DEFINITION

1. The term **peptic ulcer** is used to describe gastrointestinal ulceration of mucosal surfaces which are exposed to gastric acid and pepsin secretion.

2. Some authorities still consider peptic ulcers in terms of their most typical anatomical location and discuss the condition as two separate entities, namely duodenal ulcer and gastric ulcer. This practice reflected the former belief that the two processes had distinct aetiologies and pathognomonic clinical patterns.

3. However while there remain epidemiological, clinical and management differences between the two, increasing evidence suggests there is a basic injurious process which is common to both gastric and duodenal ulcers.

4. Peptic ulceration is not confined to the stomach or duodenum but may occur at many sites. The majority of peptic ulcers occur in the stomach or proximal duodenum but other sites include the lower end of the oesophagus, the jejunum, where there is a gastroenterostomy stoma and the ileum near a Meckel's diverticulum with its heterotopic gastric mucosa. In the Zollinger Ellison Syndrome where there is gastric hypersecretion, multiple ulcers may develop throughout the duodenum and jejunum.

5. Peptic ulcers may be acute or chronic. Histopathologically, acute peptic ulcers are confined to the mucosa and submucosa. Chronic ulcers penetrate into and often through the muscularis propria.

ACUTE PEPTIC ULCER

6. This condition occurs most commonly in the stomach but if it is severe the duodenum may also be involved. Pathologically, there is a progression from acute haemorrhagic gastritis through gastric erosions, to multiple small peptic ulcers, which are usually less than 1 cm in diameter.

CLINICAL MANIFESTATIONS

7. Acute peptic ulcers commonly present as acute severe dyspepsia with epigastric pain, acid reflux and waterbrash. Haemorrhage is often considerable, even life-threatening, and this may be the first manifestation. They may also present as an acute peritonitis.

AETIOLOGY

8. Acute peptic ulcers usually arise in relation to acute gastritis secondary to therapeutic drugs (especially the non-steroidal anti-inflammatory group). They may also occur in patients severely ill from burns, sepsis, trauma, surgery or rapidly progressive intracranial disease.

CHRONIC PEPTIC ULCER

9. The incidence in the UK is falling. It remains however a very common condition which is estimated to affect 10% of the UK population at some time in their lives. The disease falls into two main groups based on the clinical findings, pathology and epidemiology.

    9.1 Ulcers occurring in the first part of the duodenum and the most distal part of the gastric antrum. Typically the affected individual is male, under 50 and of a higher socio-economic class. Often these ulcers are multiple.

    9.2 Ulcers of the proximal part of the stomach, most often on the lesser curvature. These are solitary ulcers which affect men and women equally and are commoner in the older age groups and lower socio-economic groups.

    9.3 A third overlapping group is recognised with features of both of the above groups.

PATHOLOGY

10. The macroscopic appearance of a chronic peptic ulcer is similar regardless of its site. It is a well demarcated, oval or round punched out ulcer ranging in size from 2 to 10 cm. The base of the ulcer may be fibrosed and the surrounding mucosa typically flat and atrophic. This surrounding mucosa often shows histological features of active chronic gastritis or duodenitis. When healing of the ulcer occurs there is replacement of the normal epithelium by immature glandular epithelium. Specialized epithelial cells and the muscularis mucosae do not regenerate.

CLINICAL MANIFESTATIONS

11. A chronic peptic ulcer, regardless of its site, may produce symptoms of dyspepsia with pain, heartburn, waterbrash and nausea. Endoscopic studies have shown that in a considerable proportion of chronic peptic ulcers no dyspeptic symptoms and signs occur. The condition may present with one of its complications.

12. The disease is one of relapse and remission. Numerous studies have shown that even without specific therapy, 40% of peptic ulcers will remit spontaneously in 6 weeks although there will be a recurrence rate of about 2/3 within a year.

13. The principal complications of chronic peptic ulcer are:

    13.1 Haemorrhage. This may take the form of a chronic slight ooze giving rise to iron deficiency anaemia. If the bleeding is acute, the medical emergency of haematemesis and melaena may result. Ulcers located in the duodenum bleed acutely more frequently than those in the stomach.

    13.2 Perforation. This occurs more commonly in ulcers of the duodenum or pyloric antrum and leads to a chemical, and subsequently a bacterial, peritonitis. Clinically this presents as a classic acute abdomen with pain rigidity, rebound, absent bowel sounds and shock. In the elderly, silent perforations may occur.

13.3 Penetration, when the ulcer erodes to an adjacent viscus. Most commonly the pancreas is involved leading to a low grade pancreatitis. Gastro-colic or duodeno-colic fistulae may occur and there may be invasion of the splenic artery with haemorrhage.

13.4 Fibrosis, oedema and muscular spasm leading to stenosis of the gastric or duodenal lumen. This may produce pyloric stenosis, duodenal stenosis or an hour-glass stomach.

13.5 Carcinoma. This has been a controversial issue. There is no evidence that chronic peptic ulcers occurring in the duodenum become malignant. The situation with respect to gastric ulcers is less clear. This reflects the difficulty in distinguishing an ulcer which has become malignant from a malignant tumour which has become ulcerated. Authoritative opinion holds that a carcinoma may occasionally arise at the margin of a gastric ulcer which has been present for more that 5 years. The risk of this occurring is less than 1%.

