ORIGINAL RESEARCH PAPER

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ENDOVASCULAR THERAPY OPTIONS FOR DISTAL MALPERFUSION SYNDROME IN AORTIC DISSECTION

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ABSTRACT

Malperfusion syndrome is one of the common associations with aortic dissection. The morbidity and mortality in aortic dissection are very much related to the presence of ischemic complications at clinical presentation. Visceral and extremity ischemia are the most important complications and are associated with high in-hospital mortality. The identification of malperfusion syndrome and its mechanism is very important in the management of these cases. It is also important to understand the undergoing pathology and presentation of malperfusion syndrome in dissection as treatment options also vary accordingly. In the era of mini-invasive therapy these pathologies are increasingly being treated endovascularly. The aim of this paper is to describe the possible endovascular treatment options based on the pathology of malperfusion syndrome. With the help of the following typical cases the various available endovascular treatment options to deal with distal malperfusion syndrome in aortic dissection are illustrated.

INTRODUCTION:

Malperfusion syndrome (MS) is one of the common associations in aortic dissection (AD) and is seen in 25 to 50 % of cases (around 30% in type A dissection (1-4)). The morbidity and mortality of these are related to the presence of ischemic complications at clinical presentation. Visceral and extremity ischemia are the most important complications and are associated with high in-hospital mortality.

In type A dissection, surgical repair with supracoronary aortic reconstruction is the treatment of choice, aiming at surgical closure of the proximal tear and consequent reduction of rupture risk or other complications including myocardial infarction and cardiac tamponade (3,5). The pre-existing distal malperfusion usually improves after surgery in majority of these cases (3,6). The persisting malperfusion after surgery often demands further treatment. In case of type B dissections without distal ischemia, conservative treatment with good blood pressure control is the treatment of choice. Complicated type B dissection with distal malperfusion demands further treatment, as persisting malperfusion leads to visceral organ impairment and in long term constant false lumen high pressure, which in turn possibly lead to rupture or development of false lumen aneurysms (7,8).

Conventionally MS was treated surgically. In the era of mini-invasive therapy these pathologies are increasingly being treated endovascularly. It is important to understand the undergoing pathology and presentation of MS as treatment options also vary accordingly. The aim of this paper is to describe the possible endovascular treatment options based on the pathology of MS. With the help of the following typical cases some available endovascular treatment options to deal with distal MS are illustrated.

Case 1:

A 62-year old man was admitted to the peripheral hospital for acute pain in the chest and between shoulder blades, radiating to upper abdomen intermittently. The evaluation in the cardiology department did not confirm a heart attack, but the echocardiogram performed suspected an acute AD. The computed tomography angiogram (CTA) performed revealed a Stanford type B AD. The patient was referred to our hospital for further treatment. Meanwhile the patient developed kidney function deterioration with uncontrolled hypertension and increased serum lactate levels. The CTA also revealed a significant narrowing of the true lumen of the thoracic aorta distal to the intimal tear (Figure 1) and a dynamic obstruction of the left renal artery (LRA) and superior mesenteric artery (SMA, Figure 2). The interdisciplinary consensus decided for an endovascular treatment with stent-graft implantation covering the proximal tear distal to the left subclavian artery (LSA) and aiming at improvement of blood flow in the true lumen. Under general anaesthesia with complete percutaneous approach using two Prostar XL closure devices (Abbott Medical) at the femoral access sites, an endovascular stent-graft (Valiant Captivia, 34/34/167 mm, Medtronic) was implanted with the proximal covered end starting just distal to the LSA origin. The control angiogram showed a significant true lumen expansion with consequent better flow distally. The control CT 3 days later showed significant true lumen expansion (Figure 3) and increased blood flow to the visceral arteries (Figure 4) and the patient improved significantly both clinically and with laboratory values on further follow up.

