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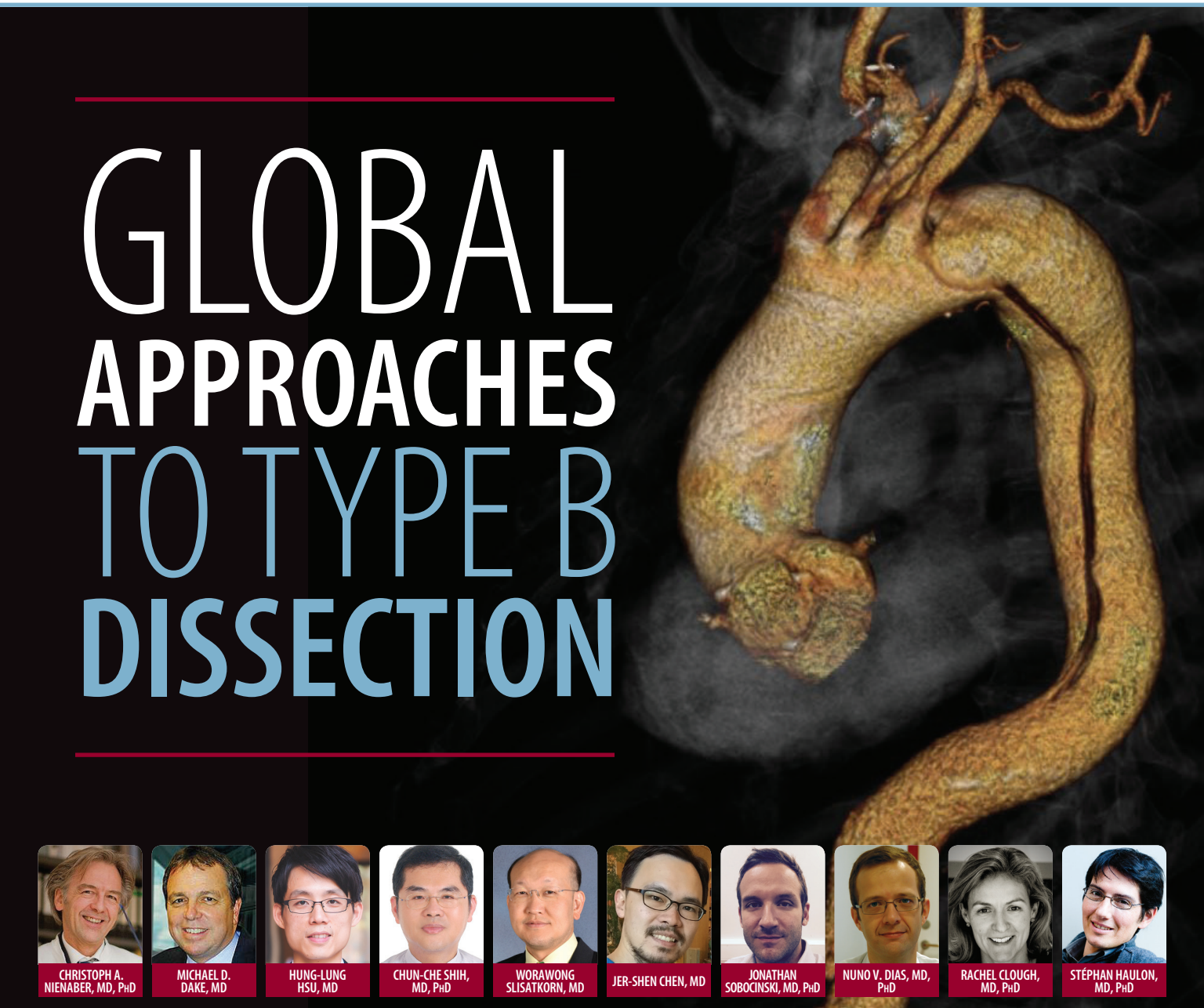
GLOBAL APPROACHES TO TYPE B DISSECTION



CHRISTOPH A. NIENABER, MD, PHD

MICHAEL D. DAKE, MD

HUNG-LUNG HSU, MD

CHUN-CHE SHIH, MD, PHD

WORAWONG SLISATKORN, MD

JER-SHEN CHEN, MD

JONATHAN SOBOCINSKI, MD, PHD

NUNO V. DIAS, MD, PHD

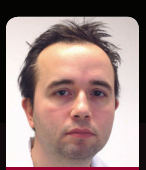
RACHEL CLOUGH, MD, PHD

STÉPHAN HAULON, MD, PHD


PETER MOSSOP, MD


IAN NIXON, MD

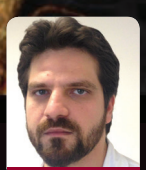

MASAAKI KATO, MD


GEORGE N. KOUVELOS, MD


TILO KÖLBEL, MD, PHD


ATHANASIOS KATSARGYRIS, MD


NIKOLAOS TSILIMPARIS, MD, PHD


KYRIAKOS OIKONOMOU, MD


ERIC L.G. VERHOEVEN, MD, PHD

Contents

3 **WHERE ARE WE HEADED WITH THE TREATMENT OF TYPE B AORTIC DISSECTION?**
By Christoph A. Nienaber, MD, PhD

4 **TIMING OF TEVAR TREATMENT**
When should treatment occur in acute, initially uncomplicated type B aortic dissection?
By Michael D. Dake, MD

7 **DISTAL STENT GRAFT-INDUCED NEW ENTRY**
The current paradigm and future management of dSINE.
By Hung-Lung Hsu, MD, and Chun-Che Shih, MD, PhD

10 **SEEING THE SIGNS**
The top precursors/predictors for earlier endovascular treatment versus medical management of type B aortic dissection.
By Worawong Slisatkorn, MD

12 **TREATING COMPLICATED ACUTE TYPE B AORTIC DISSECTION WITH PROXIMAL STENT GRAFTS AND DISTAL BARE STENTS**
Bare aortic stents can expand the true lumen immediately and result in early favorable aortic remodeling.
By Jer-Shen Chen, MD

17 **EXPLORING THE USE OF BARE STENTS IN THE TREATMENT OF TYPE B DISSECTION**
Understanding the effects on aortic remodeling and malperfusion.
By Jonathan Sobocinski, MD, PhD; Nuno V. Dias, MD, PhD; Rachel Clough, MD, PhD; and Stéphan Haulon, MD, PhD

22 **TOWARD THE UNILUMINAL STATE**
Evolving concepts in the endovascular elimination of aortic dissection.
By Peter Mossop, MD, and Ian Nixon, MD

26 **TEVAR FOR CHRONIC TYPE B AORTIC DISSECTION**
A discussion of therapeutic strategies for chronic type B aortic dissection, avoiding expansion of the false lumen, and treatment with endovascular therapy.
By Masaaki Kato, MD

30 **POSTDISSECTION ANEURYSMS**
Devices and techniques for treating this condition.
By George N. Kouvelos, MD; Tilo Kölbel, MD, PhD; Athanasios Katsargyris, MD; Nikolaos Tsilimparis, MD, PhD; Kyriakos Oikonomou, MD; and Eric L.G. Verhoeven, MD, PhD

Where Are We Headed With the Treatment of Type B Aortic Dissection?

BY CHRISTOPH A. NIENABER, MD, PHD

As a direct consequence of demographic changes and an aging population, along with an increasing awareness of the disease and better diagnostic logistics, the true (population-based) incidence of acute aortic syndrome is rising, with up to 35 cases per 100,000 persons per year in the 65- to 75-year-old age group.[1]

Whereas open surgery with cardiopulmonary bypass is the main therapeutic option when the proximal ascending aorta is involved, open surgical approaches have essentially failed in the setting of dissection confined to the distal arch or descending aorta.[2] Although type B or distal dissection does not usually present with immediate, life-threatening complications, this condition is nonetheless threatening life with delayed mortality and morbidity.

In the setting of complications, such as malperfusion from obstruction of any side branch or the aorta itself, contained rupture (with extra aortic blood collection), or inflammatory signs of impending ruptures, thoracic endovascular aortic repair (TEVAR) has emerged as the first-line therapeutic option with promising results and a recent class I level of evidence C recommendation.[2-5]

In the absence of signs of obvious life-threatening complications and with adequate response to blood pressure-lowering medications, a more elective approach appears sensible today, including a careful work-up with a focus on any signs of progression or ongoing aortic inflammation.[6] Such an approach should involve high-resolution electrocardiogram-grated imaging at discharge, with follow-up after 3 months (possibly by CT in combination with 18-fluorodeoxyglucose positron emission tomography to trace evidence of progression, expansion, or ongoing inflammation). Recent data suggest that elective, individualized TEVAR within the window of opportunity (plasticity) of approximately 100 days is the right therapeutic decision, as remodelling of the aorta is more likely to be successful within this subacute time frame.

Published evidence from registries and randomized studies confirms a long-term stabilizing effect of preemptive placement of tailored stent grafts in patients without classic criteria of complications.[7-9] The consideration of active endovascular treatment in lower-risk patients, however, requires even more careful TEVAR procedures, optimized and individualized dissection-specific endovascular devices, and a skillset to deal with complications. A hybrid intervention suite, a team approach, and an option to convert to proximal aorta/cardiac surgery if needed, should become standard in order to manage potential complications (eg, retrograde dissection, 2% to 6% in the United States).

To ensure the highest standard of care and optimal patient safety, regional centers of care for aortic diseases/dissections may be a solution. A similar network model for organized care of acute coronary syndrome has been extremely successful in fighting heart attacks in the Western world and has already been used in the management of ruptured abdominal aortic aneurysms in the United Kingdom.[10]

Along with promising and emerging treatment options for aortic dissection, an even more important concept is a lifelong surveillance effort, which must include the patients and the medical community. Any aortic center should run a follow-up clinic and offer a surveillance program.

Finally, preventive actions (such as elimination of hypertension and some form of genetic profiling for asymptomatic aortic diseases) will be needed in the near future, if not today. This is particularly relevant when we consider the mounting evidence of high incidences of dissection and its precursors in some Asian populations with a high prevalence of untreated or undertreated arterial hypertension. ■

Christoph A. Nienaber, MD, PhD, is with Royal Brompton Hospital, Interventional Cardiology & Aortic Centre, The Royal Brompton & Harefield NHS Trust in London, United Kingdom. He has stated that he has no financial interests related to this article. Prof. Nienaber may be reached at C.Nienaber@rbht.nhs.uk.

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Timing of TEVAR Treatment

When should treatment occur in acute, initially uncomplicated type B aortic dissection?

