Inflammatory phenotypes of severe asthma in India

Sir,

Severe asthma is a heterogeneous disease that warrants accurate phenotyping for optimal disease control since standard guideline-based treatment may not be effective in this subgroup of asthmatics.\[1,2\] The purpose of phenotyping is to provide an insight into the mechanism (high Th2 vs. low Th2) driving the disease and direct treatment accordingly. Among others, sputum quantitative assay is a well-established method for measuring the cellular nature of airway inflammation and even for titrating doses of corticosteroids.\[3\] Although this method has been variously criticized for its need for a wet laboratory, adequate training, and patients not being able to produce sputum despite induction, this remains the most specific and accurate method for phenotyping airway diseases so far.\[3\] Further, this is the only noninvasive method which measures the inflammatory cells directly from the airways whereas most other methods (blood eosinophils and fraction of exhaled nitric oxide) do not.

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We used sputum quantitative assay (Hamilton protocol) along with other clinical and blood parameters to phenotype 100 consecutive severe asthma patients who attended the severe asthma clinic of a referral hospital in eastern India.[2,4] The mean age of the cohort was 50.93 (standard deviation [SD]: 15.46) years. There were 57% males. The mean duration of disease was 15.18 (SD: 10.62) years. A history of allergy was present in 62%, family history of asthma was present in 32%, and 16% of the participants were smokers. There were associated sinus disease and a history of pneumonia in 47% and 32% patients, respectively. The mean prebronchodilator forced expiratory volume in 1 s was 1.06 L (47% predicted). Sixty-four percent of the participants were atopic with a raised serum immunoglobulin E.

The most common inflammatory phenotype in our cohort (sputum report available in 72 patients) was neutrophilic (59%) which is only modestly higher than earlier reports.[1] Among them, 13% had raised total cell count indicating bacterial infection and need for antibiotic therapy. The remaining had isolated neutrophilia (46%). This latter group is known to respond poorly to corticosteroids and thus needs further research for identifying possible drivers of neutrophilia.[5]

The eosinophil phenotype comprised 26% of participants while mixed pattern and paucigranulocytic sputum were present in 11% and 3%, respectively. The eosinophilic phenotype was present in 64% of participants when a blood eosinophil count of >300 cells/cu mm was used as the cutoff. Thus, a discordance between blood eosinophilia and sputum eosinophilia [Figure 1] existed which indicates that mere finding of eosinophils in blood may not be reflective of the presence of activated eosinophils in the airways. Further, India has a greater prevalence of parasitic infections than most other countries which might necessitate using a greater cutoff for blood eosinophils. This, given the recent availability of specific anti-eosinophil agents, needs urgent attention.

While being limited by a cross-sectional design, this is the first report of the inflammatory phenotypes of severe asthma from the Indian subcontinent. The various phenotype prevalences are only modestly different from those in other countries[1,2] and iterate the need to set up “state of art” severe asthma clinics for accurate phenotyping, especially when asthma is severe and biologics are contemplated.

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Conflicts of interest
There are no conflicts of interest.

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