Airway inflammation in asthma is heterogeneous. Cell types include eosinophilic, neutrophilic, paucigranulocytic and mixed cellularity. Studies have often been confounded by cigarette smoking or inhaled corticosteroid (ICS) treatment. We aimed to ascertain the prevalence of inflammatory subtypes in the absence of confounders and clarify whether steroid responsiveness is exclusive to eosinophilic asthma.

**Methods:** 78 non-smoking adults with current asthma were recruited. Inclusion criteria: hyper-responsiveness to hypertonic saline OR methacholine OR ≥12% increase in FEV1 post-bronchodilator. ICS was withdrawn until loss of control (LOC) or one month if no LOC. Patients were classified as eosinophilic (EA: ≥2% sputum eosinophils), or non-eosinophilic (NEA: <2%). Steroid responsiveness was assessed after 4-weeks fluticasone 500mcg bd.

**Results:** Baseline characteristics for EA and NEA were similar except ICS dose (872 vs. 403 mcg/day, p<0.05). After steroid withdrawal, 50 patients (64%) were eosinophilic, 26 (33%) paucigranulocytic, and 2 (3%) mixed cellularity. With steroid, the before/after outcomes for EA/NEA were (mean±SD): ACQ: EA 1.9±0.9/0.7±0.6; NEA 1.0±0.6/0.7±0.5, p-values<0.05; FEV1: EA 2.20±0.76/2.86±0.79, NEA 2.65±0.83/2.83±0.88, p-values<0.05; ΔPC20AMP doubling doses; EA 4.2±2.9; NEA 1.6±2.2, p<0.001. Cell type changed: from eosinophilic to mixed cellularity in 2/48 (4%) and neutrophilic in 2/48 (4%); and from paucigranulocytic to neutrophilic in 3/25 (12%). Overall a significant increase in sputum neutrophilia was seen after steroid (19±17% to 29±23%, p<0.01).

**Conclusions:** Steroid responsiveness occurred almost exclusively in EA and was minimal in NEA. In our population, neutrophilic asthma was absent, but inhaled steroid resulted in an increase in sputum neutrophilia. Whether steroid induced sputum neutrophilia is important is unclear.

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