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Dysphonia as an atypical presentation of gastro-oesophageal reflux

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Abstract
We present three cases of dysphonia in which gastro-oesophageal reflux (GOR) appeared to be a precipitating factor. In all cases reflux was clinically occult. We discuss the theory and implications of GOR presenting atypically with laryngeal symptoms, and outline our current approach to laryngeal symptoms of clinically evident or occult GOR.

Key words: Voice disorders; Larynx; Gastro-oesophageal reflux

Introduction

The manifestations of gastro-oesophageal reflux (GOR) in Otolaryngology are bewildering in their complexity and heterogeneity. Globus syndrome, otalgia, and even halitosis have at one time or another been ascribed to GOR (Bain *et al.*, 1983). Delahunty and Cherry (1968) were the first to connect abnormal laryngoscopic signs with GOR. However, most published work describes patients in whom the diagnosis of GOR is straightforward because of the presence of classical symptoms such as heartburn or regurgitation, whilst it is recognized that these symptoms are often absent (Koufman, 1991). In this paper we describe three patients who presented with dysphonia which recovered only after treatment for GOR. It is concluded that dysphonia may be an atypical presentation of GOR.

Case reports

Case 1

A 28-year-old woman was referred with a six-year history of severe dysphonia. In the past she had undergone two courses of speech therapy with no improvement. There were no symptoms of dysphagia or GOR. Examination revealed a structurally normal larynx but there was a wide gap between the vocal folds on phonation which disappeared on coughing. A diagnosis of functional dysphonia was made and a further course of speech therapy was commenced. She remained dysphonic despite good motivation.

In view of her poor response a fibre-optic oesophagoscopy was performed. This revealed linear erosions and erythema of the oesophagus together with free GOR and a small hiatus hernia. The patient was prescribed a three-month course of omeprazole. At follow-up her voice was normal with fully functioning vocal folds. Omeprazole was stopped, but she has required subsequently further anti-reflux medication for her dysphonia.

Case 2

A 52-year-old lady was referred with a seven-year history of fluctuating dysphonia. There were no precipitating factors in her history, although her job entailed using the telephone for long periods. She denied symptoms of GOR, and was a non-smoker.

Examination showed a minor degree of erythema of the mid portion of both vocal folds, which moved normally. Areas of vocal abuse were identified, and a diagnosis of functional dysphonia was made. A dramatic improvement was made by improving her vocal technique and hygiene, but within a few weeks her voice deteriorated again. At this point examination revealed erythema of both arytenoids. Routine haematology, thyroid function tests and a chest X-ray were all normal, but fibre-optic oesophagoscopy showed a sizeable hiatus hernia with free GOR, and erosive oesophagitis. After two months treatment with omeprazole her voice returned to normal. On two occasions since then she has tried to withdraw from omeprazole, with subsequent relapse of her dysphonia.

Case 3

A 33-year-old gentleman was referred with several years of recurrent severe dysphonia. He was a semi-professional singer who had been formally trained and still undertook regular singing lessons. His dysphonia was unrelated to periods of heavy voice use. He denied symptoms of GOR. In the past he had undergone a septoplasty and subsequently a tonsillectomy for similar symptoms with a presumed diagnosis of recurrent laryngitis.

Examination revealed an entirely normal larynx, and speech therapy was commenced for a diagnosis of functional dysphonia. Despite good motivation there was no improvement in his voice. Further examination revealed uniform erythema of the supraglottic larynx. Fibre-optic oesophagoscopy showed free GOR with erythema and oedema of the oesophagus. Treatment with omeprazole for one month resulted in complete

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resolution of his symptoms, although cessation of this treatment caused a return of his dysphonia.

Discussion

A patient who describes symptoms of GOR together with atypical laryngopharyngeal symptoms will often display the signs of what has been christened 'Acid laryngitis' (Ward and Berci, 1982). These are erythema and thickening of mucosa in the inter-arytenoid region, often with inflammation of the posterior third of the vocal fold. Jacob *et al.* (1991) showed that reflux may be forceful enough to traverse the upper oesophageal sphincter aided by a paradoxical relaxation of this sphincter particularly during sleep. This relaxation occurs independently of swallowing. Therefore, because the larynx is not elevated, the refluxate may spill into the larynx.

Koufman (1991) recognized that, by traditional symptomatology, in more than half the patients with laryngopharyngeal symptoms GOR was occult. Therefore a high degree of clinical suspicion is required in these patients. The patients described above show that GOR may present with dysphonia as its sole symptom. We believe that the association between laryngeal symptoms and GOR in our patients is valid given the marked 'on-off' effect with omeprazole shown by all three. Nor are we the first to describe such an association. Hallewell and Cole (1970) described five patients subsequently shown to have hiatus hernia who presented with dysphonia as a single symptom. All had normal laryngoscopic findings.

Common to all these patients is the considerable delay in the diagnosis of GOR, with significant social and economic morbidity. This situation is the counterpart of that described by Koufman and Blalock (1991) where functional factors co-exist with organic laryngeal disease. The latter will remain incompletely treated until the former functional deficit is addressed. Similarly in GOR-related laryngeal symptoms, the voice will not improve until the problem of reflux is addressed, as is illustrated by our patients. In the patients described above the diagnosis was unexpected, illustrating that GOR must be considered as a possibility in any patient whose dysphonia is unresponsive to standard treatments.

The diagnosis of GOR may present difficulties of its own. Radiological studies are notoriously unreliable (Richter and Castell, 1982). Short term pH manometry is a specific but unsensitive technique (Behar *et al.*, 1976). Prolonged ambulatory pH manometry is recognized as the diagnostic gold standard, but is beset with many practical difficulties, and is by its nature time-consuming (Castell, 1990). In our experience it is regarded essentially as a research tool. Fibre-optic endoscopy is a useful first line investigation although interpretation is operator dependent (Batch, 1985). The sensitivity of this investigation may be greatly enhanced by histological confirmation of oesophagitis (Brand *et al.*, 1979). Our current policy is to treat patients with classical symptomatology of GOR empirically, whilst those in whom clinically occult reflux is thought to be a possible factor are endoscoped and then treated accordingly. In this category we would now include those patients with a provisional diagnosis of functional dysphonia who do not improve with speech therapy. This presupposes a situation in which the therapist can recall patients to the voice clinic with ease.

We now treat all our GOR patients with the proton pump inhibitor omeprazole. This has been found to be superior to the H_2 antagonists both as a primary treatment and as maintenance therapy (Hallerback *et al.*, 1994).

We would urge those caring for patients with voice disorders to remember GOR in their differential diagnosis.

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