



Acute Massive Upper Gastrointestinal Bleeding Secondary to Post-Burn Stress: A Case Report

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Abstract

Severe burns can readily induce gastric and duodenal mucosal erosions and superficial ulcers. In severe cases, hemorrhage or perforation of peptic ulcers might occur, threatening the lives of patients. Currently, gastrointestinal bleeding following burns is primarily managed with medication and gastrointestinal endoscopy. However, the combined multidisciplinary approach of gastroscopy and vascular embolization remains uncommon. A teenager, aged 18 years old, was admitted to the intensive care unit after Self-inflicted burns. Upon admission, he presented with third-degree burns covering approximately 60% of his total body surface area (TBSA). On the 4th day after the injury, the patient developed significant upper gastrointestinal bleeding (massive melena), with a drop in hemoglobin from 16 g/dl to 8 g/dl, indicating a critical drop in his blood levels. Immediate intervention was necessary to stabilize the patient and address the source of the bleeding effectively. Under the guidance of a multi-disciplinary team, a gastroscopy examination was performed and showed bleeding from the oeso-gastro-duodenal tract. The gastroscopy showed that there were multiple areas of ulceration and erosion, from the esophagus to the bulb with a Curling ulcer of the bulb, likely a result of stress-induced mucosal disease exacerbated by the patient's critical condition. Prompt treatment was initiated, which included intravenous fluids and blood transfusions to restore hemoglobin levels, alongside the administration of proton pump inhibitors to manage gastric acidity and promote healing of the mucosal lining. Following the initiation of continuous intravenous proton pump inhibitor (PPI) therapy, the patient did not experience any recurrence of hemorrhage. However, the patient developed severe sepsis, which progressed to septic shock accompanied by disseminated intravascular coagulation (DIC). Effective management of gastrointestinal bleeding requires a comprehensive approach that not only addresses the immediate crisis but also involves long-term strategies to prevent recurrence and promote overall digestive health.

Subject Areas

Gastroenterology & Hepatology

Keywords

Gastrointestinal Bleeding, Post-Burn Stress, Long-Term Strategies

1. Introduction

Severe burns can readily cause erosion or ulceration of the gastric and duodenal mucosa due to significant stress. A postburn stress ulcer, often called Curling's ulcer, is a digestive system complication that occurs following severe burns. In addition to gastrointestinal mucosal edema, congestion, bleeding, and erosion, there is a risk of severe gastrointestinal bleeding, ulcers, or perforations, which can directly threaten the patient's life [1]. The incidence of gastrointestinal ulcers accompanied by bleeding is approximately 4.7%; however, in many cases, this manifests as diffuse oozing of blood within the intestinal cavity. At present, acute massive hemorrhage from gastrointestinal ulcers in burn patients is rare. A prior study indicated a 50% mortality rate among patients with ulcers or hemorrhages occurring within 27 days of admission [2].

Active management of the primary condition and the mitigation of stress factors mainly involves sedation, anti-shock interventions, acid suppression, and pharmacological hemostasis. The combined use of endoscopy and interventional embolization for achieving hemostasis is relatively uncommon. This report details a case of diagnosing and treating acute upper gastrointestinal hemorrhage in a burn patient [3].

2. Case Report

The patient was 18-year-old. He was admitted to the intensive care unit after Self-inflicted burns. At the time of admission, the patient was in poor general condition. Physical examination showed that the burn wounds were distributed on the trunk, both lower limbs, both upper limbs and the buttocks, covering a total area of approximately 60% of the total body surface area. Diagnosis on admission indicated that the depth of burns was 3rd degree with 60% total burn surface area (TBSA). The patient had no previous systemic diseases, or past medical history of gastrointestinal ulcer.

