VANDERBILT UNIVERSITY MEDICAL CENTER

Guideline: Burn Stress Ulcer Prophylaxis Guidelines

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I. Background

Swan was the first to note the relationship between burn injury and GI mucosal damage in 1823.[1] This was followed by Curling in 1842 who reported on acute duodenal ulcers (Curling's Ulcers) in 10 patients with large burns.[2] Burn injuries increase the risk of GI ulceration and mucosal damage early after the injury has occurred. Endoscopy has shown gastric mucosal irritation within hours of the burn injury. Burn shock leads to an associated splanchnic hypoperfusion and gastric mucosal ischemia which can result in mucosal atrophy, decreased capacity to neutralize hydrogen ions and impaired mucosal repair. In 1970, Puritt et al. reported the incidence of "Curling's ulcer to be as high as 40% in patients with burns over 70% TBSA.[3]

Currently, both an increase in the depth of a burn and an increased TBSA are associated with a higher occurrence of both gastric and duodenal ulceration. Duodenal ulcers appear to occur more in patients with large TBSA burns and are more frequently associated with melena than other types of ulcers. Since the advent of the use of pharmacologic prophylactic ulcer therapy and early enteral nutrition (EN), the incidence of ulcers and mortality secondarily to them (1.9% mortality) has significantly decreased. Published rates in the development of upper GI ulceration range from 0.4% - 10%. A study by Yenikomshian, et al. found that in their patient population, women developed ulcers at almost double that for men (14% versus 8%).[4] In addition, for those patients that do develop ulcers, the incidence of significant bleeding has decreased by 50%.[4]

Stress ulcer prophylaxis guidelines for critically ill adults were updated in 2024, but patients with burns were not represented in these analyses.[5]

The most definitive indications for stress ulcer prophylaxis in the general ICU population include:

1) Coagulopathy (INR ≥1.5 or platelet count <50,000)
2) Shock (requiring vasopressors, SBP <90 mmHg, MAP <70 mmHg, or lactate ≥4)
3) Chronic liver disease
4) Neurocritical care (including traumatic brain injury, spinal cord injury)

Other risk factors for GI bleeding in critically ill patients include large burns (≥ 20% TBSA), significant alcohol use, acute hepatic failure, sepsis, acute renal failure, trauma, prolonged NSAID use, and high dose steroids (defined as equivalent of hydrocortisone 250 mg/day), and mechanical ventilation.

Maintaining gastric pH above 3.5-5 has been shown to prevent gastric mucosal injury.[6] Histamine-2 receptor antagonists (H_2RAs) and proton pump inhibitors (PPIs) can both be used to decrease gastric acid secretion but have different mechanisms. H_2RAs reversibly inhibit H_2 receptors on gastric parietal cells, which disrupts the gastric acid secretion pathway, while PPIs irreversibly inhibit the H^+/K^+/ATPase pump in gastric parietal cells, which prevent gastric acid secretion. PPIs are superior to H_2RAs for treating gastrointestinal bleeding, but clinical data has indicated that they are equally effective in preventing clinically significant GI bleeding, possibly due to the more modest pH increase needed to prevent gastric stress ulcers.[5-6] The 2024 SCCM/ASHP stress ulcer prophylaxis guidelines and the EAST Practice Management Guidelines recommend either H_2RAs or PPIs may be used for stress ulcer

prophylaxis in critically ill patients.[5,7] Much debate regarding potential adverse effects of pharmacological SUP exists, but the network meta-analysis conducted for the 2024 SCCM/ASHP guidelines found no conclusive effects of PPIs on incidence of nosocomial pneumonia or *Clostridioides difficile* infection.[5,8-9] When compared directly to H2RAs, PPIs decreased clinically significant upper GI bleedings (RR 0.53; 95% CI 0.34-0.84), but with a slight increase in mortality (RR 1.05; 95% CI 1-1.10), which may be more apparent in patients with severe critical illness.[5] H_2RAs are more strongly associated with delirium than PPIs (RR 1.36; 1.25-1.47).[10] Altered mental status and delirium related to H_2RAs may be more apparent in older adults who may have reduced renal function at baseline, as H_2RAs are eliminated renally and must be dose adjusted in renal dysfunction.

