

CORRECTION

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Correction to: Clinically relevant phenotypes in chronic rhinosinusitis



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Following publication of the original article [1], the authors reported an error in Table 1. In the second columns of the 'Radiology' row, 'Normal anterolateral sinus mucosa' should read 'Normal superolateral sinus mucosa'. A corrected version of Table 1 is included in this Correction.

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Table 1 Summary of Key Findings of CRS Phenotypes

Characteristics	Phenotype		
	CCAD (IgE mediated)	eCRS (AERD)	Non-eCRS
Clinical Presentation	<ul style="list-style-type: none"> - Young onset (teens to 20s) - Rhinitis symptoms - Smell preserved - Other atopic disease: <ul style="list-style-type: none"> ° Childhood asthma ° conjunctival symptoms, dermatitis 	<ul style="list-style-type: none"> - Mid-Life “adult” onset (30–50 yo) - Occasionally post respiratory virus - “Completely well” prior to onset or if allergic, then symptoms limited to childhood - Smell loss (corticosteroid responsive) - Antibiotic seeking - Food and alcohol induced flares - Adult onset asthma linked temporally to CRS onset. 	<ul style="list-style-type: none"> - Older onset 50 yrs.+ - Female, obese - Cough - Poor corticosteroid response - “Asthma” present but often poor response to inhaled preventive therapy (corticosteroid based)
Endoscopy	<ul style="list-style-type: none"> - Middle turbinate edema - Polypoid changes from turbinates and septum - No thick mucin - Normal sinus mucosa on surgery 	<ul style="list-style-type: none"> - Polyps (small, multiple, large) from the middle meatus - Thick eosinophilic mucin - Secondary purulence 	<ul style="list-style-type: none"> - Polyps or polypoid edema - Purulent secretions - Lack of eosinophilic mucin
Radiology	<ul style="list-style-type: none"> - Central thickening of septum and turbinates, peripheral clearing (CCAD) - Mucus trapping only in sinuses - Normal superolateral sinus mucosa (“black halo”) 	<ul style="list-style-type: none"> - Pan-sinusitis (Lund-Mackay 24) - Neo-osteogenesis 	<ul style="list-style-type: none"> - Pan-sinusitis (undistinguishable from eCRS)
Histopathology	<ul style="list-style-type: none"> - Elevated tissue eosinophilia - Often without activation (no eosinophil aggregates and charcot-leyden crystals) - No serum eosinophils - Elevated total and specific IgE 	<ul style="list-style-type: none"> - Elevated tissue eosinophilia (>10eos/hpf, but often >100eos/hpf) - Evidence of eosinophil activation (eosinophil aggregates and charcot-leyden crystals) - Serum eosinophilia 	<ul style="list-style-type: none"> - Lack of tissue eosinophilia (< 10/HPP)
Allergy	<ul style="list-style-type: none"> - + allergy testing (dustmite/perennial allergens) - Often monoallergen-sensitized 	<ul style="list-style-type: none"> - Either negative IgE sensitization or multi-allergen sensitized 	<ul style="list-style-type: none"> - Negative skin prick, immunocap/RAST
Treatment	<ul style="list-style-type: none"> - Allergen directed immunotherapy - Endoscopic sinus surgery - Topical corticosteroid (spray or irrigation) 	<ul style="list-style-type: none"> - Systemic corticosteroid treatment (up to 2–3 times per year) if limited burden of disease - Endoscopic sinus surgery (Draf 3) - Topical corticosteroid irrigations (not sprays) For AERD: <ul style="list-style-type: none"> - Zileuton, Montelukast, Zafirlukast - Can take selective COX-2 inhibitors (Meloxicam) 	<ul style="list-style-type: none"> - Saline or corticosteroid irrigations - Endoscopic sinus surgery - Macrolide therapy (Clarithromycin 250 mg daily for 3 months) - Continue 3/week until 12 months if responder
Difficult to control disease	<ul style="list-style-type: none"> - Omalizumab (anti-IgE) 	<ul style="list-style-type: none"> - Mepoluzimab (anti-IL5) - Other immune-modulating therapy (Benraluzimab, Dupiliumab, Reslizumab, etc) For AERD: <ul style="list-style-type: none"> - ASA desensitization (1300 mg commencement and 350–700 mg daily maintenance) 	<ul style="list-style-type: none"> - Consider re-biopsy of a patient post-surgery and post-corticosteroid based treatment if not responding and may be re-classified under this phenotype