and has exacerbated rheumatoid polyarthritis.

A Case

A 50 year old woman who suffered for years, from artharlgia with localization to the hands, wrists, knees and ankles with motion limitations without associated elevated levels of acute phase of reactants of inflammation (C-reactive protein (CRP) and erythrocyte sedimentation rate (ESR) commonly observed to baseline in patients with rheumatoid arthritis.

The patient had carried out a rheumatological visit that had highlighted pain of the following joints: hands, wrists, knees and ankles, prescribed a therapy with antiinflammatory drugs and the research of rheumatoid factor and antibodies against cyclic citrullinate peptide.

During the month of April 2020 the patient presented fever, dry cough and dyspnea.

She performed a Chest CT showing areas of ground glass hyperdensity with an evident bilaterally at predominantly subpleuric disposition and more evident at level of the upper lobes and suspected phlogistic alterations. Fibrotic striae were noted in left basal posterior lobe (Figure 1,2,3,4 and 5).

Patient Consent: A patient approved the publication of the images of lung TC.

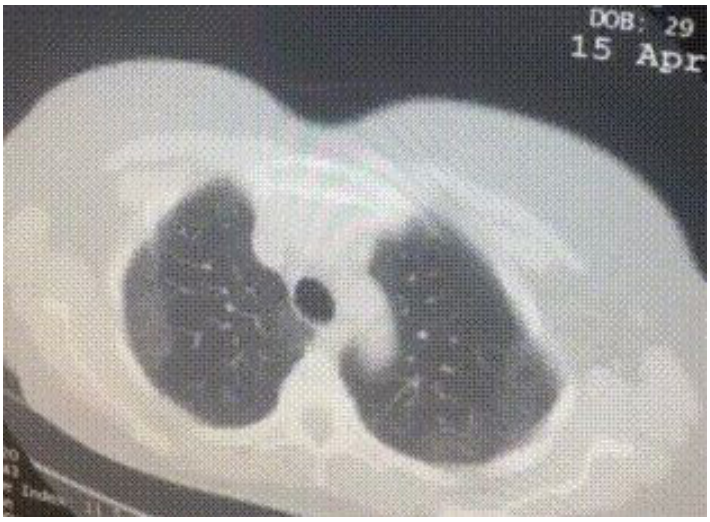

Figure 1

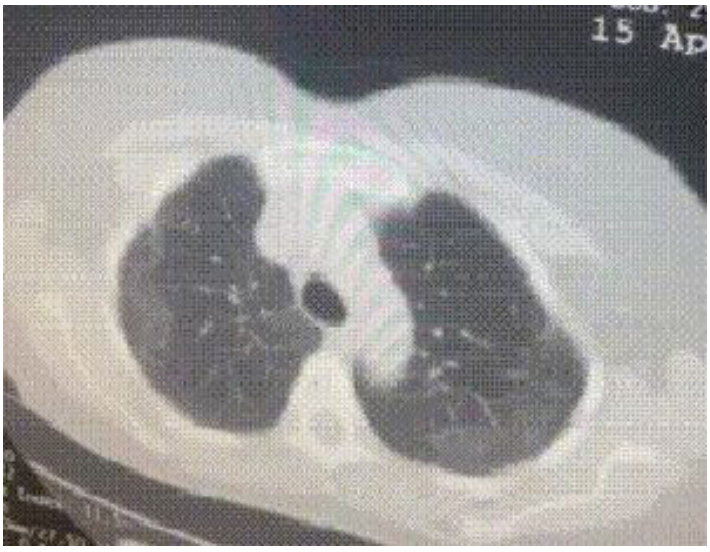

Figure 2

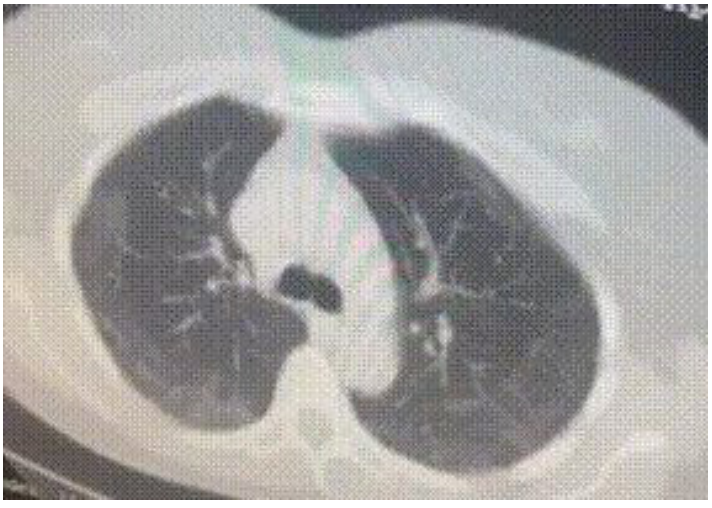

Figure 3

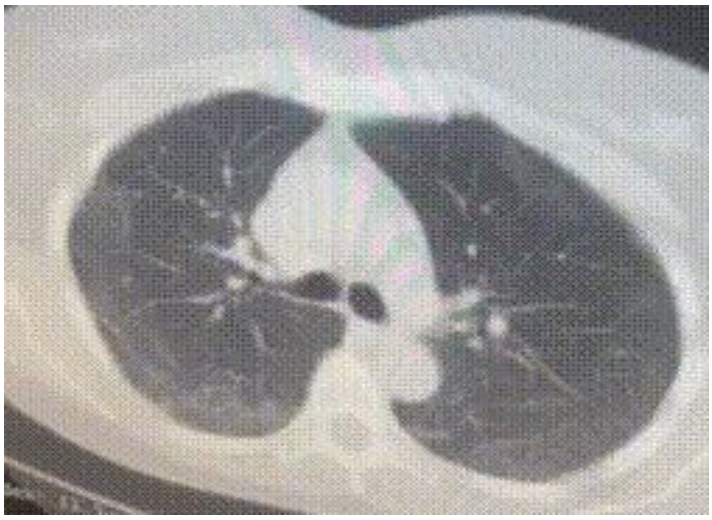

Figure 4

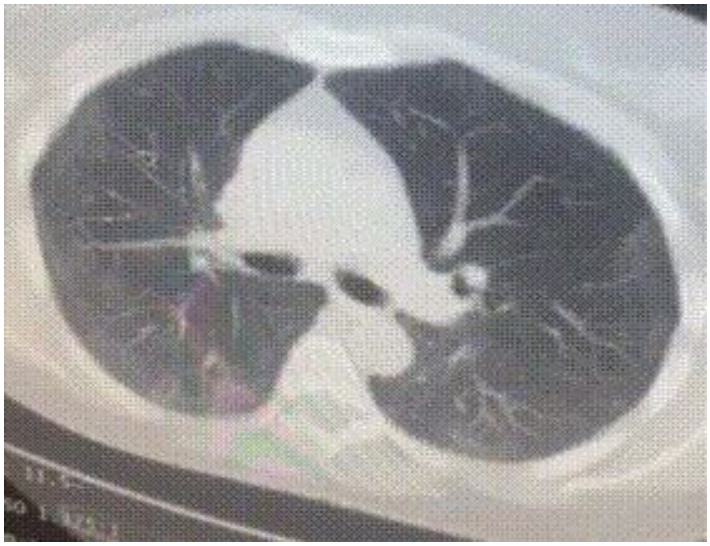

Figure 5

Legend to the Figures

COMPUTED TOMOGRAPHY (CT) show multiple areas of ground glass hyperdensity with an evident bilaterally at predominantly subpleuric disposition and more evident at level of the upper lobes and suspected phlogistic alterations.

A score TC according to Chung was applied which was 6 (LLSS4, medium lobe 1, LIS 1).

A nasopharyngeal swab was carried out which showed positive results for Sars Cov-2 and was performed a diagnosis of pneumonia SARS-COV-2 related.

She was hospitalized in intensive care, with a diagnosis of partial respiratory failure. The therapy included an antibiotic drug such as azithromycin at a dosage of 500 mg/day for 6 days, hydroxychloroquine 200 mg twice a day, low molecular weight

heparin at a dosage of 4000 Ui per day and steroids at a dosage of 8 mg/day.

After 15 days the patient improved clinically but showed considerable polyarthicular and simmetric disease despite therapy with hydroxychloroquine and steroids, therapy used for treatment of pneumonia SARS-COV-2 related.

After one month from the hospitalization, the patient underwent a chest CT scan which showed no appreciable densitometric and infiltrative alterations of lung. Fibrotic striae from LIS.

