Mucosal Folds in the Upper Gastro-intestinal Tract

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SUMMARY

Scrutiny of the mucosal folds in barium-meal examinations can be rewarding not only from the viewpoint that they are irregular or destroyed but whether they are substantially increased or decreased in prominence yet regular in outline.

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Although the mucosal folds of the stomach and duodenum are always scrutinized during any barium examination, comments have been limited to their increased or decreased prominence. In recent years a little more has been learnt of their significance in terms of physiology and pathology. The important fact is that the folds do not generally mirror the condition of the mucosal lining but merely reflect the activity of the muscularis mucosae. One exception to this is in the small bowel where enlargement of the villi can be identified separately from the appearance of the folds themselves.[1]

INCREASED PROMINENCE OF GASTRIC FOLDS

Earlier radiologists always labelled this appearance 'gastritis', meaning a chronic variety. Having regard to what histologists will accept as chronic gastritis, it is now known that there is no correlation between the X-ray and the gastroscopic findings of increased prominence of gastric folds on the one hand and histological evidence of chronic gastritis on the other.

Have these enlarged folds any pathological meaning at all? The radiologist now tends to call such a finding 'hypertrophic gastritis' and some have been chance findings at a barium meal and correlated with a constant clinical picture. However, there are others accompanied by a hypoproteinaemia and a hypokalaemia and these fall into the category of Menetrier's disease.[2] This is supposedly a specific entity with a particular anatomy and histology confined to the mucosa and rare before 20 years of age. The whole gastric mucosa except for a narrow band along the lesser curve can be involved. The wall can attain a thickness of 25 mm and the mucosa 5 mm. The glands are more numerous and longer but not usually invasive of the muscularis mucosae. X-ray features are:

1. Gross enlargement of the rugae which are not rigid, nodular or ulcerated, unlike lymphoma or polypoid carcinoma.
2. Thickened gastric wall.
3. Gross excess of mucus which is readily noted in the barium shadow because of the lack of an even coating of the mucosal surface. This condition has been known to disappear spontaneously.[3]

The abundant mucus secretion explains the chemical findings and a similar feature is noted with the villous adenoma. This lesion is better known in the colon but it occurs also in the stomach and duodenum. It can often be recognized radiologically by the fact that barium is trapped between the fronds on the surface of the adenoma.

The enlargement of the gastric folds can follow artificial histamine stimulation of acid output and it has been found that in the hands of one group,[4] assessment of the prominence of the mucosal folds in the stomach and duodenum can be as accurate as chemical laboratory tests of acid output. This accords with experience that the observation of prominent gastric and duodenal loop folds on a routine barium meal can be a sign of gastric hyperacidity.

In an extreme degree this can pinpoint the Zollinger-Ellison syndrome where the mucosal folds are very large, extending even into the upper jejunum, and there is an excess of resting fluid. In addition, there may be a steatorrhoea showing as a malabsorption pattern in the small bowel. What makes the diagnosis probable is the presence of recurrent peptic ulceration, especially if the duodenal loop is involved.

Some observers[5] have gone further and attempted to assess whether a severe pyloric obstruction is due to peptic ulceration or neoplasm. They note that if in the presence of gastric dilatation with fluid excess enlarged mucosal folds can be seen in the body of the stomach, this indicates excess acid output and hence peptic ulceration. Carcinoma at the pylorus would not be associated with excess acid output and the gastric folds would not be enlarged.

Uraemic patients on long-term dialysis have shown prominent gastric folds with some fluid retention in the stomach.[6] Also involved are the duodenal bulb and upper portion of the loop with no evidence of peptic ulceration. There is no excess acid output noted. In one series the occurrence of acute pancreatitis has been stressed and the increased prominence of duodenal folds may be due to inflammation of the adjacent tissues.

There has always been a tendency to interpret enlarged mucosal folds in the gastro-intestinal tract as being due to oedema. In the case of the postoperative stomach, particularly, it would be useful to be sure of the presence of local oedema since this is sometimes the only indication of anastomotic peptic ulceration. Such ulcers are very difficult to find because of the altered anatomy, unless they are large.

It is noted that normally there is an approximate equality in the width of the raised folds and the barium-filled valleys between them. When the raised folds are much wider and the valleys are very much narrower, this is very probably due to oedema. In the extreme case the valleys are almost obliterated and the mucosal surface is coated with a sheet if the barium adheres. It is noted that in the so-called hypertrophic gastritis the relative widths of folds and valleys are nearly equal. The difficulty arises with the recognition of very mild degrees of oedema on these criteria since lymphoma has to be differentiated and hence some oedema due to lymphatic blockage may also be present. There are also causes other than oedema and lymphoma, e.g. amyloid deposits.

DECREASED PROMINENCE OF GASTRIC FOLDS

The opposite condition of decreased prominence of gastric folds is much more accurately correlated with chronic atrophic gastritis and its end-stage, gastric atrophy. The former shows cell infiltration of the whole lamina propria, atrophy of glandular tubules, flattened surface cells and intestinal metaplasia. The latter shows mucosal atrophy without cell infiltration and is complete in pernicious anaemia.

Gastric changes of any degree are very rare before 30 years of age and severe changes are uncommon before 50 years. Atrophic gastritis occurs in 30% of patients over 50 years coming to autopsy. Age is among the most important causes.

X-ray features are consistent. The configuration of the body of the stomach is that of a tube with parallel sides disposed vertically. The mucosal folds along the greater curve are absent or diminished. The fundus is described as 'bald' in that the gastric folds are very inconspicuous there. In the fundus and body they are very thin.

The interpretation of the X-ray appearances of the folds is that they are thrown up by contractions of the underlying muscularis mucosae. In pernicious anaemia there can be atrophy of the muscle coat and this is possibly an explanation. Gastric atrophy may precede development of pernicious anaemia and conversely pernicious anaemia without gastric atrophy may indicate a non-Addisonian pernicious anaemia.[7]

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