Current Survey

STENOSIS DUE TO DUODENAL ULCERATION

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THE lumen of the first part of the duodenum is a comparatively narrow segment of the alimentary canal so that when the surgeon passes his finger along its length at operation he finds that its mucosa hugs him comfortably. Placing an ulcer into this canal and surrounding its crater by scar tissue would, one might think, produce stenosis in a high percentage of cases, yet the somewhat surprising fact is that obstruction due to duodenal ulceration is comparatively uncommon. When stenosis does occur, it is rarely produced simply by fibrous scar tissue. Usually obstruction occurs in the presence of active ulceration and a considerable part of the obstructive factor is produced by associated inflammatory oedema and pylorospasm. Indeed, if the ulcer heals under intensive medical treatment, the clinical features of the stenosis regress and there may even be long term relief of the obstructive symptoms (Brown, 1959).

Incidence

It is difficult to give a true incidence of this complication of duodenal ulceration. Figures vary with the criteria accepted for the diagnosis of pyloric stenosis and with the particular sample considered, since they may be based on autopsy or surgical series, or on the general hospital population, or on the peptic ulcer population as a whole.

At autopsy on patients with peptic ulcer, Hurst and Stewart (1929) recorded a 15.6%, and Portis and Jaffe (1938) an 11.8% incidence of stenosis. However it is difficult to assess the presence of duodenal obstruction at post mortem examination except in advanced cases. At operation on patients with duodenal ulcer, Moynihan (1912) reported a 19.5%, Berkman (1923) a 22.6% and Moody, Cornell and Beal (1962) a 28.9% incidence of stenosis. Here again their figures can be criticised since the patients submitted to surgery are, of course, selected, and one might argue also that the more complicated problems might, to some extent at least, be referred to surgical units with a particular interest in gastroenterological problems.

There have been some excellent reviews of the incidence of stenosis in peptic ulcer patients requiring hospital admission, although again these are somewhat selected groups. Haubrich (1963)

noted that 7% of 4,971 patients with duodenal ulceration admitted to hospital had some element of obstruction and this was in contrast with a 3.1% incidence of acute perforation and 16.1% of haemorrhage in the same series. Kozoll and Meyer (1964) found that 11.1% of 6,085 patients admitted with duodenal ulceration to the Cook County Hospital, Chicago had pyloric stenosis.

It is difficult to estimate the incidence of pyloric obstruction in the general peptic ulcer population, but Balint and Spence (1959) have calculated that this probably amounts to some 2% of the patients with peptic ulceration in North London.

The Metabolic Consequences of Duodenal Obstruction

The loss of gastric juice in the vomit, or even its sequestration within the dilated stomach, may produce extensive metabolic disturbances. These are not explained entirely by the loss of fluid and electrolytes in the gastric secretion since there are also extensive changes produced by alterations both in the urinary excretion of electrolytes and in renal function. These complex alterations have only been demonstrated comparatively recently by careful metabolic balance studies (Clark and Norman, 1964; Howe and Le Quesne, 1964; Parsons and Watkinson, 1954).

Initially there is loss of gastric fluids which contains chloride in excess of sodium and potassium, due, of course, to the hydrochloric acid present in gastric juice; hydrogen ion is also lost unless there is achlorhydria. The initial disturbance is therefore a fall in plasma chloride and a rise in plasma bicarbonate. At this early stage, the alkalotic tendency is compensated by renal excretion of sodium bicarbonate which maintains the pH within normal limits. At this phase, the dehydration results in diminished volume and concentration of the urine whose chloride content is first diminished and then disappears (in response to increasing chloride deficit) and whose reaction is alkaline.

If vomiting continues, a large sodium deficit now becomes manifest. This loss of sodium is partly accounted for by loss in the vomitus but it is mainly due to urinary excretion consequent upon the bicarbonate excreted in the urine as sodium bi-

carbonate. This sodium loss leads to shrinkage of the extracellular fluid volume which may indeed eventually produce peripheral circulatory collapse. As the plasma volume shrinks so too the glomerular filtration decreases, urea clearance falls and the blood urea commences to rise.

As the body's sodium reserves become depleted, hydrogen and potassium ions are substituted for sodium as the cations which are excreted with the bicarbonate. This results in the paradox that the patient with alkalosis in the advanced state now excretes an acid urine. The potassium loss in the urine, as in the case of sodium, is indeed greater than in the vomit.