In July 2020 the patient performed a rheumatological examination during which were prescribed: a magnetic resonance (RMN) of the hands and feet and blood tests including inflammation indices, rheumatoid factor and antibody against cyclic citrullinated peptide.

The results of the resonance showed a synovitis at the metacarpo-phalangeous joints and metatarsophalangeal joints bilaterally.

The patient began therapy with a low dosage of steroids and sulfasalazine at a dosage of 2 g/day.

Conclusions

Infection by coronaviruses is characterized by the activation of inflammatory activity in an irregular manner.

A majority of the studies demonstrating the effects of neutrophils and inflammatory monocytes-macrophages (IMMs) during lung coronavirus infection [4]. Following virus infection, infact, infiltrating neutrophils and monocyte-macrophages promote a pro-inflammatory status with high levels of IL-1B, ILRA and TNF-α mainly higher levels of IL-2, IL-10 and TNF-α are detected in intensive-care-unit patients [5].

Many AA. suggested that Covid-19 might damage lymphocytes and confirmed decrease of CD4+ T cells but no significant change in the number of CD8+ cells [2].

As we discussed here, several mechanisms can explain how coronavirus might activate the immune system.

In lung target cells might be located in the lower airway leading to activate inflammatory mediators from cells of immune system and subsequent tissue damage.

The chest CT scans show bilateral ground glasses opacities.

Early studies have shown that increased amounts of proinflammatory cytokines in serum (IL1B, IL6, IL12, IFNγ, IP10 and MCP1) were associated with pulmonary and extensive lung damage in SARS patients [4].

So our patient presented an active form of Rheumatoid Artrhtis that has been slatentized during infection SARS COV-2 and has determined a cascade of proinflammatory cytokines similar to those involved in RA pathogenesis.

This overexpression of cytokine's storm has resulted in disease severity.

During the hospitalization the patient took therapy with hydroxychloroquine and steroids without taking advantage of the joint ivolvement evidence of significant inflammatory effect after coronavirus infection.

Probably the infection caused a chain of events leading a cytokines cascade to have an active form of rheumatoid arthritis that did not respond to the initial treatment with steroids and antimalarial drugs.

Subsequently a more robust therapy by sulfasalazine was undertaken which took into account the previous infection and a good clinical response was made.

Of course, this is a case report and more studies can be conducted.

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