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Management of Acute Type B Aortic Dissection with Malperfusion via Endovascular Fenestration/Stenting

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Abstract

Objective: To evaluate the management of malperfusion in acute type B aortic dissection (ATBAD) with endovascular fenestration/stenting.

Methods: From 1996–2018, 182 patients with an ATBAD underwent fenestration/stenting for suspected malperfusion based on imaging, clinical manifestations, and lab findings. Data were obtained from medical record review and the National Death Index database.

Results: The median age was 55-years-old. Signs of malperfusion included abdominal pain (61%), lower extremity weakness (27%), non-palpable lower extremity pulses (24%), and abnormal lactate, creatinine, liver enzymes, and creatine kinase levels. Confirmed hemodynamically significant malperfusion affected the spinal cord (2.7%), celiac (24%), superior mesenteric (40%), renal (51%), and iliofemoral (43%) arterial distributions. Of the 182 patients, 99(54%) underwent aortic fenestration/stenting, 108(59%) had one or multi-branch vessel fenestration/stenting, 5(2.7%) had concomitant TEVAR, 17(9.3%) had additional thrombolysis or thromboembolectomy, and 48(26%) received no intervention. Following fenestration/stenting, 24(13%) required additional procedures for necrotic bowel or limb and 9(4.9%) had subsequent aortic repair (TEVAR, open repair) before discharge. The new-onset paraplegia was 0%. The in-hospital mortality was 7.7% over 20+ years, 0% in the last 8 years. The 5- and 10-year survival was 72% and 49%, respectively. The significant risk factors for late mortality were age and acute paralysis (HR=3.5), both $p<0.0001$. Given death as a competing factor, the 5- and 10-year cumulative incidence of reintervention was 21% and 31% for distal aortic pathology.

Conclusions: ATBAD patients with malperfusion can be managed with endovascular fenestration/stenting with excellent short- and long-term outcomes. This approach is particularly helpful to patients with static malperfusion of aortic branch vessels.

Graphical Abstract

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Conflict of Interest: Dr. David Williams is on the Medical Advisory Board of Boston Scientific and Drs. David Williams and Himanshu Patel are consultants with Gore on an unrelated device.

INTRODUCTION

Malperfusion, a feared complication of aortic dissection, is present in around 20% of type B aortic dissections^{1,2} and is a risk factor for mortality² (mortality ranging from 17–64%)^{2–5}. An International Registry of Acute Aortic Dissection (IRAD) study showed 28% mortality in ATBAD with malperfusion compared to 9.6% in those without malperfusion². Malperfusion is defined as inadequate flow to a vascular territory, while malperfusion syndrome (MPS) is decreased flow to a vascular territory resulting in tissue/organ necrosis and end-organ dysfunction, both because of dissection-related obstruction of the aorta and its branch vessels. Patients with malperfusion more often undergo thoracic endovascular aortic repair (TEVAR) than open repair^{6–8} to alleviate the end-organ ischemia. However, TEVAR in ATBAD cannot reliably resolve static malperfusion of aortic branch vessels which results from thrombosis of the false lumen and compression of the true lumen of the aortic branch vessels, has the potential risk of acute paraplegia due to false lumen thrombosis of the descending thoracic aorta and its intercostal arteries when the entire descending thoracic aorta is covered, and carries approximately a 2%–5%^{9,10} risk of retrograde type A dissection.

Since 1996, we have adopted the approach of endovascular reperfusion via fenestration/stenting by interventional radiology (IR) of the critically malperfused organ systems for patients with ATBAD and malperfusion, which can resolve both dynamic and static malperfusion of aortic branch vessels. Previously we reported our 10-year experience of treating malperfusion in ATBAD¹¹. In this study, we report the short- and long-term outcomes of endovascular fenestration/stenting to treat malperfusion in ATBAD over the past 20 years.

MATERIALS AND METHODS

This study was approved by the Institutional Review Board at the University of Michigan, Michigan Medicine (Ann Arbor, MI).

Study Population

From February 1996 to May 2018, 182 patients presented with spontaneous ATBAD and suspected malperfusion and went to angiography for diagnosis and fenestration/stenting. Patients with ATBAD and malperfusion due to trauma (n=4) or malperfusion treated with isolated TEVAR (n=8) or open surgical repair were excluded. ATBAD was defined as onset within 14 days of admission with dissection confined to the aorta distal to the left subclavian artery. Investigators utilized medical record review to obtain pre-, intra-, and post-procedural characteristics. Reinterventions included open or endovascular aortic repair of the aorta distal to the left subclavian artery and were collected from a thorough medical record review. The National Death Index database through December 31, 2015¹² and medical record review were utilized to determine survival. Loss of follow-up was treated as a censor during the time to event analysis.

