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Editorial

Autoimmune Thyroiditis in Rheumatology Practice: Should we be Looking?

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EDITORIAL

Chronic lymphocytic thyroiditis (CLT), a variant of autoimmune thyroiditis (AIT) is the commonest autoimmune disease with a prevalence of about 10% worldwide, rising to about 20% after age 50 years with a female to male preponderance of about 9:1 [1,2]. Although it is generally asymptomatic, severe disease can lead to failure of the thyroid gland and hypothyroidism in about 20% of cases [2]. Less commonly known is the fact that CLT and its goitrous form, Hashimoto thyroiditis, are associated with a multitude of other clinical syndromes including nervous system, psychological, dermatologic, metabolic, endocrine and musculoskeletal conditions [3]. A variety of causative mechanisms are probably involved including metabolic, endocrine, autoimmune and perhaps inflammatory processes. The association of CLT with other autoimmune conditions including connective tissue diseases is also likely responsible for the tremendous diversity of clinical presentations [4].

Despite the mounting evidence in the literature of rheumatic diseases associated with CLT, the rheumatology community remains largely unaware of the importance of thyroid autoimmunity. The purely rheumatic disease associations reported include chronic widespread pain syndromes, fibromyalgia syndrome, osteoarthritis and erosive osteoarthritis, inflammatory arthritis resembling rheumatoid arthritis, calcium pyrophosphate dihydrate deposition disease (CPPD), myositis, spinal degenerative disc disease, carpal tunnel syndrome, Raynaud's phenomenon, sicca symptoms, prolonged morning stiffness, chronic fatigue syndrome, undifferentiated connective tissue disease and several well-defined connective tissue diseases [5]. The high prevalence of CLT and the high proportion of CLT sufferers reported as having rheumatic disease associations suggest that the numbers involved reach epidemic proportions. However there are no well-designed population-based studies examining the true prevalence of these CLT-related clinical syndromes. Part of the problem is the very fact of unusually high prevalence of thyroid autoimmunity. Thus the "background" against which statistical significance must be shown is extraordinarily high. Indeed because of that high threshold only well-designed population-based data could yield unequivocal results of disease causation. Coupled with that is the fact that autoimmunity seldom occurs as an isolated phenomenon making it difficult to separate the true causes of the rheumatic associations. Current knowledge suggests that up to 50% of adult CLT patients have another well-defined autoimmune process [4]. Of course the spectrum of autoimmunity is so broad and comprises several less clinically apparent conditions which can go undiagnosed for prolonged periods, that the co-occurrence of CLT with other autoimmune processes is probably higher still. These less commonly diagnosed autoimmune processes include autoimmune cytopenias, celiac disease, pernicious anemia, myasthenia gravis and autoimmune neuropathies. Despite these concerns regarding the difficulties with proving causation there is growing and overwhelming evidence of the association of CLT with several rheumatic phenomena.

Therefore the clinical relevance of finding CLT in patients or their families is that it strengthens our suspicion for an autoimmune etiology underlying their disease process. It also provides an assessment of risk for several rheumatic conditions associated with CLT including chronic widespread pain and osteoarthritis. In some situations it probably provides the only identifiable risk factor for that disease process as in the case of osteoarthritis or chronic widespread pain in young patients for whom no other reasonable explanations exist. Placing CLT squarely within the pantheon of autoimmune diseases, which preoccupy rheumatologists in particular and other healthcare providers in general, would allow better definition of the problems associated with it, a more scientific understanding of the pathogenic mechanisms involved and more rational therapies for patients with these conditions. It would also lay the ground work for better classification of the associated syndromes and encourage basic research into how thyroid autoimmunity mediates the panoply of findings including arthritis, chronic widespread pain and neuropathy. The high accessibility of the thyroid gland relative to other organs should facilitate any basic research efforts into its role in autoimmunity. Several questions need to be addressed including whether or not most of the conditions mentioned before are part of a more generalized autoimmune process in CLT or are caused by separate mechanisms of injury. We still do not know why the thyroid gland is caught up in the autoimmunity associated with rheumatoid arthritis or systemic lupus erythematosus in up to 25% of cases [4]. Most baffling is the fact that some patients

with CLT appear to have isolated thyroid autoimmunity but display several systemic correlates of connective tissue diseases including arthritis, Raynaud's phenomenon, morning stiffness, fibromyalgia, elevated serum acute phase reactants and chronic fatigue [6]. Indeed it might not be premature to speculate that autoimmunity is perhaps more fluid and promiscuous in terms of its end organ involvement than fixed and that understanding the role of the thyroid in the evolution of the autoimmune process could be a crucial part of understanding autoimmunity as a whole. Thyroid autoimmunity should be investigated and the assessment for thyroid autoantibodies performed in all patients with unclear diagnoses in whom autoimmunity is thought to underlie their disease pathology. This would be particularly helpful when patients exhibit symptoms of chronic widespread pain, spinal degenerative disc disease, excessive fatigue and early-onset osteoarthritic changes. The presence of such findings in those with well-defined connective tissue diseases should also trigger a search for CLT since its presence could influence our understanding of the overall prognosis. A significant proportion of patients currently misdiagnosed or inadequately diagnosed could receive better explanations of the factors generating their symptoms and signs. Thus, previous diagnoses of osteoarthritis in young patients or primary fibromyalgia, would be reclassified to include a clearer understanding of the risk factors and underlying driving forces if defined in the context of their CLT. This shift from a normative to a descriptive mode of diagnosis would strengthen disease classification and nosology in rheumatology. Although effective therapies to mitigate the autoimmune process in the thyroid gland do not exist at this time, increased awareness of the disease associations of CLT would channel our healing energies into providing better comprehensive care for affected patients who until now have been fighting a hidden enemy in urgent need of exposure and better definition.

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