Gastroesophageal Reflux Causing Stridor*

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We describe an infant with stridor associated with gastroesophageal reflux (GER). This is the first report in which there is clear documentation by pH probe of a temporal association between individual episodes of GER and stridor. We review the literature and speculate on the relationship between these two phenomena. GER should be considered in patients with stridor.

Gastroesophageal reflux has been implicated in the production of a variety of respiratory problems, including aspiration pneumonia and chronic cough,1,4 asthma,2,3 bronchiolitis,6 bronchitis,7 apnea,7,8 and sudden infant death syndrome (SIDS).9 Although the association of GER and laryngospasm is likely,6 a close temporal relationship between individual episodes of GER and episodes of stridor has never been documented. We report here an infant with episodic inspiratory stridor clearly associated temporally with GER episodes and relieved by pharmacologic treatment of GER. This case provides support for the hypothesis that reflex laryngospasm may cause stridor in infants with GER and also suggests an explanation for the rarity of stridor in patients with reflux.

CASE REPORT

A nine-day-old black girl had had several episodes of vomiting and “choking,” as well as intermittent mild-to-moderate stridor. She was the term product of an uncomplicated pregnancy. There was no history of anemia, abnormal weight loss, apnea, or cyanosis.

Physical examination showed a normal infant, except that mild micrognathia was present, and stridor with intercostal, suprasternal and substernal retractions was noted intermittently. The stridor could often be relieved by anterior traction on the lower jaw.

Barium swallow showed gastroesophageal reflux to the level of the hypopharynx, without aspiration. Chest x-ray film findings were normal. Esophageal manometry documented a short (0.5 cm) lower esophageal sphincter 18-18.5 cm from the nares, coinciding with the point of reversal of intraesophageal inspiratory pressure, which is the diaphragmatic division between abdomen and thorax. The mean lower esophageal sphincter pressure was 9.7 mm Hg (normal in our lab >10 mm Hg). Recording of distal esophageal pH over 48 hours using an indwelling pH probe8 showed considerable reflux (21 percent of the time spent with esophageal pH <4) with delayed clearance of acid (19 episodes longer than five minutes, and the longest episode 63 minutes). Episodes of stridor occurred only during episodes of reflux.

During the next nine days she was treated with betahanechol, 1.25 mg 20 minutes before feedings, and prone head-elevated positioning at home and had a marked reduction in stridor. She returned to the clinic for a repeat two-hour pH probe evaluation 12 hours after her last dose of betahanechol. The study again revealed stridor associated with episodes of gastroesophageal reflux; it was again relieved by spontaneous clearance of refluxed acid from the esophagus (Fig 1). A subcutaneous injection of betahanechol temporarily prevented recurrence of gastroesophageal reflux; stridor was absent during this reflux-free period. In these instances, stridor cleared without manipulation of the jaw.

Stridor disappeared completely within the next month, and did not recur during the succeeding five months of betahanechol treatment, nor after betahanechol was withdrawn.

DISCUSSION

This report documents a clear temporal association between individual episodes of gastroesophageal reflux and individual episodes of stridor in an infant, and thus expands our understanding of this interesting phenomenon. The immediate onset of stridor with the onset of reflux episodes and their resolution with acid clearance were consistent and striking.

Two prior reports implicate gastroesophageal reflux in the pathogenesis of stridor.10,11 The relationship between gastroesophageal reflux and stridor in these two reports was not as clear-cut as in our infant. None of the ten infants previously reported is described as having intermittent stridor, and the purported relationship between gastroesophageal reflux and stridor is made on the basis of worsening respiratory symptoms after reflux episodes or resolution of stridor after treatment of gastroesophageal reflux. Six of

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FIGURE 1. pH measured at 2 cm above the manometrically-determined position of the lower esophageal sphincter. S: stridor.

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the infants had increase in retractions five to ten minutes after onset of pH probe-documented reflux, suggesting that their upper airway obstruction had increased. Two of five infants treated medically and six of six treated surgically had resolution of their stridor within two to three weeks. These temporal relationships are suggestive, but not incontrovertible, evidence of a causal relationship between gastroesophageal reflux and stridor. Our case is stronger proof of such a relationship.

Four logical alternative explanations could account for stridor and gastroesophageal reflux occurring together: 1) the reflux causes the upper airway obstruction (eg, by reflex laryngospasm with the sensory input either esophageal or laryngeal); 2) the upper airway obstruction causes the gastroesophageal reflux (eg, by excessive negative intrathoracic pressure overcoming a low esophageal sphincter pressure); 3) the upper airway obstruction and gastroesophageal reflux are both caused by a third phenomenon (episodic fluctuations in autonomic smooth muscle tone in the larynx and lower esophageal sphincter); and 4) the upper airway obstruction and gastroesophageal reflux occur together by chance alone. The fourth explanation is very unlikely because of the frequent, documented, clear relationship between the two phenomena in the 11 infants reported to date. The second and third explanations may contribute to the association between gastroesophageal reflux and stridor, although there is little specific evidence in their favor at present. The fact that the relief of the stridor followed the treatment of the reflux in all patients reported is strong evidence in favor of the first explanation. Our patient's repeated relief of stridor with spontaneous clearance of refluxed acid indicates that it was the acid clearance rather than any direct effect of a treatment that relieved the stridor.

Herbst et al.15 have documented laryngospasm as a response to intraesophageal instillation of acid in some infants. The exact mechanism of this response is unknown, but vagal reflexes have been implicated. Similarly, gastroesophageal reflux causing laryngospasm has been implicated in some instances of apnea and SIDS.

Many people have gastroesophageal reflux, but only rarely does stridor result. All reported patients with gastroesophageal reflux and stridor have been infants, perhaps because their normally smaller upper airways predispose to stridor, or because laryngospasm as a reflex response to gastroesophageal reflux is the response of an immature nervous system. Three of these individuals, including our patient, had an upper airway anomaly (laryngomalacia or micrognathia) further predisposing to stridor, although apparently insufficient alone to cause it. The four other reported patients who underwent bronchoscopy had laryngeal inflammation, perhaps also a cause of airway narrowing. We suggest that stridor in infants with gastroesophageal reflux may only occur in infants who also have upper airway narrowing, which may be primary, or secondary to laryngeal inflammation caused by refluxed acid. Whether the contribution of the gastroesophageal reflux to laryngospastic events is mediated through vagal reflexes from the esophagus or through laryngeal aspiration is unknown, although several studies implicate the former.

We recommend that gastroesophageal reflux, as well as causes of upper airway narrowing, be considered in patients with stridor.

REFERENCES

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Serial Angiographic Evidence of Rapid Resolution of Coronary Artery Stenosis

Timothy A. Sanborn, M.D.; David P. Faxon, M.D.; Mirle A. Kellett, M.D.; and Thomas J. Ryan, M.D.

An example of rapid, spontaneous resolution of an eccentric coronary luminal narrowing from 95 percent to 80 percent and subsequently to 50 percent stenosis over a six-week time period is presented. Spontaneous thrombolysis is proposed as the explanation for these changes and is discussed with reference to existing experimental and clinical observations.

Spontaneous resolution of coronary stenosis after myocardial infarction has been demonstrated previously; however, the pathophysiology of this process remains unclear. The angiographic appearance of single or multiple thin

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