Diagnosis of Dynamic and Static Malperfusion

Malperfusion, inadequate blood flow to the end organs, could be diagnosed with radiographic findings consistent with reduced or absent flow to an end organ or complete true lumen collapse on computed tomography (CT), including disappearance of the aortic double lumen indicating elimination of the true lumen with the dissection flap being pushed against the aortic wall causing obstruction of flow to branch vessels, continuation of dual lumen patency with absent flow in a branch vessel (dynamic malperfusion), and dissection into a branch vessel or a thrombosed false lumen in a branch vessel (static malperfusion), with or without clinical evidence of end-organ dysfunction (Figure 1). Malperfusion syndrome involves tissue/organ necrosis and end-organ dysfunction as a result of inadequate blood flow (malperfusion) and requires clinical features (abdominal pain, bloody diarrhea, tenderness to palpation, decreased urine output, absence of peripheral pulses, motor or sensory deficit of the lower extremity) and laboratory findings (elevated lactate, liver enzymes, metabolic acidosis, elevated creatinine) in addition to radiographic findings. The etiology of the malperfusion can be static, dynamic, or both static and dynamic obstruction of a branch vessel¹³. Dynamic malperfusion results from the dissection flap of a collapsed true lumen prolapsing across the origin of the branch vessel and obstructing flow and can vary in severity depending on variations of pressure in the false lumen. Dynamic obstruction can usually be resolved with restoration of the true lumen with a TEVAR endograft covering the intimal tear or aortic fenestration/stenting. Static obstruction results from extension of the dissection flap into a branch vessel, frequently accompanied by false lumen thrombosis due to no or very small re-entry tear and occlusion of the true lumen, and is present throughout the cardiac cycle. Total occlusion of a vessel, like the superior mesenteric artery (SMA), by a thrombosed false lumen can lead to thrombosis of the true lumen distal to the dissection. Furthermore, collateral flow to an obstructed SMA is often compromised by dissection-related compromise of the celiac trunk or inferior mesenteric artery. Static obstruction usually requires stenting or other intervention (fenestration/thromboembolectomy/thrombolysis) of the affected branch vessel to restore flow. Both static and dynamic obstruction can be resolved with endovascular reperfusion via fenestration/stenting. In contrast to our management in type A aortic dissection, where MPS is the indication for IR procedures, in type B aortic dissection suspected malperfusion (not MPS) is an indication for IR.

Endovascular Techniques

Angiography was completed a median of one day following hospital admission. The angiographic evaluation of malperfusion has been previously described in detail^{13–16}. In angiography, treatable malperfusion was indicated by ongoing arterial obstruction and was confirmed by a systolic blood pressure gradient >15 mmHg between the ascending aorta and the branch vessel. If a branch artery is dissected, branch artery manometry is performed distal to the dissection (confirmed by intravascular ultrasound [IVUS]). The gradient of 15 mmHg was chosen as the criterion for malperfusion based on the customary acceptance of a blood pressure differential of >20 mmHg as indicating hemodynamic significance in patients with aortic coarctation¹⁷. Fenestration and stenting were performed by creating a tear in the dissection flap 2–4 cm above the celiac artery using a 16 mm diameter balloon, thereby permitting flow between the true and false lumens, followed by deployment of a 16–18 mm diameter closed-cell self-expanding stent (Wallstent, Boston Scientific Corporation, Marlborough, MA, off-label application) exclusively in the aortic true lumen (Central Picture), as previously described^{13,16,18,19} if the true lumen remains collapsed or a gradient between the aortic root and abdominal aorta persists following aortic fenestration. Blood pressure gradients between the aorta and the branch vessels were determined both before and after fenestration/stenting (Wallstents). If after aortic fenestration/stenting a significant gradient persisted between the aorta and a branch vessel, then branch vessel fenestration/stenting, thrombolysis, or thromboembolectomy was performed as appropriate, based on angiographic and IVUS findings. (Central Picture) Complete resolution of malperfusion was defined as blood pressure gradient decreased to <15 mmHg. In dissected vessels with thrombosed false lumens, gradients after stenting might exceed 15 mmHg, defined as partial resolution of malperfusion, but as long as absolute perfusion pressure was viable, i.e., >60 mmHg, post-dilation of stents was not performed.

Concomitant ($n=5$) or post-IR TEVAR ($n=4$) or open aortic repair ($n=5$) were indicated for pending rupture, refractory back pain, uncontrollable severe hypertension, and aortic aneurysm. Concomitant TEVAR includes patients who initially had TEVAR for back pain/impending rupture with persistent post-operative static malperfusion subsequently treated by fenestration/stenting. All open procedures were performed before 2004. Post-procedure management consisted of aspirin therapy, blood pressure control, standard management of end-organ dysfunction, and adequate analgesia and sedation. When bowel ischemia or extremity ischemia was present, general or vascular surgery was consulted, respectively, to determine if exploratory laparotomy or fasciotomies were indicated.

