A Case of Disseminated Intravascular Coagulation after Thoracic Endovascular Aortic Repair

Shinsuke Kotani, MD

I report a hemorrhagic complication due to disseminated intravascular coagulation after thoracic endovascular aortic repair for a dissecting aortic aneurysm. A 74-year-old man underwent thoracic endovascular aortic repair and carotid-carotid artery bypass to close the primary entry site of the dissecting aortic aneurysm. Postoperatively, he developed a gradually expanding cervical hematoma. Laboratory data showed disseminated intravascular coagulation. He could not be extubated until postoperative day 6 because of the risk of airway obstruction. He was treated with transfusion to replenish the coagulation factor. Disseminated intravascular coagulation may occur secondary to thrombus formation in the false lumen after thoracic endovascular aortic repair.

Keywords: disseminated intravascular coagulation, aortic aneurysm, blood vessel prosthesis

Introduction

It is well known that aortic dissection or aneurysm may result in disseminated intravascular coagulation (DIC) due to consumption coagulopathy. However, DIC is relatively uncommon after endovascular surgery for aortic dissection or aneurysm.1,2) I report here a hemorrhagic complication due to DIC after thoracic endovascular aortic repair (TEVAR) for dissecting aortic aneurysm.

Case Report

A 74-year-old man with a 10-year history of left-sided hemiplegia after a stroke was admitted to our hospital with a chronic dissecting aortic aneurysm of the distal aortic arch. The aneurysm had been diagnosed 10 years previously, but had not been followed up. Computed tomographic angiography showed a Stanford type B chronic aortic dissection extending from the distal aortic arch to the abdominal aorta, with the false lumen entirely patent (Fig. 1a). The primary entry site was 20 mm distal to the origin of the left subclavian artery (Fig. 1b), and the re-entry site was at the origin of the left renal artery. The maximum diameter of the distal aortic arch was 72 mm, and the diameter of the descending aorta was 65 mm. The celiac axis, superior mesenteric artery and right renal artery originated from the true lumen. Laboratory data showed mild coagulopathy: fibrinogen 262 mg/dl, prothrombin time international normalized ratio 1.39, activated partial thromboplastin time 34.5. Liver function test was within normal limits. It would have been difficult to perform conventional replacement of the descending aorta via left thoracotomy because the patient had been bedridden for a long time and could not even stretch the left arm and could not be placed in the left lateral position due to joint contracture. To avoid the risk associated with conventional open surgery, I decided to perform TEVAR to close the primary entry site.

The procedure was performed under general anesthesia using the Zenith TX2 TAA Endovascular Graft (Cook Medical, Bloomington, Ind). To ensure enough length of the healthy aorta at the proximal neck, the stent graft was positioned so that it covered the origins of the left common carotid artery and left subclavian arteries after carotid-carotid artery bypass (Fig. 2a). The left subclavian artery was not revascularized because preoperative magnetic resonance imaging showed communication between the right and left vertical arteries. The origin of the left subclavian artery was embolized with metallic coils to prevent a type 2 endoleak. The primary entry site in the distal aortic arch was covered with the stent graft, and postoperative angiography did not show contrast medium in the false lumen of the thoracic aorta (Fig. 2b). The re-entry site at the origin of the left renal artery was not closed. The operation time was 276 min and no blood transfusion was needed.

After surgery, the patient was transferred to the intensive care unit in a stable condition, with continued endotracheal intubation. He subsequently developed gradually expanding hematomas and oozing in all surgical wounds. Although the oozing was immediately stopped by the
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Compression hemostasis, extubation was delayed because of the risk of airway obstruction due to swelling in his neck. The preoperative platelet count of $17.6 \times 10^4$/mm$^3$ decreased to $6.7 \times 10^4$/mm$^3$ immediately after surgery, and the initial fibrinogen level of 262 mg/dL decreased to 150 mg/dL. On postoperative day 1, the platelet count decreased to $4.1 \times 10^4$/mm$^3$, and the fibrinogen level to 116 mg/dL. D-dimer was 35.2 µg/ml (normal range, 0–1 µg/ml) and prothrombin time international normalized ratio was 1.48. Clinical and laboratory findings suggested DIC associated with consumption of coagulation factors in the large false lumen. He was treated with transfusion of red cell concentrates, fresh frozen plasma and platelet concentrates to replenish the coagulation factors. The hematoma gradually reduced in size as the levels of coagulation factors improved, and he was extubated on postoperative day 6. The remaining postoperative course was uneventful. Computed tomography on postoperative day 15 showed thrombus occupied the entire false lumen of the thoracic aorta (Fig. 3). The false lumen of the abdominal aorta remained partly patent with blood flow from the re-entry site at the left renal artery. The patient was transferred to another hospital for rehabilitation on postoperative day 31. The hematoma had resolved by date of hospital discharge. After 1 year, he had recovered well without an increase in the diameter of the aneurysm.

