Case Report

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A Case Report of non Hodgkin Lymphoma Presenting With Upper Gastrointestinal Tract Bleeding

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Abstract

Possible causes of upper gastrointestinal bleeding include peptic ulcer, esophageal ulcer, variceal bleeding and malignancies. A patient presenting with gastrointestinal bleeding should be evaluated as an emergency and treatment should be started as soon as possible. Hemodynamically stable patients should undergo endoscopy for both diagnostic and therapeutic purposes. In cases where bleeding cannot be stopped by endoscopic methods, invasive vascular methods and surgery may be tried. A 48-year-old male patient presented to the emergency department with complaints of nausea, vomiting in the form of coffee grounds and black stools. Gastrointestinal endoscopy revealed bleeding from a large, fragile mass in the stomach. Our patient was diagnosed as non-hodgkin lymphoma with early biopsy and the bleeding could not be stopped by endoscopic and surgical methods. With the chemotherapy given to our patient, the mass shrunk significantly and bleeding was under control. It should be kept in mind that early biopsy diagnosis and early chemotherapy may be a life-saving option in gastrointestinal bleeding caused by malignancy.

Keywords: Upper gastrointestinal tract bleeding, non hodgkin lymphoma, failure of medical treatment

Introduction

Initial evaluation of patients with acute gastrointestinal (GI) tract bleeding includes assessment of hemodynamic stability and resuscitation if necessary. Diagnostic studies (usually endoscopy) follow diagnostic goals and, when possible, treatment of the specific disorder(1). Non-Hodgkin's lymphomas (NHL) can have nodal or extranodal onset. NHL is seen in patients of all ages, races and socioeconomic levels(2)T cell progenitors, mature B cells, mature T cells, or (rarely. Patients with lymphoma with gastrointestinal system (GIS) involvement may present with anorexia, early satiety, weight loss, vomiting, ileus, perforation or bleeding. In this case report, we will try to present a case of lymphoma presenting with acute upper gastrointestinal tract bleeding refractory to treatment.

Case

A 48-year-old male patient was admitted to the emergency department with complaints of dizziness, intense weakness, vomiting in the form of coffee grounds and black diarrhea. On admission to the emergency room, blood pressure arterial: 80/55 mm-Hg, pulse rate: 130/min, temperature: 36.8 C°. Blood tests revealed hemoglobin (hb):8.5 gr/dl, ldh:642 U/L, hemostasis parameters were normal, urea 80 mg/dL, creatinine 1.3 mg/dl. The patient's rectal palpation was melena smear. An intravenous line was opened rapidly and saline (SF) infusion was started at 200 cc/h. 80 mg pantoprazole intravenous (IV) puff followed by infusion at 8 mg/h was started. The patient was ordered 3 units of erythrocyte suspension and hospitalized in the intensive care unit. The patient was admitted to the internal medicine outpatient clinic about 2 weeks ago with complaints of nausea, vomiting and abdominal pain. Blood tests performed at the outpatient clinic revealed hb:13.2 gr/dl and no pathologic values in biochemistry parameters. The patient had no special history. There was no history of regular medication and gastrointestinal surgery. Abdominal tomography (CT) was ordered. In the abdominal CT scan, it was reported as; "a lobulated contoured space-occupying solid mass lesion starting from the fundus level of the stomach and extending to the antrum, filling the lumen of the stomach almost completely at these levels and exceeding the stomach wall at the level of the great curvature, showing

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heterogeneous contrast uptake, measuring approximately 125x91 mm in the coronal plane at its widest point". During upper GI endoscopy, multiple biopsies were taken from the mass at the level of the great curvature. Although the patient was advised to continue hospitalization for the mass that allowed partial passage of the expected pathology result, the patient was discharged voluntarily. After admission to the emergency department, the patient was hospitalized in the intensive care unit, a central venous catheter was inserted and 1 unit of erythrocyte suspension was given without complications. During this period, the patient's arterial blood pressure and tachycardia improved slightly and urgent upper GI endoscopy was performed. It was observed that the patient had diffuse bleeding from the mass starting from the fundus and extending to the antrum. Endoscopic intervention (such as sclerotherapy, band ligation) could not be performed. The patient was then referred to oncologic surgery. The patient was taken to emergency surgery and was told that no surgical procedure could be performed for the mass that disrupted hemodynamics. The previous biopsy result of the patient was plasmablastic lymphoma and the Hb value was 7.7 g/dl in the control examinations. The remaining 2 units of erythrocyte suspension were also administered. Tranexamic acid was started 3x250 milligram IV. Since the hemostasis panel was normal, TDP (fresh frozen plasma) or cryoprecipitate was not given. The patient's relatives were interviewed. All possible risks including gastric perforation were explained. Consent for chemotherapy was obtained. DA-EPOCH chemotherapy protocol was started. With the treatment, the patient's GI bleeding stopped, her melena decreased, her vital signs improved and the need for erythrocyte suspension replacement disappeared. The patient, whose general condition improved, was transferred to the hematology service approximately 2 weeks later.

