Esophageal Perforation in a Patient With Kidney Transplantation

Manouchehr Amini, 1 Ali Ghorbani Abdegah, 2 Behnam Molavi, 2 Amir Reza Radmard, 4 Ali Askari 5

Esophageal perforation is a rupture of the esophageal wall, caused by iatrogenesis in 56% of cases. Perforation of the esophagus remains a challenge, and its incidence has increased as the use of endoscopic procedures has become more frequent. We report a 54-year-old woman with esophageal perforation 8 days after kidney transplantation. She had received a gastrointestinal consultation prior to her transplantation. This report highlights the fact that perforation may occur after any organ transplantation, especially during the initial 2 weeks after transplantation, when mycophenolate mofetil and cyclosporine as well as high doses of corticosteroid are administered. If there is a delay in passage and a swallowing difficulty, high doses of immunosuppressive drugs are likely to cause ulceration and perforation. Preventive strategies including intravenous steroids for the first 2 to 3 weeks and divided doses of pills should be considered for such patients.

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INTRODUCTION

Severe complications in the gastrointestinal system occur in 10% of the cases after kidney transplantation and are 2nd in rank after infection.1,3 Survival rate significantly decreases when complications occur in the 1st year.2,4 Esophageal perforation is a severe gastrointestinal complication. It can have multiple causes including prescription of immunosuppressive drugs.2 These drugs, especially corticosteroids, could hide and delay the diagnosis and treatment of serious complications.2,5 Differential diagnoses of esophageal perforation are acute myocardial infarction, acute pancreatitis, and peptic ulcer perforation.4,6 Abdominal and chest pain, subcutaneous emphysema, pleural effusion, pneumomediastinum, tachypnea, and abdominal rigidity could be seen in these patients.2,4 We report a patient with esophageal perforation 8 days after kidney transplantation.

CASE REPORT

The patient was a 54-year-old woman who presented with odynophagia, heartburn, and hematemesis 8 days after kidney transplantation. Medical history evaluations revealed prior difficulty in swallowing and feelings of blocking. She reported eating small pieces of food and chewing food more than usual. Mycophenolate mofetil, cyclosporine, and prednisolone (1mg/kg) had been administered during the period after transplantation. A diagnosis of esophageal perforation was confirmed by the chest computed tomography scan (Figures 1 and 2). Endoscopy was performed before any symptoms appeared. However, the fear that another endoscopy would be harmful to the patient prevented a repeat of the procedure.

Vital signs and laboratory finding are shown in the Table. Esophagectomy, esophagostomy, gastrostomy, and jejunostomy procedures were
performed. Afterwards, the patient was admitted to the intensive care unit. Since mediastinitis threatened the patient’s life, the immunosuppressive drugs were stopped except for steroids. Serum creatinine level increased after a few days and transplant rejection established and the patient underwent nephrectomy. During the hospitalization, the patient was diagnosed with sepsis. A collection under the abdominal wall was discovered and drained and antibiotics were prescribed. Furthermore, due to dyspnea and pleural effusion, a chest tube was inserted.

The patient was affected by rectorrhagia and the examinations indicated the origin as the jejunostomy feeding stoma which was then brought under control. The patient was finally discharged after 68 days of being hospitalized. After 6 months, when her general condition became stable, the patient underwent gastric pull-up surgery and was discharged after 1 week.

**DISCUSSION**

Perforation of the esophagus constitutes a true emergency and most commonly occurs following diagnostic or therapeutic procedures. Spontaneous perforation, referred to as Boerhaav syndrome, occurs after vomiting or retching with pressure.7 Another cause for perforation is foreign bodies.