BY MICHAEL D. DAKE, MD

During the last 5 to 10 years, we have witnessed an increasingly sharp focus on many aspects of aortic dissection. This concentration is not solely directed at treatment strategies; rather, it has produced important new insights into diagnosis, imaging, classification, prognostic features of disease progression, and follow-up regimens.

The impetus for this heightened understanding is fueled by a broader range of interested specialists and is based on the emergence of endovascular procedures, including thoracic endovascular aortic repair (TEVAR), that provide less invasive alternatives to open surgical repair to address manifestations of the disease.

Around the world, TEVAR is now acknowledged as the treatment of choice for acute, complicated type B aortic dissection. Traditionally, this includes a type B dissection associated with rupture, symptomatic branch vessel involvement, persistent pain, or difficult-to-control hypertension.

DISEASE PROGRESSION

Recently, attention has been directed to the risk of disease progression within the first 3 to 5 years after diagnosis of a type B dissection initially considered uncomplicated. There is now awareness of a variety of disease features observed at the time of diagnosis that appear to represent risk factors for subsequent disease progression.

The majority of these high-risk features are anatomically based, often related to aortic dimensions measured on imaging studies. In the future, it is likely that additional prognostic factors from physiological, hemodynamic, or aortic wall biological studies will be recognized and will contribute to additional understanding of which patients with initially uncomplicated dissection may be at increased risk of early disease progression, including rupture or aneurysm formation.

In the meantime, one of the frequent topics of discussion and debate currently featured at cardiovascular meetings and in articles that provide a perspective on current management of type B dissection is whether we should offer TEVAR treatment to patients who have an initially uncomplicated process but harbor multiple high-risk features for progression. And, if so, when should treatment occur?

Obviously, immediate TEVAR will be performed on patients who present with a life-threatening complication (rupture or branch vessel ischemia) at the time of diagnosis, if anatomically suitable and feasible. Current controversies focus on whether it's possible to identify a subgroup of patients who are initially deemed uncomplicated, but who would benefit from an essentially prophylactic TEVAR procedure to potentially prevent subsequent complications, which may or may not present emergently.

If we examine what we know in the current snapshot in time, the answer to this question is unclear, but there exists an abundance of opinions. Given the best available data, how can we begin to analyze the risks and benefits for such a strategy?

WHAT DO WE KNOW?

First, in patients managed with what is currently the best medical therapy, the risk of death within the first 30 days after diagnosis of acute type B dissection is approximately 10% to 11%.[1,2] We can assume that an overwhelming majority of these patients were deemed initially uncomplicated; otherwise, endovascular or open surgical interventions would have been performed to manage any complications. The majority of the early deaths in this group are due to aortic rupture that occurs within the initial 14 days after diagnosis. Could this early mortality rate be improved by early TEVAR therapy in a subgroup of patients with a high-risk profile based on some composite of features that can predict disease progression or early complications? Currently, we don't know.

What we do know is that early treatment of type B aortic dissection within the first 48 to 72 hours, or even within a week, is associated with an increased risk of retrograde type A dissection—at least when TEVAR is used to manage patients with an acute complicated process (Figure 1). This dreaded catastrophe, which is not universally fatal and not exclusively due to the endoprosthesis, may occur at a rate as high as 3% to 4%.[3-5] Based on published meta-analyses, approximately 23% of these cases were diagnosed during the procedure (8%) or immediately periprocedurally (15%), with associated mortality rates of 70% and 50%, respectively.[3] This is compared to an estimated mortality rate of 30%

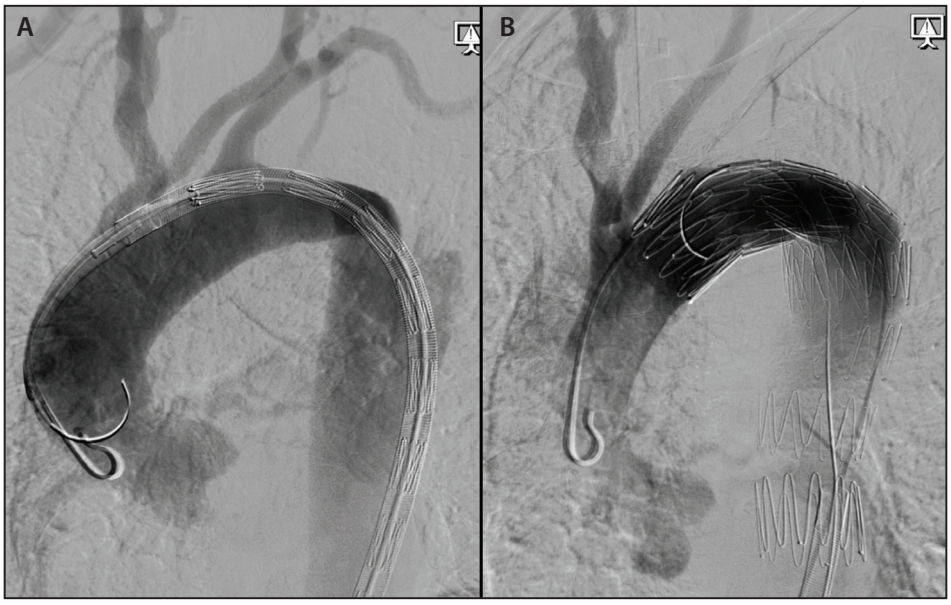

Figure 1. A 68-year-old woman with an acute type B aortic dissection. The left anterior oblique aortogram with an endograft in the aortic arch just beyond the left carotid artery origin prior to deployment. The ascending aorta is normal with a guidewire and flush catheter against the outer anterior wall (A). Aortogram postdeployment of the endograft with a flush catheter within the true lumen of the ascending aorta displaced away from the anterior wall by a false lumen caused by retrograde type A aortic dissection. The patient went to the operating room for open repair of the ascending segment and recovered uneventfully (B).

for cases of retrograde type A dissection diagnosed after hospital discharge.

With this risk in mind, what other signposts can we look to in order to direct our future management strategies? The much-publicized results of the INSTEAD trial raised awareness of the frequency of late aortic-related events in patients with type B dissection deemed initially uncomplicated who were treated more than 14 days after the onset of symptoms (median of approximately 8 weeks).[6] The so-called INSTEAD-XL extension of the original protocol provided follow-up results between 2 and 5 years after the initial randomization of treatment to endograft placement plus medical therapy (n = 72) or medical therapy alone (n = 68).[7]

The landmark analysis of the outcomes from 2 to 5 years in the two groups detailed 15 deaths in the optimal medical therapy arm over this time period and none in the TEVAR-plus-medical-therapy group. Of the 15 deaths in the optimal medical therapy arm, all but two were due to a known aortic rupture or sudden death (defined as a death within 1 hour in patients with known absence of coronary or structural heart disease).

During the course of the 5-year study, 26% of the medical therapy patients underwent crossover to TEVAR placement (14 cases, including five emergencies) or conversion to open repair (four cases), both for enlarging false lumen diameters. Conversely, in the TEVAR group, additional stent graft placement was required in seven cases and conversion to open repair in three cases, for a total reintervention rate of 13% over the same time period. Notably, there was no periprocedural mortality after crossover to TEVAR or conversion to open repair.

Over the 5-year study, the difference in all-cause mortality was not statistically significant (P = .13) between TEVAR plus optimal medical therapy (11.1%) and optimal medical therapy alone (19.3%); however, the difference in aortic-specific mortality at 5 years was statistically significant (6.9% versus 19.3%, P = .04). In terms of disease progression through 5 years, there was a 19.1% absolute risk reduction with TEVAR (27.0%) when compared to medical therapy (46.1%). This difference between the outcomes in the two groups was statistically significant (P = .04). Of note, in the TEVAR group, there was one case (1.4%) of retrograde type A dissection.

So, given these data, what can we make of the opportunity for reducing the mortality rates and disease progression by early TEVAR intervention in patients with initially uncomplicated type B aortic dissection?

MORE QUESTIONS THAN ANSWERS

Well, the trend of podium opinions around the world indicates that if we could confidently define a group of patients with a high-risk profile for disease progression, based on various clinical and anatomic manifestations of their dissections, a strategy of early TEVAR may be warranted to prevent complications, including rupture and false lumen dilatation. A number of criteria composed of high-risk features have been shown to predict those patients who are likely to progress from initially uncomplicated to a complicated type B status within the early- to mid-period after diagnosis.

Unfortunately, no one criterion or composite of features has been consensually agreed upon or proven to precisely define such a group and their specific risks of complications, or to predict within what time frame after diagnosis they are most susceptible. Consequently, we proceed much like a jury weighing each proposed

high-risk feature until we accumulate a preponderance of evidence that meets a threshold and triggers consideration of TEVAR in a patient with an initially uncomplicated disease process.

Therefore, the question at present is whether the procedural risk of TEVAR is outweighed by the benefit of a prophylactic or preventive therapy applied to a group of patients that is yet to be strictly defined. Suffice it to say, no one knows for sure.

Another unknown that could influence our decision making if better understood, is the appearance of the aorta on the most recent imaging surveillance in patients who experienced rupture or a sudden death in the midterm (2 to 5 years) after diagnosis. Regrettably, this particular imaging follow-up was not available from the INSTEAD-XL data. There were 13 cases of rupture or sudden death in the optimal medical therapy group in INSTEAD-XL between 2 and 5 years, but we did not know whether these patients had progressive false lumen dilatation on successive surveillance imaging exams, and if so, to what degree.

The lack of these results from a well-controlled, prospective clinical trial highlights the difficulty of strict monitoring for possible disease progression at intervals frequent enough to identify patients at high risk for catastrophic or impending complications. Clearly, this is even more challenging in a real-world setting where patient compliance with prescribed follow-up protocols, including CT imaging, is even more difficult to achieve.

The bottom line is that we just don't know if a patient at risk for catastrophic events is following a personal trajectory of disease progression that reaches a threshold, such as a cutoff in the aneurysm diameter, that can predict a high risk of mortality. By tracking with vigilant imaging surveillance, we could potentially avoid rupture by crossing over to TEVAR at a time that minimizes procedural risks. Also, if a monitoring strategy to securely minimize late aortic-specific mortality is possible, at what point of follow-up would we lose the ability to achieve the same desirable aortic remodeling observed with TEVAR in the more acute setting?

Clearly, we now have many more questions than answers.

GOLDILOCKS DILEMMA

For patients who have been traditionally classified as having acute uncomplicated type B aortic dissection, we are slowly growing comfortable with a consensus view that their conditions are actually only initially uncomplicated. Rather, they exhibit certain anatomic and clinical features that predict a high risk for disease progression and aortic-related events sometime within 60 months after diagnosis.

