CASE REPORTS

Hiatus hernia and Mallory-Weiss syndrome

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THE ASSOCIATION of a small hiatus hernia with laceration of the oesophagogastric junction has been recognized in recent years, but the significance of this condition with a large hiatus hernia has not been commented on, and such a case is here reported.

Case report

A 74-year-old woman was admitted to Hammersmith Hospital on 12 August 1965. She had been well until that day when, 3 hr after her evening meal, she felt nauseated and then vomited blood spontaneously. She had two further haematemeses during the next 4 hr. There was no abdominal pain or melaena and nothing in her past history to suggest peptic ulceration or cirrhosis with varices as the cause. She had been taking small quantities of salicylates for relief of pain from osteoarthritis. In the past she had suffered from heartburn and a chest X-ray taken in 1962 had shown a large hiatus hernia (Fig. 1).

On examination she was pale and shocked. Her systolic blood pressure was 50 mmHg. There were no abnormal findings except for slight epigastric tenderness. A diagnosis of haematemeses from a previously symptomless peptic ulcer was made.

She was initially treated conservatively but over the course of 10 hr she had repeated haematemeses, losing 6.5 litres of blood. A transfusion of 3 units of plasma and 8 units of blood was given but her condition was not adequately maintained and a laparotomy was performed. Investigations done just prior to this revealed haemoglobin 61%, PCV 37%, and WBC 4000/mm³. Electrolytes: sodium 146, potassium 3.0, chloride 95, bicarbonate 36 mEq/l, and urea 105 mg/100 ml.

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FIG. 1. Large hiatus hernia with fluid level seen on chest X-ray.

At operation through a right upper paramedian incision a large hiatus hernia was found. Considerable traction was required to reduce the stomach fully, which was distended and contained about 1 litre of blood. Extending down the lesser curve from the cardia for approximately 6 cm was an area of subserous haemorrhage. There was no evidence of gastric or duodenal ulceration and so an anterior gastrotomy 10 cm long was made extending to within 5 cm of the cardio-oesophageal junction. The contained blood clot was evacuated and it was then evident that bleeding was coming from the region of the cardia. The

source was a longitudinal tear 5 cm long extending through the cardia into the lower oesophagus, situated posterolaterally on the right side, and involving mucosa, submucosa and muscle coats. It was underrun with a continuous suture but the haemorrhage did not cease until sutures were placed through the muscular layers. The gastrotomy was then closed.

Her progress following operation was uneventful except for a chest infection which resolved with physiotherapy and antibiotics. An attempt was made to assess gastric acid secretion but this was abandoned because of the discomfort it produced. The nasogastric tube was seen on screening to be curled up in the intrathoracic stomach. A Diagnex Blue test showed the presence of free acid. A gastrograffin swallow performed 9 days after operation showed a large hiatus hernia (Fig. 2). The oesophagus was slightly dilated, but there was no gross narrowing in the distal oesophagus. There was abnormal neuro-muscular activity with inefficient emptying and tertiary contractions.

FIG. 2. Gastrograffin swallow showing most of the stomach to be intrathoracic. Arrows mark the level of the diaphragm.

Comment

Mallory & Weiss (1929) considered that following repeated vomiting neuromuscular inco-ordination of the lower oesophagus resulted with failure of relaxation and suggested that propulsion of gastric contents against the unrelaxed oesophagus led to a sudden rise in the intragastric pressure and lacerations in the region of the cardia. They showed that similar lesions could be produced in the cadaver by raising the intragastric pressure and occluding the pylorus and lower oesophagus. These lacerations have, however, been described with the initial episode of vomiting, as in this patient, and they have been associated with conditions other than vomiting that raise intra-abdominal pressure, e.g. paroxysms of coughing, status asthmaticus and epileptiform convulsions (Atkinson *et al.*, 1961). It has been suggested that atrophic gastritis is a predisposing factor (Decker, Zamcheck & Mallory, 1953), but it was not present in this patient. At laparotomy the only indication of the underlying condition was the subserous haemorrhage along the lesser curve. This has rarely been described before (Freeark *et al.*, 1964), and indicates a deep tear involving the muscular coats. Fleischner (1956) first recognized the significance of a hiatus hernia with this condition and considered that the difference between the raised intragastric pressure on vomiting and the lower intrathoracic pressure acting at the cardia displaced above the diaphragm was partly responsible for the tears. It has been shown that transient herniation may occur on vomiting in normal subjects. Atkinson & Mitchell (1962) found a hiatus hernia, which was nearly always small and reducible, in six of their fourteen patients with Mallory-Weiss syndrome, and Degradi (1966) demonstrated a hernia in all of their thirty cases. Atkinson & Mitchell (1962) showed that lacerations limited to the cardia could be produced when the stomach of a cadaver was distended *in situ* and the pressure raised to between 130 and 150 mmHg. When the stomach was removed from the body the tears occurred in all regions. They considered that the pressure gradient acting on the cardia displaced into the thorax following a sudden rise of intra-abdominal pressure was responsible and they showed that this pressure gradient varied between 60 and 100 mmHg in healthy subjects during straining, retching and vomiting. These values fall short of those found necessary to produce a tear in the cadaver and the fact that the Mallory-Weiss syndrome is uncommon following vomiting suggests another factor may be necessary for its development. The increased pressure gradient in this patient must have been present over most of the stomach wall