Statistical Analysis

Continuous variables were summarized by median (25%, 75%) and categorical variables were reported as n (%) in frequency tables. Crude survival curves since admission were estimated using the non-parametric Kaplan-Meier method. Multivariable logistic regression was performed to calculate the odds ratio (OR) of risk factors for in-hospital mortality adjusting for age, gender, coronary artery disease (CAD), acute myocardial infarction (MI), acute renal failure on dialysis, acute paralysis, celiac malperfusion, mesenteric malperfusion, renal malperfusion, and extremity malperfusion. Cox proportional hazard regression was performed to calculate the hazard ratio (HR) for late mortality by stepwise selection of

variables including age, gender, coronary artery disease, chronic renal failure on dialysis, acute myocardial infarction, acute paralysis, acute renal failure requiring dialysis, malperfusion syndrome found, bowel resection, amputation, and fasciotomy. As patients may experience death before reintervention was indicated, cumulative incidence curves adjusting for death as the competing risk were generated to assess cumulative incidence of reintervention over time. Cox regression was used to calculate the risk factors of reintervention adjusting for age, gender, connective tissue disease, aortic flap fenestration without TEVAR or open aortic repair, and hypertension. All statistical calculations used SAS 9.4 (SAS Institute, Cary, NC) and were considered significant at $p < 0.05$.

RESULTS

Demographics and Pre-procedural Data

The median age was 55-years-old and most patients (88%) had hypertension. The majority (93%) of patients were transferred from another hospital. Patients frequently presented with abdominal pain (61%), lower extremity weakness (27%) with non-palpable pulses (24%), and elevated creatinine and liver enzymes. Some already had certain vascular procedures for limb ischemia prior to presentation (Table 1).

Procedural Data

Renal, extremity, and mesenteric malperfusion were the most frequently suspected and confirmed malperfusion in ATBAD. Frequently, multiple vascular beds were affected. Overall, 74% of patients had interventions in the angiography suite, including aortic fenestration/stenting (54%), branch vessel fenestration/stenting (59%), or thrombolysis/thrombectomy/embolectomy (9.3%) for static malperfusion. (Table 2, Figure 1) Branch artery stenting was performed in the iliac, renal, superior mesenteric, and celiac arteries 56, 47, 37, and 8 times, respectively. Overall, 93% of malperfusion was completely resolved and 5% was partially resolved (Table 3). Following endovascular fenestration/stenting, 13 patients required laparotomy for suspected bowel ischemia, including 9 patients who had bowel resection, and 12 patients required additional vascular procedures during the current hospital stay. Nine patients required additional TEVAR ($n=4$) or open aortic repair ($n=5$) due to aortic aneurysm, pending rupture, and persistent symptoms. (Table 2)

Postoperative Outcomes

Post-procedural new-onset paraplegia (0%), retrograde type A dissection (0%), and new-onset acute renal failure requiring dialysis (1.6%) were low. In five patients with spinal cord malperfusion, two had complete resolution and one had partial resolution of paraplegia. Overall in-hospital mortality was 7.7% over 20 years, 11.3% in the first decade (1996–2007), 3.5% in the second decade (2008–2018), and 0% in the last 8 years. (Table 4) The significant risk factors for in-hospital mortality were age ($OR=1.15$), acute MI ($OR=8.6$), acute paralysis ($OR=11.5$), and extremity malperfusion ($OR=8.8$) (Table 5). Detailed causes of death included aortic rupture, extensive necrotic intestine, arrhythmia, and stroke. (Supplemental Table 1)

Long-term Outcomes

The 5- and 10-year survival was 72% (95% CI: 64%, 78%) and 49% (95% CI: 39%, 58%) respectively. (Figure 2A) The significant risk factors for late mortality were age [HR=1.04, (95% CI: 1.02, 1.06)] and acute paralysis [HR=3.5, (95% CI: 1.8, 6.8)], $p<0.001$.

