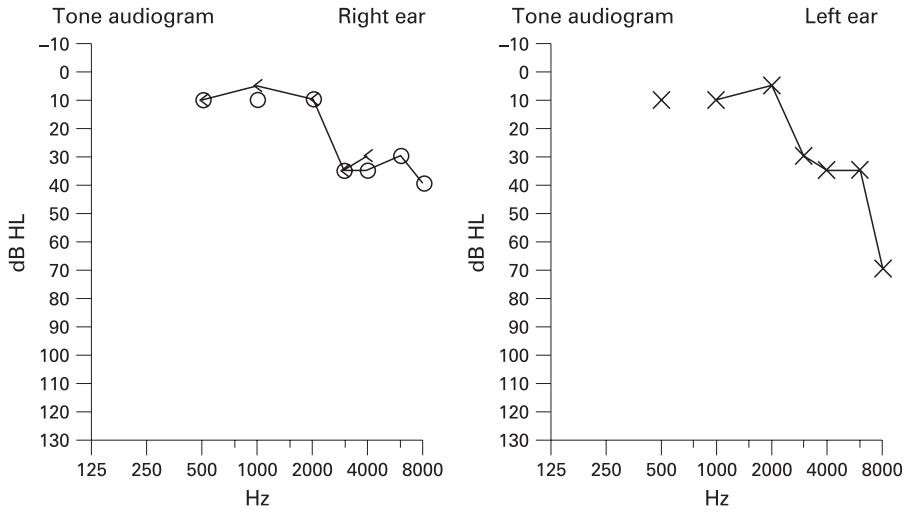

Figure 1 Audiogram showing bilateral high frequency hearing loss.

mutation. The prevalence of the m.1555A>G mutation in the UK is quoted as 1 in 40 000, but this figure is based on studies in children who are born deaf. Variable gene penetrance and aminoglycoside exposure means the actual prevalence is likely to be higher.[3]

The use of aminoglycoside antibiotics is widespread in CF care. First isolation of *P aeruginosa* in a patient with CF necessitates treatment to eradicate it. Nebulised and oral antibiotics or intravenous antibiotics are used.[4] For chronic *P aeruginosa* infection, intermittent intravenous antibiotics are frequently required. To prevent antibiotic resistance, two agents are used—usually a β-lactam and an aminoglycoside.[5] Ototoxicity and renal toxicity with aminoglycosides are well recognised, but these factors need to be balanced against clinical need.

The cost-effectiveness of screening for the m.1555A>G mutation in individuals who will potentially receive aminoglycosides makes financial sense, given the costs to the health service and to society of meeting the needs of profoundly deaf individuals.[3] This case highlights the potential need for more widespread screening for the m.1555A>G mutation in patients with CF. This will ensure that we can protect against aminoglycoside ototoxicity where possible, or at least fully inform our patients of the potential risk of deafness with these antibiotics.

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REFERENCES

1. **Usami S,** Abe S, Shinkawa H, *et al*. Sensorineural hearing loss caused by mitochondrial DNA mutations: special reference to the A1555G mutation. *J Commun Disord* 1998;**31**:423–34.
2. **Hu DN,** Qui WQ, Wu BT, *et al*. Genetic aspects of antibiotic induced deafness: mitochondrial inheritance. *J Med Genet* 1991;**28**:79–83.
3. **Bitner-Glindzicz M,** Rahman S. Ototoxicity caused by aminoglycosides. *BMJ* 2007;**335**:784–5.
4. **Frederiksen B,** Koch C, Høiby N. Antibiotic treatment of initial colonization with Pseudomonas aeruginosa postpones chronic infection and prevents deterioration of pulmonary function in cystic fibrosis. *Pediatr Pulmonol* 1997;**23**:330–5.
5. **Cheng K,** Smyth RL, Govan JR, *et al*. Spread of beta-lactam-resistant Pseudomonas aeruginosa in a cystic fibrosis clinic. *Lancet* 1996;**348**:639–42.

Anti-IgE in Churg–Strauss syndrome

The cases reported by Grohé *et al* are interesting, but they need to be better debated.[1] As far as we know, we were the first group to administer anti-IgE to a patient with Churg–Strauss syndrome (CSS) with asthma.[2 3]

In our patient, CSS had shown remission with systemic corticosteroid but he still presented with asthma which was difficult to control. The asthma did not improve despite the use of high-dose inhaled corticosteroids, long-acting β_2 agonists and several courses of steroid pulse therapy. Omalizumab was given and his asthma was controlled as well as could be expected, but a marked improvement in eosinophilia was also observed. The patient was not taking systemic corticosteroids or immunosuppressive agents at this time.

Besides the IgE blockage effect, studies have shown antiallergic and anti-inflammatory properties of anti-IgE, among which is the reduction in circulating and tissue eosinophils.[4] On the other hand, there are at least three cases of temporal association between anti-IgE use and the development of CSS.[5] CSS was probably caused by systemic steroid tapering in these cases, rather than anti-IgE use.

In the report by Grohé *et al*, it was not clear if the patients showed only improvement in respiratory symptoms or also in other CSS manifestations after introduction of anti-IgE. Moreover, at the time omalizumab was administered, the patients were also taking corticosteroids and immunosuppressives (all these drugs were acting).

Together, these cases suggest that omalizumab can be used in patients with CSS to treat and control asthma, but they also show that anti-IgE alone is not enough to control CSS activity. Systemic corticosteroids are the drug of choice to treat this disease.[5]

Although we are getting more evidence about the safety of omalizumab in patients with asthma and CSS, larger long-term studies are needed before the widespread use of anti-IgE can be recommended and also to verify if it is effective in the treatment of CSS.

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REFERENCES

1. **Grohé C,** Tiyerili V, Pabst S. Apparent response to anti-IgE therapy in two patients with refractory "forme fruste" of Churg Strauss syndrome. *Thorax* 2008;**63**:747–8.
2. **Giavina-Bianchi P,** Giavina-Bianchi M, Agondi R, *et al*. Administration of anti-IgE to a patient with Churg-Strauss syndrome. *Int Arch Allergy Immunol* 2007;**144**:155–8.
3. **Giavina-Bianchi P,** Agondi R, Kalil J. One year administration of anti-IgE to a patient with Churg-Strauss syndrome. *Int Arch Allergy Immunol* 2008;**146**:176.
4. **Noga O,** Hanf G, Brachmann I, *et al*. Effect of omalizumab treatment on peripheral eosinophil and T-lymphocyte function in patients with allergic asthma. *J Allergy Clin Immunol* 2006;**117**:1493–9.
5. **Giavina-Bianchi P,** Giavina-Bianchi M, *et al*. Omalizumab and Churg-Strauss syndrome. *J Allergy Clin Immunol* 2008;**122**:217.

Authors' reply

We thank Giavina-Bianchi *et al* for their response to our letter. Treatment of patients with Churg–Strauss syndrome (CSS) remains a challenge owing to the heterogeneity of the underlying symptoms. We have followed our patients since early 2006 and have noticed an overall improvement in both pulmonary (obstructive airway disease) and systemic (ie, congestive) heart failure as a result of the addition of omalizumab. In particular, compared with the prior worsening, the clinical course of both patients