X-RAY APPEARANCES OF GASTRIC, DUODENAL AND JEJUNAL ULCER

by H. C. H. Bull, M.A., M.B., B.Ch., M.R.C.P.(Lond.)
Hon. Radiologist, Southend General Hospital

A gastric ulcer is first an erosion of the mucous membrane which subsequently penetrates the deeper layers of the submucosa, muscle and peritoneum. Radiology is shadow diagnosis and so the burrowing and eroding ulcer becomes to us a niche or projection from the clearly defined stomach wall demonstrated in shadow by the contrast medium filling it.

Of gastric ulcers 90 per cent. are found on or about the lesser curvature above the incisura angularis. The lesser curvature differs anatomically from the rest of the stomach, and is the direct pathway of food and fluids entering, but just why it is the seat of ulceration is not known.

Of the remaining 10 per cent. some are found on the lesser curvature between the incisura and pylorus; the others—and they are few, probably less than 1 per cent.—are on the anterior wall of the pyloric end of the stomach.

Simple ulcers have been recorded on the greater curvature. I have not seen one other than a malignant ulcer, but there are a few published cases.

The outstanding fact and diagnostic feature—the 'direct evidence' of gastric ulcer—is the niche seen in profile or tangential view projecting outwards from the cavity of the stomach illustrating in shadow picture the identical form seen in post-mortem pathology (Fig. 1).

These gastric ulcers may be small or large, recent or of long standing. The size is some estimate of chronicity, but any size above one inch diameter carries with it a suggestion of malignancy. Despite the work, pathological and surgical, devoted to study of the early malignant changes in gastric ulcer there is direct evidence that chronic gastric ulcers often develop malignant change. When large the ulcer is usually old and certainly penetrating in the sense that it has broken through and passed beyond the natural boundaries of the stomach. Once an ulcer has reached the peritoneum, established adhesions between it and an adjacent viscus, usually the pancreas or the liver, and broken down the peritoneal boundary, erosion proceeds rapidly and the crater becomes a large cavity of irregular outline. Such an ulcer tends to show a relatively narrow passage at the point of exit through the peritoneum, expanding into a large cavity beyond.

In shape gastric ulcers vary a good deal according to chronicity and position. The small ulcer projects from the lesser curvature as a blunt point or a rounded bud and its outline is relatively smooth. The larger ulcer which has penetrated through the peritoneum will often show a fluid level with a gas bubble above when the patient is in the erect position.

An ulcer situated between the incisura and pylorus on the lesser curvature has the same general characters as that above the incisura but, as gravity plays a part, it does not hold barium so well in the erect position. Examined under the screen, barium can be pushed up into the crater, but when the pressure is released a certain amount only sticks on to the rough surface, and is better demonstrated on the films taken with the patient prone.

Carman, in 1920, described the saddle ulcer lying astride the lesser curvature between incisura and pylorus, an ulcer with rolled edges holding gutters of barium when pressure was applied so that the appearance was that of detachment. To this he gave the name 'meniscus' and he said it was always malignant. Such an ulcer is not common. Even a simple ulcer often has thick rolled edges, thus retaining barium, and it has been my experience that an innocent ulcer can show the meniscus sign. If we add to Carman's dictum that the meniscus sign in an ulcer of one inch or more diameter spells malignancy, then we are approaching nearer the truth.

Thick rolled edges, to which mention has been made in connection with malignancy, is a feature of chronic ulcer and not necessarily a sign of malignancy.

The radiologist, particularly whilst screening, may find many indirect signs or accompaniments of gastric and duodenal ulcer useful as pointers to show that he is on the trail of an abnormal variation. Such may be:

1. Excess secretion.
2. Thickening of the mucous membrane.
3. Spasm.
4. Alterations of peristalsis.

1. Excess Secretion. Normally the amount of resting fluid in the empty stomach is so small it can and does pass unnoticed when the initial bolus of barium tracks down the lesser curvature to pool in the most dependent part of the body of the stomach. For satisfactory radiological examination of it, the stomach should be physiologically empty and to ensure this the patient must neither eat nor drink during the prior 12-hour period. There are always some patients unwilling or
**Fig. 1.**—Large penetrating ulcer of the lesser curvature with spastic contraction of the greater curvature opposite.