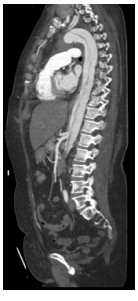

Figure 1

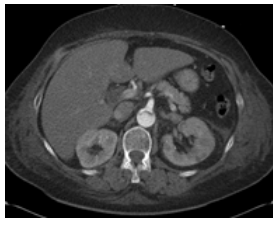

Figure 2

Figure 1, 2: Sagittal and axial reconstructions of the CTA showing dynamic compression of SMA (superior mesenteric artery).

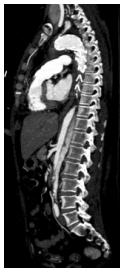

Figure 3

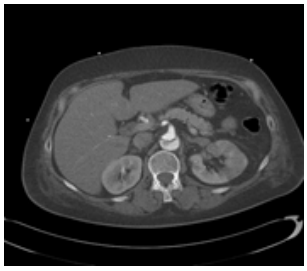

Figure4

Figure 3,4: Sagittal and axial reconstruction of the CTA showing increased blood flow in the celiac trunk and SMA after closure of the proximal tear with stent-graft implantation.

Case 2:

A 61-year old man was admitted to the hospital for acute severe pain in the neck, sternum and intermittently radiating to the upper abdomen region. On admission the patient was weak but otherwise hemodynamically stable. CT investigation of the chest and abdomen revealed a Standford type A dissection extending to the aortic bifurcation. The CT also revealed a static obstruction of the LRA. Dissection of the ascending aorta was then managed operatively with supracoronary aortic and arch reconstruction and a Frozen Elephant Trunk prosthesis. After 2 days the re-evaluation CTA revealed a persistent static occlusion of LRA (Figure 5) with signs of renal ischemia and deteriorating renal functions. The perfusion to right renal artery was exclusively from the false lumen. The interdisciplinary discussions decided for an endovascular treatment. Through a right transfemoral access (6F), the true lumen of LRA was recanalised and a balloon expandable stent (Figure 6, Hippocampus 6/20 mm, Invatec, Medtronic) was implanted at the origin extending to the aortic true lumen. The control CTA the next day showed better perfusion of the left kidney with improvement of the renal functions and urine output on further follow-up.

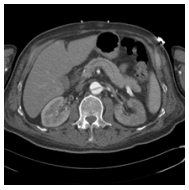

Figure 5

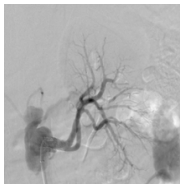

Figure 6

Figure 5: CTA showing a static occlusion of the LRA; visible hypoperfusion of left kidney in the arterial phase.

Figure 6: Digital substraction angiography (DSA) after stent-implantation for static occlusion of the LRA.

Case 3:

A 55-year old man presented with severe pain in the chest and intermittently in upper abdomen. Investigations in the emergency department with an echocardiogram suspected an acute AD. The CTA confirmed a Standford type B dissection. The CTA also showed an aberrant right subclavian artery (RSA) with origin from the descending aorta and a posterior course in relation to the oesophagus. The intimal tear was observed just distal to the origin of LSA and before the origin of the RSA. Because of the complex anatomy and absence of ischemic complications at presentation, a conservative management was decided with good blood pressure control after interdisciplinary discussions. About 7 days later the patient presented again with intermittent abdominal pain and bloody diarrhoea, with increased serum lactate levels on admission. A repeated CT abdomen showed a static occlusion of SMA with a co-existing dynamic component, because of the narrowing of the aortic true lumen. Preliminary ischemic changes of the ascending colon were also suspected on the CT-scan, which demanded further intervention.