In this group of patients, especially those who may not comply with prescribed follow-up protocols, a more aggressive treatment approach incorporating early TEVAR, if anatomically suitable, may be considered. The timing of such a procedure may need to be individualized based upon patient factors. In order to minimize the risk of retrograde type A dissection associated with the procedure, acute TEVAR may not be advisable. Rather, a strategy of delayed TEVAR performed from 1 week to 3 months after diagnosis has been advocated by some authorities.

This approach acknowledges our current understanding of the evolving spectrum of the acute type B aortic dissection and the importance of stratification of management strategies based on certain anatomic and clinical features of the disease. So, in the end, today, we find ourselves facing the proverbial Goldilocks dilemma in terms of deciding the optimal time to intervene with TEVAR.

We don't want to intervene with TEVAR too soon in the acute phase when the risk of fatal type A retrograde dissection may be the highest, but we don't want to wait too long and lose the opportunity to prevent a catastrophic rupture in poorly compliant patients who become lost to follow-up, or the chance to optimally remodel the aorta post-TEVAR when disease progression is too advanced or too chronic. We want to mitigate all these risks and perform TEVAR at just the right time.

CONCLUSION

The dilemma is all too real, and identifying the right time to intervene is currently an unmet challenge. Successfully defining the risks/benefits regarding the timing of TEVAR will undoubtedly contribute greatly to improved outcomes for our patients with acute, initially uncomplicated type B dissection. ■

Michael D. Dake, MD, is the Thelma and Henry Doelger Professor in the Department of Cardiothoracic Surgery, Stanford University School of Medicine, and Medical Director, Catheterization and Angiography Laboratories, Stanford University Hospital in Stanford, California. He has stated that he has no financial interests related to this article. Dr. Dake may be reached at mddake@stanford.edu.

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Distal Stent Graft–Induced New Entry

The current paradigm and future management of dSINE.

BY HUNG-LUNG HSU, MD, AND CHUN-CHE SHIH, MD, PHD

During the past 15 years, the treatment of type B aortic dissection has evolved after the introduction of thoracic endovascular aortic repair (TEVAR). With high success rates and acceptable clinical outcomes, stent grafting has been widely accepted as the treatment of choice for acute, complicated type B aortic dissection. The basic concept of this technique is to cover the proximal primary intimal tear of the aorta, to exclude the false lumen, and to initiate thrombosis of the false lumen and expansion of the true lumen. However, the distal landing zone of the stent graft is still often located in the diseased aorta, and potential intimal injury by the endograft is always a major concern. New distal intimomedial injury by the stent graft, so-called distal stent graft–induced new entry (dSINE), has been increasingly observed.[1,2] dSINE may lead to a new patent false lumen, then aneurysmal degeneration, and eventually aortic rupture.

THE INCIDENCE, ONSET, AND MORTALITY OF dSINE

The incidence of dSINE ranged from 3.4% to 27%.[1,3] A higher incidence was noted in patients treated for chronic aortic dissection. Our published data revealed an incidence of 18.9% in patients with acute type B aortic dissection and 35.7% in chronic cases.[2] One recent study also reported that 89% of dSINE was noted in patients with chronic aortic dissection.[4] In addition, dSINE usually develops late after endografting and patients may be asymptomatic for a long time. Our previous study showed that the average time from TEVAR to dSINE onset was 24.8 ± 5.9 months.[2] Others reported onset times ranging from 11 ± 16 months[1] to 31.5 ± 28.6 months.[4] With a mortality rate as high as 28.6%,[1] dSINE can be insidious in character and needs to be monitored by a long-term CT follow-up protocol.

THE MECHANISM OF dSINE DEVELOPMENT

The potential mechanisms for the development of dSINE are complex. Besides the natural progression of the aortic disease, aortic wall fragility and stent grafting–related factors may also play important roles. The pulsatile stimulus from the rigid end of the stent graft against the fragile intimal flap could eventually cause a tear and create a new entry. In chronic dissection, the intimal flap is more fibrotic and less mobile than in the acute phase. The rigid membrane would be less compliant to the expansion of the stent graft and thus carry a higher risk of a new intimal break.

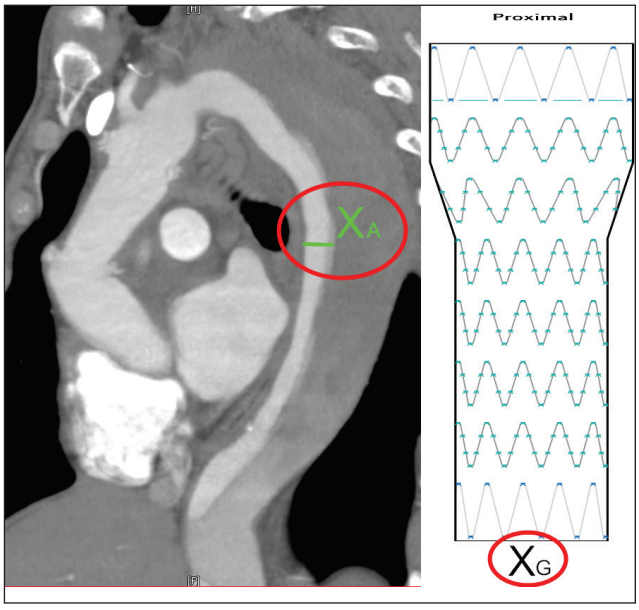

Figure 1. Oversizing ratio = (X_G/ X_A) – 1. X_G: the distal size of the selected stent graft before the procedure; X_A: the size of the true lumen at the presumed level of the distal end of the stent graft before the procedure. An oversizing ratio ≧ 4 is predictive of dSINE formation (*P* = .031).[6]

Another important factor is distal stent graft oversizing (the ratio between the distal size of the selected stent graft and the true lumen size at the presumed level of the distal end of the stent graft before the procedure; Figure 1). Previous studies have shown that greater distal stent graft oversizing, in either area or diameter measurements, is related to the formation of dSINE.[3,4] The possible mechanism is that the true lumen is usually narrower at the presumed distal landing zone than at the proximal landing zone, so stent grafts chosen according to the proximal landing zone will lead to substantial oversizing at the distal landing zone. This will create an excessive radial force and pose a risk

of dSINE. Some studies demonstrated that a higher taper ratio (the ratio between the sizes of the true lumen at the proximal landing zone and that at the distal landing zone before the procedure) was seen in patients with dSINE.[1,4,5] However, this phenomenon was not observed in our published studies. This means that distal oversizing may contribute more in the formation of dSINE than the discrepancy between the sizes of the proximal and distal landing zones.

Elastic recoil, the tendency of the stent graft to revert to its initial straight form, may also lead to intimal injury at the distal landing zone. In most cases of aortic dissection, the stent graft needs proximal landing in the aortic arch and is passively bent to conform to the curvature of the aorta. However, the endograft has the inherent tendency to spring back to its initial straight form, which generates stress along the outer curve and leads to an angulated aorta at the distal landing zone, posing a risk of dSINE formation.[4]

PREDICTION AND PREVENTION OF dSINE

Because the incidence of dSINE is high, and it is associated with potentially life-threatening outcomes, prediction and prevention are crucial in its management. Our published study revealed that the oversizing ratio between the sizes of the selected stent graft and the true lumen at the presumed distal landing zone is a significant preoperative predictive factor of dSINE (Figure 1).[6] This study also reported that the expansion mismatch ratio between the true lumen sizes at and adjacent to the distal landing zone is an important postoperative predictor of dSINE (Figure 2).[6] Noticeably, these predictive factors were measured by area because the true lumen is usually elliptic or even crescent in shape, and the definition or calculation of the ratios by diameters is more complex.

Several preventive procedures have been adopted at our institution to reduce the formation of dSINE. First, endografts with a tapered configuration, such as Zenith Dissection Endovascular Grafts with 4-, 8-, or 10-mm-diameter tapering (Cook Medical), can be used to avoid excessive distal oversizing. Alternatively, a bottom-up technique can also produce the effect of tapering in diameter or area. This technique is composed of deployment of a smaller stent graft distally, followed by deployment of a larger stent graft proximally. We have used this approach since 2010. Our unpublished analysis showed that the area oversizing ratio in the distal landing zone was reduced, and the incidence of dSINE decreased from 34.7% to 8.3% with the use of this endografting technique.

Second, restrictive bare stenting (RBS) or the modified PETTICOAT (provisional extension to induce complete attachment) technique may protect the intima at the distal edge of the stent graft from the excessive radial force of the stent graft. This technique involves the placement of a properly sized bare stent in the intended distal landing zone of the stent graft, prior to deployment of the stent graft. RBS was reported to be associated with a lower incidence of dSINE (0% vs 2.9%; $P = .033$) and fewer secondary interventions (3.9% vs 9.3%; $P = .040$).[7] A recent study also demonstrated favorable results of the modified PETTICOAT technique.[4]

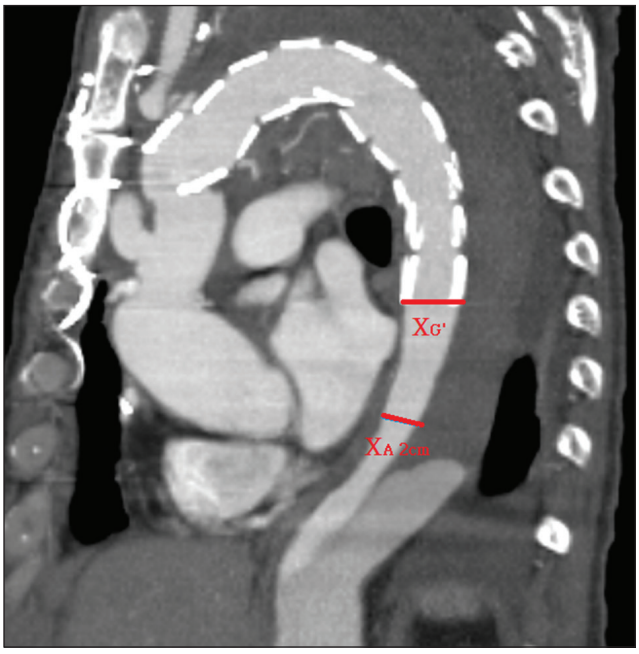

Figure 2. Expansion mismatch ratio of true lumen: $X_G' / X_{A\ 2cm}$. X_G': the distal size of the limitedly expanded stent graft within the true lumen after the procedure; $X_{A\ 2cm}$: the size of the true lumen 2 cm distal to the distal end of the stent graft after the procedure. An expansion mismatch ratio ≧ 2.4 is predictive to dSINE formation ($P = .031$).[6]

