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PROGNOSTIC IMPLICATIONS OF AUTOIMMUNE THYROIDITIS IN THE CLINICAL COURSE OF RHEUMATOID ARTHRITIS

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✓ Resume

The literature review examines various aspects of the combination of rheumatoid arthritis (RA) with autoimmune thyroid diseases (Hashimoto's thyroiditis and Graves' disease). The prevalence, genetic predisposition, similarity of pathogenesis and clinical symptoms, as well as the ability of organ-specific autoantibodies (antibodies to thyroid peroxidase, antibodies to thyroglobulin) and hypothyroidism to influence the course of RA and the risk of cardiovascular complications are discussed.

Keywords: rheumatoid arthritis; autoimmune diseases of the thyroid gland; Hashimoto's thyroiditis; Graves' disease; antibodies to thyroid peroxidase; antibodies to thyroglobulin; hypothyroidism; cardio-vascular diseases.

ПРОГНОСТИЧЕСКОЕ ЗНАЧЕНИЕ АУТОИММУННОГО ТИРЕОИДИТА В КЛИНИЧЕСКОМ ТЕЧЕНИИ РЕВМАТОИДНОГО АРТРИТА

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✓ Резюме

В обзоре литературы рассматриваются различные аспекты сочетания ревматоидного артрита (PA) с аутоиммунными заболеваниями щитовидной железы (тиреоидит Хашимото и болезнь Грейвса). Обсуждаются распространенность, генетическая предрасположенность, сходство патогенеза и клинических симптомов, а также способность органоспецифических аутоантител (антитела к пероксидазе щитовидной железы, антитела к тиреоглобулину) и гипотиреоза влиять на течение PA и риск сердечно-сосудистых осложнений.

Ключевые слова: ревматоидный артрит; аутоиммунные заболевания щитовидной железы; тиреоидит Хашимото; болезнь Грейвса; антитела к тиреоидной пероксидазе; антитела к тиреоглобулину; гипотиреоз; сердечно-сосудистые заболевания.

REVMATOID ARTRITNING KECHISHIDA AUTOIMMUN TIROIDITNING PROGNOSTIK TA'SIRI

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Adabiyot sharhida revmatoid artrit (RA) ning qalqonsimon bezning avtomatik immun kasalliklari (Hashimoto tiroiditi va Graves kasalligi) bilan kombinatsiyasining turli jihatlari ko'rib chiqilgan. Tarqalishi, genetik moyilligi, patogenezi va klinik belgilarining o'xshashligi, shuningdek, organlarga xos autoantitelalarning (qalqonsimon peroksidazaga autoantitelalar, tiroglobulinga autoantitelalar) va gipotiroidizmning RA kechishiga ta'sir qilish qobiliyati va yurak-qon tomir asoratlari xayfi muhokama qilinadi.

Kalit so'zlar: Revmatoid artrit; qalqonsimon bezning autoimmun kasalliklari; Hashimoto tiroiditi; Graves kasalligi; qalqonsimon peroksidazaga autoantitelalar; tiroglobulinga autoantitelalar; gipotiroidizm; yurak-qon tomir kasalliklari.



Relevance

The co-occurrence of rheumatoid arthritis (RA) and autoimmune thyroiditis (AIT) is a fascinating clinical entity attracting substantial research interest. RA, characterized by chronic joint inflammation and autoantibody production, affects roughly 1% of the population. AIT, encompassing Hashimoto's thyroiditis and Graves' disease, also involves autoimmunity targeting the thyroid gland, with a much higher prevalence of 5-10%. The association between these two seemingly distinct autoimmune diseases raises intriguing questions about shared mechanisms, clinical implications, and potential therapeutic overlap (1).

Rheumatoid arthritis (RA), a chronic autoimmune disease characterized by inflammatory synovitis and joint destruction, affects roughly 1% of the population. While often considered an isolated condition, RA frequently co-occurs with other autoimmune diseases, particularly autoimmune thyroiditis (AIT) (4). AIT, encompassing Hashimoto's thyroiditis and Graves' disease, affects around 5-10% of the general population, and its association with RA has sparked significant research interest (3).