For the adequate coverage of the proximal dissection tear and improvement of blood flow in true lumen, coverage of origins of both the subclavian arteries with a stent-graft would have been mandatory, which in turn would be of high risk for a posterior circulation stroke (because of vertebral artery origin closure). A stent-graft implantation would have been possible only after surgical carotid-subclavian bypass on both sides. With clear clinical and laboratory signs of on-going malperfusion an immediate endovascular approach with an infrarenal fenestration of the dissection membrane was decided. Access to both the femoral vessels was obtained (7F on right side, 5F on left side) and the true lumen was catheterised from the right side and the false lumen from the left side. The DSA through the true lumen confirmed the static obstruction of SMA with the dissection extending into the vessel. The true lumen was also significantly narrowed showing the dynamic compression (Figure 7, 8). The false lumen DSA did not show any perfusion of the SMA. The dissection membrane was exposed in profile in 30-degree angulation, after which an endovascular infrarenal fenestration was performed from true to false lumen with an Outback LTD Re-entry catheter device (Cordis, Johnson & Johnson Company, Bridgewater, NJ). The fenestration was first ballooned with a low profile balloon (6/40 mm, Advance Low profile, Cook Medical) followed by a XXL Esophageal Ballon (18/60 mm, Boston Scientific). The control angiograms showed better perfusion to SMA. No signs of complications were observed. The intervention was brought to an end with decision to follow the patient clinically as well with laboratory values.

The laboratory values next day did not show significant improvement and the patient still complained of bloody diarrhoea and intermittent abdominal pain without any change. Femoral access was obtained on the right side (5F) and the true lumen was catheterised for a second DSA. This showed a similar situation as before with significant true lumen narrowing and persistent static occlusion of SMA, which demanded further intervention. The SMA was then catheterised with a Cobra catheter (Terumo Corporation, Japan) following which a self-expanding stent (Cobalt Assurant, 8/60 mm, Medtronic) was implanted at the proximal course 'till the origin' of the vessel and was stabilized with a 7 mm balloon (Advance, Cook Medical). After exchanging to a 12F catheter sheath at the right femoral access, a self-expanding stent (Wallstent, 20/55mm, Boston Scientific) was introduced to the abdominal aorta. This was then deployed in the true lumen of the abdominal aorta with the proximal end placed just distal to the origin of the SMA. The control angiogram showed a significant improvement of SMA perfusion with timely portal venous filling in the late phase. Improved perfusion to the true lumen from the false lumen through the fenestration was also observed. Primary hemostasis was then achieved at the right femoral access site with Proglide closure device (Abbott Medical). The patient improved drastically with improvement of the clinical symptoms and fall in the serum lactate values on follow-up.

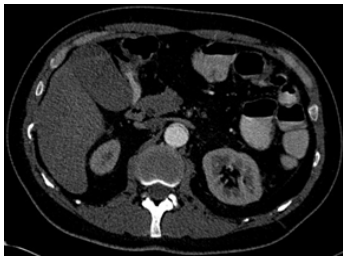

Figure 7

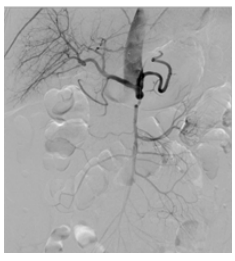

Figure 8

Figure 7:CT showing static & dynamic occlusion of SMA.

Figure 8:DSA through the true lumen showing perfusion of the coeliac trunk and SMA – so called "Hanging vessels".

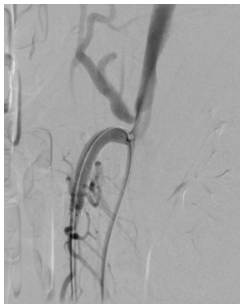

Figure 9

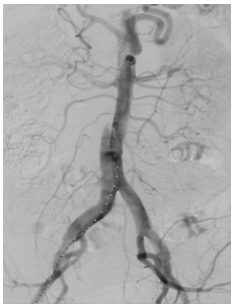

Figure 10

Figure 9:DSA after true lumen stenting of SMA.

Figure 10: DSA after creation of an infrarenal fenestration and true lumen stenting of the aorta with better perfusion of SMA and LRA.

DISCUSSION

In dissection MS results from the aortic branch compromise leading to secondary end organ ischemia. The morbidity depends on the involved vascular territory. It is important to understand the ongoing pathologies in these cases.