Third, some studies suggested that avoidance of distal landing in a tortuous portion of the aorta[1] or placement of several stent grafts until the distal end of the last stent graft is oriented parallel to the aortic wall would reduce the stress of the stent graft against the curvature of the aorta.[8]

MANAGEMENT OF dSINE

Medical treatment with optimal blood pressure control is preferred upon confirming the diagnosis of dSINE. The indications for reintervention include persistent enlargement of the false lumen, contained rupture, pseudoaneurysm formation, malperfusion, or symptoms. In our institute, the procedure for the secondary intervention is performed in the same way as the standard TEVAR with proper device sizing. The artery of Adamkiewicz is located by CT preoperatively and is preserved as much as possible during the secondary procedure to reduce the risk of paraplegia, and cerebrospinal fluid drainage is set up immediately if there is any symptom of paraplegia. Our data showed that reendografting seems effective to treat complicated dSINE.[3]

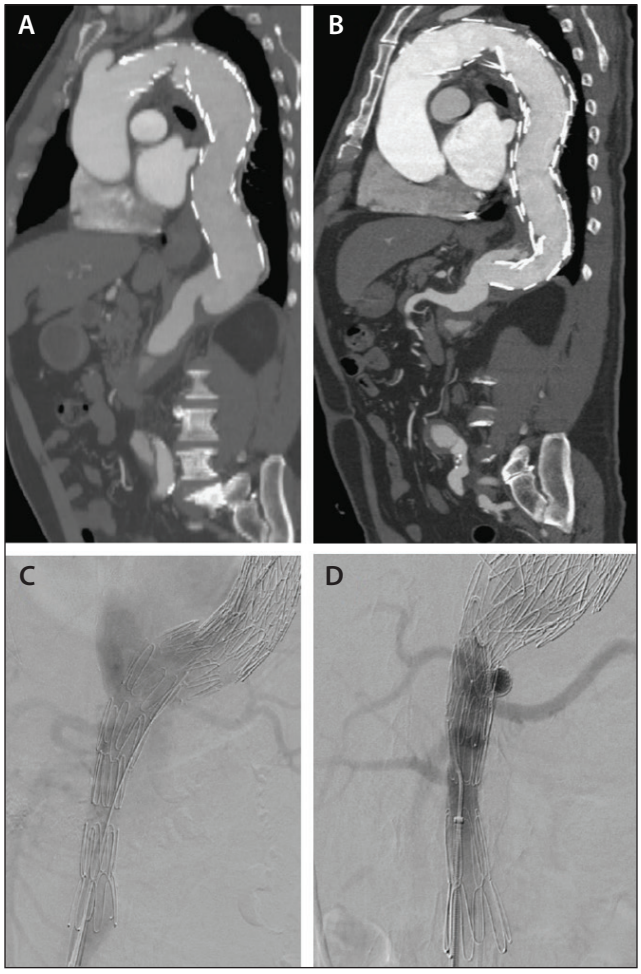

Figure 3. Recurrence of dSINE and management with a modified PETTICOAT technique. The first dSINE was noted 2 years after TEVAR (A); recurrence of dSINE 2 years after reendografting (B); a modified PETTICOAT technique with the placement of the Zenith Dissection Endovascular Stent (C); followed by endografting and celiac trunk chimney to exclude the intimal tear (D).

ROLE OF AORTIC BARE STENT IN dSINE

The Zenith Dissection Endovascular System (Cook Medical) is designed for treatment of aortic type B dissection utilizing the PETTICOAT technique. This device comprises stent grafts to cover the primary entry tear proximally and uncovered metal stents to promote true lumen expansion distally. Previous studies have demonstrated favorable clinical and anatomic results in the management of type B aortic dissection.[9,10] Interestingly, it may help reducing the risk of dSINE formation. In our initial experience with this device, in nearly 50 cases of complicated type B aortic dissection, only one dSINE formation was noted in a 2-year follow-up period. In addition, we started to use the modified PETTICOAT technique to treat recurrence of dSINE (Figure 3). No further redissection was noted in a 6-month follow-up period, but the effectiveness of this management needs further investigation.

CONCLUSION

dSINE is not rare and is possibly life-threatening. Stent grafts with a dissection-specified tapering design, a bottom-up technique, restrictive bare stenting, PETTICOAT, or modified PETTICOAT technique can be used to reduce the risk of dSINE. The preoperative distal oversizing ratio and postoperative expansion mismatch ratio appear to be predictive of the formation of dSINE. Endovascular reintervention seems an effective management for complicated dSINE. However, recurrence can occur, and long-term follow-up is mandatory. ■

Hung-Lung Hsu, MD, is with the School of Medicine, National Yang Ming University in Taipei, Taiwan; the Department of Cardiovascular Surgery, Far Eastern Memorial Hospital in New Taipei City, Taiwan; and the Division of Cardiovascular Surgery, Department of Surgery, Mennonite Christian Hospital in Hualien, Taiwan. He has disclosed that he has received speaker fees, research grants, or consulting fees from Cook Medical. Dr. Hsu may be reached at dennishsu906@gmail.com.

Chun-Che Shih, MD, PhD, is with the Division of Cardiovascular Surgery, Department of Surgery, Taipei Veterans General Hospital in Taipei, Taiwan; the Institute of Clinical Medicine and the School of Medicine, at National Yang Ming University, in Taipei, Taiwan. He has disclosed that he has received speaker fees, research grants, or consulting fees from Cook Medical. Professor Shih may be reached at ccshih@vghtpe.gov.tw.

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Seeing the Signs

The top precursors/predictors for earlier endovascular treatment versus medical management of type B aortic dissection.

BY WORAWONG SLISATKORN, MD

Acute aortic dissection is a life-threatening condition with an incidence of 5 to 30 people per million per year in Western countries and 43 people per million per year in Asian countries.[1-3] These numbers include both type A and type B aortic dissections in which mortality is high if it is an aortic dissection involving an ascending aorta (type A) or a type B dissection with complications. Forty percent of these patients have acute type B dissection. The standard treatment for uncomplicated acute type B dissection is optimal medical therapy using blood pressure and pain control. Survival rates at 1 month, 1 year, 5 years, and 10 years are 89%, 84%, 60% to 80%, and 40% to 45%, respectively.[2,4,5] Complicated acute type B dissection is associated with high mortality (50%–85%) if left untreated. Surgery for this complex disease is associated with significant mortality of 18.2% to 50%.[2]

Since 1996, endovascular repair with stent grafts has emerged as a less invasive procedure for the treatment of aortic dissection by covering the primary entry tear in the proximal descending aorta, with a 30-day mortality of 16%, as reported in an early series.[6] Pooled outcomes of endovascular treatment for complicated type B dissection (malperfusion or rupture) from five physician-sponsored investigational device exemption clinical trials demonstrated a mortality rate of 10.8% at 30 days and 29.4% at 1 year.[7] Data from the International Registration of Aortic Dissection showed a significantly lower mortality rate after endovascular treatment compared to open surgery (10.2% vs 33.9%; $P = .002$) for complicated type B dissection.[8] There is increasing evidence that endovascular treatment demonstrates a significant advantage over open surgery in patients with complicated type B dissection, even though there is no prospective randomized controlled trial, which is unlikely to happen.

There is enthusiasm to broaden the indication of thoracic endovascular aortic repair (TEVAR) to treat uncomplicated type B dissection. The objectives are to obliterate the false lumen by covering the proximal intimal tear and to stabilize the dissected aorta to prevent late complications. The INvestigation of STEnt Grafts in Aortic Dissection (INSTEAD) trial is a randomized study that compared outcomes of TEVAR to optimal medical therapy for the treatment of subacute (2–52 weeks after onset), uncomplicated type B dissection. At 2-year follow-up, TEVAR failed to improve survival and adverse event rates despite leading to favorable aortic remodeling.[9] However, TEVAR significantly decreased aorta-related mortality and disease progression at 5 years compared to medical therapy alone, but there was no difference in total mortality.[10] This study has influenced some physicians, who started changing their clinical practices. Currently, the European Society of Cardiology recommends that, "TEVAR should be considered in uncomplicated type B aortic dissection, class IIa level B."[11]

TOP PREDICTORS FOR EARLIER ENDOVASCULAR TREATMENT OF TYPE B AORTIC DISSECTION

Some special conditions of type B aortic dissection carry significant mortality risk if treatment is delayed. These included aortic dissection with rupture, malperfusion syndrome, persistent pain, and uncontrolled hypertension. Early endovascular treatment is a crucial management strategy in these situations.

1. Rupture or leakage: Acute type B aortic dissection with rupture is an emergency. Rupture in the thoracic segment usually presents as massive left pleural effusion, but some patients may present with right pleural effusion especially if they have a tortuous descending aorta or right-sided aortic arch. After TEVAR, the pleural effusion still needs to be monitored, as some patients may develop persistent hypotension from ongoing bleeding. In this situation, open surgery via left thoracotomy is crucial to secure the bleeding point.
2. Malperfusion symptom: Organ malperfusion is indicated for endovascular treatment. TEVAR is able to restore blood flow to the true lumen in the majority of cases. In some patients, peripheral stenting may be performed adjunctively to resume specific organ perfusion. In case of spinal cord ischemia, it is very difficult to predict whether the neurologic outcome will be improved after the procedure.

