**Chronic rhinosinusitis and immunodeficiency**

We write with regards to the recent article on sinusitis by Morcom et al (AFP June 2016).¹ We wish to highlight that recurrent or chronic rhinosinusitis (CRS) may be a sign of an underlying immunodeficiency, which is an important consideration for general practitioners (GPs) and other clinicians in the assessment of patients with rhinosinusitis. Immunodeficiency is more likely if rhinosinusitis persists or recurs despite adequate medical and/or surgical management,² and it is of the ‘without nasal polyposis’ subtype.³

CRS may indicate dysfunctional humoral immunity, as this part of the adaptive immune system is particularly important in the control of bacterial infections. Any humoral immunodeficiency, for example, common variable immunodeficiency (CVID), may predispose to chronic or recurrent acute rhinosinusitis. Patients with a humoral deficiency are also more susceptible to other sinopulmonary infections, such as otitis media and pneumonia. Retrospective studies found approximately one-fifth of patients with CRS have evidence of low/deficient antibody levels⁴ and two-thirds had a depressed antibody titre response to vaccine challenge, indicative of a specific antibody defect.⁵

Consequently, we believe GPs should consider testing patients with CRS for immunodeficiency, including serum immunoglobulins, lymphocyte subset analysis and vaccine antibody responses, particularly if there are other clinical red flags such as recurrent, deep-seated, unusual or prolonged infections, or a suggestive family history. Consideration must also be given to secondary causes of immunodeficiencies (eg human immunodeficiency virus (HIV), immunosuppressive drugs) and, where possible, managed accordingly. A referral to a clinical immunologist or other specialist may be in order as appropriate.

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**References**


**Reply**

Individuals with pre-existing or acquired immunodeficiencies are at risk of developing CRS. In a study by Chee et al⁶ of 79 patients with CRS (with or without nasal polyps) refractory to medical treatment, 30% had decreased T-cell function and 20% had an immunoglobulin deficiency. Common variable immunodeficiency was present in 10%.

A meta-analysis by Schwitzguébel et al⁷ revealed immunoglobulin deficiencies in 13% of patients with refractory CRS, and 23% of patients with difficult-to-treat CRS (persistent symptoms despite surgery and ongoing medical therapy). As such, investigation of possible immunodeficiencies should be considered if adequate medical treatment has failed, or for individuals with multiple infections (otitis media, bronchitis, pneumonia).

The benefits of surgery in individuals with a non-acquired immunodeficiency are similar to that experienced by individuals with normal immune function⁸ and, as such, treatment should be approached in a similar fashion.

The presence of a sinus mycetoma (fungal ball) is not suggestive of an underlying immunodeficiency, with no difference in immunoglobulin levels detected compared to CRS.⁴ Acquired immunodeficiencies (HIV infection, haematopoetic stem cell transplants) may predispose to acute invasive fungal rhinosinusitis, with a significant risk of mortality. Treatment in this setting should be multimodal, with surgical debridement and antifungal treatment, along with management of the underlying immunodeficiency state.

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**References**


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