Of the 182 patients, 14 patients died in the hospital before discharge and 12 (6.6%) were lost to follow-up for reintervention. The mean follow-up time was 6.2 years. The 5- and 10-year cumulative incidence of reintervention for pathology of the descending or thoracoabdominal aorta adjusted for death as a competing factor was 21% (95% CI: 15%, 28%) and 31% (95% CI: 23%, 40%) (Figure 2B). The significant risk factors for reintervention were connective tissue disease [HR=3.4 (95% CI: 1.4, 8.3), $p=0.007$] and male gender [HR=3.2, (95% CI: 1.3, 8.0), $p=0.014$]. Fenestration without TEVAR or open repair was not a significant risk factor [HR=0.8, (95% CI: 0.4, 1.5), $p=0.49$]. The 5- and 10-year cumulative incidence of reintervention for patients undergoing fenestration/stenting only, without TEVAR or open repair during the hospital stay, was 20% (95% CI: 13%, 28%) and 31% (95% CI: 21.5%, 41%) (Figure 2C). The primary indication for reintervention was aortic aneurysm (91%) and was primarily done through open repair (76%). The median interval time to reintervention was two years.

DISCUSSION

In this study, we managed ATBAD complicated by malperfusion with endovascular fenestration/stenting, which enables resolution of both dynamic and static malperfusion. The in-hospital mortality was 7.7% over 20+ years and 0% in the last 8 years; the cumulative incidence of reintervention was 21% at 5 years and 31% at 10 years; and the 5- and 10-year survival was 72% and 49% respectively.

Aortic dissection, both type A and B, can be complicated by malperfusion due to dissection-related obstruction of aortic branch vessels. In acute type A aortic dissection, because of the risks of aortic rupture, acute heart failure and aortic insufficiency, acute myocardial infarction, pericardial effusion and tamponade, and neurologic complications^{20,21}, we pursue upfront angiography with endovascular reperfusion only for patients with malperfusion syndrome (malperfusion with tissue/organ necrosis and end-organ dysfunction). In ATBAD patients, we extend this strategy to patients with malperfusion unresponsive to blood pressure and heart rate control and patients with a documented history of poor compliance with anti-hypertensive medication in addition to patients with MPS. Patients with acute type B aortic dissection have a lower risk of aortic rupture with adequate blood pressure management^{11,22} and a small risk of the proximal aortic complications commonly seen with type A dissections²³. Malperfusion due to dynamic obstruction, which is generally corrected by proximal aortic repair in ATAAD patients, may persist in ATBAD patients unless dealt with directly by fenestration/stenting or TEVAR. In summary, malperfusion, in addition to malperfusion syndrome, is an indication for angiography for diagnosis and potential treatment in ATBAD patients. Because clinical manifestation of mesenteric and renal malperfusion may lag their CT demonstration and unsuspected vascular beds with malperfusion are frequently identified when we investigate malperfusion of suspected vascular beds, we consider the endovascular evaluation of ATBAD patients an

angiographic emergency and have a low threshold for performing it. The angiographic evaluation of these patients typically involves manometry, IVUS examination, and hand injection of 7 ml of contrast material diluted 1: 1 with normal saline into the SMA, bilateral renal arteries, and external iliac arteries. In the case of SMA or renal artery dissection associated with a significant pressure deficit, IVUS examination is performed to determine radiographic landmarks of the dissection terminus, in order to aid in stent placement.

Management options for ATBAD complicated with malperfusion have generally included open surgical repair and more recently TEVAR. Although TEVAR has reduced early mortality to around 10% in all ATBAD^{1,7,24–26}, we still use fenestration/stenting as our main stream treatment for malperfusion in ATBAD for the following reasons: (1) TEVAR has risks of retrograde type A dissection²⁷ (1.6% in large registry¹⁰) and spinal cord ischemia and paraplegia (2–10%^{28–30}), (2) TEVAR alone cannot reliably resolve static malperfusion, (3) TEVAR sometimes has to cover the left subclavian artery to cover the primary intimal tear, which requires additional procedures (such as left carotid artery-subclavian artery bypass) to preserve blood flow to the left subclavian artery, and (4) When patients suffer from necrotic bowel or limb and sepsis, TEVAR has a higher risk of graft infection. With endovascular fenestration/stenting, we can avoid all the risks from TEVAR and adequately treat static malperfusion with branch vessel stenting, fenestration, thromboembolectomy, or thrombolysis.

In this study, 59% of patients had fenestration/stenting of an aortic branch vessel and 9.3% had thrombolysis or thromboembolectomy for static malperfusion which could not be resolved by TEVAR alone. Since we did not cover any intercostal arteries, protecting the spinal cord from ischemic injury, our post-procedural new-onset paraplegia due to ischemic spinal cord injury was 0%, which is lower than those treated with TEVAR alone (2–10%^{28–30}). The in-hospital mortality rate was 7.7% with this approach in this sick sub-population (ATBAD with malperfusion) with 0% mortality in the last 8 years as we became more experienced with fenestration/stenting and with utilization of TEVAR for aortic pending rupture or rupture, which is lower than that seen with open repair and with TEVAR alone^{1,6,7,24–26,31}, possibly because TEVAR alone does not reliably correct static obstruction. Other reasons for improved mortality include better imaging, prompt diagnosis, treating suspected malperfusion in an acute dissection as an angiographic emergency, and better ICU management (blood pressure control). Endovascular fenestration/stenting of ATBAD with malperfusion combined with TEVAR and open repair achieved favorable survival (5-year and 10-year survival: 72% and 49%) which was better than or similar to those treated with TEVAR or open repair alone^{7,8,24,25,31,32}. The significant risk factors for late mortality were age and acute paralysis (HR=3.5). By decreasing the risk of new-onset paraplegia, endovascular fenestration/stenting could potentially decrease the late mortality.

