A life-threatening respiratory complication of gastro-oesophageal reflux in a patient with tetraplegia

I.P. Donald, M.W.L. Gear and S.P. Wilkinson

Gloucestershire Royal Hospital, Great Western Road, Gloucester, UK.

Summary: Recurrent attacks of life-threatening dyspnoea and choking occurred in a patient with tetraplegia. Conventional investigations for gastro-oesophageal reflux were normal, but 24-hour oesophageal pH recording revealed gross reflux in association with an attack of dyspnoea. Surgical correction of the reflux abolished the attacks. The possibility of autonomic dysreflexia as the mechanism linking reflux and respiratory symptoms in this patient is discussed

Introduction

Nocturnal asthma and other respiratory problems are now recognized as complications of gastro-oesophageal reflux.^{1–3} Here we describe a case occurring secondary to tetraplegia with associated autonomic failure. In this situation the syndrome of autonomic dysreflexia may arise; this is a mass reflex in the sympathetic and parasympathetic nervous systems which lack the normal modulation from higher centres in the brain.⁴ Manifestations include parasthesiae, headache, tightness in the chest and dyspnoea, hypertension, a facial flushing and cold peripheries. It may be triggered by a wide variety of stimuli, but most commonly by urinary outflow obstruction, rectal enemas, or pressure sores. In this patient, autonomic dysreflexia appeared to be precipitated by gastro-oesophageal reflux. Elucidation of the nature of the clinical problem was only possible by use of 24-hour oesophageal pH monitoring.

Case report

This 28 year old man sustained an injury to the spinal cord when aged 18 resulting in tetraplegia complete at the level of C6. He presented to a spinal injuries unit 9 years later with a 2 year history of attacks of epigastric pain, nausea, sweating and headache. These symptoms were interpreted as probable autonomic dysreflexia. Investigations into the likely cause of dysreflexia showed urinary outflow obstruction. Internal membranous urethrotomy was performed but this

resulted in loss of control over micturition with no improvement in his symptoms.

Six months later he presented to this hospital with attacks of dyspnoea, choking and coughing with frothy sputum in addition to the previous symptoms. Because of difficulty with expectoration the attacks were often life-threatening. He had also developed persistent anorexia and loss of taste. He was a non-smoker but had a family history of atopy. He was receiving baclofen and dantrolene for spasticity.

Several attacks of breathlessness were observed by us: he appeared pale, cyanosed, sweaty and tachypnoeic. Widespread crepitations were heard on chest auscultation, while the pulse rate and blood pressure were modestly elevated. Chest radiographs following attacks were normal except on one occasion when a partial right lower lobe collapse was seen. Arterial blood gas during an attack showed severe respiratory failure – pH = 7.29, PCO_2 = 8.0 kPa, PO_2 = 5.5 kPa. There was no improvement from intravenous diuretics or atropine, or inhaled salbutamol, but physiotherapy and inhaled ipratropium were of benefit. One episode occurred an hour after a barium swallow, but aspirated barium was not demonstrated radiologically.

Upper endoscopy and barium meal were normal with no evidence of reflux. A technetium swallow showed an oesophageal transit time of 15 seconds (normal <15 seconds). Twenty-four-hour oesophageal pH monitoring using a naso-oesophageal antimony electrode catheter (Synectics Medical) was grossly abnormal. While in a wheelchair the reflux duration (pH <4.0) was 14% (normal while upright <4.2%)⁵ and 21% while in bed (normal <0.8%). There were seven prolonged episodes of reflux, one lasting almost two hours and clearly associated with an attack of his usual respiratory

Correspondence: I.P. Donald M.A., M.R.C.P., Department of Health Care of the Elderly, Queen's Medical Centre, Nottingham NG7 2UH, UK.

Accepted: 25 November 1986

symptoms (Figure 1). Investigations included: serial electrocardiograms (normal); gated cardiac scan (left ventricular ejection fraction = 77%, normal > 45%); and skin sensitivity testing (negative). Lung function tests showed the expected restrictive defect with remarkable improvement following inhaled ipratropium but none after salbutamol (Table I).

The integrity of his autonomic nervous system was also briefly studied: there was only 2 beats/minute sinus arrhythmia on deep breathing, and carotid sinus and ocular pressure had no effect on heart rate. Valsalva manoeuvre caused a tachycardia in Phase II from 58 beats/minute to 86 beats/minute, but diastolic blood pressure fell 35 mm Hg with no subsequent overshoot or bradycardia at the end of the procedure.

The attacks continued and it was decided that he warranted anti-reflux surgery. An Angelchik prosthesis⁶ was inserted under spinal anaesthesia; no repair of the hiatus was required. The prosthesis was chosen in preference to a fundoplication because of the speed and simplicity of the procedure, with excellent reflux control.⁷

Following surgery there was complete resolution of

upper gastro-intestinal symptoms and his sense of taste returned. There were two mild attacks of breathlessness in the first week post-operatively but none in the subsequent year. The manifestations of autonomic dysreflexia also disappeared. Four months after surgery repeat lung function tests and oesophageal pH monitoring were performed. The former showed a marked improvement from pre-operative values (Table I), while the latter showed only three episodes of reflux, none of which lasted longer than 5 minutes.