**Laboratory Findings at Admission and After Esophageal Perforation**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>On Admission</th>
<th>After Perforation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Leukocyte count, × 10⁹/L</td>
<td>5.4</td>
<td>16.4</td>
</tr>
<tr>
<td>Hemoglobin, g/dL</td>
<td>11.2</td>
<td>10.2</td>
</tr>
<tr>
<td>Platelet count, × 10⁹/L</td>
<td>188</td>
<td>250</td>
</tr>
<tr>
<td>Prothrombin time, s</td>
<td>14.6</td>
<td>40.7</td>
</tr>
<tr>
<td>Partial thromboplastin time, s</td>
<td>28</td>
<td>&gt; 110</td>
</tr>
<tr>
<td>Aspartate aminotransferase, U/mL</td>
<td>25</td>
<td>23</td>
</tr>
<tr>
<td>Alanine aminotransferase, U/mL</td>
<td>28</td>
<td>21</td>
</tr>
<tr>
<td>Alkaline phosphatase, U/mL</td>
<td>248</td>
<td>162</td>
</tr>
<tr>
<td>Bilirubin, mg/dL</td>
<td>...</td>
<td>0.6</td>
</tr>
<tr>
<td>Albumin, mg/dL</td>
<td>3.9</td>
<td>3.8</td>
</tr>
<tr>
<td>Blood urea nitrogen, mg/dL</td>
<td>79</td>
<td>51</td>
</tr>
<tr>
<td>Creatinine, mg/dL</td>
<td>8.3</td>
<td>2.3</td>
</tr>
<tr>
<td>Troponin, mg/dL</td>
<td>...</td>
<td>45.5</td>
</tr>
<tr>
<td>Amylase, mg/dL</td>
<td>88</td>
<td>323</td>
</tr>
<tr>
<td>Blood pressure, mm Hg</td>
<td>130/80</td>
<td>180/100</td>
</tr>
<tr>
<td>Pulse rate, /min</td>
<td>81</td>
<td>105</td>
</tr>
<tr>
<td>Respiratory rate, /min</td>
<td>21</td>
<td>24</td>
</tr>
<tr>
<td>Body temperature, °C</td>
<td>37.1</td>
<td>36.8</td>
</tr>
</tbody>
</table>

**Figure 1.** Computed tomography scan of the patient with oral contrast, which shows extraluminal leakage of oral contrast (curved arrow) from a defect (arrow) in left anterolateral distal esophageal wall extending downward to left inferior mediastinum medial to left lower lobe. Top, coronal view; Middle and Bottom, axial view.
Effective factors for viscus perforation after kidney transplantation are uremia, steroids, cytomegalovirus, autoimmune diseases, digestive system hormones, hypercalcemia, and hyperparathyroidism. High doses of prednisolone in the initial stages after transplantation may cause damage and lead to viscus perforation, increasing the mortality and morbidity rate of patients. Decreasing the dose may lower these complications. Corrosive injury due to gastric contents in the setting of motility disorder and gastric outlet obstruction in a malnourished patient, can cause acute esophageal necrosis.

Pain is a striking and consistent symptom. If subcutaneous emphysema is present, the diagnosis is almost certain. Tachycardia, diaphoresis, fever, and hypotension are common. Hematemesis is a rare finding in esophageal perforation. The chest radiography is abnormal in 90% of cases. It could be normal in the beginning of the process. Contrast esophagography with water-soluble media such as gasterographin is the gold standard for esophageal perforation diagnosis. If the perforation is diagnosed within the initial 12 to 24 hours, edges of the injury are primarily trimmed and closed. If there is a delay of more than 24 hours, the mortality rate increases from 36% to 64% and esophagectomy, esophagostomy, gastrostomy, and jejunostomy are recommended. Our patient underwent esophagectomy for the abovementioned reasons and gastric pull-up was performed thereafter.

Strict observation after transplantation in patient with swallowing difficulty, especially in the initial period, should be mandatory. Swallowing difficulty due to motility disorder cannot be diagnosed through endoscopy. For a diagnosis of motility disorder manometry and barium swallow are essential. If the problem is not solved, the transplant patient must be carefully observed. This important matter must be considered in all cases of organ transplantation, and in patients who are receiving high doses of oral immunosuppressive drugs. Preventive strategies such as using intravenous steroids for the first 2 to 3 weeks and divided doses of pills should be considered.

CONFLICT OF INTEREST
None declared.

REFERENCES


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