Eventually the patient may develop tetany as a result of a shift of the ionised, weakly alkaline calcium phosphate to its un-ionised state, in attempted compensation for the alkalosis. The concentration of calcium ions in the plasma therefore falls although the total calcium concentration is not affected.

We may sum up the metabolic disturbances as follows:—

The patient is dehydrated and the haematocrit level is raised.

The urine is scanty, concentrated, initially alkaline but later acid; its chloride content is considerably reduced or indeed absent.

Serum chloride, sodium and potassium are all lowered and the plasma bicarbonate and urea are raised.

Clinical Picture

The diagnosis of obstruction due to duodenal ulceration is usually easy enough to make. There is often a long preceding history of typical peptic ulcer-type pain which we found to be 10 years or more in half our own patients. Every now and then a patient will present with only mild dyspepsia or even with painless vomiting which is found to be due to benign stenosing ulcer but he will prove to be a placid individual of low intelligence and a high threshold of pain. With advanced stenosis the character of the ulcer pain may change and may become more of a generalised upper abdominal discomfort which is relieved by a massive vomitus which may well be self-induced.

Vomiting is a common and important feature of stenosis. It is typically projectile, voluminous and may well contain food which is recognisable as having been eaten many hours, or even two or three days, previously.

If the vomiting is copious and frequent, there may be anorexia, loss of weight, thirst and weakness. Profound metabolic disturbances may rarely be accompanied by tetany, mental disturbance, convulsions or even coma.

TABLE 1

LENGTH OF HISTORY OF PAIN	
Under 1 year	5
1 - 4 years	14
5 - 9 years	19
10 - 19 years	28
20 years +	8
?	1
Total	75

Constipation as a result of dehydration and lack of roughage in the diet is a common complaint although occasionally diarrhoea is a feature, as stressed by Balint and Spence (1959); its aetiology is conjectural.

A history of previous gastrointestinal haemorrhage occurs in about a quarter of the patients with stenosis and it is not unusual for there to have been a preceding repair of a perforation.

On examination the patient with pyloric stenosis due to peptic ulceration is usually thin and indeed it is unusual to see a patient with this condition who is overweight. There may be signs of dehydration and electrolyte disturbance but fortunately these are not often encountered in modern practice. Abdominal examination may reveal a gastric splash which is of significance if detected three or four hours after the last meal or drink. As the stomach dilates, a visible swelling may be seen in the upper abdomen and peristalsis may be noted passing from the left to the right. Finally, the grossly dilated stomach, loaded down by its retained gastric contents, may not only be audible and visible but also palpable. The presence of a mass in the region of the pylorus is suggestive of a carcinoma and indeed occurs in about half the cases of obstructing pyloric cancer; however, rarely a duodenal ulcer may produce a large enough inflammatory mass to produce a tender palpable swelling.

The clinical diagnosis of pyloric obstruction can be clinched by passing a stomach tube following an over-night fast. A residue of 500 ml. or more of foul-smelling gastric fluid is quite typical.

The incidence of the salient clinical features of obstructing duodenal ulcer can be demonstrated by considering the last 75 consecutive examples of this condition undergoing surgical treatment at Westminster Hospital. There were 51 males and 24 females in this series and approximately half had a typical story of peptic ulcer pain for ten or more years (Table 1). However, in 5 patients the history was less than one year and in another 14 it was between one and four years. Vomiting was the

commonest presenting symptom, occurred in 68 patients (91%) and was typical projectile vomiting of large amounts in 57 of these. In addition, 34 of the patients noted stale food in the vomitus. Weight loss was a prominent feature and occurred in 59 patients (78%). Recent constipation was reported in 21 patients and diarrhoea in 3 cases. 18 patients had had previous episodes of gastro-intestinal bleeding, 5 had had a previous perforation repaired and one unfortunate man had had both of these complications on separate occasions.

On clinical examination 46 of the patients were thin or even emaciated (61%) and only 3 were obese. Abdominal examination revealed a gastric splash in 51 patients (66%) in 18 of whom, in addition, visible peristalsis was seen and in 8 of whom the gastric distention was great enough to produce an obvious palpable mass.

Radiological Findings

The radiological features of stenosis due to duodenal ulceration can be sub-divided into those indicating the presence of the obstruction and those which demonstrate its site and suggest its nature.

