Multiple Endovascular Treatments of Aortic Rupture Secondary to an Acute Aortic B Dissection

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Abstract
Ruptures of the thoracic aorta in acute type B dissections are currently treated with Endoprosthesis at the proximal tear level. On the other hand, problems occur when one or more visceral vessels arise from the false lumen, because the bleeding through the ostium of the vessel in the lamella of dissection can continue. Eventually, the simple exclusion of proximal tear might not preserve the patient from the next progression to an aneurysm. This case shows both complications.

Case Report
In May 2014 a 72 year-old man was admitted to the Emergency Room for chest pain radiating to the back, acute dyspnea and hypotension. Blood samples presented mild anemia (Hb: 9 g/dl) and leucocytosis (WBC: 19.500). He immediately underwent a Computed Tomography (CT) which showed a massive left hemothorax due to a rupture of a chronic post-dissection aneurysm (Figure 1). The aneurysm diameter in the proximal descending aorta was 57 mm (true/false lumen ratio: 4) and 47 mm at the thoraco-abdominal passage (true/false lumen ratio: 2). Right Renal, Superior Mesenteric and Inferior Mesenteric arteries originated from the true lumen, while the Celiac trunk and the left renal artery originated from the false lumen. Renal and hepatic functions were normal. Associated chronic diseases were: atrial fibrillation treated with oral anticoagulation, coronary artery disease, heart failure and dilatative cardiomyopathy with severe left fraction ejection dysfunction (LVFE: 30%). After coagulation factors infusion, INR resulted 1.4, and he underwent an emergency treatment.

Under general anesthesia, the exposure of the left Common Femoral Artery (CFA) and percutaneous canulation of the left Brachial Artery were performed. First angiographic acquisition (with a pig-tail as marker for LSA) showed that rupture of false lumen was widely perfused from an entry tear just below LSA. For this reason a Valiant Captivia® 42×42×200 mm covering LSA was deployed. Free-Floo® edge landed proximally to the left Common Carotid Artery (CCA) (Figure 2). Good exclusion of entry tear was documented with angiography, but false lumen and rupture were still perfused through the origin of the left Renal Artery (Figure 3, left). Its ostium was incanulated with a guide wire reaching the renal artery through the false lumen, and a 6 mm Viabhan® was placed between the true aortic lumen and the main trunk of the renal artery. Therefore, while the left kidney resulted as normally perfused, the ascending flow towards the aortic rupture from reperfusion of the false lumen was interrupted (Figure 3, right). Operative procedures ended with the ballooning of Viabhan® transition zone through the dissection layer. The final check highlighted the patency of every visceral artery and a good exclusion of the aortic rupture. A thoracic drainage was placed in order to reduce hemothorax. The patient spent his post-operative course in ICU for 24 hours then he was transferred to the Vascular Division. He received 4 blood units transfusion. CT was performed after 48 hours and displayed the false lumen thrombosis with true lumen expansion, no progression of dissection and LSA perfusion from Vertebal Artery (Figure 4) were detected. The thoracic drainage was removed after 12 days. Following postoperative days were uneventful. The discharge occurred after 17 post-operative days.

After 6 months, check with CT Scan showed a new wide entry tear just distally to the aortic endoprosthesis, the reperfusion of false lumen and its enlargement from 6.5 to 8.5 cm (Figure 5, left).

The patient was immediately hospitalized, and the next day he underwent a second endovascular treatment. Under local anesthesia, through the femoral isolation, two new Valiant Captivia® endoprosthesis (42×38×150 and 38×38×150 mm) were caudally overlapped to the first TEVAR.

Completion angiography and pre-discharge CT scan check showed the absence of reperfusion of the false lumen. The patient was discharged in good health conditions on the fourth postoperative day. Clinical and CT follow up is good after 16 months.

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Discussion

About a third of patients with acute aortic B dissection presents a complication at the diagnosis such as malperfusion syndrome or hemodynamic instability [1,2], and aortic rupture is a catastrophic event with a high mortality rate ranging between 14 and 67%. The endovascular treatment is the gold standard but visceral branches could generate technical problems when they originate from the false lumen. For instance, in our case a second tear was the ostium of the left renal artery, causing a large retrograde reperfusion of false lumen and rupture, even if the previous upper entry tear coverage had been successful. In these cases, you have to cover that visceral vessel with other aortic endografts (with high risks of visceral infarction) or try to perform a similar technique as the one we had - of course as an alternative to open (Hybrid) surgery to revascularize visceral vessels [3,4].

Moreover, although the acute phase of rupture is overcome, the 20 to 40% possibility of an aneurysmatic evolution must be taken into consideration in spite of various variables considered as predictors of that (age, gender, race, radiological features) [5,6]. For example, in our case there was a very sudden progression of the aortic diameter (5 months only). Therefore, it is fundamental to plan a close follow up, in order to recognize a possible, and common, new treatment [7].

References