After admission, the patient received intensive care, anti-infection treatment, stabilization of the internal environment, and regular wound dressing changes. No acid suppression therapy was started. On Intensive care unit admission, standard stress-ulcer prophylaxis was not initiated because initial management prioritized hemodynamic resuscitation, infection control, wound care, and early enteral nutrition. At the time of admission, the electrocardiogram (ECG) and chest X-ray showed no abnormalities. Procalcitonin (PCT) levels were positive, the white

blood cell count (WBC) was $13 \times 10^9/L$, the neutrophil percentage (Neu%) was 70.00%, and C-reactive protein (CRP) levels (measured using the colloidal gold method) exceeded 250.00 mg/L. The elevated infection markers in the patient were likely due to the absorption of toxins from the burn wounds. At day 4 of his admission, the patient was found to have tarry black stool, with an important volume. Given the bleeding tendency of gastrointestinal stress ulcers, the intravenous push administration of anti-acid was immediately used. An emergency complete blood count (CBC) showed a hemoglobin (HGB) of 8 g/dL, and a platelet count (PLT) of $110.000/mm^3$.

The patient was diagnosed with upper gastrointestinal bleeding. Treatment included intravenous omeprazole for acid suppression, fluid infusion, and blood transfusion to prevent shock. However, the patient continued to pass large amounts of tarry black stool, his blood pressure dropped to 90/40 mmHg, and his heart rate increased to 115 beats per minute, despite continuous rehydration and blood product supplementation. Repeated complete blood count (CBC) tests indicated a continued decline in hemoglobin (HGB) and platelets. Due to persistent gastrointestinal bleeding, an emergency gastroscopy was performed. Oeso-gastro-duodenoscopy revealed a severe hemorrhage esophagitis (Table 1), active bleeding Bulbo-gastritis with bulbar Curling's ulcer without active bleeding (Figure 1). Due to the diffuse lesions, there was any endoscopic intervention to control the bleeding. The patient was stabilized post-procedure with close monitoring in the intensive care unit, with continuous intravenous PPI to ensure effective management of his condition and prevent further complications. Following the initiation of continuous intravenous proton pump inhibitor (PPI) therapy, the patient did not experience any recurrence of hemorrhage. However, the patient developed severe sepsis, which progressed to septic shock accompanied by disseminated intravascular coagulation (DIC). Despite the intensive care provided, the patient succumbed to these complications and passed away 10 days after admission.

Table 1. Clinical course with key vital signs, transfusion volumes and laboratory values.

Day (ICU stay)	Blood pressure (mmHg)	Heart rate (bpm)	Hemoglobin (g/dL)	Platelets ($\times 10^9/L$)	INR	Transfusion (units)	Notes
Admission (Day 0)	Stable	Stable	16	—	—	0	Third-degree burns, 60% TBSA
Day 4 – GI bleeding onset	90/40	115	8	110	—	2 RBC units	Massive melena, ICU resuscitation started
Post-transfusion (same day)	100/60	105	9.5	95	1.5	+1 FFP	Emergency gastroscopy performed
Day 5	100/55	110	8.5	90	1.6	1 RBC	Continuous IV PPI infusion
Day 7	95/50	120	7.9	85	1.8	2 RBC units	Persistent sepsis, DIC evolving
Day 10	80/40	130	7.2	60	2.0	—	Septic shock, DIC, fatal outcome

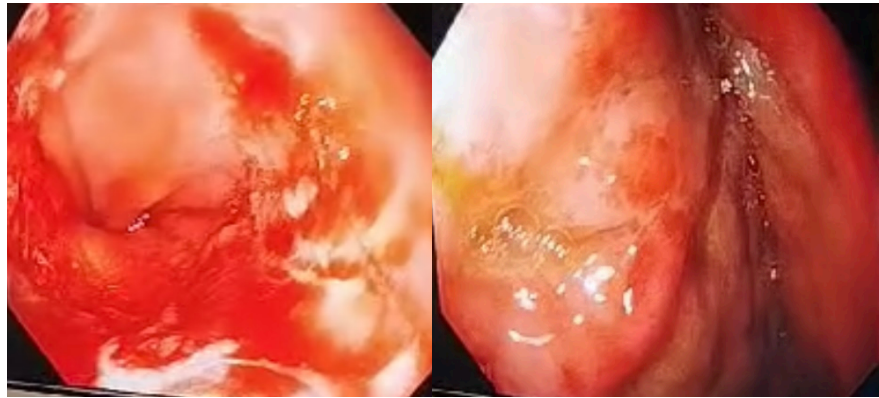


Figure 1. Severe esophagitis, and Bulbar Curling's ulcer.