This introduction delves into the intricate relationship between RA and AIT, focusing on the potential prognostic implications of AIT in the clinical course of RA. We will explore: The prevalence and clinical characteristics of RA-AIT: How often does AIT occur in RA patients; Does it influence the presentation, severity, or extra-articular manifestations of RA; Possible mechanisms behind the RA-AIT link: What shared genetic, environmental, or immunological factors might contribute to the co-occurrence of these diseasese; Clinical implications of AIT for RA prognosis: Does AIT influence disease progression, response to treatment, or risk of complications in RA patients; Current research challenges and future directions: What limitations exist in our understanding of the RA-AIT relationship, and what research avenues hold promise for advancing our knowledge (10).

Throughout this exploration, we will critically analyze existing literature, highlighting key findings and potential controversies. Ultimately, we aim to provide a comprehensive understanding of how AIT shapes the clinical course of RA and identify critical gaps in knowledge that future research should address (2).

Additionally, consider further enriching the introduction by:

- Briefly mentioning the potential economic and social burden associated with RA-AIT compared to RA alone.
- Highlighting the importance of early diagnosis and monitoring of both RA and AIT in patients with this comorbidity.
- Underscoring the ongoing efforts to develop personalized treatment strategies that consider the presence of AIT in RA patients.

By crafting a thorough and engaging introduction, you can set the stage for a compelling investigation into the prognostic implications of AIT in RA and its potential impact on clinical practice and research.

Prevalence: Studies report a significantly higher prevalence of AIT in RA patients compared to the general population, ranging from 10% to 30%. This suggests a strong association exceeding mere chance (1-4).

Clinical Differences: Compared to RA alone, RA-AIT patients might exhibit:

Less severe joint damage: Some studies suggest slower radiographic progression of joint destruction in RA-AIT, possibly due to lower pro-inflammatory cytokines;

higher prevalence of extra-articular manifestations: Sjögren's syndrome, interstitial lung disease, and vasculitis seem more common in RA-AIT, indicating broader autoimmune dysregulation; increased risk of secondary amyloidosis: This rare complication of chronic inflammation appears more frequent in RA-AIT, potentially due to combined inflammatory burden (6).

The exact cause of the RA-AIT link remains elusive, but several possible explanations have been explored: Genetic susceptibility: Genome-wide association studies have identified several shared genetic loci between RA and AIT, suggesting common genetic vulnerability for both diseases. Molecular mimicry: Similarities between certain thyroid and joint antigens could lead to immune cross-reactivity, with antibodies targeting both tissues. Environmental triggers: Viral infections, smoking, and psychological stress have been implicated in the onset of both RA and AIT, suggesting they might act as common environmental triggers (11).

Intestine microbiome dysbiosis: Alterations in the gut microbiome composition have been linked to both RA and AIT, hinting at a potential role in immune system activation.

Early diagnosis is crucial: Routine screening for thyroid function in RA patients and vice versa is recommended due to the potential for subclinical involvement. Disease-specific treatment remains the mainstay: Conventional therapies like methotrexate for RA and levothyroxine for AIT are employed.

Emerging therapeutic options: Some studies suggest immunomodulatory therapies like abatacept or rituximab might benefit both RA and AIT components, but further research is needed (6-8).

Limitations and Future Directions: Current research on RA-AIT is mostly observational, limiting causal inferences and definitive conclusions. Larger, prospective studies with standardized methodologies are needed to confirm the reported clinical features and clarify the underlying mechanisms. Investigating shared therapeutic targets in RA and AIT could pave the way for personalized treatment strategies tailored to individual patients. Unraveling the gut microbiome's role in both diseases might yield novel therapeutic insights for managing both RA and AIT (9).

Conclusion

The co-occurrence of RA and AIT presents a complex clinical picture with intriguing associations beyond chance. Understanding the shared mechanisms, clinical implications, and potential therapeutic avenues for this disease intersection holds significant promise for improving patient outcomes and developing personalized management strategies. Continued research in this field is crucial to unlock the secrets of this fascinating medical puzzle.

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