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A national survey of ankylosing spondylitis in Iran

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Objective: Ankylosing spondylitis (AS) is a chronic systemic inflammatory disease with variable clinical expression. Ethnic, racial, geographical and gender-related factors have been associated with disease incidence and clinical manifestations. The study intended to describe clinical characteristics and assess disease severity, gender influences, and treatment status in the Iranian population.

Subjects and methods: 320 patients diagnosed with primary AS throughout Iran were evaluated for baseline characteristics, clinical manifestations, HLA-B27 status, disease severity, functional indices, quality of life, and treatment status.

Results: A gender ratio of 3.8:1, average age onset of 27±7.3, and a mean diagnostic delay of 8 years was observed. The incidence of juvenile-onset cases was 11%. Positive family history was higher than that observed in most other countries. Both gender groups were similar in terms of ethnicity, positive family history, juvenile-onset AS, and diagnostic delay. Enthesitis was present in more than two-thirds of patients with females demonstrating a higher rate ($p<0.05$). Uveitis was the leading extra-articular manifestation. Overall HLA-B27 positivity was 73% and four HLA-B27 subtypes were found. HLA-B27 positivity was higher among males (78.3% vs. 55.2%; $p<0.001$). Disease activity was high and the functional status was poor as indicated by mean Bath AS disease activity, functional, and metrology indices. Female disease was at least as severe as male disease and more severe impairment was present in some aspects. Extra-articular manifestations and treatments modalities presented similar frequencies among genders. Biological medication was utilized less frequently than disease modifying anti-rheumatic drugs and corticosteroids. Quality of life was considerably impaired.

Conclusion: The study exhibits a broad characterization of Iranian AS patients providing better understanding of the disease status and allowing for healthcare development and advancement of earlier diagnostic and treatment strategies. Early detection and specialized care would be of great practical importance.

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Silencing of microRNA-21 confers silica-induced cytotoxicity through inhibition of the PI3K/AKT pathway and enhancing autophagy in RAW264.7 cells

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Silica is a core part of the cause of Coal Workers' Pneumoconiosis (CWP), which is a chronic occupational lung disease characterized by irreversible pulmonary fibrosis. However, the underlying immunologic mechanisms of CWP are poorly understood. Macrophages constitute the first line of cellular defense against pathogens. Autophagy is a fundamental cellular homeostasis pathway that operates with the intracellular degradation/recycling system. In response to invading pathogens, induction of the autophagic process in macrophages constitutes a crucial mechanism in innate immunity. Here, it is demonstrated that microRNA 21 (miR-21), an elevated miRNA in silica-induced pulmonary fibrosis, is one of the main players in silica- cytotoxicity. Silica-cytotoxicity in RAW264.7 cells measured by cytotoxic cell survival assays was closely associated with the expression of miR-21. P62, indicator for autophagic degradation, increased in a dose/time-dependent way in cultured RAW264.7 cells due to silica exposure. Blocking miR-21 with anti-miR-21 led to a decreased expression of p62 in 200 μg/ml silica-treated RAW264.7 cells for 12 hours and the suppression of phosphor-Akt (ser473) was as well observed. In a cell cycle analysis, a significant increased arrest in the G1/G0 phase by anti-miR-21 was found at 48 hours after silica-treatment. Notably, obtained results demonstrated that anti-miR-21 raised factors involved in autophagosome formation. Moreover, augmented autophagy by anti-miR-21 resulted in an increase in the apoptotic population after silica exposure. The findings show that miR-21 is a crucial molecule for alleviating silica-induced cell death in RAW264.7 cells by inhibiting the PI3K/AKT pathway and enhancing autophagy, providing a new insight into the mechanisms of CWP.

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