

Arteriomesenteric Duodenal Compression Following Head Injury

V. Naraynsingh, F.R.C.S. and R. U. Adam, F.R.C.S.(C)

Departments of Surgery and Neurosurgery, Port-of-Spain General Hospital, Port-of-Spain, Trinidad

ABSTRACT

A patient who lost 15 kg in weight while recovering from severe brain injury started vomiting after 7 weeks. Barium meal confirmed vascular compression of the duodenum as the cause of his vomiting. Vomiting persisted in spite of high calorie liquid feeds and hyperalimentation. Duodenojejunostomy, performed 4 weeks later, cured his vomiting. Arteriomesenteric duodenal compression must be considered when vomiting occurs in the severely brain-injured patient with weight loss, and indeed in any patient with prolonged recumbency and weight loss.

INTRODUCTION

Although vascular compression of the duodenum was first recognised by Rokitansky in 1842, the only description of its occurrence in the patient with severe brain injury was in 1973 (Bouzarth et al, 1973). Awareness of the association between these two conditions is important to the neurosurgeon as vomiting may be wrongly attributed to a central nervous system disorder.

We herein report on a patient who developed arteriomesenteric duodenal compression following severe brain injury, prolonged recumbency and weight loss.

CASE REPORT

A 17-year-old right-handed student was admitted on February 12, 1982, following a motor vehicle accident. He was restless with purposeless response to deep painful stimuli, increased tone on left, left hyperreflexia and left hemiparesis. He had left parietal scalp lacerations, blood in both nostrils and the left ear. The left pupil was moderately dilated with minimal light reaction. Skull and cervical spine X-rays showed no fractures. At emergency craniotomy, a left acute 75 ml fronto-parietal subdural haematoma was evacuated and a bone flap removed because of intense brain swelling. Post-operatively, he improved with fluid restriction, mannitol, dilantin and antibiotics. After initial intravenous therapy, he was fed by nasogastric tube, 2000 ml (1 Cal/ml) per 24 hours. In the 3rd week post-operatively, he showed mild deterioration, and hydrocephalus was demonstrated. He improved following a low pressure ventriculo-atrial shunt and would follow objects with his eyes, would attempt to vocalise and was responsive in coordinated fashion to painful stimuli. This neurological improvement, however, was accompanied by progressive weight loss. Attempts to increase his nasogastric intake resulted in profuse vomiting despite anti-emetics, and the use of higher caloric nasogastric feeds (more than 1 Cal/ml) further worsened his vomiting and threatened aspiration vomitus. On March 28, six weeks after injury, having progressively lost weight from an initial 55 kg to 40 kg, he developed persistent vomiting. Physical examination showed abdominal distension, normal bowel sounds, an empty rectum and a functioning shunt with a concave scalp flap. Intravenous fluids were started, but vomiting promptly returned whenever nasogastric feeding was resumed in the ensuing week. Intravenous hyperalimentation was started with approximately 2000 Cal/24 hrs. A barium meal done on April 24 showed an abrupt vertical midline obstruction of the transverse duodenum with proximal bowel distension (Fig.). After correction of his hyponatremia and hypokalemia, surgery was performed.

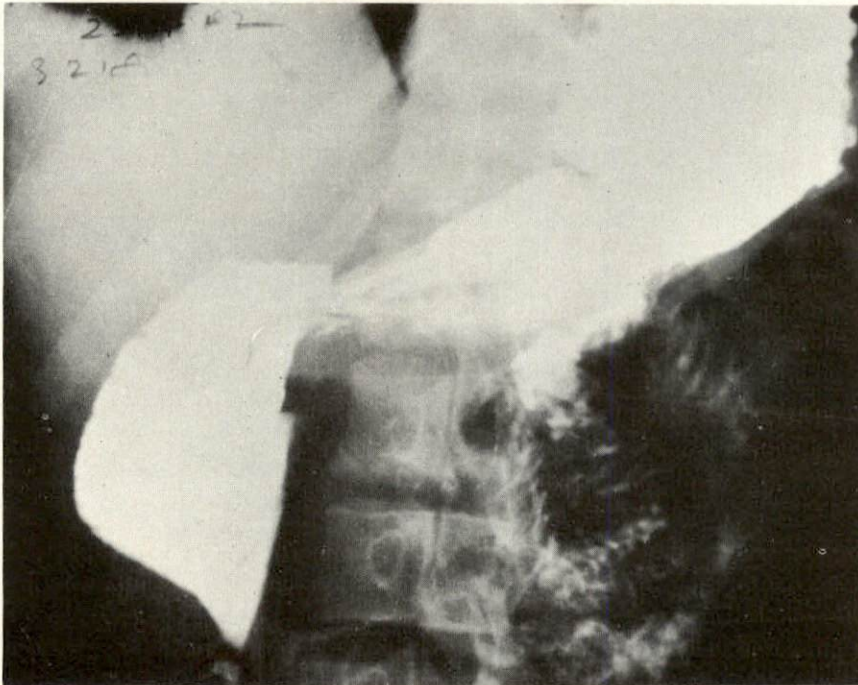


Fig. Barium meal showing an abrupt vertical midline obstruction of the transverse duodenum

At laparotomy, the dilated duodenum, obstructed by the superior mesenteric artery, was anastomosed to the collapsed jejunum. After resumption of nasogastric feeding of 2000 ml (1Cal/ml) per 24 hours he gained weight steadily and maintained a stable neurological state.

