Endovascular treatment of visceral malperfusion in acute type B aortic dissection: our experience

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Purpose

Acute type B aortic dissection complicated by visceral malperfusion is a life-threatening condition with very high in-hospital mortality and morbidity rates, but also during follow-up after hospital discharge (1-2). Emergency therapy is mandatory to avoid clinical status deterioration. Aortic fenestration is often used as a first-line treatment option in this clinical condition, but it is still affected by very high perioperative mortality and morbidity rates, not only as a surgical traditional act, but even as an endovascular manoeuvre (3-5). Moreover, during follow-up, aortic fenestration does not favour a positive aortic remodeling, especially at abdominal level, with high risk of aneurysmatic infrarenal and subrenal evolution (5,6). Emergency endovascular treatment by aortic stent-graft placement could be another less invasive therapy option. Being its rationale to close the entry tear at thoracic aorta level with subsequent positive aortic remodeling (true lumen expansion, false lumen thrombosis and shrinkage) at both thoracic and abdominal aorta, thoracic endovascular treatment (TEVAR) could join an immediate or fast release from malperfusion syndrome without paying too much in terms of mortality and morbidity. Secondly, TEVAR may possibly avoid aneurysmatic evolution of the aortic dissection in the mid and long-term. Our purpose is to demonstrate the short and mid-term efficacy and the relative safety of TEVAR for the treatment of malperfusion syndrome as a first-line approach and standard of care, through the analysis of our ten-years experience in the management of acute type B aortic dissection.
Methods and Materials

From 1999 to 2011 19 consecutive patients (mean age 59.9±10) underwent TEVAR for acute type B dissection complicated by visceral malperfusion. 7 patients presented with signs of acute renal failure. We performed a retrospective analysis of CT-angiography, performed in all patients before and after the procedure during hospital stay and later during imaging follow-up in all but 5 patients. We evaluated the procedure success in terms of true lumen expansion at all aortic levels distal to stent-graft site and opacification of visceral vessels. Aortic remodeling was considered, besides true lumen expansion, as false lumen thrombosis and shrinkage at three levels: thoracic aorta (stent-graft level), suprarenal abdominal aorta (A1) and infrarenal abdominal aorta (A2). Aortic diameters were measured on CT at all these levels both before the procedure and at last CT-imaging follow-up. We also analyzed clinical evolution both during hospital stay and after discharge. Adverse events considered were: death (dissection and not-dissection related), neurological complications (paraplegia), endoleak (type I or III), renal failure and bowel ischemia. Statistical analysis was performed to compare aortic dimensions before and after the procedure (last imaging follow-up) at all aortic levels considered (Wilcoxon signed-rank test).
Results

Technical success with thoracic FL thrombosis and TL expansion was achieved in all patients, but one. Immediate release form malperfusion syndrome was achieved in 74% of cases (Fig.1-2). Mean hospital stay was 10.9 days. There were 2 in-hospital deaths (dissection-related) and 3 deaths after hospital discharge (one dissection-related) with an overall survival rate of 74% at 22 months (Fig.3), 2 renal failure and 2 bowel ischemia. 3 patients (15.7%) suffered from major neurological complications (2 transitory). Only 1 patient had a type I proximal endoleak (5%), successfully treated. The same patients was affected by a retrograde type A dissection, surgically treated.

5 (26%) patients needed a secondary endovascular procedure (one case in the acute phase), 4 successfully performed. Neurological morbidity were constantly associated to left subclavian artery overstenting without revascularization (due to emergency setting).

At last CT imaging follow-up all patients showed signs of positive aortic remodeling, with 100% false lumen thrombosis at thoracic level, complete thrombosis in 84% of cases. 63% false lumen thrombosis at abdominal aorta level, with 42% of complete thrombosis at suprarenal level and 21% at infrarenal level. False lumen shrinkage was achieved in 63% of cases at thoracic level, 26% at suprarenal abdominal level and 15% at infrarenal level. Statistical analysis with Wilcoxon signed-rank test (Fig.3a-c) shows no significant increment of aortic diameter at all levels, except for infrarenal abdominal aorta were it shows a slightly statistical significance due to true lumen expansion after stent-graft placement. As a whole it demonstrates the absence of aneurysmatic evolution of the distal aorta after TEVAR.
**Fig. 1:** Pre-operative angiography in acute type B aortic dissection with a severe malperfusion syndrome: at the injection in the true lumen at thoracic level, note the severe true lumen compression in infrarenal abdominal aorta, the absent opacification of mesenteric artery and the severe size reduction of right iliac artery (limb ischemia).

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Fig. 2: Same patient: angiography post stent-graft placement in thoracic aorta show a complete resolution of true lumen compression in abdominal aorta, right iliac artery and appearance of both mesenteric artery and left iliac artery, previously obstructed

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Fig. 3: Kaplan Meyer survival estimates of our series with a mean follow-up of 22.5 months. Note that all deaths occurred in the acute phase or at least in the first three months of follow-up.

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Fig. 4: No significant variation in thoracic diameters after stent procedure. Note that acute type B dissection with malperfusion do not generally have severe dilation at thoracic level, while stent positioning can explain slightly greater post-operative diameters.

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Aortic remodeling at Suprarenal abdominal aorta

\[ \text{Diameter (mm)} \]

\[ \begin{align*}
\text{A1 pre} & \quad \text{A1 post} \\
\end{align*} \]

\[ p = 0.35 \]

Fig. 5

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Aortic remodeling before and after Tevar

\[ \text{Diameter (mm)} \]

\[ \begin{align*}
\text{A2 pre} & \quad \text{A2 post} \\
\end{align*} \]

\[ p = 0.02 \]
**Fig. 6:** Slightly statistically significant elevation of abdominal diameters after stent-graft procedure, partly due to the true lumen expansion, but anyway no evident aneurysmatic evolution of the distal aorta even in the mid-long term.

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Conclusion

Malperfusion syndrome complicating acute type B aortic dissection is a life-threatening clinical condition that, despite advancing medical and surgical management, still carries a severe prognosis, as confirmed by our survival rate and rate of complications in the acute phase (1).

Anyway emergency TEVAR reduces mortality and morbidity in this setting showing a comprehensive minor invasivity regarding other approaches like aortic fenestration. Moreover, our results demonstrates that TEVAR also achieve a positive aortic remodeling even at the level of distal aorta (Fig.7), avoiding the aneurysmatic evolution of abdominal aorta. Consequently, TEVAR should be considered as a first line therapy and the standard of care of type B acute aortic dissection complicated by visceral malperfusion. Aortic fenestration must be performed only in specific cases where TEVAR alone does not resolve completely malperfusion or when TEVAR is not available. In our experience only one patient would have taken advantage form other approaches in addition to TEVAR, but in this case the problem was due to a static compression on celiac trunk and mesenteric artery by the false lumen, already thrombosed even from clinical onset.
Images for this section:

![CT scan of aortic dissection immediately after TEVAR](image)

**Fig. 7:** Aortic dissection immediately after TEVAR. Note perfusion of both lumen at suprarenal level

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**Fig. 8:** Same patient at 10 months after TEVAR: note complete false lumen thrombosis at suprarenal level

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**Fig. 9:** and at infrarenal level

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References