**Discussion**

DIC secondary to consumption coagulopathy is a relatively rare complication after TEVAR, and there are few reports of DIC after endovascular treatment for aortic dissection or aneurysm. Shimazaki et al. reported that consumptive coagulopathy did not occur after TEVAR for thoracic aortic dissection or aneurysm, even though activation of coagulation and fibrinolysis were observed.\(^1\) Monaco et al. reported that coagulation disorders after endovascular repair did not influence clinical outcomes and did not cause hemorrhagic syndromes.\(^2\) However, both these studies found a significant correlation between aneurysm diameter and the degree of fibrinolysis in patients who underwent endovascular repair for aortic dissection. Several cases of postoperative DIC due to endoleaks after endovascular repair for aortic aneurysms have been reported.\(^3\)–\(^5\) These reports suggested that blood flow from endoleaks or from patent re-entry sites may have caused consumption of the coagulation factors.

In the present case, DIC may have been caused by thrombogenesis in the large false lumen and the remaining

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**Fig. 1** (a) Preoperative computed tomography showing a large dissecting aortic aneurysm extending from the distal aortic arch to the descending aorta. (b) The primary entry site was 2 cm distal to the origin of the left subclavian artery (arrow).

**Fig. 2** (a) Debranching with carotid–carotid artery bypass via the retropharyngeal was performed to ensure enough length at the proximal neck. (b) Postoperative angiography did not show contrast medium in the false lumen of the thoracic aorta.

**Fig. 3** Computed tomography on postoperative day 15, showing the stent graft positioned from the distal aortic arch to the descending aorta, and complete thrombosis of the false lumen of the thoracic aorta.
blood flow through the patent re-entry site at the left renal artery. Debranch technique with carotid-carotid artery bypass resulted in cervical hematoma associated with DIC, resulting in a need for prolonged intubation.

The treatment of DIC in patients with aortic dissection or aneurysm is still controversial. It is common sense that treatment of the underlying condition is the most important aspect of management of DIC. Previous studies reported that treatment of endoleaks after endovascular aortic repair resulted in resolution of DIC.3–5 Although anticoagulation therapy such as recombinant human soluble thrombomodulin may be effective for the treatment of chronic DIC caused by consumption of coagulation factors with stagnation of blood flow in the patent false lumen or large aneurysm,6,7 there is no evidence that anticoagulation therapy is effective for the treatment of postoperative DIC after endovascular repair. Antifibrinolytic therapy may be applicable to treat DIC after endovascular repair. Uzuka et al. reported tranexamic acid was effective for the treatment of persistent endoleak and coagulopathy after endovascular repair.8 Transfusion of platelet concentrate or fresh frozen plasma has been reported to be effective for the treatment of acute DIC with severe bleeding or hemostatic abnormality.9 In the present case, the primary entry site was closed and only the blood flow through the re-entry site remained. I believed that the consumption of coagulation factors would stop when the large false lumen was filled by thrombus, and therefore immediately initiated blood transfusion to replenish the coagulation factors. After transfusion, the platelet count and fibrinogen level stabilized, and the hematoma gradually reduced in size. Administration of tranexamic acid might accelerate thrombogenesis of the large false lumen. If there had been more prolonged consumption of coagulation factors, closure of the re-entry site would have been required.

Conclusion

I consider that DIC owing to consumption coagulopathy may occur after TEVAR for dissecting aortic aneurysm because of thrombus formation in the false lumen. Anticoagulation therapy may be unnecessary in this setting, because the consumption of coagulation factors stops after the false lumen is completely filled with thrombus.

Disclosure Statement

All authors have no conflict of interest.

References