Our approach is based on risk stratification to determine best management. In the setting of ATBAD with malperfusion without signs of rupture (persistent or increasing back pain), we think the malperfusion is the most immediate concern and elect to treat the malperfusion with percutaneous fenestration/stenting. This approach accomplishes the goal of resolving the malperfusion and essentially “converts” a complicated ATBAD to an uncomplicated ATBAD and allows patients to recover with medical management afterwards. If patients had

rupture/pending rupture, refractory back pain, uncontrollable hypertension, or large aortic aneurysm, a concomitant or delayed TEVAR or open aortic repair was performed as is seen in a small portion of this cohort (n=14, 7.7%) (Table 2). Managing ATBAD with malperfusion via fenestration/stenting does not prevent aortic rupture, as would open surgery or TEVAR. In this study, seven patients (3.8%) possibly died from aortic rupture a median of 3 days (IQR: 2, 4.5 days) following angiography with fenestration/stenting, which all happened before 2011 when TEVAR was not commonly used at our institution. Aortic rupture may have been prevented in these patients if they had undergone TEVAR, although cases of rupture have been reported during²⁴ and following TEVAR^{1,24,25,33}. After 2010, we applied endovascular fenestration/stenting, combined with TEVAR and open repair as appropriate, to all patients with ATBAD and malperfusion, with a 0% aortic rupture rate and 0% in-hospital mortality. In summary, endovascular fenestration/stenting is a very effective tool to treat malperfusion (dynamic and static) in ATBAD and is a valuable adjunct to both medical and surgical therapy (TEVAR and open repair). The fenestration/stenting approach does not exclude TEVAR or open repair of the aorta. If needed, all three approaches can be used to treat ATBAD with different complications.

For patients with malperfusion syndrome (late stage malperfusion with tissue/organ necrosis and dysfunction), endovascular fenestration/stenting resolves the malperfusion with minimal operative trauma and provides the opportunity for patients to recover from malperfusion syndrome. Additional intervention may be needed in order to recover from severe malperfusion syndrome. In our study, 24 patients (13%) required general and/or vascular surgery intervention for bowel and extremity necrosis following angiography, including bowel resection, fasciotomy and amputations (Table 2). Of these 24 patients, 20 patients had branch vessel stenting during angiography for static malperfusion, which could not have been treated with open repair or TEVAR alone. This highlights the gravity of the malperfusion syndrome; despite initial reperfusion of affected vascular territories, patients may still suffer complications of the pre-existing malperfusion and subsequent reperfusion. With prolonged static malperfusion, as would be with initial open repair or TEVAR, it is suspected that more patients would suffer from irreversible, unsalvageable end-organ death.

One concern about leaving a patent false lumen after fenestration/stenting is that it could increase the risk of reintervention. TEVAR could facilitate aortic remodeling by thrombosing and stabilizing the size of the thoracic false lumen due to closure of the primary intimal tear with a covered stent graft³³. We think the aortic flap fenestration/stenting approach achieves the same goal by creating a distal fenestration as outflow for the false lumen to decompress and prevent the dilation of the false lumen even though the proximal primary intimal tear is open. Burris et al.³⁴ found that in chronic type B aortic dissection the false lumen dilates very quickly with a large proximal primary intimal tear and a small distal re-entry tear due to high pressure in the false lumen during diastole evident as regurgitant blood flow from the false into the true lumen through both proximal and distal intimal tears. If the distal re-entry tear is large, there is no regurgitant flow from false to true lumen and there is minimal growth of the false lumen and the dissected aorta³⁴. Our approach, endovascular fenestration of the distal aortic flap, serves exactly the same purpose. As a result, the 5- and 10-year cumulative rate of reintervention with fenestration/stenting alone was 20% and 31%, respectively, adjusting for death as a competing factor (Figure 2B, 2C),

which is similar if not better than that reported with TEVAR^{24,32,35} and open repair⁷ alone; and we had longer follow-up than most studies since TEVAR is a more recent technology. Most of the studies using TEVAR in the literature use freedom from reintervention and do not adjust for late death as a competing factor, which could underestimate the rate of reintervention. Taken together, we do not think endovascular fenestration/stenting alone treating malperfusion in ATBAD increases the risk of reintervention compared to TEVAR.

