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Risk stratification and definitive hemostasis of nonvariceal upper gastrointestinal bleeding with blood flow detection and combination techniques

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ABSTRACT

Nonvariceal upper gastrointestinal (UGI) hemorrhage remains a significant health and economic burden. As the use of urgent endoscopy for UGI hemorrhage has increased, there has been a decline in associated mortality. Endoscopic hemostasis is based on risk stratification of stigmata of recent hemorrhage. A Doppler endoscopic probe can provide further risk stratification by detecting arterial blood flow under the lesion and as a guide to successful endoscopic treatment. Standard treatment options for endoscopic hemostasis include submucosal injection therapy usually in combination with either thermal coagulation or through-the-scope clips. A large over-the-scope clip, which has been used to close fistulas and perforations, has been shown to be effective in cases of refractory nonvariceal UGI hemorrhage, and might also be useful in other types of gastrointestinal bleeding.

1. Introduction

Upper gastrointestinal (UGI) bleeding continues to be a significant health and economic burden. Over the past 2 decades there has been a decline in hospitalization for nonvariceal UGI hemorrhage, as well as a reduction in nonvariceal UGI bleeding-associated mortality. Concurrently, there has been an increase in the rate of in-hospital upper endoscopy—including early endoscopy—and endoscopic therapy [1]. Peptic ulcers are the most common cause of nonvariceal UGI hemorrhage [2]. A number of techniques are available to treat bleeding nonvariceal UGI lesions. However, it has been difficult to demonstrate a mortality improvement, thus other clinical outcomes—primary hemostasis rate, rebleed rate, length of hospital stay, need for red blood cell transfusions, and rate of angiographic embolization or surgery—are used to assess a particular intervention's effect.

This review will examine endoscopic risk stratification of nonvariceal UGI bleeding (eg, ulcers, Dieulafoy lesions and Mallory-Weiss tears), including the use of the Doppler endoscopic probe (DEP). Additionally, we will discuss the various endoscopic techniques—both standard and emerging—for definitive endoscopic hemostasis.

2. Endoscopic approach

2.1. Preparation

Patients with severe, active hemorrhage (ie, a high-volume bloody gastric lavage or ongoing hematemesis, melena, or hematochezia) should undergo emergency endoscopy soon after medical resuscitation, usually in the intensive care unit. Hemodynamically stable patients can undergo endoscopy often in the endoscopy unit rather than the intensive care unit. For UGI bleeding, therapeutic single- or double-channel endoscopes with large-diameter suction channels are especially useful to allow quick removal of fresh blood and clots from the gastrointestinal (GI) tract during endoscopy. Additionally, a water pump can be used to target irrigate lesions through the accessory channel and dilute blood for suctioning.

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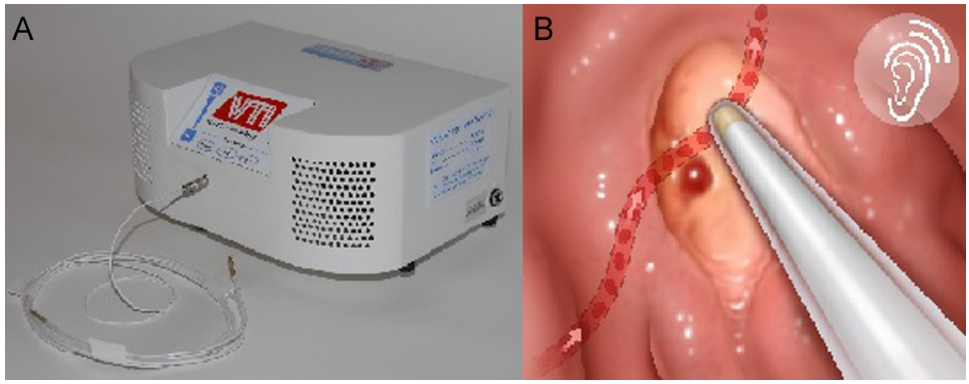

Fig. 1. Doppler endoscopic probe (DEP). (A) The device and (B) schematic of interrogating the direction of blood flow underneath an ulcer with stigmata of recent hemorrhage (SRH). (*Images courtesy*: Rachana Suchdev from VTI.) (Color version of figure is available online).

2.2. Endoscopic evaluation and risk stratification

In addition to detecting peptic ulcers and other nonvariceal UGI lesions, the endoscopist can categorize stigmata of recent hemorrhage (SRH) that are associated with an increased risk of rebleeding. The Forrest classification has been used to classify ulcers with stigmata according to risk of rebleeding. These include active spurting bleeding (Forrest IA), oozing blood (Forrest IB), pigmented protuberance or nonbleeding visible vessel (Forrest IIA), adherent clot (Forrest IIb), flat pigmented spot (Forrest IIC), and clean-based ulcer (Forrest III) [3,4].

