

# Massive Gastrointestinal Bleeding as an Initial Presentation of Hodgkin Lymphoma

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## ABSTRACT

Hodgkin lymphoma is the most common malignant neoplasm affecting adolescents and young adults. Patients with Hodgkin lymphoma typically present with lymph node enlargement and systemic symptoms such as fever, weight loss, and night sweats. In addition, 40% of patients present with mediastinal mass-related symptoms such as cough and shortness of breath. Herein, we report a case of Hodgkin lymphoma with an unusual symptom, massive upper gastrointestinal bleeding. The bleeding persisted despite multiple endoscopic and hemostatic management. However, initiation of chemotherapeutic regimen stopped the bleeding. This posed the question of whether HL is a potential cause of massive upper GI bleeding.

**Keywords:** Gastrointestinal hemorrhage; Hodgkin lymphoma, Children

## INTRODUCTION

Massive upper gastrointestinal (GI) bleeding is rare in children and is not described as an initial symptom of Hodgkin lymphoma (HL)<sup>1</sup>. The approach to addressing such bleeding is to identify the underlying causes and commence urgent medical and endoscopic interventions if needed<sup>1</sup>. This approach is usually curative. We encountered a case of refractory massive upper GI bleeding in a newly diagnosed case of HL.

## CASE REPORT

**Clinical Findings:** A 12-year-old female was diagnosed with stage IIIB HL after months of lymphadenopathy, weight loss, and fever. After diagnostic and staging workup (before chemotherapy), the patient experienced massive upper GI bleeding (hematemesis and melena) without bleeding elsewhere. Planned chemotherapy was not initiated, and the patient was transferred to the intensive care unit for further management and monitoring.

**Diagnostic Assessment:** The initial workup assessed hemostasis but did not reveal the cause of the bleeding. The patient had no history of bleeding or excessive use of non-steroid anti-inflammatory drugs (NSAIDs) or steroids. Laboratory tests revealed normal platelet counts, coagulation, and renal and hepatic profiles. Neither a computed tomography angiogram nor a red blood scintigraphy scan identified the source of the bleeding. Diagnostic and therapeutic endoscopies revealed diffuse bleeding from the duodenal mucosa. Initial endoscopy showed a large clot in stomach, however, in duodenum an ulcer with oozing of blood of its site in the bulb (Figure 1). Due to the patient's clinical instability, we could not perform a diagnostic positron emission tomography scan.

**Therapeutic Intervention:** Intravenous proton pump inhibitors, sucralfate, tranexamic acid, and octreotide were administered to control the bleeding. The patient experienced repeated bouts of upper GI bleeds that required multiple endoscopic interventions including epinephrine injections, argon plasma coagulation, gold probe thermal therapy and endoscopic clips. The patient was transiently stable for one day but experienced multiple massive GI bleedings over the following

two weeks. During this period, the patient underwent six endoscopic sessions and 90 ml per kg of blood transfusions. Of note, the last endoscopy showed a large ulcer > 1cm with visible blood vessel that needed gold probe thermal therapy, (Figure 2). Bleeding has improved but did not slow completely.



**Figure 1:** Initial endoscopy showed an ulcer in the duodenal bulb that was oozing blood. Injection of epinephrine and clipping were done, and hemostasis was achieved. Histology was showed duodenitis and no evidence infiltration of abnormal cells.

Given the repetitive nature of the upper GI bleeds, short transient stability of the endoscopy, and potential of lymphoma, chemotherapy was initiated after the sixth endoscopy. A steroid-free chemotherapeutic regimen, adriamycin, bleomycin, vinblastine, and dacarbazine (ABVD), was administered to avoid the detrimental effect of steroid use on the already fragile gastric mucosa. The bleeding stopped after initiation of chemotherapy. Subsequently, we weaned the patient off medical management for GI bleeding before the second ABVD cycle.

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**Follow-up and Outcomes:** The patient completed six ABVD cycles. A positron emission tomography scan performed at the end of the therapy revealed a complete metabolic response. Subsequently, the patient underwent a follow-up endoscopy, which confirmed normal gastric and duodenal mucosa, (Figure 3). Approximately one year post-operatively, the patient was well and in complete remission.



**Figure 2:** The latest endoscopy was done before initiation of chemotherapy and showed another ulcer just lateral to the previously clipped ulcer with obvious vessel (shown in arrow). Gold probe cautery, clipping and injection of epinephrine were done.



**Figure 3:** Follow up endoscopy showed a completely normal mucosa and normal histology.

## DISCUSSION

Patients with HL typically present with progressive lymph node enlargement with or without systemic symptoms such as fever, night

sweat, or weight loss<sup>2</sup>. This is the first report of massive upper GI bleeding as an initial symptom of HL. This rare presentation poses diagnostic and management challenges, especially when bleeding is refractory to medical and endoscopic management.

Causes of GI bleeding can include peptic ulcer disease, with or without *Helicobacter pylori* infection, NSAID use, Mallory Weiss Syndrome, esophageal varices, severe thrombocytopenia, coagulopathies and rarely lymphoma<sup>3</sup>. Multiple reports described different types of lymphoma causing massive GI bleeding in adults<sup>4-8</sup> and children<sup>9</sup> but never to our knowledge in patients with HL. The most common affected locations are described in the stomach and rarely duodenum<sup>4-9</sup>.

It is unlikely that the upper GI bleeding in our patient was related to NSAIDs (or any other causes) for several reasons. Firstly, our patient had no history of regular or excessive NSAID or steroid use. Secondly, endoscopy revealed no signs of drug- or infection-induced mucosal damage in the esophagus, stomach, or duodenum. Thirdly, laboratory tests confirmed the absence of infection, thrombocytopenia, or coagulopathy. Finally, despite appropriate medical and endoscopic management of upper GI bleeding, recurrent bouts occurred.

The negative endoscopic biopsy results cannot rule out HL because most biopsies are small and often lead to the acquisition of false-negative results, which is the same finding in our case. In HL, approximately 10% of lymph node tissues represent malignant tumors<sup>10</sup>. Therefore, there is a high chance of obtaining false-negative results during screening. This is consistent with previous studies demonstrating that small biopsies obtained from lymph nodes by fine-needle aspiration present false-negative results<sup>11</sup>. Thus, fine-needle aspiration has been discouraged as a diagnostic procedure for lymphoma<sup>11</sup>.

The refractory course of our patient's upper GI bleeding to medical and endoscopic interventions posed the possibility of HL-related GI bleeding. However, the fear of worsening GI bleeding by initiating chemotherapy with a fragile gastric mucosa coupled with lack of evidence of metastatic or paraneoplastic phenomena related to HL led to a delay in initiating chemotherapy. Therefore, we initiated chemotherapy, steroid-free regimen (ABVD), after a two-week for upper GI bleeding. The patient's symptoms stabilized after initiation of chemotherapy, and no further bleeding occurred. The patient required no further endoscopic intervention or blood transfusions. We postulate that HL played a role in the upper GI bleeding in our patient and should be considered as a cause of bleeding during differential diagnosis to initiate immediate chemotherapy.

## CONCLUSION

**Based on our report, HL should be considered a potential cause of massive upper GI bleeding. Conventional medical and endoscopic treatment should be used to target common causes of massive upper GI bleeding in HL patients; however, chemotherapy should be initiated when the source of bleeding cannot be identified or controlled.**

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