Know Case of Hyperthyroidism with Newly Diagnosed Adult Onset Stills Disease

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Abstract

To our knowledge, the possible unveiled interaction between adult-onset Still’s disease (AOSD) with autoimmune thyroid disease (AITD) has never been reported although it is well established that systemic autoimmune disease may usually occur in relation to AITD. As increasingly clear links of AITD with other autoimmune disease such as systemic lupus erythematosus (SLE), rheumatoid arthritis (RA), and primary Sjögren’s syndrome (pSS) have been reported, and the incidence of AOSD concurrent AITD draws our attention rapidly. In this study, we searched relevant literatures published in the past 30 years to explore that condition.

Keywords: Adult-onset Still’s disease, thyroid disease.

Introduction

Adult-onset Still’s disease (AOSD) is a rare systemic autoimmune syndrome of unclear etiology. In 1971, fourteen adults with arthritis and systemic symptoms in accordance with the features of juvenile rheumatoid arthritis or Still’s disease were described by Bywaters. From then on, AOSD was regarded as a different disorder gradually by us [1], which was characterized by spiking fever, pruritic or nonpruritic evanescent rash, arthritis, pharyngitis, leucocytosis and lymphadenopathy, and less frequently, hepatitis and polyserositis [2]. There is no single diagnostic test for AOSD and it is mostly based on clinical and laboratory criteria with the exclusion of other autoimmune diseases, neoplasms and infection. With several diagnostic criteria developed over the years, Yamaguchi’s criteria has been the most widely used as a result of higher sensitivity (96.2%) and specificity (92.1%) [3]. Thyroid disease of autoimmune origin is a frequent condition affecting 1% to 5% of general population, especially mostly seen in women of their third to fifth decades. Autoimmune thyroid disease (AITD), usually includes Graves’ disease (GD) and Hashimoto’s thyroiditis (HT), which are the most common reasons for thyroid disorders. An interplay among some conditions such as immune mechanism, environmental (for example, infection, iodine and stress), genetic and constitutional factors contributes to the mechanism of AITD. GD, the most common cause of thyrotoxicosis, is more prone to a family history with thyroid disease, especially with GD. GD can be incited by the combination of autoantibodies with TSH receptor which is activated then to stimulate thyrocyte growth and function.

Case

A 23-year-old male patient without evident disease previously presented to our hospital with a 2
years of intermittent wandering arthralgia on the left side, and also with spiking fever (the highest body temperature up to 103°F), thoracalgia, sputum rashes over chest and upper back for 3 months. According to the examinations of the local hospital, X ray of the chest showed left sided pleural effusion leading to oppressive lung parenchyma; mild inflammatory infiltration of both lungs; and swelling lymph nodes on the right cardiophistic angle. Distinctive laboratory findings, easily played a role in establishing the following diagnoses: chronic symmetrical inflammatory polyarthritis with appendicular involvement and no axial involvement with systemic manifestation of fever, anemia, hepatomegaly, lymphadenopathy and hyperthyroidism. On physical examination, he appeared irritable, general fatigue and underweight. And palpable lymph nodes at bilateral inguinal region and supraclavicular fossa were also found. According to the laboratorical results of the local and our hospital, hyperthyroidism was diagnosed. An abdominal ultrasound revealed normal study. Thyroid ultrasonography revealed heterogeneous in echoic distribution and rich blood perfusion.. According to the laboratory results, imaging studies, necessary exclusion and Yamaguchi et al. criteria, this case fulfilled the 1, 2 and 4 items of major criteria and 2 and 4 items of minor criteria. AOSD was therefore diagnosed.

Discussion

As far as we know, the association between thyroid disorders and AOSD has never been reported. Nonetheless, the association of AITD with rheumatoid arthritis, systemic lupus erythematosus, Sjögren’s syndrome and Myasthenia gravis has usually been reported [4-6]. And the systemic autoimmune disease related to AITD has been widely known. Moreover, AOSD is considered as a variant of rheumatoid arthritis, so the similar condition of thyroid disorders with AOSD may exist. In our study, coincidental link could not be ruled out.

In this case, the examination outside our hospital showed hyperthyroidism and anti-thyroid drugs were prescribed, which led to the normal FT3, FT4, TT3 and TT4 level and decreased TSH in our hospital. Therefore, the anti-thyroid drugs were discontinued. But the spiking thyroid hormone changes occurred during hospitalization. Two explanation might be correlated to this relapse. Firstly, methimazole was stopped and the recurrence happened; secondly, AOSD and thyroid gland might interact with each other. An early report by Chen et al. was similar to our case. In Chen’s case, elevated thyroid hormone concentrations and a palpable goiter had not been observed prior to admission. Although hydroxychloroquine was prescribed, Graves’ disease appeared on the ten-day hospitalization and during the course of the disease, the hyperthyroidism had been aggravated whenever AOSD was in active stage.

So far, only a few reports have discussed the coexistence of AOSD and thyroid disorders. Literature research revealed five relevant reports (one in Chinese, one in French and one in Japanese, and two in English). Among them, two studies disclosed AOSD and thyroid disorders were diagnosed simultaneously; two indicated AOSD which was found before thyroid dysfunctions; one did not provide more information. In addition, only two reports provided the definite
types of ITD but the remaining three studies had no information of detailed type of AITD and we also failed to obtain the type from the relevant antibodies level. As a result, from the three reports we only diagnosed hyperthyroidism or hypothyroidism. At present, we only believe the two diseases can interact with each other by their shared pathogenesis; however, the clear link between them fails to demonstrate the coexistence of AOSD and ITD is rarely published. With an eye to the limitation of this study, it would be better that when physicians diagnose their patients as one of the two, screen the another. Then we will have more chances to explore the correlation further and with the implementation of large-scale trials, it will be greatly advanced our understanding of the interplay and therapy.

**Conclusions**

In conclusion, coexistence of AOSD and ITD is so rarely published that the explicit relationship is hard to acquire, but we can believe they could interact with each other through their common pathogenesis. As more cases are reported, the hidden association will become clear. Polyserositis is very scarce in the manifestation of AOSD. When, we, physicians encounter this sign, it becomes necessary to screen the relevant laboratory and imaging abnormalities. Circumstances permitting, a thyroid gland screening is recommended, especially in female patients. Finally, it is reasonable to reconsider the various manifestations of AOSD.

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**References**