3. Persistent pain: Ongoing pain/symptom despite optimal medical therapy is a risk factor of threatened rupture. Endovascular repair is indicated and can prevent aortic rupture and alleviate severe pain/symptoms.
4. Uncontrolled hypertension: Poor control of blood pressure is associated with a risk of aortic growth and needs close monitoring.
5. Progressive dilatation of the aorta: The dissected aorta with a diameter ≥ 5.5 cm or with an expansion rate > 0.5 cm per year is an indication for surgical intervention.[12]

The following precursors indicate that endovascular treatment should be considered:

1. Young age: Patients younger than 60 years are known to have a significantly higher aortic growth rate.[13,14]
2. Aortic dilatation: A maximal aortic diameter ≥ 40 mm during the acute phase is a predictor of aortic growth.[15]
3. Large false lumen diameter: The risk of aneurysm formation and mortality is increased in patients with a false lumen diameter ≥ 22 mm in the upper descending thoracic aorta (aneurysm formation was 42% vs 5%; $P < .001$; mortality was 17% vs 5%; $P = .09$).[16]
4. Patency of the false lumen: A study from Japan showed that patency of the false lumen is a risk factor for dissection-related deaths and events (hazard ratio, 2.59 and 1.8, respectively). Freedom from aortic enlargement (≥ 55 mm) at 1 year, 5 years, and 10 years was 100%, 94.7%, and 89.2%, respectively, in patients with an aortic size < 45 mm and a thrombosed false lumen. However, in patients with an aortic size > 45 mm and a patent false lumen, freedom from aortic enlargement at 1 year, 5 years, and 10 years was decreased to 72.6%, 66%, and 42.8% respectively.[17] The aortic growth rate among patients with a partially thrombosed false lumen appears to be higher than that in patients with a completely thrombosed or patent false lumen.[18] Tsai et al reported that patients with a partially thrombosed false lumen had a higher mortality rate than those with a patent or completely thrombosed false lumen at a mean follow-up of 3 years (31.6% ± 12.4% for partial thrombosis, 13.7% ± 7.1% for patent false lumen, and 22.6% ± 22.6% for complete thrombosis).[19]
5. Configuration of false lumen: An elliptical configuration of the true lumen with a circular formation of the false lumen is a result of high pressurization in the false lumen and was associated with increased aortic growth.[18]
6. Large primary entry tear: Patients with a large primary entry tear (> 1 cm) had more dissection-related events and a higher rate of aortic growth than those with a smaller entry tear.[20]

CONCLUSION

Endovascular repair is indicated in acute complicated type B dissection and is playing an increasing role in uncomplicated dissection. Many predictors of aortic growth and complications are useful warning signs that may indicate early endovascular treatment to prevent future catastrophic events. ■

Worawong Slisatkorn, MD, is with the Division of Cardio-Thoracic Surgery, Department of Surgery, Faculty of Medicine at Siriraj Hospital, Mahidol University, Bangkok, Thailand. He has stated that he has no financial interests related to this article. Dr. Slisatkorn may be reached at (662) 419-7998; wslisatkorn@yahoo.com

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Treating Complicated Acute Type B Aortic Dissection With Proximal Stent Grafts and Distal Bare Stents

Bare aortic stents can expand the true lumen immediately and result in early favorable aortic remodeling.

BY JER-SHEN CHEN, MD

Thoracic endovascular aortic repair (TEVAR) has been widely used to treat aortic dissection in recent years. Although TEVAR covers the entry tear in the descending aorta, there are still some concerns, including stent graft–induced new entry (SINE) tears[1] and persistent distal false lumen expansion.[2] The bare aortic stent is a part of the Zenith Dissection Endovascular System (Cook Medical). After proximal stent grafting, bare aortic stents are deployed distal to the stent graft to support the dissected distal aorta. The STABLE trial, a prospective, nonrandomized multicenter study, reported favorable clinical and anatomic results.[3] A recent systematic review article that analyzed four studies on proximal stent grafting and distal bare stenting also clearly demonstrated

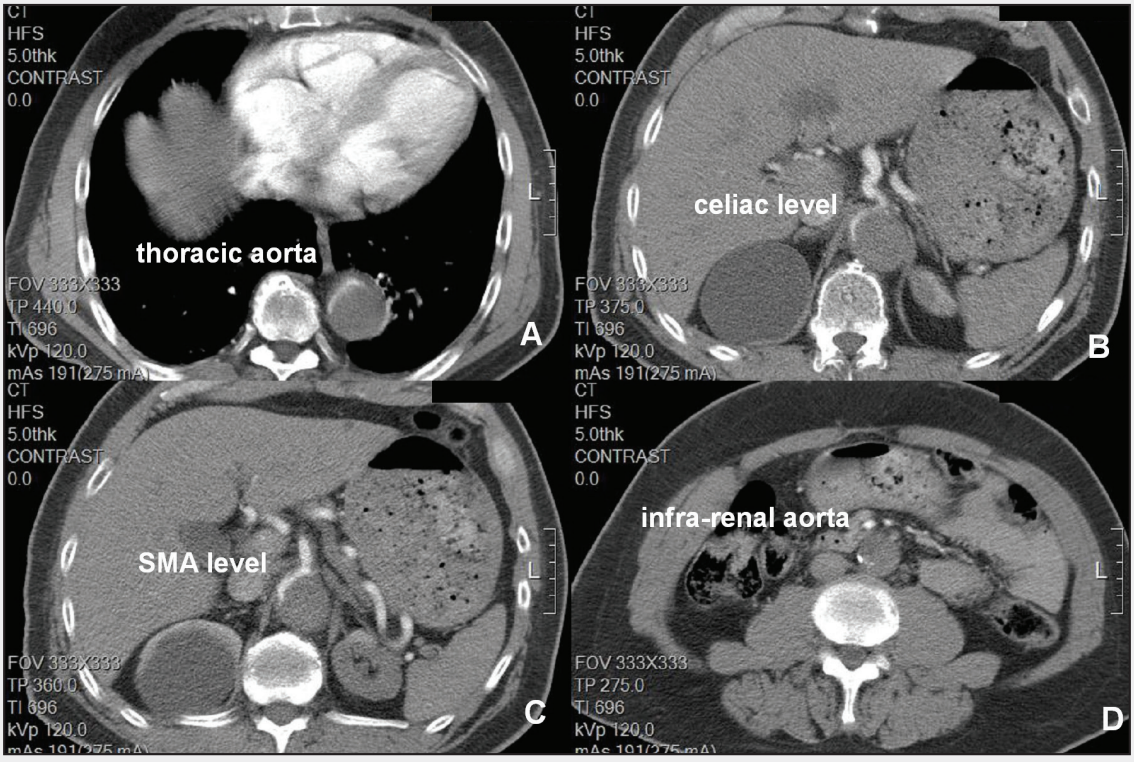

Figure 1. The true lumen was severely compressed by the false lumen, starting from the level of the mid-thoracic aorta (A). The celiac artery and superior mesenteric artery (SMA) were compromised (B, C). In the infrarenal aorta, virtually no blood flow was seen in the true lumen, which resulted in bilateral critical limb ischemia (D).

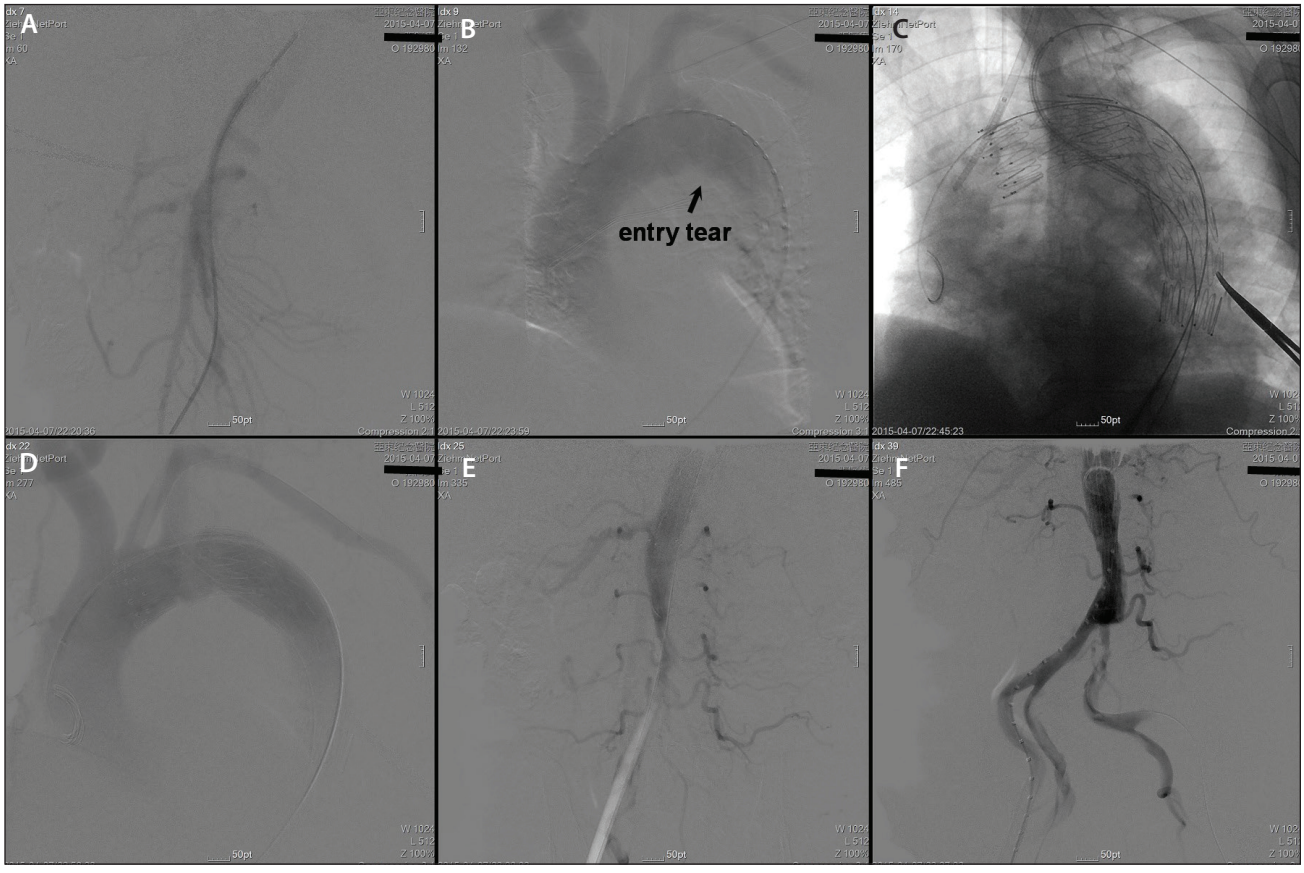

Figure 2. A thin true lumen was demonstrated in the abdominal aorta (A). The single entry tear was located just distal to the left subclavian artery (B) so that a zone 1 landing was required. In order to maintain the arch vessels' patency in the shortest time, a chimney graft was inserted from the left common carotid artery (11- X 50-mm Viabahn), and a periscope graft (13- X 100-mm Viabahn) was inserted from the left brachial artery between Cook TX2 aortic stent grafts (C). Aortography showed good seal of the entry tear without compromise of the arch vessels (D). However, after proximal stent grafting, the infrarenal aorta did not expand (E). Two Cook bare aortic stents (diameter: 36 mm, length: 180 mm) were placed down to aortic bifurcation, and the true lumen was opened by the stents (F). Thrombi in the left common iliac artery were removed.

improved true lumen perfusion and diameter.[4] Moreover, in patients with extremely malperfused branch vessels, the bare aortic stents have the advantage of immediate true lumen expansion, which can alleviate the malperfusion actively, rather than just waiting for true lumen expansion.