**Fig. 2.**—Ulcer near the lesser curvature with marked spastic incisura.

**Fig. 3.**—Ulcer of the pyloric end of the stomach.
2. The next of the indirect signs is thickening of the folds of mucous membrane. We still do not know the real meaning of thick folds of mucous membrane, but the gastroscope suggests that they are inflammatory in nature. The folds are always more or less thickened in the presence of ulcer of the stomach or duodenum, but often occur in conditions in which no ulcer can be demonstrated.

3. Spasm. Ulcers are inflammatory, and spasm is the natural answer of unstriped muscle to inflammation, spasm is nearly always found with an ulcer. But since the stomach may go into spasm on behalf of its remote friends—gastritis, mental inability to cooperate who will surreptitiously eat or drink within that time. When the radiologist finds excess fluid he has to judge between physical and mental pathology since these sinners do not always confess. Apart from food or fluids introduced within the forbidden limit the amount of resting fluid should be insignificant. Excess fluid means pyloric obstruction, gastritis, prolonged alkaline treatment, duodenal ulcer with partial obstruction or delay in the small intestine. It becomes an indirect sign of gastric ulcer because of the associated gastritis, or because it follows reflex pyloric spasm.

Fig. 4.—Hyperperistalsis of the stomach with ulcer of the duodenum.
bladder, kidney, appendix and colon as well as on its own account, this sign is in no way diagnostic.

Spasm accompanying gastric and duodenal ulcer takes many forms. The simplest and most helpful is the local indrawing of the greater curvature opposite an ulcer on the lesser curvature pointing fingerlike to the crater (Fig. 2). The spasm is not always like that but there is nearly always some degree of contraction, shallow or deep on the greater curvature when an ulcer is situated on the lesser curvature above the incisura. Spastic contraction of the pyloric end of the stomach is common, but only rarely does it mean an ulcer of the pyloric end of the stomach (Fig. 3). It may accompany an ulcer in the body of the stomach away from the pylorus or an ulcer of the duodenum. Even more often the pyloric end of the stomach contracts because of some peritoneal irritation not gastric in origin. True pyloric ulcers are a rarity and there is no difference in the type of spasm to distinguish its origin. But when due to an intrinsic lesion it is more fixed and less yielding to the persuasion of massage or Belladonna. Spasm of the longitudinal and oblique muscle bands can occur and it causes foreshortening of the stomach dividing it into two loculi at different levels. Thus the barium enters the upper loculus and spills over, cascading into the lower. This form of spasm is nearly always reflex.

4. Peristalsis. Peristalsis is disturbed whenever there is gastritis from any cause. Deep and frequent peristaltic waves to be seen in the stomach at the same time is called hyperperistalsis and is usually a sign of partial pyloric obstruction of which duodenal ulcer is the commonest cause (Fig. 4).

**Duodenal Ulcer**

The direct sign of ulcer of the duodenum is deformity of the first part which from its shape and smooth round outline is called the cap (Fig. 5). There are two causes of deformity—peristalsis from within and inflammation from without. Ulcer, whether shallow or deep, recent or chronic, gives rise to a characteristic regional contraction which is in part spastic and in part fibrotic and permanent. The vastly greater number of deformed duodenal caps are due to ulcer from within; the few which are deformed by inflammation from without come from adhesions, sometimes
Fig. 7.—Duodenal ulcer, deformity of the cap, the projecting niche may or may not be the ulcer crater.

Fig. 8.—Gastric and duodenal ulcers.

Fig. 9.—Large jejunal ulcer adjacent to the stoma with gastritis on the proximal and jejunitis on the distal sides.
to an adjacent pathological gall-bladder, sometimes residual from operation or peritonitis.

The deformity assumes many forms from simple linear spastic contraction of the wall to total destruction of shape so that the term of cap no longer fits (Fig. 6). Most positive when it can be demonstrated is the ulcer crater, but it must be demonstrated in two views at right angles to each other, and this can only be done in a few selected cases. The irregularities of outline of a deformed cap are not necessarily craters and should not be accepted as such (Fig. 7).