Gastric stasis may be hinted on the plain abdominal film if a large gastric shadow is seen which contains the typical patchy translucencies of retained food material. The erect film may show a large, high gastric fluid level.

Gastric outlet obstruction is suggested on the barium meal examination if a large gastric residue is present. This is demonstrated in the erect position by the presence of three media: a large gastric air bubble which overlies a thick layer of resting juice which, in turn, floats on the underlying pool of barium. In the early stages of stenosis the stomach demonstrates excessive peristalsis on screening but as the pathological process becomes more severe and the stomach enters into an atonic state these giant waves fade and the stomach appears relatively inert during the screening examination. Delay in emptying can be demonstrated on a four or six hour film using barium sulphate as the contrast medium; in pyloric obstruction there is more than 50% retention. Pylorospasm can be distinguished from organic stenosis by giving an intravenous injection of propantheline bromide which effectively relaxes the spasm.

When the site of obstruction is beyond the pylorus — and this is nearly always due to duodenal ulceration — the pyloric canal demonstrates abnormal mobility (Kreel and Ellis, 1965). During the screening of the duodenal cap region in the course of the barium meal examination the pyloric canal may dilate intermittently up to 2.5 cm. in diameter and even appear to be part of the gastric antrum; during further observation it is then seen to contract down again to its usual size. We have come to

regard this as a reliable sign of duodenal bulb obstruction.

That the stenosis is produced by duodenal ulceration is indicated first by the demonstration of the presence of an active ulcer crater, which is often of the penetrating type and most frequently occurs on the postero-superior aspect of the duodenum; second by the demonstration of scarring of the duodenal cap. This scarring may be central, which results in an irregular star-shaped appearance, or eccentric, with the formation of a pre-stenotic diverticulum of Akerlund, this latter being the more common appearance.

It is important, of course, (and by no means always an easy matter), to differentiate between obstruction due to a stenosing duodenal ulcer and a carcinoma of the pyloric region of the stomach. It is helpful to remember that malignant disease of the stomach very rarely encroaches radiologically upon the duodenal cap and furthermore that carcinoma is rare in association with active duodenal ulceration. Therefore, deformity proximal to the duodenal cap is strongly in favour of malignant disease whereas deformity of the cap itself suggests the presence of duodenal ulceration.

Pyloric obstruction in the adult is most commonly due to duodenal ulceration but is occasionally produced by pyloric or pre-pyloric benign ulcers. Second to this is carcinoma of the gastric antrum. Other causes are rare and include benign gastric tumours, adult pyloric hypertrophy (Keynes, 1965), the reticuloses (Cornes and Jones, 1962), ectopic pancreatic tissue at the pylorus (Tonkin, Field and Wykes, 1962) pre-pyloric mucosal diaphragm (Conway, 1965; Gerber, 1965), adhesions between the duodenum and either an inflamed gall bladder or the liver bed following cholecystectomy (Ger, 1964) and invasion of the duodenum from an adjacent carcinoma of the pancreas.

Differentiation between benign and malignant obstruction can often be made clinically. The patient with the benign peptic ulcer usually has a long history, and has a considerably dilated stomach (because the chronic obstruction has given this organ time to distend and hypertrophy). The patient with carcinoma typically has a relatively short history, may in fact have experienced no pain at all and will frequently demonstrate an abdominal mass at the pylorus without evidence of gross dilatation of the recently obstructed stomach. Certainly exceptions occur to these generalisations and it may not be possible to be certain of the diagnosis even after careful radiological investigation.

The pre-operative diagnosis of the rarer conditions producing obstruction which we have listed above is usually only possible radiologically and even then may not be at all accurate.

Treatment

The treatment of advanced mechanical obstruction due to a stenosing duodenal ulcer is inevitably surgical but must be preceded by careful medical preparation; the more severe the obstruction the longer one must be prepared to spend on this. The aims of the medical treatment are to decompress the dilated stomach and to restore its normal peristalsis, to control ulcer activity, to correct the fluid and electrolyte balance and to improve the patient's general condition. Once the stomach has been decompressed by twice-daily gastric aspiration and saline lavage, and once the ulcer has begun to heal under strict medical treatment, the surrounding inflammatory oedema and associated gastritis subside, the obstruction lessens and soon considerable amounts of fluid can be taken and absorbed readily by mouth. Sieved foods are allowed but solid material is forbidden since particles will simply clog up the narrowed duodenal lumen.

