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Nonspecific Interstitial Pneumonia Development after Fire Extinguisher Dust Inhalation

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Assessment of Treatment Response using the Multi-Biomarker Disease Activity Score in Rheumatoid Arthritis Patients Initiating Methotrexate

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Program: Internal Medicine

Background/Purpose: The Multi-Biomarker Disease Activity (MBDA) score was developed to provide an objective measure of rheumatoid arthritis (RA) disease activity. The ability of the MBDA to predict treatment response may vary among disease-modifying agents, and has not been evaluated in patients initiating methotrexate (MTX), a cornerstone therapy in RA.

Methods: We conducted a secondary analysis of an open-label study of RA patients initiating MTX. The association of MBDA scores at baseline (N=130) and week 16 (N=95) with treatment response was assessed using multivariable regression models. Convergent validity and responsiveness were determined by calculating correlations of the MBDA with disease activity measures and through the calculation of standardized response means (SRM).

Results: Patients achieving an ACR50 response demonstrated greater reductions in MBDA than those failing to achieve an ACR50 response (p=0.01). However, baseline MBDA scores (OR 1.01, 95% CI 0.99, 1.04) and categories (moderate: OR 1.55, 95% CI 0.45-4.20; high: OR 2.55, 95% CI 0.50-14.20) were not predictive of response. Higher baseline MBDA scores were associated with greater improvement in DAS28-ESR (p=0.01), as were baseline DAS28-ESR values (p<0.001). The MBDA demonstrated moderate correlations with DAS28-ESR and ESR, but weaker correlations with the HAQ and PtGA. Treatment responsiveness was greater for DAS28-ESR (SRM -1.32) than MBDA (SRM -0.65).

Conclusions: Baseline MBDA was associated with greater reductions in DAS28-ESR but was not a robust predictor of ACR50 response. Although demonstrating moderate convergent validity with standard disease activity assessments, the MBDA yielded lower responsiveness than the DAS28-ESR in the context of MTX treatment.

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Introduction: Nonspecific interstitial pneumonia (NSIP) is a form of chronic inflammatory idiopathic interstitial pneumonia. It typically presents with months of dyspnea. NSIP is frequently associated with connective tissue disease, HIV, and drug-induced lung disease.

Case Presentation: A middle-aged patient, non-smoker with hypertension and OSA on CPAP presented with six months of exertional dyspnea and right-sided chest pain. The patient developed a new exertional 10 LPM supplemental oxygen requirement after significant smoke and fire extinguisher dust inhalation. The patient denied a family history of lung disease or connective tissue disease, had faint inspiratory crackles, and no leg edema.

ANA, RF, ANCA, IgE, CK, aldolase, anti-CCP, HIV, and hypersensitivity pneumonitis panel were unremarkable. Chest CT revealed diffuse ground glass sparing the costophrenic angles without traction bronchiectasis and honeycombing. Pulmonary function testing revealed FEV1/FVC of 82%, TLC was 56% predicted, and DLCO was 63% predicted. EF was normal and pulmonary artery systolic pressure was 45-50mmHg. Right lower lobe wedge biopsy demonstrated alveolar wall thickening with focal lymphocytic inflammation in a fairly uniform distribution without honeycombing, consistent with NSIP. The patient underwent pulmonary rehabilitation and was started on Prednisone and transitioned to Azathioprine resulting in improvement of his dyspnea.

Discussion: Diagnosis of NSIP is dependent on multidisciplinary interactions among pulmonology, radiology, and pathology to differentiate it from other interstitial lung diseases and guide treatment. The patient developed hypoxic dyspnea within months of fire extinguisher dust exposure and lacked other risk factors for development of NSIP. Fire extinguisher exposure is typically associated with desquamative interstitial pneumonia, and per our literature review, there are no reported cases of fire extinguisher exposure related to NSIP, making this case unique.

Conclusions: It is probable that exposure to smoke and fire extinguisher dust inhalation triggered the development of NSIP.

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