Aorto-enteric fistula: A case report

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Abstract

An aorto-enteric fistula (AEF) is a fistula formed between the aorta and the GI tract that causes severe rectal bleeding or large amounts of blood in vomit. It is a rare form of gastrointestinal hemorrhage and generally has a poor prognosis. AEF is often accompanied by a herald bleed. Immediate diagnosis and treatment is required once a herald bleed has been detected. Here, we report on a case of unsuccessful treatment of secondary AEF following stomach cancer. AEF is commonly referred to a lethal, and because of the interesting symptomatic and treatment aspects of the case, we have chosen to report it.

Introduction

An aorto-enteric fistula is a fistula formed between the aorta and the GI tract that causes severe rectal bleeding or large amounts of blood in vomit. It is a rare form of gastrointestinal bleeding, and with a mortality rate of nearly 100%, it usually has a very poor prognosis.¹ Treatment in the present case was unsuccessful despite the fact that the patient was conscious and lucid at the time of admission to the hospital. Though the outcome was extremely regrettable, the progress of this case is typical of AEF. We report on this case in the hopes that it will lead to improved survival rates for this disease, as knowing the distinctive characteristics and proper methods of treating the disease is particularly important in cases of AEF.

Case Presentation

The patient was a 60-year old male. The patient underwent a total gastrectomy by his local physician one year prior. The procedure was a type IV, 7x5 cm SE HO PO N1MO CY1, R-Y reconstruction. Thereafter, radiation 30 Gy treatment was performed. The patient was followed up for one month after the completion of treatment. He was brought to the hospital by ambulance after experiencing epigastric discomfort and hematemesis with blood clots. He vomited blood several times while in the ambulance. The total volume of this blood was approximately 500 ml. At the age of 54 he had undergone bypass surgery and was taking warfarin 0.5 mg and Cilostazol 100 mg.

The patient was conscious and lucid at the time of his admission to the hospital. Body temperature
was 35.9°C, blood pressure was 78/58 mmHg, heart rate was 145/min, SpO2 was 96% (O₂ 6L), and facial color was pallid. He reported peripheral coldness, presented with a soft, flat abdomen, and reported no pain upon pressure. Laboratory findings were as follows: pH 7.261, HCO₃ 20.5 mmol/l, BE -5.8 mmol/l Lac 10.5 mmol/l, WBC 6.1 x 1000/μ, RBC 287 x 10000/μ, Hb 10.1 g/dl, PLT 16.6 x 10000/μ1, BUN 12.1 mg/dl, and CRE 0.85 mg/dl. The patient presented lactic acidosis and anemia.

After arrival at the hospital the patient was given a massive transfusion with colloidal solution. Though he was vomiting blood with clots and passing dark red blood per rectum, his blood pressure was stable and 20 minutes after arrival at the hospital an upper endoscopy was performed. There was bright red hemorrhaging from the anastomosis site of his total gastrectomy and the blind end of the R-Y reconstruction. Hemostasis was attempted by clipping, but this proved difficult. Because the bleeding seemed to be arterial in origin, the endoscopy was ended after approximately 30 minutes. One hour after arrival at the hospital, the patient vomited a large amount of blood, his level of consciousness dropped, and he entered a state of shock. We therefore performed an endotracheal intubation. We then gave him a MAP transfusion with pumped colloidal solution, but his blood pressure could not be maintained. One hour and 20 minutes after arrival at the hospital he went into hemorrhagic shock and he exhibited pulseless VT. CPR was then begun. He was given two IVs of epinephrine 1 mg and after nine minutes his heartbeat was reestablished. Massive transfusions including blood transfusions were continued, but his blood pressure remained unstable and reached a low of Hb 3.8 g/dl. However, two hours after arriving at the hospital his blood pressure stabilized at around 90/40 mmHg, although abdominal CT images could not identify a source of hemorrhage. Two hours and 10 minutes after arriving at the hospital the patient was transferred to an OR and a laparotomy was performed. Coagulum, staining and marked distension was observed throughout the small intestine, but the source of the hemorrhage could not be identified. Because the patient remained in shock, a left thoracotomy was performed and the descending aorta was clamped. Two hours later the surgery was completed and the patient was transferred to the ICU without identifying the source of the hemorrhage. Four hours later he died.

Permission was obtained from family members to perform a pathological autopsy. This revealed that there was communication between the aorta and the blind end of the duodenum, and arterial rupture was confirmed as the cause of the hemorrhage in the GI tract. A macroscopic examination did not reveal a recurrence of cancer, but a histological examination revealed undifferentiated tumor cells in the vicinity of the rupture and differentiated tumor cell growth over a wide area of a portion of glandular tissue. These cells were similar to the scirrhous stomach cancer tumor cells identified histopathologically by the previous physician. Accordingly, we were able to confirm that this was a case of aortic rupture caused by tissue necrosis induced by the gastric cancer and radiation therapy.
Discussion

AEF is a form of aortic hemorrhaging into the GI tract with an extremely poor prognosis. It most commonly occurs in the third and fourth portions of the duodenum, followed by the jejunum and the ileum.1 AEF has three classic signs: massive GI tract hemorrhaging, a palpable mass in the abdomen, and abdominal or back pain. However, all three symptoms present in only 11% of cases2 and there are no specific symptoms. Two-thirds of AEF cases present with a herald bleed varying from a small to moderate amount of blood3. Presentation may occur as intermittent hemorrhaging, as any coagulum formed may temporarily close off the fistula. Thus, regardless of the fact that there is an arterial fistula, this first bleed is usually not critical and tends to stabilize. In the present case, the patient was conscious and lucid at the time he arrived at the hospital, and the fact that his blood pressure initially stabilized is thought to have been due to the progress of a herald bleed. Treatment of this life-threatening condition hinges upon early diagnosis and treatment at the herald bleed stage.

AEF is categorized as primary AEF or secondary AEF according to the cause. Most primary AEF are caused by an aortic aneurysm, but may also be caused by syphilis or tuberculosis.4,5,6,7 Most cases of secondary AEF are caused by abdominal vascular grafts,8,9 but may also result from malignant infiltration or radiation therapy. There are also reports of cases of AEF accompanying trauma.10 The present case is thought to have been caused by vulnerabilities in the GI tract brought on by infiltration of scirrhous stomach cancer and radiation therapy. However, the patient also underwent surgery for occlusive arterial disease, which suggests the possibility that his aorta was also compromised.

AEF is most commonly diagnosed by endoscopy, but as the most common sites for AEF are beyond the third portion of the duodenum, endoscopic diagnosis can be difficult. CT and angiography are used for diagnosis but these techniques are less reliable.1,5 AEF in the present case occurred after stomach cancer reconstructive surgery, and this factor compounded diagnosis by laboratory tests. Many cases require exploratory laparotomy, as was required in the present case.

The main treatment for AEF is surgery, but there are few cases in which both early and accurate diagnosis and laparotomy are both possible. Because most cases of AEF are preceded by a herald bleed, early and accurate diagnosis via endoscopy can be performed at this stage of the progression of the disease. The decision to perform CT and other tests and laparotomy should be made without first attempting to perform endoscopic hemostasis.

Conclusion

This report detailed a fatal case of AEF, caused massive GI tract hemorrhaging and which was preceded by a herald bleed. It demonstrates the necessity for clinical physicians to recognize causes of potentially fatal GI tract hemorrhaging such as AEF.

References


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