Epidemiology and Demographics of Upper Gastrointestinal Bleeding: Prevalence, Incidence, and Mortality

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KEYWORDS

• Upper gastrointestinal bleeding • Epidemiology • Mortality
• Time trends

Although there have been major therapeutic advances, improved diagnostics, and prevention in bleeding peptic ulcer disease (PUD) in recent years, this condition still poses a significant problem in clinical practice. Upper gastrointestinal bleeding (UGIB) is commonly related to PUD and is also the major cause of mortality. As a consequence of major changes in the treatment of peptic ulcers and their complications with the introduction of potent acid inhibitors, endoscopic therapy, and eradication of *H pylori*, a rapid decrease in both incidence and mortality was expected. However, published data show contradictory conclusions. Opposing trends in peptic ulcer complications such as bleeding or perforation have been reported in different countries, and no decrease or increase in hospitalizations because of peptic ulcer bleeding (PUB) complications has been observed. It has been proposed that the widespread consumption of nonsteroidal antiinflammatory drugs (NSAIDs) also influenced the more recent trends in the occurrence of ulcers, especially those resulting in bleeding complications and death. This article reviews all epidemiologic aspects of UGIB, especially those related to PUD.

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CAUSES OF UGIB

Several studies confirm that peptic ulcer is responsible for 28% to 59% of UGIB.[1–7] In the United States and Greece, the percentage of PUB is high compared with other European populations.[2,4] Percentages of PUB as high as 59% in the United States and Greece versus 28% to 37% in some European countries (UK, France, Netherlands) have been reported. The reasons for these differences are unclear, but it is suspected that the high proportion of NSAID use and/or the prevalence of *H pylori* infection may be behind these differences. However, there are no enough data from these countries to confirm that this hypothesis is based on solid grounds. Recent evidence suggests, however, that the incidence of PUB as a cause of acute UGIB either may be decreasing or is underreported. The Analysis of the Clinical Outcomes Research Initiative found that between December 1999 and July 2001 endoscopy performed for acute UGIB found a duodenal or gastric ulcer in only 1610 (20.6%) of the patients included.[8] In this study, the presence of mucosal abnormality was the most common cause of UGIB (40%). Probably, widespread proton pump inhibitors (PPIs) prescribing and *H pylori* eradication protocols also contribute to this downward incidence trend of PUD causing nonvariceal UGIB. Another important factor might be the use of cyclooxygenase 2 (COX-2) inhibitors, which are associated with both decreased episodes of UGIB and endoscopic lesions when compared with nonselective NSAIDs.[9–12]

There are few epidemiologic data focusing on bleeding in cirrhotic patients.[13–15] UGIB is, however, an important cause of morbidity and mortality in liver cirrhosis. Lecleire and colleagues[15] evaluated cirrhotic and noncirrhotic patients using the same data set as in the survey of Czernichow and colleagues.[3] A total of 2133 patients presented with UGIB in a 6-month period in 1996 in 4 French geographic areas, including 468 patients with cirrhosis. Variceal bleeding was the cause of bleeding in 59% of the cirrhotic patients, followed by PUB in 16%. The inhospital mortality rate was significantly higher in cirrhotic patients compared with noncirrhotic patients (34% vs 11%, respectively). In a French study comparing data from 1996 and 2000 concerning endoscopic variceal bleeding management, it was shown that endoscopic variceal ligation therapy is now more often used as the first-choice therapy compared with endoscopic sclerotherapy. Furthermore, there are also increasing trends in the use of terlipressin and somatostatin, 2 vasoactive drugs that have shown to be beneficial in cirrhotic patients with this complication.[16] Epidemiologic studies are still needed to evaluate the most recent outcome trends in cirrhotic patients.

There are other less frequent causes of UGIB. It is estimated that 5% to 15% of all cases of acute UGIB are secondary to Mallory-Weiss tears.[17–19] Vascular ectasias or angiodysplasias are another source of both acute and chronic nonvariceal UGIB[20] and are estimated to be the cause of UGIB in approximately 5% to 10% of cases. Dieulafoy lesion represents the cause for nonvariceal UGIB in less than 5% of all cases.[21,22] Malignant and benign neoplasms are another infrequent cause of nonvariceal UGIB and represent less than 5% of all causes.[23] More than one potential cause of UGIB is recorded in 16% to 20% of cases.[5,24] Eventually, in 7% to 25%, no lesions were found that could have explained the bleeding episodes, despite one or more endoscopic procedures. It is unclear how many of these episodes represent bleeding events from the small bowel, beyond the angle of Treitz.

RISK FACTORS FOR UGIB

H pylori and NSAID independently increase the risk of gastroduodenal ulcer and ulcer bleeding. The prevalence of gastroduodenal ulcers in patients taking NSAIDs regularly is approximately 15% to 30%,[25] although it has been reported to be up to 45% at 6