A CTA of complete aorta in the arterial and venous phase is very important in this regard (9-10). Electrocardiography (ECG) gated acquisition in CT is also reported as standard technique by some authors (11) for suspected AD. A differentiation between true and false lumen in CTA is of great relevance. True lumen is very often compressed, in 80 % of cases, while false lumen has a larger cross-section area. Hypodense thin strands of incompletely separated tunica media layer in the false lumen are often referred as "Cobweb sign" (12).

The complete tear of the dissection membrane at the origin of visceral arteries results in visceral artery perfusion exclusively from the false lumen. On the contrary, the incomplete tear results in an intimal tag formation hanging at the visceral artery origin. By the absence of a re-entry distally, the false lumen is constantly under high pressure and thereby leading to its expansion. Because of the fact that the false lumen wall is composed of tunica adventitia and only a part of tunica media, this lumen is relatively fragile and has a high risk of rupture in the acute or chronic phase. These can also lead to aneurysm development of the false lumen on follow-up (13). There are two mechanisms responsible for aortic branch vessel compromise in case of malperfusion as first described by Williams et al (14), each of which has specific treatment implications - dynamic and static obstructions (Figure 11).

Common among these is the dynamic obstruction, resulting from the prolapse of the dissection flap into the vessel ostium, covering it like a curtain. This accounts for 80 % of MS and is usually obvious during the aortic systole. Dynamic obstruction is influenced by blood pressure, heart rate, cardiac output, peripheral resistance of the outflow vessel as well as circumference involvement of dissected aorta. The restoration of adequate branch inflow by increasing blood volume in the true lumen with coverage of proximal entry tear using endovascular stentgraft implantation in type B AD (as in case 1) or surgical ascending aorta repair in type A dissections is usually the mode of management in these patient groups.

In contrast the static obstruction is caused when the intimal flap extends into the branch vessel origin propagating into the vessel wall and constricting the lumen. The obstruction may be related to compression and subsequent thrombosis of the aortic branch vessel secondary to the blind end of the false lumen. In extreme cases the true lumen thrombosis beyond the compressed ostia leads to further degradation of blood flow into the vessel. In case of static obstruction restoration of blood flow into the aortic branch vessel includes management of the vessel itself. These include stent implantation of the true lumen (as in case 2), fenestration of the dissection membrane or surgical bypass graft. Often static and dynamic obstructions exist on the same patient (as in case 3).

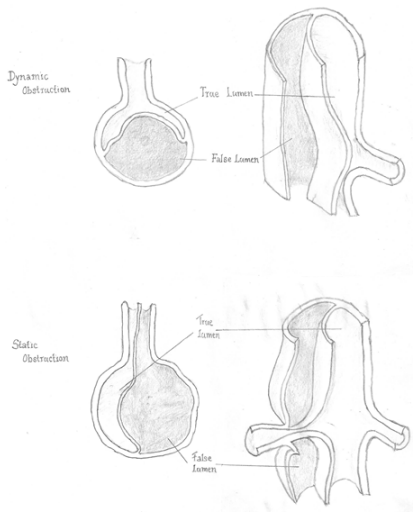

Figure 11: Schematic diagram explaining static and dynamic obstruction.

In cases of type A AD the malperfusion symptoms improve drastically after the surgical repair of closure of proximal tear (3,4). Some authors delayed surgery in the absence of cardiac complications in favour of early endovascular treatment of malperfusion (15,16).

The MS was treated in the past surgically with creation of fenestration or bypass to the ischemic vessels. The surgical fenestration creating a single lumen in AD was first reported in 1935 by Gurin and colleagues (17) and later further developed and improved by others (18,19). The operation mortality in this patient group is reported to be between 25 to 51 %, especially in patients with renal or intestinal malperfusion (19,20). In the era of mini-invasive interventions, ischemia complicating AD can be effectively treated by percutaneous interventions resulting in good early and mid term outcomes (13). These include percutaneous angiogplasty with true lumen stenting of involved visceral artery or thrombolysis, balloon endovascular fenestration and true lumen stenting of aorta.