If the patient is not dehydrated and his blood biochemistry is normal, it is sufficient to give copious fluids orally, but if electrolyte depletion has occurred then intravenous therapy is necessary and depends primarily upon replacement with isotonic saline. This restores the extra-cellular fluid volume and hence produces a normal renal blood flow. It provides chloride in excess of its plasma concentration, thus replacing the chloride deficit, and the sodium content allows the kidney to excrete an alkaline urine. Potassium chloride is required in addition in the severe case.

Attention is paid to the correction of anaemia, if necessarily by blood transfusion, carious teeth are removed and ascorbic acid prescribed, since these patients are often vitamin C depleted in consequence of their long period of restricted diet.

Usually seven to ten days are required in this preliminary phase of preparation.

The conventional operation for stenosing duodenal ulcer is a high partial gastrectomy with gastro-jejunal reconstruction. Although vagotomy combined with gastric drainage has become a popular procedure in the treatment of duodenal ulceration it has often been condemned when pyloric stenosis exists (Bergin and Jordan, 1959; Harper, 1966; Kraft, Fry and De Weese, 1964; Mialaret, 1964). It has been argued that the decompensated stomach musculature might be more prone to the atonic effect of gastric denervation and that prolonged gastric stasis is likely to result. However, Feggetter and Pringle (1965) found that vagotomy and gastro-jejunostomy produced similar satisfactory long-term results both in uncomplicated cases of duodenal ulceration and in those with stenosis. We have carried out vagotomy and either pyloroplasty or gastro-jejunostomy in the last 32 consecutive

cases of gross pyloric stenosis due to duodenal ulcer with uniformly good immediate and late results. In no case had post-operative gastric retention occurred and none required post-operative gastric aspirations. All had good functional results and long-term subsequent barium meal studies in the first 21 of these patients (Ellis, Starer, Venables, and Ware, in press) showed return of the dilated stomach to normal size and tone with efficient emptying through the drainage stoma.

Gastro-jejunostomy alone, which was once the standard procedure advised for pyloric stenosis (Reid and Marcus, 1948), still has a part to play in the treatment of the frail, elderly and ill patient.

I would like to thank my colleagues at Westminster Hospital for access to their notes of patients with pyloric stenosis.

REFERENCES

- BALINT, J. A., and SPENCE, S. (1959): Pyloric Stenosis, *Brit. med. J.*, i, 890.
- BERGIN, W. F., and JORDAN, P. H. (1959): Gastric Atonia and Delayed Gastric Emptying after Vagotomy for Obstructing Ulcer, *Amer. J. Surg.*, 98, 612.
- BERKMAN, D. M. (1923): Gastric Retention: its Treatment and Surgical Mortality, *Coll. Pap. Mayo Clin.*, 15, 92.
- BROWN, C. H. (1959): Duodenal Ulcer Complicated by Obstruction: Results of Medical Treatment, *Amer. J. Dig. Dis.*, 4 (n.s.), 940.
- CLARK, R. G., and NORMAN, J. N. (1964): Metabolic Alkalosis in Pyloric Stenosis, *Lancet*, i, 1244.
- CONWAY, N. (1965): Pyloric Antral Mucosal Diaphragm, *Brit. med. J.*, i, 970.
- CORNES, J. S., and JONES, T. G. (1962): Leukaemia Lesions of the Gastrointestinal Tract, *J. clin. Path.*, 15, 305.
- ELLIS, H., STARER, F., VENABLES, C. W., and WARE, C. C. (in press): Vagotomy and Gastric Drainage in the Treatment of Pyloric Stenosis, *Gut*.
- FEGGETTER, G. Y., and PRINGLE, R. (1965): The Relationship Between the Severity of Duodenal Ulceration and the Results of Bilateral Vagotomy and Gastro-jejunostomy, *Brit. J. Surg.*, 52, 691.
- GER, R. (1964): Postoperative Extrinsic Pyloric Stenosis, *Brit. med. J.*, ii, 294.
- GERBER, B. C. (1965): Prepyloric Diaphragm: an Unusual Abnormality, *Arch. Surg. (Chic.)*, 90, 472.
- HARPER, F. B. (1966): Gastric Dysfunction after Vagotomy, *Amer. J. Surg.*, 112, 94.
- HAUBRICH, W. S. (1963): Obstruction at or near the Pylorus. In Bockus, H. L. (Ed.) *Gastroenterology* (2nd ed.) Vol. 2. Philadelphia: Saunders.
- HOWE, C. T., and LE QUESNE, L. P. (1964): Pyloric Stenosis: the Metabolic Effects, *Brit. J. Surg.*, 51, 923.
- HURST, A. F., and STEWART, M. J. (1929): Gastric and Duodenal Ulcer. London: Oxford University Press.
- KEYNES, W. M. (1965): Simple and Complicated Hypertrophic Pyloric Stenosis in the Adult, *Gut*, 6, 240.
- KOZOLL, D. D., and MEYER, K. A. (1964): Obstructing Gastroduodenal Ulcers, *Arch. Surg. (Chic.)*, 88, 793.
- KRAFT, R. O., FRY, W. J., and DE WEESE, M. S. (1964): Post-vagotomy Gastric Atony, *Arch. Surg. (Chic.)*, 88, 865.