A CASE STUDY

A 67-year-old man was sent to the emergency room due to sudden onset of severe back pain and cold sweating, followed by bilateral lower leg numbness. On examination, his heart rate was 60 beats/min, and his blood pressure was 209/119 mm Hg. Bilateral lower leg sensations were severely impaired. No pulsation over both feet could be detected by a handheld Doppler ultrasound probe. A CT scan revealed an acute Stanford type B aortic dissection with severe compromise in the visceral arteries and bilateral lower legs (Figure 1). The patient was sent to the operating room immediately.

Bilateral groin, left elbow, and left neck cutdowns were performed. Initial aortography demonstrated a severely compressed true lumen of abdominal aorta (Figure 2A). The patient had a single entry tear, which was just distal to the left subclavian artery (LSA) (Figure 2B). Therefore, a zone 1 landing was required. In order to restore the patient's perfusion in the shortest amount of time, we decided to maintain the patency of the left common carotid artery (LCCA) and LSA by a chimney and periscope method. The first Cook TX2 stent graft (diameter: 28–32 mm, length: 162 mm) was inserted via the right femoral artery and deployed just distal to the LSA. A left brachial wire was inserted into the aortic stent graft, and a 13- X 100-mm Viabahn stent graft (Gore & Associates) was inserted into the LSA as a periscope graft. A second 11-X 50-mm Viabahn was inserted via the LCCA as a chimney graft. Then, the second Cook TX2 stent graft (diameter: 36 mm, length: 77 mm)

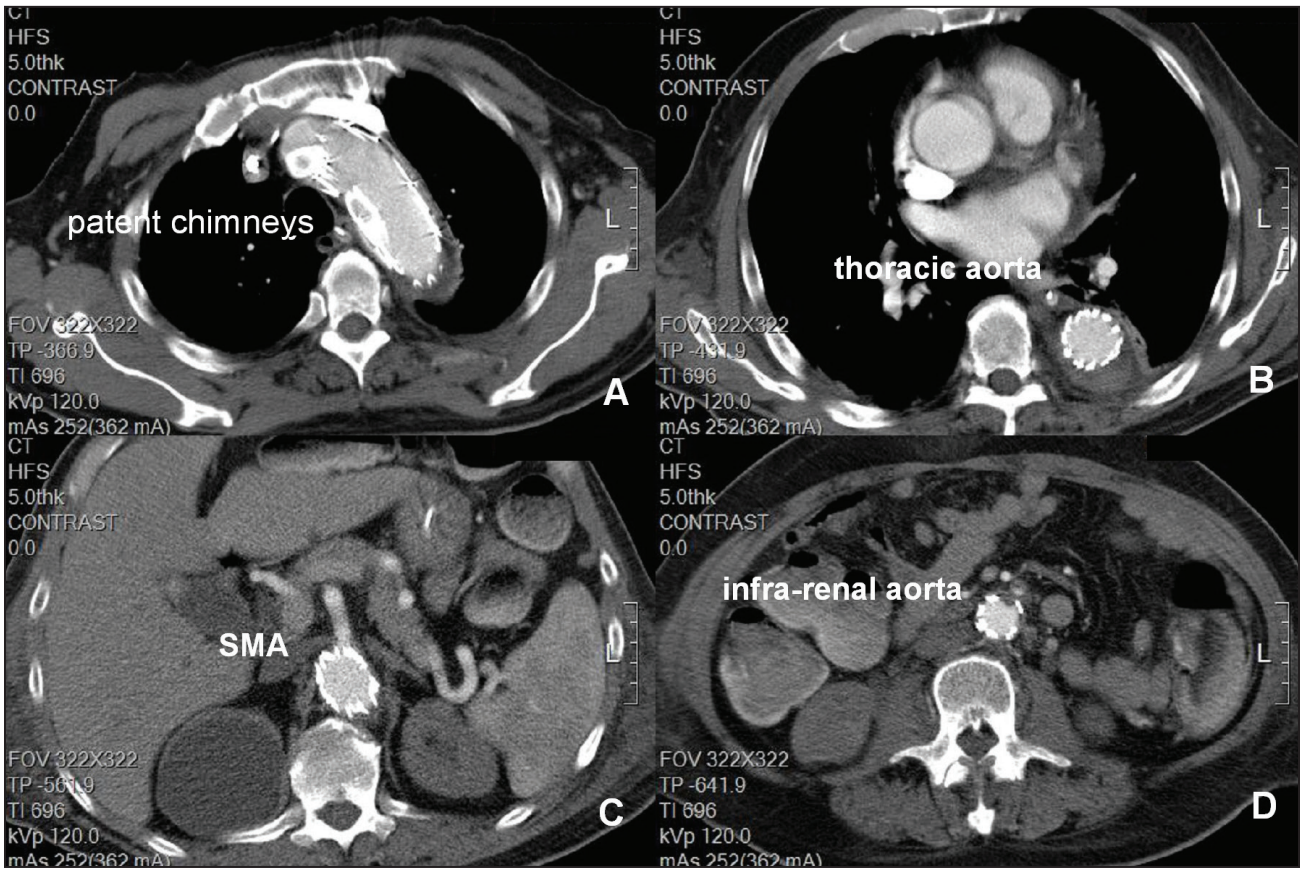

Figure 3. A follow-up CT was performed 1 month later. The chimney and periscope grafts were patent (A). The true lumen fully expanded all the way down (B–D).

was inserted into zone 1 (Figure 2C). After deployment of all devices, the entry tear was well sealed, and the LCCA and LSA were patent (Figure 2D). Unfortunately, the infrarenal aorta still did not expand (Figure 2E). Bilateral femoral artery pulsation still could not be felt via the cutdown wounds. Therefore, two Cook dissection bare aortic stents (diameter: 36 mm, length: 180 mm) were deployed down to the aortic bifurcation, and the true lumen was successfully expanded by the stents (Figure 2F). Right femoral pulsation recovered, and left femoral pulsation also recovered after removal of the thrombi in the left common iliac artery.

After the operation, the patient's peak creatine phosphokinase was 56,440 IU/L. He received hemodialysis for 2 months for rhabdomyolysis and acute renal failure. Ileus resolved quickly without complications. A follow-up CT 1 month later showed patent chimney and periscope grafts (Figure 3A) and fully expanded aortic true lumen all the way down (Figure 3B–D).

OUR DATA

From February 2014 to November 2014, nine consecutive patients (eight men and one woman; mean age of 58.6 ± 9.2 years) with complicated acute type B dissection

TABLE 1. OUTCOMES OF PROXIMAL STENT GRAFT AND DISTAL BARE STENTING FOR COMPLICATED ACUTE TYPE B DISSECTION

Technique success, n (%)	9 (100%)
Resolution of dissection-related complications, n (%)	
Ileus and bowel ischemia (n = 5)	5/5 (100%)
Hepatic dysfunction (n = 1)	1/1 (100%)
Refractory pain (n = 2)	2/2 (100%)
Lower limb ischemia (n = 1)	1/1 (100%)
Extubation in operation room, n (%)	6 (66.7%)
Intensive care unit stay (days), mean ± SD	1.3 ± 1.6
Hospital stay (days), mean ± SD	5.8 ± 2.2
Complications, n (%)	
Upper gastrointestinal bleeding	2 (22.2%)
Jaundice	1 (11.1%)
Renal failure requiring hemodialysis	1 (11.1%)
Paraplegia	1 (11.1%)
Operative mortality/30-day mortality	0 (0%)

underwent proximal stent grafting and distal stenting with the Cook Zenith Dissection Endovascular System. The technical success rate was 100%. All preoperative dissection-related complications, which were also surgical indications, resolved quickly after the operation. Six patients (66.7%) were extubated in the operating room. The mean intensive care unit stay was 1.3 ± 1.6 days (range, 0-4 days). The mean hospital stay was 5.8 ± 2.2 days (range, 3-9 days) (Table 1).

Follow-up CT was arranged accordingly, generally around 3 to 6 months after the operation. To assess aortic remodeling, we measured the true lumen, false lumen, and total aortic diameter at five levels: immediately after the orifice of the LSA, T9 vertebra, T11 vertebra, celiac artery, and superior mesenteric artery (SMA). The T9 level was measured in addition to the T11 level because it was immediately below the end of the proximal stent graft. The decreases in the false lumen diameter ranged from 53.9% to 86.8% at different levels, which were statistically significant at all levels except for the SMA level (Table 2). The increases in the true lumen diameter ranged from 130% to 226%, which were also statistically significant at all levels except for the SMA level (Table 3). In terms of the total aortic diameter, although the false lumen regressed and the true lumen expanded, the total aortic diameters were unchanged at the T9, T11, celiac, and SMA levels. Only the diameter of the thoracic aorta beyond the LSA level decreased significantly from 44.1 ± 7.2 mm to 31.8 ± 5.1 mm (P = .002).

TABLE 2. AORTIC REMODELING IN THE FALSE LUMEN

	False lumen			
Diameter	**Pre-OP (mm), mean ± SD**	**Post-OP (mm), mean ± SD**	**Regression[b] (%)**	***P***
Beyond LSA	25.0 ± 5.1	3.3 ± 6.2	86.8%	< .001[a]
T9	24.5 ± 8.0	8.3 ± 8.6	66.8%	.001[a]
T11	22.8 ± 9.5	8.8 ± 7.0	61.4%	.005[a]
Celiac	17.0 ± 4.7	7.1 ± 6.6	58.2%	.004[a]
SMA	10.2 ± 8.3	4.7 ± 6.8	53.9%	.16

Abbreviations: OP, operation; LSA, left subclavian artery; SMA, superior mesenteric artery.
[a]Statistically significant, $P < .05$
[b]Regression percentage was defined as [1-(Post-OP/Pre-OP diameter)]*100%

TABLE 3. AORTIC REMODELING IN THE TRUE LUMEN

	True lumen			
Diameter	**Pre-OP (mm), mean ± SD**	**Post-OP (mm), mean ± SD**	**Expansion[b] (%)**	***P***
Beyond LSA	19.1 ± 6.0	28.4 ± 3.3	149%	.002[a]
T9	10.9 ± 3.3	24.6 ± 3.5	226%	< .001[a]
T11	12.8 ± 4.4	24.1 ± 3.7	188%	< .001[a]
Celiac	11.2 ± 3.0	21.4 ± 3.9	191%	< .001[a]
SMA	15.1 ± 4.8	19.6 ± 4.3	130%	.05

Abbreviations: OP, operation; LSA, left subclavian artery; SMA, superior mesenteric artery.
[a]Statistically significant, $P < .05$
[b]Expansion percentage was defined as (Post-OP/Pre-OP diameter)*100%

DISCUSSION

Complicated aortic dissections occur in 25% of patients with acute type B dissection.[5] In the interdisciplinary expert consensus on the management of type B dissection, TEVAR was considered to carry lower mortality than open surgery.[6] Today, TEVAR is used widely in the treatment of patients with complicated acute type B dissection. However, persistent distal false lumen expansion[7] remains one of the unsolved problems of TEVAR for acute type B dissection.