Our study is limited by a single-center and retrospective experience. The management strategy of angiography evaluation and endovascular fenestration/stenting has a learning curve. Because the follow-up of reintervention was 93.4% complete, we could underestimate the rate of reintervention. This study is also limited by lack of a direct comparison group, such as TEVAR alone.

CONCLUSION

In patients with ATBAD complicated by malperfusion, endovascular fenestration/stenting can effectively resolve the malperfusion and achieve favorable short- and long-term results with additional indicated TEVAR or open aortic repair. We recommend endovascular fenestration/stenting when treating ATBAD with malperfusion, especially in patients with static malperfusion.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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GLOSSARY OF ABBREVIATIONS

ATAAD	acute type A aortic dissection
ATBAD	acute type B aortic dissection
CAD	coronary artery disease
CI	confidence interval
CT	computed tomography
HR	hazard ratio
IQR	interquartile range
IR	interventional radiology

IRAD	International Registry of Acute Aortic Dissection
IVUS	intravascular ultrasound
MPS	malperfusion syndrome
OR	odds ratio
SMA	superior mesenteric artery
TEVAR	thoracic endovascular aortic repair

Biography

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Central Picture Legend:

A) Aortic flap balloon fenestration; B) Thoracic aortic true lumen, and C) SMA stenting.

Central Message:

Endovascular fenestration/stenting can effectively resolve dynamic and static malperfusion in acute type B aortic dissection with favorable short- and long-term outcomes (survival and reoperation).

Perspective Statement:

Endovascular fenestration/stenting effectively and timely resolves dynamic and static malperfusion in acute type B aortic dissection with minimal risk of paraplegia and retrograde type A dissection, and excellent in-hospital mortality, cumulative incidence of reintervention, and long-term survival in this sick patient population as combined with TEVAR or open repair when they are indicated.

Figure 1:

Computed tomography (CT) angiogram of a 55-year-old man with an acute type B aortic dissection and static malperfusion of the celiac and superior mesenteric arteries before and eight years after endovascular fenestration/stenting. Axial CT at the level of the superior mesenteric artery (SMA) (A) shows thrombosed false lumen (arrowhead) just beyond the SMA origin. Sagittal CT of the upper abdomen through the false lumen of the aorta (B) again shows thrombus in the terminal portion of the false lumen of the celiac trunk and the SMA, resulting in arterial occlusion. 3D reconstruction of the abdominal aorta (C) shows the dissection flap cleaving the celiac and SMA origins with thrombosed false lumen (red), with the inferior mesenteric artery (IMA) supplied by the false lumen. Superior mesenteric arteriogram (D), approximately 28 hours after symptom onset, shows proximal occlusion of the SMA trunk (arrowheads), absent filling of jejunal and ileal branches, and retrograde filling of the celiac distribution through pancreatic collaterals. Following stenting of the SMA and celiac trunk, distal SMA pressure was 57/43 mmHg, 32 mmHg lower than aortic true lumen pressure. Sheath injection at the celiac origin after stenting (E) fills hepatic and splenic arteries and refluxes into the abdominal aorta, filling stented SMA (arrowhead) with jejunal and ileal branches (asterisks). CT with 3D-reconstruction 8 years later (F) shows the celiac artery (C) stent extending into the common hepatic artery (H) with jailed but patent splenic artery (S). The SMA stent (arrowhead) is patent down to the ileocolic artery. Several small jejunal and ileal branches continue to fill through stent interstices. A prominent IMA

(not shown) supports flow through the middle colic artery (MC). T= true lumen, F = false lumen, H = hepatic artery, IMA = inferior mesenteric artery, MC = middle colic artery, S = splenic artery.

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Figure 2A:

Kaplan-Meier survival analysis of patients with an acute type B aortic dissection and malperfusion syndrome undergoing endovascular fenestration/stenting. The 5- and 10-year survival was 72% (95% CI: 64%, 78%) and 49% (95% CI: 39%, 58%) respectively.

Figure 2B:

The cumulative incidence of reintervention of the whole cohort (n=182) for pathology of the descending thoracic or thoracoabdominal aorta following hospital discharge, adjusting for death as the competing event. The 5- and 10-year cumulative incidence of reintervention was 21% (95% CI: 15%, 28%) and 31% (95% CI: 23%, 40%) respectively.

Figure 2C:

The cumulative incidence of reintervention for patients who had only fenestration/stenting (n=125) following hospital discharge, adjusting for death as the competing event. The 5- and 10-year cumulative incidence of reintervention was 20% (95% CI: 13%, 28%) and 31% (95% CI: 21.5%, 41%) respectively.

