We report lethal hemorrhage from the kidney after thoracic endovascular repair for chronic type B dissection complicated by disseminated intravascular coagulation (DIC). A 70-year-old woman underwent thoracic endovascular repair to treat chronic DIC. Two weeks after surgery, refractory shock suddenly occurred and computed tomography showed a massive hematoma around the left kidney. Emergent renal artery angiography showed multiple bleeding points in the renal cortex. Immediate embolization of the renal artery was performed and her hemodynamic condition recovered. Physicians should be aware that massive hemorrhage from visceral organs is possible during the perioperative period of endovascular intervention for treatment of DIC.

Keywords: disseminated intravascular coagulation (DIC), hemorrhage, kidney

Introduction

Aortic dissection or aneurysm is possibly associated with disseminated intravascular coagulation (DIC) due to consumption coagulopathy. Subcutaneous and deep intramuscular hemorrhage, such as iliopsoas hemorrhage, is sometimes observed in this pathology. Administration of low-weight heparin, antithrombin-III, and blood transfusion, such as platelets and clotting factors, are effective for treatment of DIC. In cases that are resistant to medical therapy, surgical intervention should be taken into account. The efficacy of endovascular surgery for treatment of DIC has been reported. Additionally, postoperative DIC after thoracic endovascular repair (TEVAR) has been reported. We report a case of fatal hemorrhage from the left kidney, requiring emergent embolization of the renal artery, after TEVAR for chronic type B dissection for treatment of DIC.

Case Report

A 70-year-old female patient received conservative therapy for acute type B dissection 14 months before the present episode. She underwent superior mesenteric artery stenting because of ischemic colitis at that time. Her past clinical history was systemic lupus syndrome and she took 11 mg of oral prednisolone daily for 30 years. She underwent graft replacement of the infrarenal abdominal aorta 7 months later. Abnormal findings of a blood test were observed 2 months after abdominal surgery and she was hospitalized. Her platelet count was $4.4 \times 10^4$ mm$^3$, the fibrinogen level was 600 mg/L, and the D-dimer level was 39.1 µg/mL (normal range, 0.1–1 µg/mL) on admission. Her DIC score, as defined by the International Society on Thrombosis and Hemostasis, was 7. This score was classified as overt DIC. After hospitalization, she suffered from repeated deep intramuscular bleeding in the right hip. This condition was treated with coil embolization. Administration of heparin improved her blood test results, but bleeding, including subcutaneous bleeding, appeared. Therefore, surgical intervention was selected. A dissected thoracoabdominal aorta (Fig. 1a), in which mural thrombosis and reentry were observed, was resected. After this operation, the false lumen was almost completely thrombosed and the DIC score improved from 7 to 3 (Fig. 1b), but then became worse (5) 2 months later. The cause of worsening in DIC score was considered to be attributed to residual primary entry of the distal arch in which an ulcer-like projection increased and partial thrombosis was observed (Fig. 1c). Therefore, we performed a second surgical intervention of right-left axillo-axillary artery bypass.
Fatal Hemorrhage after Endovascular Repair

Two weeks after surgery, she suddenly lost consciousness because of refractory shock. Her systolic blood pressure was 50 mmHg. With intravenous bolus administration to maintain her blood pressure, computed tomography showed a massive hematoma around the left kidney and in the abdominal cavity (Fig. 3a). She had no evidence of trauma to the left kidney. Although her body weight was only 32 kg, more than 5000 mL of fluid replacement for 1 h maintained her systolic blood pressure at approximately 60–70 mmHg. Emergent left renal artery angiography showed multiple bleeding points from the surface of the cortex (arrow).

operation (Fig. 2). Two weeks after surgery, she suddenly lost consciousness because of refractory shock. Her systolic blood pressure was 50 mmHg. With intravenous bolus administration to maintain her blood pressure, computed tomography showed a massive hematoma around the left kidney and in the abdominal cavity (Fig. 3a). She had no evidence of trauma to the left kidney. Although her body weight was only 32 kg, more than 5000 mL of fluid replacement for 1 h maintained her systolic blood pressure at approximately 60–70 mmHg. Emergent left renal artery angiography showed multiple bleeding points from the surface of the cortex (arrow).
abdominal cavity the next day. The massive bleeding from the capsular tear on the left kidney in the retroperitoneal space penetrated into the abdominal cavity, resulting in fatal hemorrhage. She was fully recovered without impairment of renal function and discharged. Her DIC score improved to 3 from 5 and no evidence of hemorrhage was observed during 6 months in the follow-up period.

