The sense of smell in primary ciliary dyskinesia

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Olfactory dysfunction is common in chronic rhinosinusitis (CRS); indeed, it is one of the key diagnostic symptoms in both the European Position Paper on Rhinosinusitis¹ and the American Clinical Practice Guidelines on Sinusitis.² Objective evidence of hyposmia/anosmia has been reported in 30%–78% of patients with CRS, depending on which test is used.³ Subjective complaints of a loss of or reduction in the sense of smell have been shown to correlate well with objective measures.⁴ Loss of the sense of smell is an important symptom from a quality of life perspective and is known to correlate with depressive symptoms.

In inflammatory CRS, the cause of olfactory dysfunction is often multifactorial; nasal polyps are a physical barrier to odorant molecules reaching the olfactory epithelium, whereas oedematous mucosa also contributes to this ‘conductive’ problem. Motile cilia on the nasal epithelium may be impaired as a result of the disease process, as can the non-motile (primary) olfactory cilia on the specialised olfactory epithelium that are essential for initiation of the olfactory signalling cascade.⁵

Primary ciliary dyskinesia (PCD) is a congenital motile ciliopathy, associated with chronic sinusitis due to impaired mucociliary clearance. Pifferi *et al*⁶ hypothesise that olfactory function might be impaired in this condition due to primary ciliopathy affecting the olfactory cilia. There certainly seems to be growing

evidence of this in other ciliopathies.⁷ The idea is an interesting one and would certainly be useful to know from a clinical point of view, if only to better counsel patients as to the likelihood of improvement of this very important symptom with treatment.

This study found that patients with PCD were more likely to experience hyposmia and anosmia than their non-PCD sinusitis counterparts (74% vs 24%, respectively). Patients with PCD had significantly worse olfactory scores than non-PCD sinusitis patients, even taking into account the radiological severity of the sinus disease. The CT score has previously been shown to correlate well with the level of olfactory dysfunction,⁸ suggesting that in the patients with PCD there is an additional cause for their loss of smell. The fact that olfactory dysfunction was worse in patients with PCD with major ultrastructural abnormalities adds further support to this theory.

Patients with PCD seem to experience olfactory dysfunction more commonly and more severely than the general CRS population. It would seem worthwhile to obtain baseline smell measurements as routine in PCD clinics, using Sniffin’ Sticks or a similar easy-to-use objective test, along with baseline lung function and hearing tests. Patients can then be counselled appropriately, which is so important when a symptom adversely affects quality of life as much as this one does.

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