Table 1.**Demographics and Characteristics before Procedures by Interventional Radiology**

Variables	Total (n=182)
Patient age (years)	55 (48, 65)
Sex (male)	139 (76)
BMI	30 (26, 33.5)
Hypertension	160 (88)
Hyperlipidemia	47 (26)
Diabetes	15 (8.2)
COPD	23 (13)
CAD	40 (22)
PVD	18 (9.9)
History of Smoking	
None	61 (34)
Former	43 (24)
Current	78 (43)
History of MI	17 (9.3)
History of Stroke	12 (6.6)
History of Renal Failure	20(11)
On Dialysis	3 (1.6)
Connective Tissue Disorder	12 (6.6)
Acute MI	25 (14)
Acute Stroke	1 (0.5)
Worsening Renal Function	91 (50)
Requiring Dialysis	5 (2.7)
Acute Paralysis	12 (6.6)
Prior Cardiac Surgery	30 (16)
Prior Aortic Intervention	29 (16)
Location of Initial Admission	
University of Michigan	12 (7)
Outside Hospital	170 (93)
Admission to IR (days)	1 (0, 2)
<u>Presenting Signs and Symptoms</u>	
Abdominal Pain	111 (61)
Lower Extremity Weakness	49 (27)
Lower Extremity Pulses Non-palpable	43 (24)
Lab Values	
Creatinine on Admission (mg/dL)	1.2 (0.9, 1.6)
Maximum Creatinine before IR (mg/dL)	1.5 (1.0, 2.1)
Maximum lactate before IR (mmol/L)	1.5 (1.0, 2.2)
Elevated AST/ALT	66 (37)

Variables	Total (n=182)
Max CK (U/L)	124 (58.5, 232)
<u>Procedures During Hospital Stay before IR</u>	
Exploratory Laparotomy for Suspected Ischemia	0 (0)
Vascular Surgery for Suspected Ischemia	6 (3.3)
Thrombolysis/Stenting of SMA	1 (0.5)
Thrombectomy/Embolectomy	2 (1.1)
Fem-Fem Bypass	4 (2.2)
Fasciotomy	2 (1.1)
Amputation	0 (0)
Aortic Surgery	3 (1.6)
TAVR	1 (0.5)
Median Sternotomy *	1 (0.5)
Open TAA	1 (0.5)

Data presented as median (25%, 75%) for continuous data and n (%) for categorical data. ALT = alanine aminotransferase; AST = aspartate aminotransferase; BMI = body mass index; CAD = coronary artery disease; CK = creatine kinase; COPD = chronic obstructive pulmonary disease; MI = myocardial infarction; PVD = peripheral vascular disease; SMA = superior mesenteric artery; TAVR = transcatheter aortic valve replacement; TAA = thoracic aortic aneurysm.

* Acute type B aortic dissection noted intraoperatively during aortic root repair.

Table 2.**IR Indications and Procedures and Subsequent Procedures During Hospital Stay**

Variables	Total (n=182)
<u>IR Indications</u>	
Malperfusion Suspected	182 (100)
Spinal Cord	8 (4.4)
Celiac	22 (12)
Mesenteric	81 (45)
Renal	119 (65)
Extremity	71 (39)
Malperfusion Found	140 (77)
Spinal Cord	5 (2.7)
Celiac	43 (24)
Mesenteric	73 (40)
Renal	93 (51)
Extremity	79 (43)
<u>IR Procedures</u>	
No Intervention in IR	48 (26)
Aortic Fenestration/Stenting	99 (54)
Branch Vessel Fenestration/Stenting	108 (59)
Concomitant TEVAR	5 (2.7)
Additional Procedures *	17 (9.3)
<u>Subsequent Procedures During Hospital Stay</u>	
Exploratory Laparotomy for Suspected Ischemia	13 (7.1)
Bowel Resection	9 (4.9)
Vascular Surgery for Suspected Ischemia	12 (6.6)
Thrombectomy/Embolectomy	6 (3.3)
Fem-Fem Bypass	2 (1.1)
Fasciotomy	5 (2.7)
Amputation	3 (1.6)
Aortic Surgery	9 (4.9)
TEVAR	4 (2.2)
Open TAA/A	4 (2.2)
Open AAA	1 (0.5)
Time from IR (days)	12 (9, 14)

Data presented as n (%).

* Thrombolysis, thrombectomy, embolectomy of aortic branch vessels.

AAA = abdominal aortic aneurysm; IR = interventional radiology for fenestration/stenting; TAA/A = thoracic aortic aneurysm (TAA) or thoracoabdominal aortic aneurysm (TAAA); TEVAR = thoracic endovascular aortic replacement. TEVAR was performed by both IR faculty and cardiac surgeons together.