Discussion
DIC is a rare and serious complication of aortic aneurysm and dissection. The exact incidence of DIC in cases of chronic aortic dissection is unclear. The reported incidence of DIC complicated by abdominal aortic aneurysm is 2.4%–4%. Sasaki et al. reported 10 DIC cases (five cases of dissection and five of non-dissection) among 547 patients with aortic aneurysm. The location of the bleeding site varies. From our personal experience, subcutaneous and deep intramuscular hemorrhage, such as iliopsoas hemorrhage, appears to be a relatively frequent bleeding site. To the best of our knowledge, non-traumatic fatal hemorrhage from the kidney, as found in our case, has not been reported in this pathology. Generally, a certain degree of bleeding from the kidney in the retroperitoneal space appears to be localized, whereas massive bleeding can easily penetrate the retroperitoneal space and result in catastrophic bleeding into the abdominal cavity.

The classical medical treatment for DIC in cases with aortic aneurysm is administration of heparin and transfusion of platelets, coagulation factors, and antithrombin-III. Relatively newly reported medical treatments are long-term administration of low molecular heparin, anti-fibrinolytic therapy with tranexamic acid, and oral direct Factor Xa. In cases that are refractory to medical treatment, surgical intervention should be taken into account. Oba et al. recommended surgical treatment without evidence of improvement in DIC after 2 weeks of meticulous treatment. Sasaki et al. reported excellent results of eight open surgical cases for this pathology. In the era of endovascular repair, successful endovascular repair for treatment of DIC has been reported. The findings from our case suggest that catastrophic bleeding may occasionally occur, even during the recovery phase of laboratory data for determining the status of DIC after endovascular surgery. Long-term oral intake of steroids and her collagen disease might have affected the fatal clinical course in the present study. Kotani et al. reported a case of worsened coagulopathy resulting in a gradually expanding cervical hematoma after TEVAR for primary entry closure of chronic type B dissection. The authors speculated that DIC might occur secondary to thrombus formation in the false lumen after TEVAR. Sasaki et al. reported that prothrombin time, platelet count, and fibrinogen levels tended to be normalized by the 7th postoperative day. However, some cases showed recurrence of hematological disturbance and bleeding tendency in the follow-up period. Regardless of whether open surgical repair or endovascular repair is performed for treatment of DIC, a large change in coagulation factors during the perioperative period might lead to a bleeding tendency, and sometimes this leads to catastrophic bleeding. The degree of fibrinolysis and aneurysmal diameter is significantly correlated in patients who undergo TEVAR for aortic dissection. In the present case, the un-thrombosed portion in the false lumen was relatively small, and the postoperative decline in platelet and coagulation factors appeared to be relatively low. However, even a small decline in coagulation factors might induce a major bleeding tendency in patients who are preoperatively complicated by DIC with previous history of bleeding tendency. The findings in the present case suggest that aggressive supplementation of platelets or coagulation factors during the early phase after TEVAR might prevent this catastrophic event. Therefore, aggressive transfusion should be considered during the early phase of the postoperative period after endovascular repair for treatment of DIC.

Conclusion
Physicians should be aware that massive hemorrhage from visceral organs might occur during the early stage of the postoperative period of endovascular intervention in cases complicated by DIC. In such cases, postoperative aggressive supplementation of platelets and coagulation factors, and preparation of vascular interventions, such as coil embolization, for possible fatal bleeding might be necessary.

Disclosure Statement
All authors have no conflict of interest.

Author Contribution
Writing: TY
Critical review and revision: all authors
Final approval of the article: all authors
Accountability for all aspects of the work: all authors

References
1) Mendes BC, Oderich GS, Erben Y, et al. False lumen embolization to treat disseminated intravascular coagulation after