NSAID-exacerbated respiratory disease, dupilumab and aspirin tolerance

Donald W. Cockcroft

Respiratory Medicine, Division of Respirology, Critical Care and Sleep Medicine, Department of Medicine, University of Saskatchewan, Saskatoon, SK, Canada.

Corresponding author: Donald W. Cockcroft (don.cockcroft@usask.ca)

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Schneider *et al*. add to the growing evidence that dupilumab is very effective in N-ERD inducing increased tolerance to ASA in 57% of patients (complete in 23%), accompanied by improvements in both upper and lower airway clinical features of disease https://bit.ly/3jqs1DC

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Nonsteroidal anti-inflammatory drug (NSAID)-exacerbated respiratory disease (N-ERD), an intriguing and incompletely understood condition, is characterised by a triad of asthma, chronic rhinosinusitis with nasal polyps (CRSwNP) and hypersensitivity to aspirin (acetylsalicylic acid; ASA) and other cyclooxygenase-1 inhibiting NSAIDs [1, 2]. Dysregulation of arachidonic acid metabolism leads to chronic overproduction of the cysteinyl leukotrienes and the inflammatory prostaglandin (PG) D2 along with underproduction of anti-inflammatory PGE2; these are further increased acutely on exposure to NSAIDs [1, 2]. Intense T2 eosinophilic inflammation, involving both CRSwNP and asthma, precede the onset of clinical NSAID sensitivity and persist in the absence of NSAID exposure [1]. N-ERD is also frequently accompanied by alcohol intolerance [3]. N-ERD prevalence is estimated to approach 1% in the general population and 7% in adults with asthma, a prevalence that increases in more severe asthma, particularly when accompanied by CRSwNP [4]. While more than 50% of N-ERD patients are atopic [5], IgE does not appear to be involved in the usual antigen-specific fashion in the pathogenesis of responses to structurally different NSAIDs [1, 2].