Mini Review

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Autoantibodies Specific for Rheumatoid Arthritis (RA) In Patients with Interstitial Lung Disease but No Clinically Apparent Articular RA

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Abstract

The goal of this study was to find rheumatoid arthritis-related autoantibodies in people who had interstitial lung disease but no particular findings to support the idea that RA-related autoimmunity may arise in non-articular locations like the lung. This was a review outline survey using center information bases of patients with ILD to recognize cases with lung sickness, RA-related autoantibody inspiration, and no clinical proof of articular RA. There were four patients who had ILD, RF, and anti-CCP positivity but no particular RA findings. The average age at diagnosis of ILD for all four patients was 70 years old, and they were all male. All of them had smoked in the past. Three patients were diagnosed with ILD and died within two years, but none of them ever developed articular symptoms consistent with RA; after ceasing immunosuppressive treatment for several months, the final case fulfilled all criteria for articular RA. Smokers can have RF and anti-CCP even if there is no clinical evidence of articular RA. In one case, symptomatic autoantibody positivity came before articular RA. These findings suggest that immunologic interactions in the lung may cause RA-specific autoimmunity and may be linked to environmental factors like smoking.

Keywords: Anti-cyclic citrullinated ● Antibodies ● Interstitial lung disease ● Rheumatoid arthritis ● Rheumatoid arthritis pathogenesis ● Rheumatoid factor (RF)

Introduction

Although the location of these initial immune deregulations is unknown, the presence of autoantibodies prior to articular manifestations of RA suggests that the initial immune deregulations in RA occur years before symptomatic disease onset [1]. However, the high prevalence of lung disease in early RA and the association of inhaled environmental agents like tobacco smoke and silica dust with the development of RA suggest that the lung may be the location of the initial immune deregulations associated with RA [2]. The goal of this study was to find people who had RA-related autoantibodies and symptomatic lung disease but had no clinical evidence of articular RA. This would back up the idea that RA-specific autoimmunity could be made in the lung even if there was no particular disease [3].

Materials and Methods

Between January and December, patients with a clinical, radiographic, or histologic diagnosis of ILD were identified using clinic databases from National Jewish Health (NJH) and clinics affiliated with the University Of Colorado Denver School Of Medicine [4]. As RF was routinely performed as part of an ILD evaluation in these subjects, the initial inclusion criteria for this study were a diagnosis of ILD and RF positivity. Anti-CCP testing was performed if RF was positive. Patients who had an established diagnosis of RA simultaneously or prior to the onset of pulmonary symptoms were excluded after these inclusion criteria were applied. According to the Revised ACR Criteria for RA diagnosis or symptoms suggestive of lupus, scleroderma, Jorgen's syndrome, or a known aetiology for lung disease patients were also excluded if chart review identified any of the following positivity for any particular, nodule, or radiographic criteria for RA: and a diagnosis

of mycobacterial infection because RF and/or anti-CCP positivity have been linked to active tuberculosis [5]. The specific type of ILD, age at diagnosis, sex of ILD, smoking status, silica dust exposure, pulmonary radiographic findings, lung pathology, and treatment and outcome of lung disease were among the additional data gathered.

Concentrate on constraints

Additionally, we were unable to link the presence or levels of RA-related antibodies to specific types of ILD or prognosis [6]. Additionally, these individuals may have had mild articular RA at the time of their ILD diagnosis, which was either missed during evaluation or suppressed by their lung disease treatments. Last but not least, it's possible that the three cases of RA-related autoantibodies that did not progress to articular RA are simply non-specific responses to chronic lung immune deregulations [7].

Discussion

There is a period of asymptomatic autoimmunity, as shown by circulating autoantibodies, following the eventual onset of clinically apparent RA once tolerance to self-antigens has been broken. This model is supported by the known pre-arthritis positivity for RF and anti-CCP in subjects who eventually develop articular RA and the known genetic and environmental factors that are associated with an increased risk of RA [8]. However, the location of the initial genetic and environmental interactions that may lead to RA is unknown. The association of HLA alleles containing the shared epitope, smoking, and the development of anti-CCP positive RA, the known high prevalence of lung disease even in early RA, and the association of RF and anti-CCP positivity with extra-articular manifestations of RA, including lung disease, support the hypothesis that the lung is the site of initial immune deregulations in RA [9]. Four smokers have ILD, RF, and anti-CCP positivity, but no particular RA findings, with one developing articular RA after presenting with lung disease, supporting this hypothesis. The finding that RF positivity and symptomatic lung disease precede clinically apparent articular RA is not new. However, mechanistic arguments for the initiation of RA-related immunity in the lung on the basis of RF positivity alone are difficult to support due to the lack of RF specificity for RA. Conversely, the high explicitness of hostile to CCP antibodies for RA particularly with associative RF energy, recommends that enemy of CCP and RF energy in patients with ILD and no clinically clear articular RA is connected with RA-explicit immunologic deregulations [10].

Conclusions

Smokers with ILD but no evidence of articular RA can have autoantibodies specific to RA. The hypothesis that RA-specific autoimmunity can be caused by environmental factors like smoking in the absence of articular disease is supported by these findings. To learn more about the connection between lung disease and the development of RA-related autoimmunity, additional research is required. To further investigate the connection between RA-related autoantibody generation and lung inflammation and citrullination, bronchoscopy with lavage or lung biopsy in subjects with lung disease and RA-related antibodies but no particular findings of RA or similar studies in subjects with early articular RA are necessary.

References

1. Nielen MM. Schaardenburg DV, Reesink HW *et al*. Specific autoantibodies precede the symptoms of rheumatoid arthritis: a study of serial measurements in blood donors. *Arthritis Rheum*.50, 380–386 (2004).
2. Metafratzi ZM. Pulmonary involvement in patients with early rheumatoid arthritis. *Scand J Rheumatol*. 36, 338–444 (2007).
3. Gabbay E. Interstitial lung disease in recent onset rheumatoid arthritis. *Am J Respir Crit Care Med*.156, 528–535 (1997).
4. Oliver JE, Silman AJ. Risk factors for the development of rheumatoid arthritis. *Scand J Rheumatol*. 2006; 35(3):169–174.
5. Arnett FC. The American Rheumatism Association 1987 revised criteria for the classification of rheumatoid arthritis. *Arthritis Rheum*. 31, 315–324 (1988).
6. Elkayam O. Auto-antibody profiles in patients with active pulmonary tuberculosis. *Int J Tuberc Lung Dis*.
7. 11, 306–310 (2007).
8. Bowes J, Barton A. Recent advances in the genetics of RA susceptibility. *Rheumatology*. 47, 399–402 (2008).
9. Klareskog L. A new model for an ethology of rheumatoid arthritis: smoking may trigger HLA-DR (shared epitope)-restricted immune reactions to auto antigens modified by citrullination. *Arthritis Rheum*.54, 38–46 (2006).
10. Alexiou I. Anti-cyclic citrullinated peptide-2 (CCP2) autoantibodies and extra-articular manifestations in Greek patients with rheumatoid arthritis. *Clin Rheumatol*.27, 511–513 (2008).
11. Gottlieb AJ. Serologic factors in idiopathic diffuse interstitial pulmonary fibrosis. *Am J Med*.39, 405–410 (1965).