Bare aortic stents have at least two theoretical advantages in the management of complicated type B dissection. First, they can expand the true lumen immediately, which will increase the blood flow in the true lumen and provide a prompt improvement of malperfusion. Second, bare aortic stents can support the distal aorta with evenly distributed radial force, and this may provide better remodeling in the long run. In our series, all cases of malperfusion improved quickly after proximal stent grafting and distal stenting. The prompt improvement of malperfusion resulted in short durations of intensive care unit stay and hospital stay after the operation. In the case presented earlier, without the bare aortic stents, we would have had to perform axillofemoral and femorofemoral bypasses, which would have increased the patient's limb ischemic time and resulted in more severe rhabdomyolysis.

For acute type B dissection, medical therapy is still the first-line treatment. Our patients had a mean duration of 81.3 ± 61.1 hours between symptom onset and operation. This meant that medical therapy of more than 3 days was carried out to stabilize the patients but

failed. For these truly "complicated" patients, we wanted to minimize the surgical trauma. Therefore, we did not perform any extra-anatomic bypass for the five patients whose safe landing zone was at zone 1 or zone 2. Chimney and periscope methods were used in two patients, while in three patients, the LSA was just covered and embolized. The technique of proximal stent grafting and distal stenting was largely the same with the TEVAR procedure. To insert bare aortic stents, the operator just had to locate the position of the aortic bifurcation. The technique success rate was 100% in our series, which was comparable with the results of TEVAR.[2]

Two patients in our series suffered from upper gastrointestinal bleeding, which was partly caused by celiac artery malperfusion. The bleeding was controlled by intravenous proton pump inhibitors and did not affect the patients' recovery. One patient had progressive jaundice, due to a comorbid condition of a gallbladder stone. ERCP with lithotripsy solved the problem smoothly. The most devastating complication was paraplegia that occurred in one patient, who also had bilateral common iliac artery compromise. When the patient presented to the emergency department, he had severe bilateral lower limb numbness. We could not differentiate whether the numbness was a result of limb ischemia or spinal cord ischemia. The patient's lower limb perfusion recovered immediately after the operation. However, the paraplegia persisted without much improvement. We suspected that the spinal cord ischemia was a complication of the acute dissection per se and was not the consequence of thoracic aorta coverage by stent grafts.

On the follow-up CT, smooth alignments between the proximal stent grafts, distal bare stents, and thoracic aorta were observed in all patients. We believe that a smooth alignment will preclude SINE in the future, although this needs longer follow-up results to prove. Moreover, the aorta had very favorable remodeling. Statistically significant true lumen expansion and false lumen regression were observed at different levels including the thoracic aorta beyond the LSA, T9 vertebra, T11 vertebra, and celiac artery. At the SMA level, false lumen regression of 53.9% (P = .16) and true lumen expansion of 130% (P = .05) were quite obvious, although not statistically significant. We believe that if we collected data from more patients, the changes at the SMA level would also become statistically significant.

In conclusion, proximal stent grafting and distal bare stenting for complicated acute type B dissection are very effective at resolving dissection-related complications. The operation was not more complex compared with standard TEVAR. This approach may facilitate favorable aortic remodeling, which appears early after the operation. ■

Jer-Shen Chen, MD, is with the Department of Cardiovascular Surgery, Far Eastern Memorial Hospital, Banqiao, and the Faculty of Medicine, National Yang-Ming University School of Medicine, in Taiwan. He has disclosed that he has received speaker fees, research grants, or consulting fees from Cook Medical. Dr. Chen may be reached at jershen@gmail.com.

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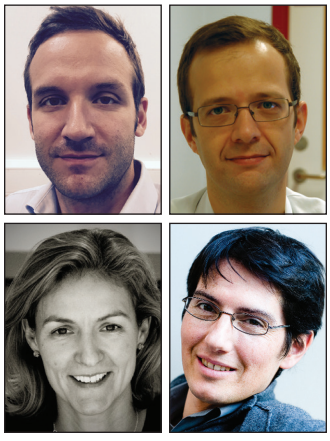
Exploring the Use of Bare Stents in the Treatment of Type B Dissection

Understanding the effects on aortic remodeling and malperfusion.

BY JONATHAN SOBOCINSKI, MD, PHD; NUNO V. DIAS, MD, PHD; RACHEL CLOUGH, MD, PHD; AND STÉPHAN HAULON, MD, PHD



Malperfusion of aortic branches and aortic rupture are the two most feared complications in the acute phase of a type B aortic dissection. When such complications occur, stent graft deployment should be considered with the proximal landing zone in a healthy, nondissected aorta.[1-3] The coverage of the main proximal intimal tear redirects the aortic flow toward the true lumen and thus promotes a drop of pressure within the false lumen. Thoracic stent grafting (TEVAR) has been associated with encouraging early outcomes for the treatment of acute complicated type B aortic dissection,[4,5] but questions still remain regarding the mid- and long-term results.[6] Initial successful treatment with TEVAR is not necessarily associated with favorable remodeling of the dissected aorta during follow-up.[7] Only few exhaustive anatomical analyses of the aorta following TEVAR for acute type B aortic dissection have been performed so far.[7-11] TEVAR generally induces positive aortic remodeling, but this is usually limited to the aortic segment covered by the stent graft; frequently, the outcomes of the distal thoracic and abdominal aorta remain of concern.[12]

The additional implantation of a self-expanding bare stent in the aortic true lumen, distally to the proximal stent graft(s), was proposed in 2005. This composite device design approach, also known as STABLE (staged aortic and branch vessel endoluminal repair), aimed to enhance global aortic remodeling, especially in the area of the abdominal aorta, and to improve the management of visceral/renal/lower limb malperfusion in the acute phase.[13] First, the proximal stent graft is positioned, then intraprocedural angiography is performed to assess if there is insufficient expansion of the true lumen, continuous retrograde perfusion of the false lumen, or evidence of malperfusion of arterial branches originating from the true lumen. If any of these features is present, then a distal bare stent may be used (Figure 1).[14]

Variations of the endovascular techniques described in the STABLE trial have been reported. He et al have suggested that if the proximal stent graft is deployed first, then the distal end of the stent graft is landed in the diseased aorta, which has the risk of causing further aortic dissection or other structural damage. They therefore advocate that the distal bare stent is placed first, at the intended distal landing zone, followed by the proximal stent graft.[15] Hofferberth et al reported the use of balloon-driven expansion of the true lumen following placement of the bare-metal stent below the stent graft to remove any residual flow in the false lumen and achieve complete true lumen expansion. This technique is called the stent-assisted balloon-induced intimal disruption and relamination in aortic dissection repair (STABILISE).[16]

AORTIC REMODELING

Long-term results from the IRAD registry indicate that at 5 years, more than 60% of patients develop aortic growth or formation of a new aneurysm after endovascular repair of acute complicated type B aortic dissection.[17] The implantation of a bare stent distal to the stent graft, to support true lumen expansion within the thoracoabdominal aorta, can be performed to promote remodeling of the dissected aorta. Improved aortic remodeling has been associated with a reduced risk of late aortic complications during follow-up. In a prospective, nonrandomized, multicenter feasibility study (STABLE 1 study), 86 patients with acute and subacute type B aortic dissections (within 90 days of symptom onset) underwent endovascular repair with a composite device design (Zenith TX2 Endovascular Graft and Zenith Dissection Endovascular Stent, Cook Medical).

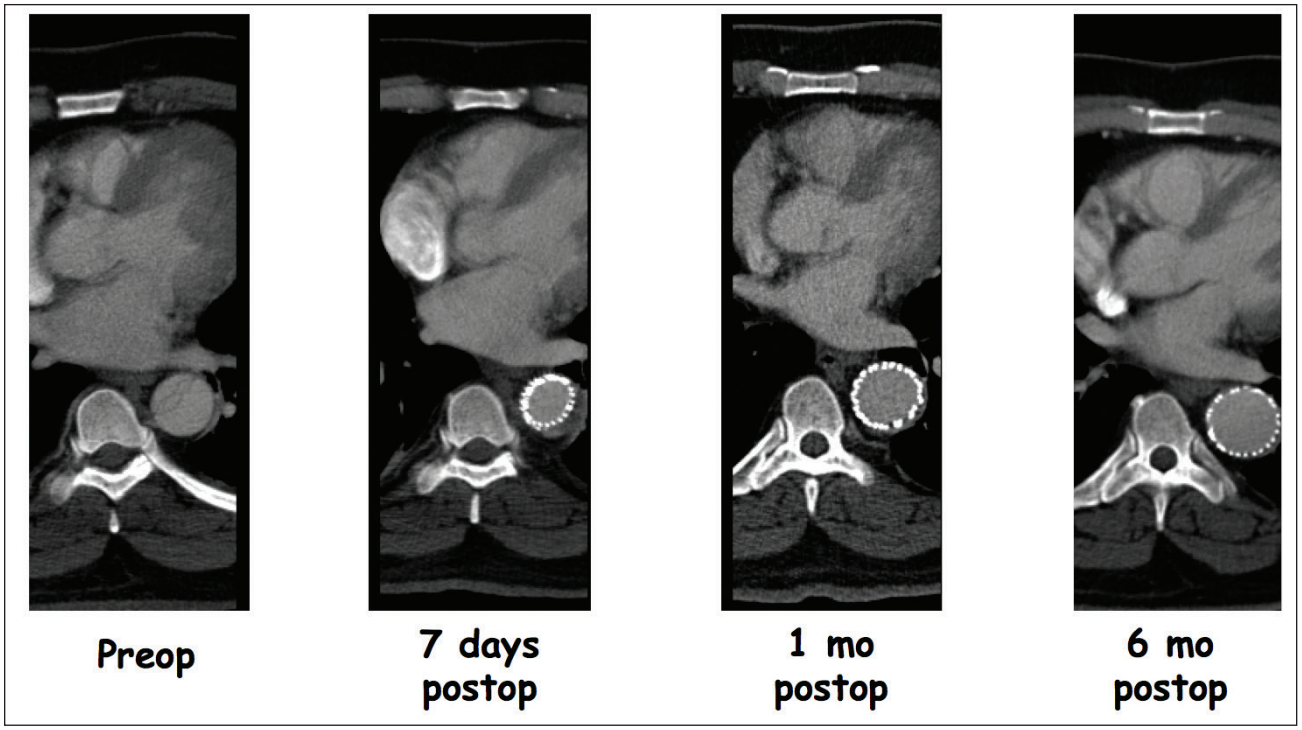


Figure 1. A patient with acute complicated type B aortic dissection was treated with a composite device design (stent graft and bare stent). Successive CTAs showing remodeling of the thoracic aorta, including expansion of the true lumen and thrombosis of the false lumen.