for it was largely intra-thoracic and yet the tear was limited to the oesophago-gastric junction. It is suggested here that there is occasionally a failure of relaxation of the lower oesophagus which raises the pressure gradient to a sufficient degree to cause a tear, for the lower oesophagus normally relaxes during vomiting and in this patient the gastrografin swallow showed evidence of abnormal neuromuscular activity. It is probable that these tears result from the increased pressure gradient acting on the cardio-oesophageal junction that is displaced above the diaphragm, following a sudden rise in intra-abdominal pressure, and it is postulated that these only occur when a pressure wave is directed against an incompletely relaxed lower oesophagus.

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References

- ATKINSON, M., BOTTRILL, M.B., EDWARDS, A.T., MITCHELL, W.M., GADSBY PEET, B. & WILLIAMS, R.E. (1961) Mucosal tears at the oesophagogastric junction (the Mallory-Weiss syndrome). *Gut*, **2**, 1.
- ATKINSON, M. & MITCHELL, W.M. (1962) The mechanism whereby Mallory-Weiss mucosal tears at the cardia are produced. *Surgical Physiology of the Gastro-intestinal Tract. Proceedings of a Symposium held in the Royal College of Surgeons of Edinburgh* (Ed. by A. N. Smith), pp. 33-39. Royal College of Surgeons of Edinburgh, 1963.
- DECKER, J.P., ZAMCHECK, N. & MALLORY, G.K. (1953) Mallory-Weiss syndrome. Haemorrhage from gastro-oesophageal laceration at the cardiac orifice of the stomach. *New Engl. J. Med.* **249**, 957.
- DEGRADI, A.E., BRODERICK, J.T., JULER, G., WOLINSKY, S. & STEMPIEN, S.J. (1966) The Mallory-Weiss syndrome and lesion. A study of 30 cases. *Amer. J. dig. Dis.* **11**, 710.
- FLEISCHNER, F.G. (1956) Hiatus hernia complex. Hiatal hernia, peptic oesophagitis, Mallory-Weiss syndrome, haemorrhage and anaemia, and marginal oesophago-gastric ulcer. *J. Amer. med. Ass.* **162**, 183.
- FREEARK, R.J., NORCROSS, W.J., BAKER, R.J. & STROHL, E.L. (1964) The Mallory-Weiss syndrome. Increasing surgical significance. *Arch. Surg.* **88**, 882.
- MALLORY, G.K. & WEISS, S. (1929) Haemorrhages from lacerations of the cardiac orifice of the stomach due to vomiting. *Amer. J. med. Sci.* **178**, 506.

Recovery in severe glutethimide poisoning

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ALTHOUGH similar in chemical structure to phenobarbital, the sedative drug glutethimide ('Doriden') has a considerably higher mortality in cases of acute intoxication (Maher, Schreiner & Westervelt, 1962). Since the first report of the use of haemodialysis in acute glutethimide intoxication (Schreiner *et al.*, 1958) the procedure has come to have a generally accepted place in the management of this condition. The following case is reported not only to re-emphasize the features of acute glutethimide intoxication, but also because the remarkable clinical course shows that recovery is possible in cases of greater clinical and biochemical severity than those previously reported.

Case report

The patient, a 38-year-old housewife, had been prone to recurrent bouts of depression since her concentration camp experiences in her early teens. Two weeks prior to admission these depressive symptoms became more severe and on 18 August

1965 she was admitted to the Royal Melbourne Hospital following the ingestion of approximately forty glutethimide tablets (10 g).

On examination she was unconscious, but reacted in a semi-purposeful manner to painful stimuli. She had a temperature of 33.5°C, dilated and equal pupils, reacting sluggishly to light, pulse rate of 88/min, respiratory rate of 24/min and BP of 98 mmHg systolic. She was flaccid and areflexic, but would not tolerate an endotracheal tube. There was no other abnormality.

The usual regime for management of patients unconscious due to barbiturate intoxication was followed but within 36 hr her blood pressure had fallen despite intravenous metaraminol ('Aramine') in a dosage of up to 5 mg half-hourly. The pupils were fixed and dilated and she was unresponsive to painful stimuli although respiration was spontaneous and adequate. The serum glutethimide level was estimated by the method of Goldbaum, Williams & Koppányi (1960) to be 11.9 mg/100 ml