Discussion

Gastrointestinal bleeding originating proximal to the ligament of Treitz is called upper gastrointestinal bleeding and bleeding originating distal to the ligament of Treitz is called lower gastrointestinal bleeding. The initial evaluation of a patient with suspected clinically significant acute upper gastrointestinal bleeding includes history, physical examination and laboratory tests. The goal of the assessment is to determine the severity of the bleeding, its potential sources, and the presence of conditions that may affect subsequent management. The information collected as part of the initial assessment is used to guide decisions regarding triage, resuscitation, empirical medical treatment and diagnostic testing(1). Hematemesis is suggestive of upper GI bleeding. The majority of melena is also caused by upper GI bleeding (90%) but can also be caused by bleeding from the oropharynx or nasopharynx, small bowel or colon(3). Possible causes of upper gastrointestinal bleeding include peptic ulcer, esophageal ulcer, Mallory-Weiss tear, variceal bleeding, portal hypertensive gastropathy and malignancy. Physical examination is an important component in the assessment of hemodynamic stability. Tachycardia, orthostatic hypotension, and hypotension in the supine position are among the signs of hypovolemia(1). Our patient presented to the emergency department with hematemesis and melena. These findings suggested that the bleeding probably originated from the upper gastrointestinal tract. Hypotension, tachycardia and dizziness were findings suggesting the severity of bleeding. Laboratory tests that should be obtained in patients with acute upper gastrointestinal bleeding include complete blood count, serum chemistry, liver tests and coagulation studies(1). The initial hemoglobin level in patients with acute upper GI bleeding may be at baseline because the patient has lost whole blood. Over time, the hemoglobin level will decrease due to the entry of extravascular fluid into the vascular cavity and dilution of the blood due to the fluid administered during resuscitation. The hemoglobin level should initially be monitored every two to eight hours depending on the severity of bleeding(1). Because blood is absorbed as it passes through the small intestine and patients may have reduced renal perfusion, patients with acute upper GI bleeding typically have a high blood urea nitrogen (BUN)-creatinine or urea-creatinine ratio (values >30:1 or >100:1, respectively) (4). The higher the ratio, the more likely the bleeding is from an upper GI source (5). At the time of diagnosis, hb was 8.5 g/dl, urea was 80 mg/dL, creatinine was 1.3 mg/dl and hemostasis panel was normal. The urea/creatinine ratio was also high. Although the principles are similar in the treatment of all patients with upper GI bleeding, there are some special considerations when it comes to patients presenting with hemodynamic instability (shock, orthostatic hypotension) (1). Adequate peripheral access should be provided with two 18-gauge or larger intravenous catheters and/or a large-diameter, single-lumen central cordis.

For patients with active/live bleeding and hypovolemia, transfusion should be guided by hemodynamic parameters (e.g. pulse and blood pressure) rather than serial parameters, the rate of bleeding, estimated blood loss and ability to stop bleeding. If the initial hemoglobin level is low (<7 g/dL), transfusions should be initiated (6). However, transfusion support should not be delayed while waiting for laboratory test results in a patient with acute bleeding. Patients admitted to the hospital with acute upper GI bleeding are typically treated with a proton pump inhibitor (PPI)(1). After 80 milligrams of PPI, pantoprazole infusion was started at 8 mg/h. A wide vascular access was opened and iv SF infusion was started. Since the patient had active bleeding and hemodynamic instability, erythrocyte suspension replacement was performed even though the patient's hospitalization Hb value was <7 g/dl. Since the