Discussion

The presence of gastro-oesophageal reflux in the absence of any endoscopic abnormality of the lower oesophagus has been increasingly recognized with the advent of ambulatory oesophageal pH monitoring.⁵ In this patient, reflux had not been demonstrated by other techniques. Ambulatory pH monitoring can also demonstrate the temporal relationship of reflux and respiratory symptoms if an attack occurs during the recording, as was the case here. Where reflux precedes

Figure 1 pH recording during the evening (top line) and the night (middle and bottom lines). The event marker indicated the onset of respiratory symptoms at 3.15 a.m.

Table I Lung function tests before and after anti-reflux surgery

	Before surgery			After surgery	
	Baseline	After salbutamol	After ipratropium	Baseline	After ipratropium
FEV ₁ (ml)	900	950	1650	1700	1900
FVC (ml)	1200	1250	1850	1800	2000

FEV₁ = forced expiratory volume in one second.

FVC = forced vital capacity.

the onset of respiratory symptoms at night, treatment of reflux is likely to improve the problem.¹ A well documented case of apparent nocturnal asthma cured by treatment of reflux was recently described² and we have six additional patients.

It is uncertain as to whether acid causes respiratory symptoms directly by aspiration or indirectly by a vagovagal reflex. We were unable to demonstrate aspiration of a radio-isotope instilled in the stomach in this patient, as has been described.⁸ Dilute acid instilled in the mid-oesophagus has been shown to alter airways resistance in some asthmatic patients,⁹ and this response can be abolished in an animal model by vagotomy.¹⁰

On the basis of limited autonomic testing, this patient showed a deficient sympathetic and parasympathetic nervous system, as is usually the case in tetraplegia. The bronchi are often more dependent upon vagal tone in tetraplegia,⁴ and wheeze is commonly severe in mild chest infections. The response to inhaled ipratropium indicated resting cholinergic bronchospasm; anti-reflux surgery may have improved resting lung function by suppression of vagal stimulation.

This patient undoubtedly had respiratory symptoms associated with gastro-oesophageal reflux, and also evidence for autonomic dysreflexia. Although gastro-oesophageal reflux can cause respiratory symptoms in any individual possibly by a vagovagal reflex stimulated by acid in the oesophagus, it is conceivable that in patients with a hypersensitive autonomic nervous system, as in autonomic dysreflexia, this respiratory response to oesophageal acid stimulation is both more likely and more severe. This patient showed other manifestations of autonomic dysreflexia during his attacks. Gastro-oesophageal reflux should therefore be considered as a cause for respiratory symptoms in tetraplegia, and as a potential trigger for autonomic dysreflexia.

Acknowledgement

We are grateful to Dr C.J. Mathias for advice in the management of this patient and in the preparation of this report.

References

1. Pellegrini, C.A., DeMeester, T.R., Johnson, L.F., Skinner, D.B. Gastroesophageal reflux and pulmonary aspiration: incidence, functional abnormality, and results or surgical therapy. *Surgery* 1979, **86**: 110–119.
2. Bengtsson, U., Sandberg, N., Bake, B. *et al.* Gastro-oesophageal reflux and night-time asthma. *Lancet* 1985, **ii**: 1501–1502.
3. Editorial. Gastric asthma. *Lancet* 1985, **ii**: 1399–1400.
4. Mathias, C.J. & Frankel, H.L. Autonomic failure in tetraplegia. In Bannister, R. (ed) *Autonomic Failure; a Textbook of Clinical Disorders in the Autonomic Nervous System*. Oxford University Press, Oxford 1983, pp 453–488.
5. DeMeester, T.R. & Skinner, D.B. Technique, indications, and clinical use of 24 hour oesophageal pH monitoring. *J Thorac Cardiovasc Surg* 1980, **79**: 656–670.
6. Angelchik, J.P. & Cohen, R. A new surgical procedure for the treatment of gastro-oesophageal reflux and hiatus hernia. *Surg Gynecol Obstet* 1979, **148**: 246–248.
7. Gear, M.W.L., Gillison, E.W. & Dowling, B.L. Randomised prospective trial of the Angelchik anti-reflux prosthesis. *Br J Surg* 1984, **71**: 681.
8. Chernow, B., Johnson, L.F., Janowitz, W.R. & Castell, D.O. Pulmonary aspiration as a consequence of gastro-oesophageal reflux – a diagnostic approach. *Dig Dis Sci* 1979, **24**: 839–844.
9. Spaulding, H.S., Mansfield, L.E., Stein, M.R., Sellner, J.C. & Gremillion, D.E. Further investigation of the association between gastro-oesophageal reflux and bronchoconstriction. *J Allergy Clin Immunol* 1982, **69**: 516–521.
10. Mansfield, L.E., Hanneister, H.H., Spaulding, H.S., Smith, N.J. & Glab, N. The role of the vagus nerve in airway narrowing caused by intra-oesophageal hydrochloric acid provocation and oesophageal distension. *Ann Allergy* 1981, **47**: 431–434.