GERD : 'Silent Refluxers'

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Gastro-esophageal reflux (GER) may cause a wide spectrum of extra-oesophageal manifestations such as non cardiac chest pain, asthma, posterior laryngitis, chronic cough, recurrent pneumonitis and even dental erosions. Many of these patients may not have the classic oesophageal symptoms of gastro-esophageal reflux disease (GERD) viz., heartburn or regurgitation and could well be termed as “silent refluxers”. In the absence of typical esophageal symptomatology, GERD may not be suspected as the root cause when dealing with the above extra oesophageal symptoms. The problem is further compounded by the fact that it may be difficult to establish a causal relationship even if GER can be established by investigations (e.g. pH studies) because individuals may simply have two common diseases which are unrelated.

GER related chest pain may exactly mimic angina pectoris in all aspects including worsening with heavy exercise. However, the majority of patients with GERD-induced chest pain have heartburn symptoms [1]. Up to 30% patients with GERD related asthma have no esophageal symptoms. GERD should be considered in asthmatics who present in adulthood, those without extrinsic allergic component and those not responding to bronchodilators or steroids [2]. The proposed mechanisms of reflux induced asthma include aspiration of gastric contents into the tracheobronchial tree with secondary bronchospasm or a vagus mediated reflex from esophagus to the lung causing bronchoconstriction. Microaspiration of gastric contents is the likely cause of a variety of laryngeal symptoms and signs of which “reflux laryngitis” is the most common. GERD is the third leading cause of chronic cough (after sinus disease and asthma), accounting for 20% of the cases [3].

The lower end of esophagus is well equipped with defense mechanisms like lower esophageal sphincter, mucosal buffers, bicarbonate production, peristalsis, etc.

to combat the periodic episodes of gastric reflux. The sensitive upper airway mucosa lacks these defenses thus rendering it vulnerable to reflux episodes extending beyond the lower esophagus. In this issue of MJAFI, Dutta et al [4] have cast light upon the atypical manifestations of GERD, especially those affecting the upper airways. Despite being increasingly recognized as a distinct clinical entity, numerous questions remain unanswered on the exact nature of laryngopharyngeal reflux (LPR).

It is still difficult to establish a correlation between GERD and airway disorders because of the common occurrence of both in the general population and the fact that a majority of patients with suspected reflux related ear, nose and throat (ENT) manifestations deny classical reflux symptoms like heartburn and regurgitation. Co-existence of other factors like smoking, drugs (anticholinesterase (ACE) inhibitors etc.), environmental irritants, chronic rhinosinusitis with postnasal drip etc. further confound the picture. The response to empiric twice daily proton pump inhibitors (PPI's) is also suboptimal in those with reflux related laryngeal disease with a significant majority reporting little or no relief even after prolonged (3 to 6 months) duration of therapy [5]. Misdiagnosis of GERD as a cause of laryngeal symptoms contributes in a large way to the non-response to PPI therapy and a recent Cochrane review found that there is insufficient evidence to conclude that treatment with a PPI is beneficial for chronic cough associated with GERD [6,7]. Hicks et al. [8] conducted a study on 105 healthy volunteers and found that 86% had one or more laryngoscopic findings included in the Reflux Finding Score (RFS) and some of the signs reached a prevalence of 70%, thereby casting a question mark on the diagnostic specificity of the laryngoscopic findings thought to indicate GERD.

The role of conventional modalities like 24 hour

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ambulatory pH monitoring and upper gastrointestinal (GI) endoscopy also remains ill defined in the algorithm for evaluation of LPR. While Dutta et al. recommend that pH metry be done prior to initiation of therapy, most published reviews recommend it only after the failure of a reasonable duration (usually 3 months) of PPI therapy [9]. Some authors recommend pH testing while on PPI and dose escalation if the reading is abnormal, others prefer cessation of therapy prior to testing. Though technically cumbersome, combining a pharyngeal pH probe with a standard lower esophageal probe to detect acid reflux in the pharynx has also been advocated recently. Those in favour argue that documenting even a single acid reflux event in the unprotected upper airway is sufficient to establish a cause-effect relationship [9]. Also, while the pH probe is capable of recording even subtle changes in pH, no available technique quantifies the volume of each refluxate. It is fair to presume that a larger volume of refluxate is more likely to overcome the anatomical and physiological barriers and cause injury. While endoscopic findings are abnormal in nearly half of the patients with typical GERD, only 20% with atypical GERD have endoscopic abnormalities [10]. Poelmans et al. [11] however reported a positive endoscopy in 45% case with atypical reflux thus reigniting the debate on the role of upper GI endoscopy.

Emerging technologies like intraluminal impedance monitoring that uses electrical impedance to detect and monitor liquid and air movement in the esophageal lumen (thus documenting all reflux episodes) and Bilitec, a spectrophotometric method to detect the presence of bilirubin in the refluxate (thereby providing evidence of duodeno-gastro-esophageal reflux), have added to the limited armamentarium to diagnose reflux related disorders [12]. Recent studies using both these modalities during PPI therapy have demonstrated non-acid reflux as a cause of persisting symptoms in a majority of patients with PPI refractory typical GERD [13]. In the absence of response to PPI's, the role of non-acid reflux, gaseous reflux and duodenal contents (bile, pancreatic enzymes) need further evaluation in those with atypical GERD. It is conceivable that future algorithms to evaluate LPR will incorporate these techniques.

While chronic upper airway disorders are well recognized atypical manifestations of GERD, in the

absence of well defined criteria one must not get lured by the temptation to attribute all undiagnosed chronic cough and hoarseness to reflux. Lack of response to PPI combined with negative pH studies/ endoscopy should necessitate a search for an alternate pathology. The role of newer diagnostic techniques in the diagnostic algorithm for LPR is still undefined.

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