Detailed IR Procedures

Table 3.

Level of aortic fenestration/stenting	Aortic Fenestration (n=87)	Aortic Stenting (n=89)	Branch Vessel Fenestration (n=2)	Branch Vessel Stenting (n=105)	Malperfusion in Vascular Bed (n=182)	Malperfusion Completely Resolved ⁺	Malperfusion Partially Resolved ⁺
Descending thoracic	6	5	-	-	-	-	-
Supraceliac	22	12	-	-	-	-	-
Celiac	17	2	0	8	43	42	1
Supramesenteric	17	45	-	-	-	-	-
Mesenteric	15	0	0	37	73	65	8
Suprarenal	1	0	-	-	-	-	-
Renal [*]	7	5	0	47	93	87	2
Infrarenal	44	62	-	-	-	-	-
Iliac [*]	-	-	2	56	79	74	4

IR = interventional radiology for fenestration/stenting. In the columns 2 –5, n means number of patients. In column 6, n means the total malperfusion found in different vascular beds, including celiac artery, superior mesenteric artery, renal arteries, and common iliac arteries and their branches (external iliac arteries, femoral arteries).

Aortic stenting: If there was a compression of the true lumen by the thrombosed false lumen in the descending aorta, we placed a 16 mm self-expanding bare stent in the descending aorta (**Descending thoracic aortic stenting**). After fenestration of aortic dissection flap, we place the same self-expanding bare stent in the distal descending thoracic aorta if needed to keep the true lumen open. The distal descending thoracic aortic stent was placed frequently above the superior mesenteric artery (**Supramesenteric aortic stenting**). If we had to place stents in the superior mesenteric artery, then we place the aortic stent above the celiac artery (**Supraceliac aortic stenting**). The goal of aortic stenting is to achieve adequate expansion of the true lumen of dissected aorta and eliminate blood pressure gradient between distal aorta and ascending aorta.

^{*} Among 47 patients receiving renal artery stenting, 38 (81%) received unilateral stenting (right renal, 38% and left renal, 43%) and 9 (19%) had bilateral renal artery stenting. Among 56 patients receiving iliac stenting, 42 (75%) had aorto-iliac stenting, 32 (57%) had common iliac artery stenting, 33 (59%) had external iliac artery stenting, and 7 (12.5%) had common femoral artery stenting. Four renal artery and one iliac malperfusion were unable to be treated due to an inability to cannulate the specific branch vessel (n=1, renal artery), anatomy unsuitable for stenting (n=3, renal artery), or a significant gradient without symptoms of malperfusion (n=1, iliac).

⁺ Complete resolution of malperfusion was defined as the systolic blood pressure gradient between the branch vessel and ascending aorta is < 15 mmHg after endovascular fenestration/stenting. Partial resolution of malperfusion was defined as systolic blood pressure gradient was still > 15 mmHg.

Table 4.

Post-procedural Outcomes

Variables	Total (n=182)
Stroke	10 (5.5)
Continued Acute Renal Failure Requiring New Dialysis	14 (7.7)
New-onset Renal Failure	10 (5.5)
Requiring Dialysis	3 (1.6)
Dialysis at Discharge	2 (1.1)
New-onset Paraplegia	0 (0)
Preoperative Paraplegia resolved *	3 (60)
GI Bleed	1 (0.5)
Groin Hematoma	8 (4.4)
Length of Stay (days)	11 (8, 18)
In-hospital Mortality	14 (7.7)

Data presented as median (25%, 75%) for continuous data and n (%) for categorical data.

* Five patients had preoperative paraplegia due to spinal cord malperfusion, two patients' paraplegia resolved completely and one patient's resolved partially.

Table 5.

Risk Factors for In-hospital Mortality (Multivariable Logistic Regression)

Variables	Odds Ratio (95% Confidence Interval)	p-value
Age	1.15 (1.06, 1.25)	0.0009
Male Gender	3.31 (0.68, 16.09)	0.14
CAD	0.65 (0.10, 4.43)	0.66
Acute MI	8.59 (1.35, 54.42)	0.02
Acute Renal Failure Requiring Dialysis	1.80 (0.11, 28.88)	0.68
Acute Paralysis	11.5 (1.83, 72.38)	0.009
Found Celiac Malperfusion	1.38 (0.20, 9.61)	0.74
Found Mesenteric Malperfusion	3.16 (0.43, 23.05)	0.26
Found Renal Malperfusion	2.88 (0.58, 14.28)	0.19
Found Extremity Malperfusion	8.83 (1.43, 54.78)	0.02

CAD = coronary artery disease; MI = myocardial infarction.