DISCUSSION

Although described Rokitansky in 1842, the first detailed account of this condition was by Wilkie in 1921. Vascular compression of the duodenum has been called a variety of names such as cast syndrome, chronic duodenal ileus, superior mesenteric artery syndrome, Wilkie's syndrome and arteriomesenteric duodenal compression (Barner and Sherman, 1963). The condition can still be regarded as uncommon since just over 300 cases have been reported to date, although some authors believe that it can be identified with greater frequency where clinicians and radiologists are aware of it (Goin and Wilk, 1956; De Fine Licht, 1956). Some even deny its existence (Cimmino, 1961). The patients have almost invariably lost weight. The associated diminution in retroperitoneal fat results in narrowing of the angle between the superior mesenteric artery and aorta as well as an increase in the anterior angulation of the duodenum as it emerges from the paravertebral gutter to cross the aorta. This causes obstruction of the transverse duodenum. The patient experiences bloating, epigastric discomfort and bile-stained vomiting which may be relieved by assuming the prone or lateral decubitus position.

Plain X-ray may suggest the diagnosis if it shows dilatation of the stomach and proximal duodenum or sometimes, especially in children, the double "bubble" sign (Burrington and Wayne, 1974). Barium meal shows a sharp, vertical, midline obstruction of the transverse duodenum due to extrinsic compression. Biplanar aortography has been recommended for accurate diagnosis (Mansberger et al, 1968) but is only rarely necessary.

Initially, in an effort to induce weight gain, a high calorie liquid diet can be administered with the patient in the prone or left lateral decubitus position under antispasmodic therapy. In our patient, however, any diet with more than 2000 calories resulted in profuse vomiting, and a high enough nasogastric caloric intake could not be obtained. Intravenous hyperalimentation can also be used. If these measures fail, as i

our case, surgical treatment offers the best hope. Duodenojejunostomy was first successfully performed for this condition by Stavely in 1908, though it was recommended a year earlier in 1907 by Bloodgood. Other operations such as division of the ligament of Treitz and gastrojejunostomy or jejunostomy are less popular, and repositioning of the duodenum to the right of the midline has been strongly advocated for pediatric cases (Burrington and Wayne, 1974). Gastrojejunostomy does not achieve direct decompression of the duodenum and is sometimes associated with persistent duodenal dilatation, recurrent symptoms or peptic ulceration (Goin and Wilk, 1956; Tyson and Keegan, 1951). Although jejunostomy will facilitate feeding, it is not a definitive procedure and does not achieve direct compression of the duodenum. Duodenojejunostomy, however, successfully bypasses the obstructed area and is regarded by most authors as the procedure of choice (Maingot, 1980; Lundell and Thulin, 1980; Appel et al, 1976).

Acute arterioesenteric duodenal compression has been described in patients following burns (Wallace and Howard, 1970), trauma (Wayne et al, 1971), immobilisation in body casts (Hall, 1974) and traction (Evarts et al, 1971). It is surprising that prior to 1973 (Bouzarth et al, 1973) it was never documented in a patient with severe brain injury since these patients are subjected to prolonged recumbency and weight loss. It is important that the neurosurgeon considers this condition in the differential diagnosis of vomiting in the patient with weight loss recovering from severe brain injury, and any surgeon or physician should also consider the condition in a patient with prolonged recumbency and weight loss.

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- Population Profiles – 20 Africa.** 1983, UNFPA, 43 pp, US\$5.00.

Book Reviews

Advanced Medicine 18

Edited by M. Sarnar, Royal College of Physicians of London, 1982, Pitman Books Ltd., London, 488 pages, £25.00

The contributions to this book are based on the 1982 Advanced Medicine Conference held at the Royal College of Physicians plus three major lectures and a clinicopathological conference. The subjects covered range from cardiovascular chest pain, antibiotics and infection, muscle diseases, therapeutic gastrointestinal endoscopy and radiology, the ageing nervous system, alcohol-related diseases through to C-reactive protein, liquid formation and absorption in foetal lung and natriuretic hormone.

I have great difficulty in understanding who could possibly find this book of review articles useful. The only potential reader is a trainee physician who needs to know what current topics to swot up for the MRCP (UK) examination. However, since for most West Indian physicians the DM examination of the University of the West Indies is more relevant to local practice, is an exit examination and of a higher standard, there is little need nowadays for UWI graduates to bother with the MRCP (UK). Thank goodness!

Perhaps the standard of the MRCP (UK) explains the quality of articles, writing and science which is disappointingly low in this book. I do not recommend it.

S. I. Terry, M.R.C.P.
Department of Medicine
U.W.I., Jamaica