hemostasis panel was normal, no blood or blood products were given for the hemostasis system. Upper endoscopy is the preferred diagnostic method for acute upper GI bleeding (7). Endoscopy has high sensitivity and specificity for detecting and identifying bleeding lesions in the upper gastrointestinal tract. In addition, once a bleeding lesion is identified, therapeutic endoscopy can provide acute hemostasis and prevent recurrent bleeding in most patients. Early endoscopy (within 24 hours) is recommended for most patients with acute upper GI bleeding. Endoscopy is recommended within 12 hours of presentation for patients with suspected variceal bleeding (1). Endoscopic therapy is indicated for the treatment of most ulcers with signs of new bleeding that increase the risk of recurrent bleeding. With appropriate treatment, high-risk lesions have recurrent bleeding rates of 5 to 20%, depending on the endoscopic appearance of the ulcer. On the other hand, ulcers with a clear base or flat pigmented spot have a low risk of recurrent bleeding (1). Surgery in peptic ulcer disease is the basis for emergency treatment of life-threatening complications such as bleeding, perforation, obstruction, and suspected malignancy, as well as disease refractory to medical therapy. Surgical options for peptic ulcer disease range from local therapies that only manage ulcer-related complications (ie, bleeding, perforation, or obstruction) to definitive ulcer operations. Generally, the least morbid procedure that will adequately manage the patient's problem should be used in each instance. Definitive ulcer operations may be directed largely or solely at reducing acid secretion (which impairs mechanisms of healing), or they may include strategies for managing the susceptibility of the ulcer bed to recurrent injury. Reconstruction is necessary following partial gastrectomy to reestablish gastrointestinal continuity. The Billroth I, Billroth II, and Roux-en-Y reconstruction techniques are the most common (8). After our case was partially stabilized, endoscopy of the upper gastrointestinal tract was performed. Although local methods (sclerotherapy) were tried because the mass was very large and fragile, they were not effective. The patient was then taken to emergency surgery and the gastrointestinal system surgeon reported that a palliative therapeutic procedure could not be performed because the mass was too large and invaded many vascular neural structures. The clinical presentation of NHL varies according to histologic subtype and sites of involvement. While some subtypes of NHL show variable lymphadenopathy for years (indolent), others are highly aggressive and can lead to death within weeks or even days if left untreated. Even within a specific NHL subtype, the clinical picture can vary greatly between individual patients (2). Aggressive lymphomas often present subacutely or acutely with a rapidly growing mass; they may present with structural symptoms such as fever, night sweats or weight loss; tumor lysis syndrome, ileus or bleeding. Examples of aggressive NHL include diffuse large B-cell lymphoma, Burkitt lymphoma, precursor B and T lymphoblastic leukemia/lymphoma, adult T-cell leukemia/lymphoma and plasmablastic lymphoma. Indolent lymphomas are usually insidious, presenting with lymphadenopathy, hepatomegaly, splenomegaly and/or cytopenias that grow slowly or increase and decrease over months or years. Examples of slow NHL include follicular lymphoma, chronic lymphocytic leukemia/small lymphocytic lymphoma and splenic marginal zone lymphoma (2). A minority of patients initially present with extranodal lymphoma (primary extranodal NHL), but many more patients develop extranodal disease (secondary extranodal NHL) during the course of their disease. Examples of extranodal manifestations of NHL include Patients with primary gastrointestinal tract lymphoma may present with anorexia, early satiety, weight loss, vomiting, ileus, perforation or bleeding (2). The most common site of primary extranodal disease is the gastrointestinal tract. Plasmablastic lymphoma (PBL) is a rare but highly aggressive lymphoma usually observed in the setting of HIV disease (9). Due to the rarity of this disease, there is no accepted evidence-based systemic treatment for PBL. CHOP remains a widely used treatment regimen, especially in resource-limited settings. However, more intensive regimens are recommended in PBL. The preferred chemotherapy regimen is the dose-adjusted (DA)-EPOCH protocol (cyclophosphamide and prednisone doses plus etoposide, vincristine and doxorubicin) (10). The HIV serology of our case was negative. All possible complications associated with chemotherapy were explained to our patient, including gastric perforation and vascular rupture. Accepting the possible risks, the patient was started on the da-epoch kt protocol. The treatment resulted in clinical improvement, hemodynamic stability, reduction and disappearance of the need for erythroid trasnfusion and improvement in laboratory values. The patient, whose general condition also improved and no longer needed intensive care, was transferred to the hematology service.

Conclusion

Aggressive non-hodgkin lymphoma may be among the etiologies of upper gastrointestinal bleeding. In cases where endoscopic and surgical methods are inadequate, chemotherapy may be a life-saving option considering the possible risks.

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