3. Discussion

Acute gastroduodenal ulceration is the most serious and life-threatening gastrointestinal complication that can arise after thermal injuries. Superficial mucosal lesions in the stomach and duodenum frequently develop within 72 hours following extensive burn injuries [4].

The precise pathogenesis of post-burn acute gastrointestinal ulcers can be linked to gastric mucosal ischemia-reperfusion injury, which is triggered by the body's stress response to burns. This process results in decreased levels of prostaglandin E2 and bicarbonate, ultimately impairing the protective mechanisms of the gastric mucosa [5]. Furthermore, an intense and excessive stress response can cause a range of harmful effects on the body, including tissue breakdown, increased energy consumption, ischemia and hypoxia, immune suppression, and endocrine imbalances [6]. Following severe burns, a cascade of neuroendocrine reactions occurs within the hypothalamic-pituitary-adrenal axis and the sympathetic nervous system, leading to heightened vagus nerve excitability. Additionally, dysregulated gastric mucosal blood flow under stress may result from sympathetic nervous system activation [7].

The reported occurrence of stress-related mucosal damage ranges from 6% to 100% among critically ill patients. This condition is due to mucosal abnormalities that can range from submucosal hemorrhage and erosion to multiple ulcers, and it can be present in the esophagus, stomach, and duodenum [8] [9]. Endoscopically evident gastritis or ulceration may be noted in up to 100% of patients, and clinically evident bleeding in 20%, with increased morbidity and mortality [10].

In 1823, Curling identified a specific type of stress ulcer in severely burned patients, caused by reduced plasma volume, leading to ischemia and necrosis of the gastrointestinal (GI) mucosa. This ulcer is more frequently associated with perforation and hemorrhage than other types of stress ulcers. Although gastrointestinal bleeding (GIB) in burn patients is rare, with an incidence ranging from 1.5% to 4%, it remains the most common life-threatening GI complication in these patients, with reported mortality rates reaching up to 50% [11].

In our case, stress-ulcer prophylaxis was not initiated on admission because in-

itial management focused on hemodynamic stabilization, infection control, wound care, and early enteral nutrition. Nevertheless, contemporary critical-care guidelines recommend early prophylaxis with a proton pump inhibitor or H₂-receptor antagonist in high-risk patients such as those with severe burn injury. Early pharmacological prophylaxis is recognized as an essential preventive strategy to reduce the incidence of stress-related mucosal disease and upper gastrointestinal hemorrhage [12] [13].

4. Conclusions

This case report highlights the critical challenges associated with diagnosis and management of gastrointestinal bleeding in patients with severe burns. The patient's development of Curling's ulcer and subsequent massive upper gastrointestinal hemorrhage underscores the need for vigilant monitoring and early intervention in burn patients.

The multidisciplinary approach employed, which included emergency gastroscopy and supportive measures, underscores the significance of timely medical intervention in managing acute complications. Although the initial management strategies were successful in temporarily stabilizing the patient, the later development of severe sepsis and septic shock highlights the inherent risks and complexities of treating critically burned patients.

This case emphasizes the need for comprehensive care protocols that not only tackle the immediate crisis but also consider long-term complications to enhance outcomes for this vulnerable patient population. Increased awareness, prompt intervention, and continual research into improved treatment methods are crucial for reducing the prevalence and impact of gastrointestinal complications following severe burns.

Main Points

- 1) Severe burns frequently lead to gastric and duodenal mucosal erosions, with a significant risk of developing Curling's ulcer.
- 2) The unique application of a multidisciplinary approach, involving emergency gastroscopy and intravenous proton pump inhibitors, demonstrates the potential for comprehensive management in stabilizing acute GI bleeding in burn patients.
- 3) This case underscores the complexities and high mortality risk associated with managing gastrointestinal complications in severely burned patients.

Ethics Statement

Publication of this case report was approved by the institutional ethics committee, which waived the requirement for informed consent in this situation.

Conflicts of Interest

The authors declare no conflicts of interest.

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