The true lumen stenting of the visceral arteries in case of static obstruction are usually done with balloon expandable stents and it is important to extend the stent at the origin of the visceral vessel till the aortic true lumen. For the iliac arteries whenever needed, a true lumen stenting is usually done with self-expanding stents. In case of acute dissections the constant compression of the true lumen can lead to the development of acute thrombosis. The presence of significant thrombus at the ostium of the visceral arteries could be treated by local thrombolysis (13) with 4-10 mg of recombinant tissue plasminogen activator (rTPA, Actilyse).

The first percutaneous membrane fenestration was reported by Williams et al. in 1990 (21) using the hard end of a 0.46 guide wire. Many other reports using puncture set and guide wire combinations exist in the literature. Saito et al used a Mullin's dilator and a transseptal needle for the membrane puncture (22) while Chavan et al (13) performed it with a TIPS-Set (Rosch-Uchida transjugular liver access set, William Cook Europe, Bjaeverskov, Denmark). In the recent years the Outback Re-entry catheter used in the subintimal recanalisations of occlusions have been increasingly used for the membrane puncture. Although many centres do the percutaneous fenestration with the help of intravascular ultrasound, this can be done without the same. The exposure of the dissection membrane in profile with different projections of DSA is required for the optimal membrane puncture. Often more than one fenestration is needed. However, if the perfusion does not improve as expected after three fenestrations, further fenestrations would not improve the results and these could lead to relevant instability of dissection membrane, which in turn could influence the perfusion of vital organs (23).

The scissor technique, which uses two rigid guidewires placed in the true and false lumens followed by advancement of an 8F, 45 cm long sheath to disrupt the intimal flap from a distal re-entry tear to the upper abdominal aorta has also been reported to carry out endovascular fenestration (24). If the perfusion of the vessels arising from the true lumen does not improve after fenestration, stenting of the true lumen of aorta between the fenestration and the ischemic vessel origin can be done. The self-expanding stents like Wallstents (Boston Scientific) or Palmaz Stents (Johnson & Johnson, Warren, New Jersey, USA) are usually used in this regard. With true lumen stenting of aorta, it is advised not to cover the origin of visceral arteries, however in an acute life threatening aortic dissection the coverage with the stent is not contraindicated (25).

CONCLUSION

In short identification of the type of MS is crucial in the management of the disease. For type A AD surgical aortic repair often improves the visceral and extremity perfusion. For type B AD percutaneous endovascular stentgraft implantation for coverage of proximal tear improves the malperfusion. The malperfusion persisting after these management options require additional treatment. In the era of mini-invasive interventions, the percutaneous endovascular treatment options including true lumen stenting, infrarenal fenestration of the dissection membrane and stenting of the aortic true lumen and selective thrombolysis are effective in the management of this group of patients.

REFERENCES:

