Splenic infarction – A rare cause of acute abdominal pain following gastric surgery: A case series

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1. Introduction

Radical gastrectomy with D2 lymphadenectomy has been recognized as the standard surgical treatment for advanced gastric cancer [1,2]. Splenic lymph node dissection is an important component of D2 radical gastrectomy, particularly for upper stomach cancer. This is often accompanied by removal of the spleen in the past few decades. During recent years, however, investigators reported that the spleen plays an important role as an immune organ, and suggested the spleen-preserving method for splenic hilar lymph node dissection [3]. But this also has its disadvantages including splenic ischemia and subsequent infarction during hilar dissection, which may result in delayed splenectomy. Splenic infarction, which has mostly clinically silent condition, is associated with numerous etiologies, mostly secondary to hematological disorders. Because many cases remain undiagnosed, the exact incidence of these cases is obscure and probably underestimated. Likewise, for gastric cancer surgery, incidence and severity of this rare entity in the postoperative period are still unknown.

We aimed to discuss this rare complication, postoperative splenic infarction, and plausible mechanisms causing this outcome following gastric cancer surgery.

2. Case presentation

2.1. Case 1

A 52 year-old male patient with gastric cancer underwent total gastrectomy and Roux-en-Y esophagojejunostomy. On the postoperative day 3, he developed mild symptoms of abdominal pain and bloating of the upper abdomen accompanied by chest discomfort. Laboratory results revealed increased levels of white blood cell (16,400/mm3) and C-reactive protein while cardiac markers were within reference range. The patient also described left shoulder pain with a much later onset. Due to slow progression of symptoms, with subfebrile fever, computed tomography (CT) of the abdomen was performed. It revealed splenic infarction with free fluid in the perisplenic and perihepatic recesses. Therefore, emergency laparotomy and splenectomy were performed due to massive infarction and hemorrhage. He was discharged on the postoperative day 15 without any complication. Histological examination confirmed complete (R0) resection of gastric cancer with a TNM score pT4 pN0 M0 (AJCC stage IIb). Retrospective examination of preoperative diagnostic methods revealed splenic atherosclerosis (Fig. 1a).

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2.2. Case 2

A 50-year-old male patient underwent total gastrectomy with D2 lymphadenectomy for adenocarcinoma located in the fundus–corpus region. On the postoperative day 6, he developed sudden onset of severe abdominal distention and pain reflecting to the left shoulder. Shortly after symptoms emerged, tachycardia and low blood pressure were observed. Since serial laboratory test results showed remarkable decrease of hemoglobin levels from 10 g/dl to 6 g/dl, abdominal imaging was performed. Abdominal CT demonstrated significant amount of free fluid in the abdomen and splenic infarction. Therefore, he was taken to the operating room for emergent exploratory laparotomy. Splenectomy was performed due to severe splenic infarction that caused rupture and severe hemorrhage (Fig. 1b and c). Postoperative day 16, he was discharged without any complication.

None of the patients had hypercoagulable state. Despite prolonged length of hospital stay, postoperative period was uneventful for both patients. In both patients, instead of classical technique of tying and knots, the bipolar vessel-sealing device was used for both dissection and division of splenic ligaments and hilar vascular structures. Dissection of the splenic hilum was performed as close as possible to the spleen to ensure better oncological outcome.

3. Discussion

Splenic infarction refers to any injury of the splenic vascular structures, particularly occlusion, leading to parenchymal ischemia and subsequent necrosis. It is associated with a heterogeneous group of diseases. Most of the cases have been reported secondary to hematological disorders or trauma [4], particularly after vascular intervention to splenic vessels [5,6]. Case reports of postoperative splenic infarction associated with atherosclerosis were also published [7]. Although latter cause, which was detected during retrospective evaluation of CT images, could have rendered the spleen more susceptible to global infarction in the first patient, it was not observed in the other patient. Therefore, vascular injury during splenic hilar dissection was seemed as the most reasonable cause for patient 2.

Splenic vascular injury may also be attributable to recent common use of bipolar vessel sealing device. Wilkinson et al. reported splenic infarct following laparoscopic fundoplication and pointed out that mobilization of tissues caused this complication [8]. Since we have encountered this complication in recent years, it seems plausible that aggressive use of tissue sealing systems can be subjected to splenic injury. Avoiding mobilization of connective tissue surrounding the vascular structures can prevent this complication. Safety and efficacy of these devices have not yet provided for splenic hilar lymphadenectomy due to restricted use of laparoscopy for gastric cancer management [9], although it was confirmed for laparoscopic splenectomy [10].

Patients with splenic infarction usually present no symptoms. Splenic infarction may be segmental or global, involving the entire organ as our patients have also experienced. In most patients, diagnosis of splenic infarct is made incidentally on CT [11]. In fact, splenic infarction alone is not an indication for surgery. In case of diagnosis of postoperative splenic infarction, nonoperative management warrants close follow-up, and surgery is indicated for persistent symptoms or in the presence of complications such as hemorrhage, rupture or abscess. In our series, hemodynamic instability and abdominal findings made us alert for prompt evaluation and emergent laparotomy for patient 2, while it showed slow progression and subsequent delayed onset of the symptoms in patient 1. In both patients, splenic infarction was attributed to vascular injury during dissection of hilum.

In conclusion, there has still been no data available about incidence and severity of splenic infarction in patients undergoing gastrectomy with D2 lymphadenectomy. In our patients, splenic infarction was probably due to aggressive mobilization of lymphoid tissue around splenic hilum and connective tissue surrounding the vessel, and subsequent injury to vascular structures by using bipolar vessel sealing device. Meticulous skeletonization of splenic hilum during D2 lymphadenectomy is required to prevent any splenic vascular injury. In the patients undergoing gastrectomy procedure, splenic infarction should be kept in mind in terms of sudden postoperative abdominal pain, particularly rising from left upper quadrant radiating to left shoulder.

Conflict of interest

None.

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Ethical Approval

This is not a research study.

Consent

Both patients have signed the consent forms for the Case reports to be published.

Author contribution

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