The indirect signs are:—
1. Thickening of the mucous membrane of the duodenum and of the stomach.
2. Irritability of the duodenal cap.
3. Excess secretion in the stomach.
4. Dilatation of the stomach with hyperperistalsis.

1. Thickened mucous membrane. Besides being thrown into folds by contraction the mucous membrane of the duodenal cap is thick and the folds coarser and deeper than those of a normal cap. In this the stomach suffers, too, as if from common cause and invidious selection. Gastric and duodenal ulcers do occur together but are uncommon (Fig. 8). But to write of a common cause is a loose expression since we have no reasonable knowledge that gastritis or deep folds of mucous membrane have anything to do with the cause; it may equally well be due to treatment, such as habitual alkalinization.

2. Irritability. The normal duodenal cap when filled holds barium for a few seconds before spilling or syphoning over into the second part. The irritable duodenal cap contracts quickly and frequently. Irritability is a sign of inflammation and in the presence of a deformed cap usually means active ulcer.


4. Dilatation and Hyperperistalsis. Excess secretion may be just an expression of gastritis, but its importance is as a sign of partial obstruction. Progressive obstruction of the outlet of the stomach from fibrotic contraction of an ulcer with oedematous swelling of the mucous membrane is, perhaps, the gravest complication of ulcer but one that can be cured by surgery. Where the excess of gastric secretion is due to obstruction it is recognized by the difficulty of forcing barium through the pylorus under the screen, and by the frequent and deep waves of peristalsis, best seen in films taken with the patient prone. In these cases the stomach appears to the eye larger than normal, and indeed is so when absolute obstruction is approaching. Such stomachs may be three times the normal size and retain half a meal for 24 hours.

3. Post-operative Ulcers. Ulcers may recur in the stomach or duodenum after local excision or cautery and a small number develop about the stoma of a gastroenterostomy within the first 12 months of operation. I have never seen an ulcer in the stomach or jejunum after successful partial gastrectomy.

4. Gastrojejunal Ulcer. Gastrojejunosotomy, popular 25 years ago, has not proved so universally satisfactory in the long run. One reason, statistically the least, is the appearance of a new ulcer in or about the stoma. These gastrojejunal ulcers develop within a few months of the operation and persist with remissions and relapses, adhesions to adjacent viscera and sometimes perforate into the colon.

Radiologically they are difficult to detect, the direct sign we want—the ulcer crater—is most difficult to identify with certainty. I used to depend on the niche of barium near the stoma which retained its relationship in all positions, could not be displaced, and was locally tender. But I have made, and seen, mistakes and do not now think the diagnosis of ulcer justified on this evidence unless the niche can be seen projecting from, in continuity with, the barium filled stomach or jejunum (Fig. 9). This applies to the smaller ulcers; large ulcers are easier to identify but not so common to meet.

The indirect signs of gastrojejunal ulcer are the same as for gastric and duodenal ulcer—spasm and thickening of mucous membranes. Spasm of the stomach around the stoma may cause delayed passage—the obstruction appearing at first but relaxing later. Spasm of the jejunum beyond the stoma is seen as a narrow contraction alternating with the spike-like appearance associated with jejunitis.

Thickening of mucous membrane of stomach and duodenum accompanies gastrojejunal ulcer, but is not really a significant sign since we find it to some degree in all cases of gastrojejunosotomy.

Conclusion
In the radiological diagnosis of gastrojejunal ulcer we are conscious of our weakness and the necessity of checking carefully against the clinical evidence. With gastric and duodenal ulcers there is more confidence and even a feeling by some that the X-ray findings alone will stand against the world. It is, indeed, true that in most cases of diagnosis the clinician agrees, however modestly retreatting from the limelight. If he does not agree, maybe the clinician is wrong, or the radiologist is wrong; in either case we are backing human judgment—and the camera can lie. One thing is certain, if the clinical and radiological findings do not agree, one is at fault and both should be rechecked.