SRH from an ulcer are shown in Figure 1. Patients at high risk of rebleeding without endoscopic treatment are those with active arterial bleeding (90%), a nonbleeding visible vessel (50%), or an adherent clot (33%) [5]. These patients, and those with the intermediate-risk stigmata of oozing bleeding [6], benefit from endoscopic hemostasis, whereas low-risk patients with a flat spot alone or clean ulcer base do not, according to past GI clinical guidelines [5,7].

2.3. Doppler endoscopic probe

The Doppler ultrasound probe for detecting arterial blood flow during GI endoscopy was first described in 1982 [8]. Since then, the DEP for emergency use has been simplified and is easy to teach GI endoscopists how to use (Figure 1) [4]. The DEP can be passed through the working channel of any diagnostic or therapeutic endoscope. The technique has been most commonly described in the evaluation of bleeding ulcers, but any GI lesion can be interrogated. The base of the ulcer should first be flushed with water to remove any exudate. The DEP tip is applied to the ulcer base with light pressure and at multiple points, including immediately adjacent to any endoscopic SRH. The direction of the artery (location relative to the stigmata) and the depth can be determined with the DEP. The artery detected by Doppler moves away from the visual SRH in a straight line. For nonvariceal lesions (such as ulcers, Dieulafoy lesions, or Mallory-Weiss tears), the blood flow detected is arterial and not venous. A positive DEP signal is defined as a repetitive and similar visual spiking waveform (or audible "swish-swish" sound) of at least 3 consecutive cycles, indicating pulsatile blood [4,9].

Although the DEP does not provide direct hemostasis, its value comes from its ability to help predict both the risk of rebleeding and success of endoscopic treatment. This has been shown in several prospective studies of patients with peptic ulcer bleeding, 2 of which we describe here in detail. In a study of 52 patients undergoing DEP, 23 underwent endoscopic therapy. Overall, 12 patients had a positive DEP signal before endoscopic therapy. Of these patients, 9 (75%) were converted to a negative DEP signal after therapy. All 3 patients with a persistent DEP-positive signal rebled within 30 days compared with only 1 patient (11%) whose ulcer had been converted to a DEP-negative signal [10]. In a more recent study, 163 patients with severe peptic ulcer bleeding underwent DEP evaluation during urgent endoscopy. Patients with major SRH (active arterial bleeding, nonbleeding visible vessel, and adherent clot) had a significantly higher DEP-positive rate than intermediate SRH (oozing alone or flat spot alone): 87% vs 42%. After standard, visually guided endoscopic hemostasis with either thermal probe or hemoclip (with or without epinephrine preinjection), there was a significantly higher DEP-positive signal in patients with major SRH vs intermediate SRH (27% vs 14%). None of the patients with oozing alone had a positive DEP signal after standard endoscopic hemostasis. The 30-day rebleed rate was 29% in patients with pulsatile bleeding ulcers and 0% in the oozing ulcer group [4]. In this prospective cohort study, rebleeding occurred in 4 of 5 (80%) patients who had residual arterial blood flow after visually guided hemostasis of a spurting ulcer and were treated medically according to current standard of care recommendations. Results of a recent randomized controlled trial (RCT) evaluating the use of DEP in nonvariceal UGI hemorrhage—including peptic ulcers, Dieulafoy lesions, and Mallory-Weiss tears—reported significantly higher 30-day rates of rebleeding, major complications, surgery, and red blood cell transfusions in the standard therapy group compared with the DEP-assisted treatment group [11]. These results indicate that residual arterial blood flow underneath ulcers is a significant risk factor for rebleeding. However, if more endoscopic treatment is applied (as in this RCT), clinical outcomes for patients with severe nonvariceal UGI hemorrhage are improved.

2.4. Hemostasis: Standard treatments

Thermal contact probes—such as heater, bipolar, and multipolar probe—have been the mainstay of endoscopic hemostasis for decades. These probes affect hemostasis through the following 2 mechanisms: tamponade of a blood vessel and interrupt underlying blood flow to stop bleeding and application of thermal energy to seal the underlying vessel, known as coaptive coagulation. They can be used for a variety of bleeding lesions, including peptic ulcers, vascular ectasias, and Dieulafoy lesions. The power setting for treating these lesions is low, ranging between 12 and 15 W, and the duration of energy application and amount of pressure applied depends on the depth and size of the vessel being treated [5,12]. The lower power setting is appropriate for thinner wall structure such as the small intestine and right colon, whereas higher power can be used more safely in other areas. In the laboratory, arteries up to about 1.5 mm in diameter could be coactively closed with thermal probes by applying firm tamponade