



**Figure 17-14** Acute gastric perforation in a patient presenting with free air under the diaphragm. **A**, Mucosal defect with clean edges. **B**, The necrotic ulcer base is composed of granulation tissue.

Peptic ulcers are solitary in more than 80% of patients. Lesions less than 0.3 cm in diameter tend to be shallow while those greater than 0.6 cm are likely to be deeper. The classic peptic ulcer is a round to oval, **sharply punched-out defect** (Fig. 17-14A). The mucosal margin may overhang the base slightly, particularly on the upstream side, but is usually level with the surrounding mucosa. In contrast, **heaped-up margins are more characteristic of cancers**. The depth of ulcers may be limited by the thick gastric muscularis propria or by adherent pancreas, omental fat, or the liver. Hemorrhage and fibrin deposition are often present on the gastric serosa. **Perforation** into the peritoneal cavity is a surgical emergency that may be identified by detection of free air under the diaphragm on upright radiographs of the abdomen.

The base of peptic ulcers is smooth and clean as a result of peptic digestion of exudate. Active ulcers may be lined by a thin layer of fibrinoid debris underlaid by a predominantly neutrophilic inflammatory infiltrate. Beneath this, granulation tissue infiltrated with mononuclear leukocytes and a fibrous or collagenous scar forms the ulcer base (Fig. 17-14B). Vessel walls within the scarred area are typically thickened and are occasionally thrombosed. Bleeding from damaged vessels within the

ulcer base may cause life-threatening **hemorrhage**. Scarring may involve the entire thickness of the wall and pucker the surrounding mucosa into folds that radiate outward.

Size and location do not differentiate between benign and malignant ulcers. However, the gross appearance of chronic peptic ulcers is virtually diagnostic. **Malignant transformation of peptic ulcers occurs rarely**, if ever, and reports of transformation probably represent cases in which a lesion thought to be a chronic peptic ulcer was actually an ulcerated carcinoma from the start.

**Clinical Features.** Peptic ulcers can be chronic, recurring lesions with significant morbidity. The majority of peptic ulcers come to clinical attention because of *epigastric burning or aching pain*, although a significant fraction present with complications such as *iron deficiency anemia, hemorrhage, or perforation* (Table 17-4). The pain tends to occur 1 to 3 hours after meals during the day, is worse at night (usually between 11 PM and 2 AM), and is relieved by alkali or food. Nausea, vomiting, bloating, belching, and significant weight loss are additional manifestations. With penetrating ulcers the pain is occasionally referred to the back, the left upper quadrant, or the chest, where it may be misinterpreted as cardiac in origin.

Current therapies for PUD are aimed at *H. pylori* eradication and neutralization of gastric acid, primarily with proton pump inhibitors. It is also important to withdraw other offending agents, such as NSAIDs, including selective COX-2 inhibitors, that may interfere with mucosal healing. While peptic ulcers were previously notorious for their recurrence, the recurrence rate is now less than 20% following successful clearance of *H. pylori*.

A variety of surgical approaches were formerly used to treat PUD, including antrectomy to remove gastrin-producing cells and vagotomy to prevent the acid-stimulatory effects mediated by the vagus nerve. However, the success of proton pump inhibitors and *H. pylori* eradication has relegated surgical intervention to treatment of bleeding or perforated peptic ulcers.

**Table 17-4** Complications of Peptic Ulcer Disease

<b>Bleeding</b>
Occurs in 15% to 20% of patients
Most frequent complication
May be life-threatening
Accounts for 25% of ulcer deaths
May be the first indication of an ulcer
<b>Perforation</b>
Occurs in up to 5% of patients
Accounts for two thirds of ulcer deaths
Is rarely first indication of an ulcer
<b>Obstruction</b>
Mostly in chronic ulcers
Secondary to edema or scarring
Occurs in about 2% of patients
Most often associated with pyloric channel ulcers
May occur with duodenal ulcers
Causes incapacitating, crampy abdominal pain
Can rarely cause total obstruction and intractable vomiting