Early enteral nutrition (EN) may be effective in the prevention of stress related ulcer formation and bleeding in critically ill patients, though definitive data with contemporary SUP practices are lacking. Intolerance to EN has been hypothesized to be the first manifestation of impending ulcer formation/upper GI bleeding. EN prevents mucosal ischemia and ulceration by increasing splanchnic blood flow and increasing gastric pH (though to a lesser degree than pharmacologic agents). Utilizing the GI tract for nutrition also assists with maintaining mucosal health and preventing bacterial translocation.[11] EN alone is not recommended for patients who are considered to have major risk factors for upper GI bleeding.[5]

II. Indications for Prophylaxis

A. High Risk Patient:
- All patients to receive prophylaxis if any high-risk factor present.

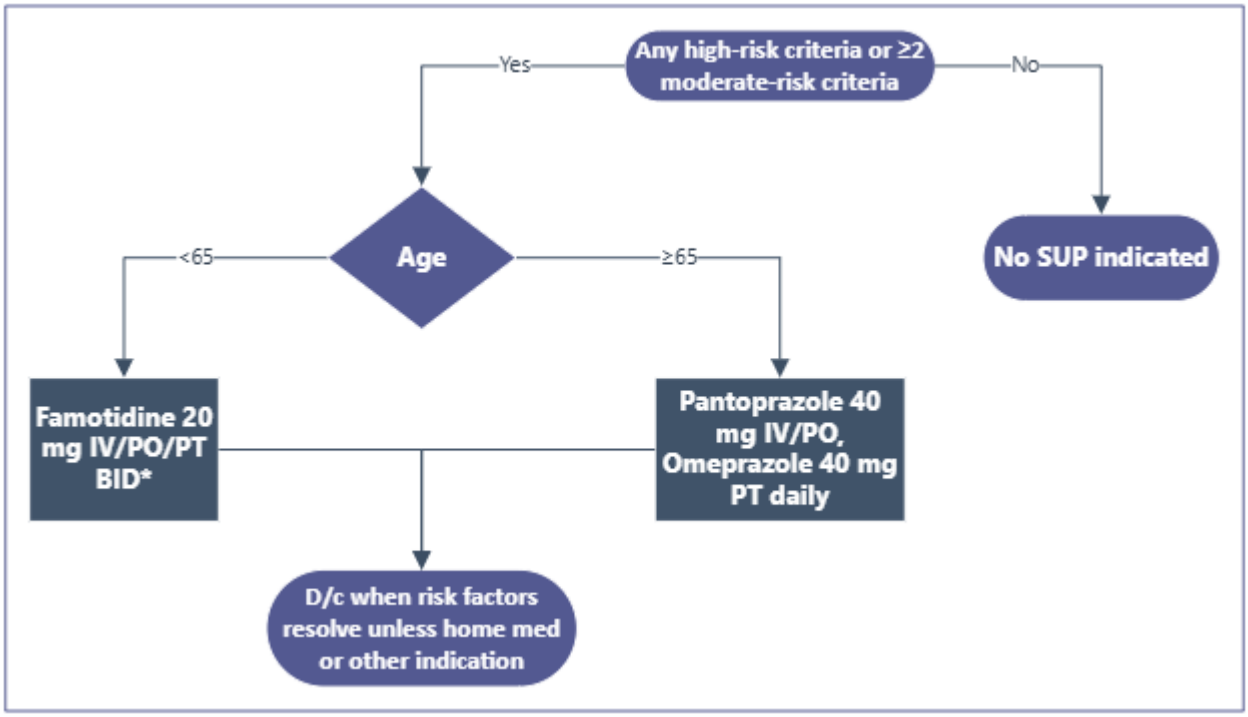
B. Moderate Risk Patient:
- Initiate prophylaxis if ≥2 moderate risk factors present

C. Low Risk Patient (≤1 moderate risk factor present)
- NO prophylaxis

HIGH RISK:
- TBSA >20% *prior to complete surgical coverage and excision of burns*
- Active shock (requiring vasopressors, SBP <90 mmHg, MAP <70 mmHg, or lactate ≥4)
- Coagulopathy (platelets <50,000 or INR ≥1.5)
- Traumatic brain injury
- Spinal cord injury (SCI)
- History of previous gastrointestinal hemorrhage
- Current outpatient treatment for peptic ulcer disease
- High dose, prolonged steroid therapy (equivalent to ≥hydrocortisone 250 mg/day)

MODERATE RISK: (≥ 2 risk factors present)
- TBSA <20% (*prior to complete surgical coverage and excision of burns*)
- Mechanical ventilation ≥48 hours
- Enteral nutrition intolerance
- Chronic NSAID or aspirin use (beyond ASA 81 mg daily)
- Current high dose NSAID therapy (ibuprofen >1200 mg/day, naproxen >1000 mg/day, all scheduled ketorolac regimens)

III. Burn Stress Ulcer Prophylaxis Algorithm



*If CrCl <50 mL/min, decrease to 20 mg daily

If using IV formulation, change to PO/PT option when patients are taking other oral medications and tolerating EN at goal

IV. References

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