Pseudo-steroid resistant asthma

The report by Thomas et al of patients whose symptoms masquerade as asthma is to be welcomed, although it is not clear how the authors selected their patients from all those referred to their clinic with “true” steroid resistant asthma. In addition, the opportunity has not been taken to see what proportion of these patients would have fulfilled the criteria for type 1 brittle asthma.

In a recent review of brittle asthma1 I discussed, along with one of the authors of this report, factors such as gastro-oesophageal reflux, psychosocial aspects, sleep apnoea, and hyperventilation/vocal cord adduction as contributory or coincident factors, and in that review we put in a plea for future studies to try to characterise phenotypically patients at the severe end of the spectrum compared with other phenotypes or genotypes.

In our patients with brittle asthma hyperventilation is common and I agree that it can be difficult to discern between pure hyperventilation and hyperventilation on a background of pre-existing asthma. The authors describe a hyperventilation test but it is perhaps surprising that they have not reported which test they used nor what objective measures were made. The tendency to hyperventilate commonly overlaps with the differential of psychosocial aspects in what asthma.3 The authors misquote our paper1 in that we did not, in that study, compare severe asthma with another chronic disease group. Equally, the authors have not read our second paper2 which, although the psychosocial impact was greater. This study also provided evidence of unusual self management strategies in this group which, in many cases, led to delay in starting or increasing oral steroids. The authors of this report assumed that the episode was not primarily severe bronchoconstriction (as presumably clinically perceived at the time), and is a major clue to the underlying functional aetiology of the problem (either vocal cord dysfunction or pseudo-severe asthma).4 High inflation pressure or a prolonged period of ventilation suggest that there is severe airways obstruction and any associated functional disease is superimposed. This leads on to the next point—namely, that severe asthma and vocal cord dysfunction/hyperventilation may, and frequently do, coexist. Diagnostic examination may help to identify those patients who have significant underlying inflammatory disease of the airways, but the sensitivity and specificity of lavage and biopsy material has not been evaluated in patients with severe asthma on steroid therapy. In addition, the identification of vocal cord dysfunction and/or hyperventilation does not necessarily result in a successful withdrawal of steroids and, indeed, inappropriately rapid or unsupervised withdrawal of treatment may have catastrophic results if severe asthma and vocal cord dysfunction are coexistent.

The readers of this article need to be aware of the practical dangers involved in the complex management of these patients. Rapid withdrawal of steroids in a patient who has been shown to have either vocal cord dysfunction or hyperventilation is hazardous as the latter conditions may be a sequel to or co-exist with severe asthma. We would recommend a staged steroid withdrawal during hospital supervision even in patients with convincing evidence of alternative pathology to explain their apparent “pseudo-steroid resistant asthma”.

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