1. Rousseau H. Thoracic aortic dissection. Springer, Berlin, Heidelberg, New York. 2006.
2. Fattori R, Botta L, Lovato L et al. Malperfusion syndrome in type B aortic dissections: role of the endovascular procedures. Acta Chir Belg 2008; 108: 192-197.
3. Borst HC, Heinemann MK, Stone CD, eds. Surgical Treatment of Aortic Dissection. Churchill Livingstone: New York, Edinburgh, London, Madrid, Melbourne, San Francisco, Tokyo; 1998
4. Cambira RP, Brewster DC, Gertler J et al. Vascular complications associated with spontaneous aortic dissection. J Vasc Surg 1998; 7: 199-209.
5. Dake MD, Kato N, Mitchell RS et al. Endovascular stent graft placement for the treatment of acute aortic dissection. N Engl J Med 1999; 340:1546-1552.
6. Miller DC. Vascular complications associated with spontaneous aortic dissection (discussion). J Vasc Surg 1988; 7:208-209.
7. Williams DM, Lee DY, Hamilton BH et al. The dissected Aorta: Percutaneous treatment of ischemic complications – Principles and Results. JVIR 1997; 8:605-25.
8. Wheat MW. Current status of medical therapy of acute dissecting aneursyms of the aorta. World J Surg 1980; 4: 563-569.
9. Cerna M, Kocher M, Thomas RP. Acute aorta, overview of acute CT findings and endovascular treatment options. Biomed Pap Med Fac Univ Palacky Olomouc Czech Repub. 161(1): 14-23, 2017.
10. Hayter RG, Rhea JT, Small A et al. Suspected aortic dissection and other aortic disorders: multi-detector row CT in 373 cases in the emergency setting. Radiology 2006; 238:841-852.
11. McMahon MA, Squirrell CA. Multidetector CT of aortic dissection: a pictorial review. Radiographics 2010; 30: 445-460.
12. Williams DM, Joshi A, Dake MD, Deeb GM, Miller DC, Abrams GD. Aortic cobwebs: an anatomic marker identifying the false lumen in aortic dissec tion—imaging and pathologic correlation. Radiology 1994; 190(1): 167–174.
13. Chavan A, Rosenthal H, Luthe L et al. Percutaneous interventions for treating ischemic complications of aortic dissection. Eur Radiol 2009; 19:488-494
14. Willias DM, Lee DY, Hamilton BH et al. The dissected aorta: part III. Anatomy and radiologic diagnosis of branch-vessel compromise. Radiology 1997; 203:37-44.
15. Patel HJ, Williams DJ, Dasika NL et al. Operative delay for peripheral malperfusion syndrome in acute type A aortic dissection: a long term analysis. J Thorac Cardiovasc Surg 2008; 135:1288-1296.
16. Fabre O, Vincentelli A, Willoteaux S et al. Preoperative fenestration for type A acute aortic dissection with mesenteric malperfusion. Ann Thorac Surg 2002; 73: 950-951.
17. Gurin D, Bulmer JW, Derby R. Dissecting aneurysm of the aorta: diagnosis and operative relief of acute obstruction due to its cause. NY State J Med 1935; 35: 1200-2.
18. Elefteriades JA, Hammond GL, Gusberg RJ et al. A safe and effective procedure for descending aortic dissection. Arch Surg 1990; 125: 786-90.
19. Heinemann MK, Buehner B, Schaefers HJ et al. Malperfusion of the thoracoabdominal vasculature in aortic dissection. J Card Surg 1994; 9:748-757.
20. Fann JI, Sarris GE, Mitchell RS et al. Treatment of patients with aortic dissection presenting with peripheral vascular complications. Ann Surg 1990; 125: 786-790.
21. Williams DM, Brothers TE, Messina LM. Relief of mesenteric ischemia in type III aortic dissection with percutaneous fenestration of the aortic septum. Radiology 1990; 174:450-52.
22. Siato S, Arai H, Kim K el al. Percutaneous fenestration of dissecting intima with a transseptal needle. A new therapeutic technique for visceral ischemia complication acute aortic dissection. Cathet-Cardiovasc-Diagn 1992; 26: 130-135.
23. Manke C, Strotzer M, Seitz J et al. Ischemic Komplikationen bei Aortendissektion – Perkutane Behandlung mit ballonfensterung und stent implantation. Fortschr Rontgenstr 1999; 198-204.
24. Midulla M, Renaud A, Martinelli et al. Endovascular fenestration in aortic dissection with acute malperfusion syndrome: Immediate and late follow-up. J Thorac Cardiovasc Surg 2011; 142:66-72.
25. Slonim SM, Miller DC, Mitchell RS et al. Percutaneous balloon fenestration and stenting for life threatening ischemic complications in patients with acute aortic dissection. J Thorac Cardiovasc Surg 1999; 117:1118-1127.