MANAGEMENT

14. The treatment of chronic peptic ulcer may be followed by a number of complications. Nowadays peptic ulcer is usually managed medically. In the event of acute complication or failure of medical management, surgical intervention may be appropriate. In the past surgical procedures have been more frequently performed.

15. The three main surgical procedures have been:

15.1 Vagotomy (including selective vagotomy)

15.2 Vagotomy with drainage (such as gastro-enterostomy)

15.3 Gastrectomy.

16. Following all forms of surgery there may be a recurrence of the ulcer. Diarrhoea may follow any surgical procedure.

17. Partial gastrectomy may be followed by the dumping syndrome. If this occurs soon after food, it is due to hypovolaemia as a result of rapid emptying of food from the gastric remnant into the intestine. Late dumping is caused by hypoglycaemia.

18. The late sequelae of partial gastrectomy include weight loss, iron deficiency anaemia, macrocytic anaemia (due to vitamin B12 and folate deficiency), mild steatorrhoea and occasionally osteomalacia. There is a slightly increased incidence of carcinoma in the gastric remnant.

AETIOLOGY

19. The aetiology and pathogenesis of chronic peptic ulcer is poorly understood. Ulceration results from a disturbance in the balance between factors that damage the mucosa, mainly gastric acid and pepsin, and the integrity of the local defence mechanism, which is maintained by the mucous-bicarbonate barrier and the surface epithelial cells. The evidence is that the relative importance of these factors is variable.

20. In general terms, gastric acid hypersecretion is a major factor in duodenal and pre-pyloric ulcers while in gastric ulcers the limiting factor is the mucosal defence system.

21. The constitution of affected individuals is also important. Family studies show that first degree relatives of peptic ulcer patients are 2 to 3 times more likely to have peptic ulcer disease than the general population. This suggested genetic predisposition is also consistent with the observed positive association between chronic peptic ulcer and blood group O and ABO(H) non-secretor status.

22. In addition to the role played by internal mucosal irritants, it might be thought that the ingestion of external mucosal irritants would lead to a chronic peptic ulcer. There is a causal association between chronic peptic ulcer and the prolonged ingestion of analgesics, including non-steroidal anti-inflammatory drugs. Other ingested substances may be important in some individuals but a causal association has not been proved. Dietary studies have been inconclusive.

23. Cigarette smoking causes delay in the healing of peptic ulcer but a role in the causation of the basic injurious process has not been established.

24. No aetiological link between peptic ulcer and stress has been confirmed.

25. Reported associations with renal failure, chronic lung disease and pancreatic insufficiency have not been explained nor confirmed.

26. Oral steroids do not cause chronic peptic ulcer but may aggravate a pre-existing ulcer.

27. Since 1983 there has been much interest in the organism Helicobacter pylori and its possible aetiological role in dyspeptic disease. International epidemiological studies have confirmed that H pylori is commonly found in the upper digestive tract even in the absence of dyspepsia. In addition its prevalance increases with age. H pylori has been found to be present in 95-100% of patients with chronic duodenal ulcer and in 60-70% of those with a chronic gastric ulcer. However only a minority of individuals with H pylori infection subsequently develop a peptic ulcer.

28. The above suggests that H pylori is a risk factor for peptic ulcer. This is further supported by the fact that eradication of the organism leads to healing of the ulcer and a reduction in the subsequent relapse rate, particularly for duodenal ulcers. However despite considerable study a direct causal role for H pylori in peptic ulcer disease has not been established.

CONCLUSION

29. Peptic ulcers arise in gastrointestinal mucosal surfaces exposed to gastric acid and pepsin secretion. They may be acute or chronic. Acute peptic ulcers are due to damage by drugs or in association with severe illness.

30. The aetiology of chronic peptic ulcer has been extensively studied and relevant factors have been identified but the precise aetiology remains unknown. The evidence is that a chronic peptic ulcer occurs when the normal balance between the mucosal defence system and factors irritating the mucosa is upset. The organism Helicobacter pylori is a major risk factor.

REFERENCES

Misiewica J J and Pounder R E. Peptic Ulcer. In: (Eds) Weatherall D J, Ledingham J G G and Warrell D A. Oxford Textbook of Medicine. Oxford. Oxford University Press. 2nd Ed. 1987. p12.64-12.77.

Mann C V and Russell R C G (Eds). Bailey and Love's Short Practice of Surgery. 21st Ed. 1992. London. Chapman & Hall Medical. p950-1001.

Williams G T. Peptic Ulcer. In: (Eds) McGee J O'D, Isaacson P G and Wright N A. Oxford Textbook of Pathology. Oxford. Oxford University Press. 1992. p1159-1164.

Baron J H. Pathophysiology of the stomach. In: (Eds) McGee J O'D, Isaacson P G and Wright N A. Oxford Textbook of Pathology. Oxford. Oxford University Press. 1992. p1173-1175.

Pounder R. Peptic Ulceration. Medicine International 1994;22:225-230.

Tytgat G N J, Noach L A, et al. H pylori infection and duodenal ulcer disease. Gastroenterology Clinics of North America 1993;22(1):127-139.

Davies G R and Crabtree J E. H pylori, trick or treat? J R Soc Med 1994;436-439.

January 1995