Understanding NTM-PD as a Host Disease

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The incidence of nontuberculous mycobacterial pulmonary disease (NTM-PD) continues to rise worldwide, with especially rapid growth in Asia. Although environmental exposure to NTM is nearly universal, only a subset of individuals develop progressive disease. This observation highlights the central role of host factors in determining susceptibility.

Distinct clinical patterns provide important clues. Fibrocavitary disease is most often observed in older men with smoking history or prior tuberculosis, whereas the nodular bronchiectatic form is typically seen in post-menopausal women who lack overt immunodeficiency but often display subtle morphologic traits such as scoliosis or pectus excavatum. Impaired mucociliary clearance, underlying bronchiectasis, and CFTR variants contribute further to vulnerability.

Immunologic pathways are also critical. The IL-12 and IFN-γ axis is essential for antimycobacterial defense. Rare genetic defects in this pathway and acquired conditions such as adult-onset immunodeficiency due to anti-IFN-γ autoantibodies, which are relatively frequent in Asia, predispose to severe or disseminated disease. Other immune mechanisms, including Th17 responses, neutrophil activity, and autophagy, influence both protection and tissue injury.

Acquired and treatment-related risk factors add further complexity. Inhaled corticosteroid therapy, especially at higher doses, has been consistently linked with increased risk of NTM disease in patients with asthma or COPD. Comorbid airway disorders further compromise host defenses and promote chronic infection.

Recognizing NTM-PD as a host-driven disease carries important clinical implications. It encourages identification of susceptible phenotypes, refinement of screening and monitoring strategies, and exploration of host-directed therapies. Early experience with inhaled GM-CSF, immunomodulatory interventions, and strategies targeting airway clearance suggests future opportunities to complement antimycobacterial regimens.

Ultimately, the rising burden of NTM in Asia reflects not only abundant environmental mycobacteria but also host susceptibility. A deeper understanding of these host determinants is essential to advance prevention, diagnosis, and treatment.