- KREEL, L., and ELLIS, H. (1965): Pyloric Stenosis in Adults: a Clinical and Radiological Study of 100 Consecutive Patients, *Gut*, 6, 253.
- MIALARET, J. In WEISS, A. G., and HOLLENDER, L. F. (1964): (Ed.) *La Vagotomie dans l'Ulceré Gastro-Duodeno-Jejunal*. Paris: Expansion Scientifique Francaise.
- MOODY, F. G., CORNELL, G. N., and BEAL, J. M. (1962): Pyloric Obstruction Complicating Peptic Ulcer, *Arch. Surg. (Chic.)*, 84, 462.
- MOYNIHAN, B. G. A. (1912): *Duodenal Ulcer* (2nd ed.). Philadelphia: Saunders.

- PARSONS, F. M., and WATKINSON, G. (1954): The Treatment of Pyloric Stenosis in Peptic Ulceration, *Postgrad. med. J.*, 30, 145.
- PORTIS, S. A., and JAFFE, R. H. (1938): Study of Peptic Ulcer Based on Necropsy Records, *J. Amer. med. Ass.*, 110, 6.
- REID, H., and MARCUS, R. (1948): Posterior Gastroenterostomy for Organic Pyloric Stenosis, *Brit. J. Surg.*, 35, 304.
- TONKIN, R. D., FIELD, T. G., and WYKES, P. R. (1962): Pancreatic Heterotopia as a Cause of Dyspepsia, *Gut*, 3, 135.

Case Reports

REVERSED INTESTINAL ROTATION

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DEVELOPMENTAL anomalies of the colon resulting from a derangement of intestinal rotation are not uncommon and include nonrotation, malrotation and failure of descent of the caecum. Reversed intestinal rotation, in which the transverse colon lies below the superior mesenteric artery, is probably the rarest and most interesting, for in 19 of the 28 recorded cases symptoms of the disorder were delayed well into adult life.

Case Report

The patient, a married woman aged 33 years, was admitted to hospital in September 1963, complaining of recurrent attacks of pain and vomiting of 10 years' duration. The attacks of pain and vomiting usually continued for several days during which time there would be absolute constipation and abdominal distension. She had 8 siblings, none of whom suffered with gastrointestinal complaints and had 4 children of her own who were alive and well.

On examination she was a thin woman who was not anaemic. The abdomen was not distended and the abdominal musculature seemed abnormally lax. In the umbilical region there was a large defect in the rectus sheath without actual herniation of peritoneal contents. No masses were felt in the abdomen, but it was noted that there was a fullness in the right iliac fossa. The bowel sounds were hyperactive and obstructive in character.

Special Investigations: Hb 80 per cent; W.B.C. 9,600/cu. mm.

Barium meal showed a normal stomach outline and a large diverticulum of the first part of the duodenum. No abnormality was detectable in the follow-through examination.

Operation: A laparotomy was performed through a right paramedian incision. An immediate striking feature on opening the peritoneum was gross varicosities of the mesenteric and omental veins. There was a volvulus of the ileum, caecum and ascending colon turning in a clockwise direction through 360 degrees (Fig. 1C.), and multiple adhesions between the loops of small bowel. The transverse colon and hepatic flexure could not be identified. Beyond the volvulus, the colon was seen to pass in a tunnel behind the third part of the duodenum and behind the superior mesenteric vessels, appearing at the left side

FIG. 1.—(a) normal intestinal rotation, (b) reversed intestinal rotation, (c) operative findings in the case described.