The procedure appeared to be safe, and good clinical outcomes were reported. Two years after the initial procedure, positive remodeling was observed in both the thoracic and the abdominal aorta.[18,19]

Recently, Patterson et al gathered 16 studies that reported the results of aortic remodeling after TEVAR with stent graft alone in aortic dissection.[20] In their study, patients with acute (≤ 14 days) and chronic (> 14 days) aortic dissections were analyzed separately. Patients with infrarenal extension of the aortic dissection experienced less aortic remodeling. This may be because TEVAR did not cover distal, secondary re-entry tear(s). The main limitation of the study was that no standard analysis was performed to evaluate aortic remodeling. The lack of clear reporting standards means that the clinical evidence is heterogeneous, making it difficult to evaluate the impact of TEVAR on aortic remodeling following aortic dissection. Also, the additional role of the bare stent in aortic remodeling has not yet been fully determined, and questions surrounding the use of the bare stent focus largely on its application and utility, compared to TEVAR alone.

We performed a retrospective analysis including 84 patients that compared aortic morphological and clinical outcomes of patients undergoing endovascular repair with stent graft(s) alone (TEVAR, 45 patients) and patients who were treated in the STABLE 1 study with the composite device design (STABLE, 39 patients).[21] The analysis focused on aortic remodeling at 1 year. Only patients with complicated acute dissection (≤ 14 days) and with available CT scans at preprocedure and 1-year follow-up were included. The study provided a thorough aortic morphological analysis including lengths, diameters, volumes, intimal tears, and lumen patency, performed on a dedicated three-dimensional workstation. Remodeling of the dissected aorta was assessed by changes in diameter and volume of the false lumen, true lumen, and total lumen and also patency of the false lumen on the preoperative and latest CT angiography performed during follow-up.

Both groups presented with largely comparable preoperative medical conditions and similar extent of aortic dissection; however, the false lumen volume was significantly larger in the STABLE group in both the thoracic and abdominal aorta. The length of aorta covered by stent grafts was not statistically different in both groups (167 ± 47 mm in STABLE patients; 184 ± 49 mm in patients with stent graft alone; $P = .11$).

Both groups exhibited extensive thrombosis of the thoracic false lumen after endovascular repair, although complete false lumen thrombosis occurred less frequently in the abdominal aorta. Although there was no statistical difference between the two groups in the proportion of patients who experienced > 10% in changes to the

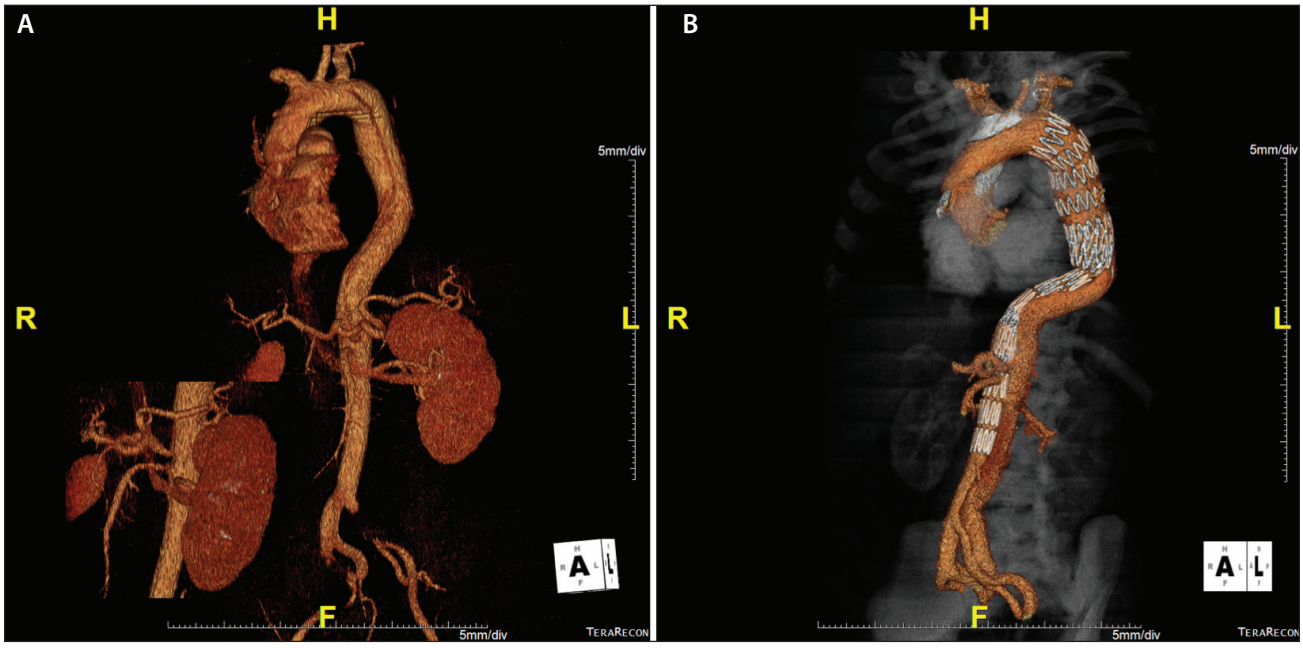


Figure 2. A patient presented with acute type B aortic dissection complicated with visceral and lower limb malperfusion (preoperative CTA) (A). The postoperative CTA showed that the use of a composite device design promoted sufficient true lumen expansion to correct the malperfusion syndrome without additional selective stenting or fenestration (B). At this early stage, the distal false lumen was still patent.

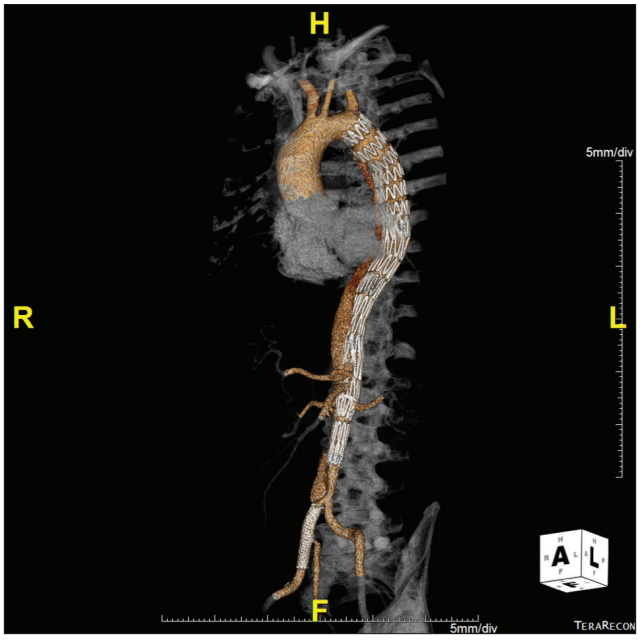


Figure 3. In the latter case, despite good reexpansion of the true lumen after the deployment of a composite device design, residual static malperfusion of the right common iliac artery required additional stenting.

thoracic or abdominal total lumen diameter or volume following endovascular repair through 12 months, there was a trend toward larger expansion of the true lumen, and more importantly, shrinkage of the false lumen in the abdominal aorta in patients treated with the composite device design. As a reminder, only patients who survived at 12 months were included in this study. Rupture, conversion, and reintervention rates were comparable between the two groups.

Previous publications have already confirmed that the deployment of a bare-metal stent within the distal aorta is safe and does not expose patients to a higher risk of complications.[2,17,22] Of note, in the setting of aneurysmal dilatation of the dissected aorta in the presence of a bare stent within the true lumen, fenestrated stent grafting can be performed.[23]

Aortic growth is frequently observed in patients who receive endovascular management in the acute phase.[17,19,22] More extensive coverage of the thoracic aorta usually results in early thoracic aorta remodeling and greater false lumen thrombosis, at least at the level of the stent graft.[24,25] The additional risks of extensive coverage (such as spinal cord ischemia) must be balanced against the risk of long-term aortic growth in each individual patient.

MALPERFUSION

Malperfusion in patients with type B aortic dissection is associated with 30-day mortality and morbidity rates of 2.7% and 51.8%, respectively.[26] Most cases of aortic branch malperfusion in aortic dissection involve a dynamic mechanism (true lumen collapse above or at the level of

the origin of the branch, with change in the flap position during the cardiac cycle); few cases of aortic branch malperfusion result from a static mechanism only.[27] In some cases, it can be difficult to make an accurate diagnosis of malperfusion due to incongruity between the clinical and imaging signs; also, there are no clear guidelines on when an intervention is warranted (Figure 2).

The correction of competitive flow and pressure between the true and the false lumens is usually obtained after coverage of the main proximal entry tear (frequently located around the aortic isthmus) with a stent graft. We only consider selective visceral/renal/lower limb artery stenting or flap fenestration after exclusion of the primary tear in the proximal descending thoracic aorta. After deployment of the stent graft, an angiogram is performed to evaluate the flow within the true lumen and whether any malperfusion remains. It will also depict large secondary tears that can generate dynamic malperfusion. In our practice, the coverage of the descending thoracic aorta is extensive only when malperfusion persists after deployment of the proximal stent graft and/or when aortic rupture is suspected. It always needs to be balanced against the risk of spinal cord ischemia. When a secondary entry tear is located in the abdominal aorta or close to the visceral/renal arteries, flap fenestration can be proposed to relieve malperfusion. In addition, the deployment of a bare stent can improve true lumen expansion and the quality of distal flow and thus reduce the risk of residual malperfusion after TEVAR and the need for additional revascularization (Figure 3).

In a previous series of 52 patients with acute type B aortic dissection treated in Lille, Caen, and Malmö between 2004 and 2011, 17 out of 22 patients presenting with malperfusion required additional revascularization after stent graft deployment.[22] In the STABLE 1 study of the composite device design, 9 of 40 patients (23%) required adjunctive branch vessel stenting during the index procedure.[18] From the morphological study of TEVAR and STABLE as previously described, we learned that the use of an additional aortic bare stent seems to promote aortic remodeling by increasing true lumen expansion and false lumen shrinkage in the abdominal aorta. Further analysis is required to determine the impact of the composite device approach on outcomes related to malperfusion. The origin of the visceral/renal/lower limb arteries should be noted before stent placement, because previous studies have shown there is reduced flow in vessels originating from the false lumen after endovascular repair.[28]

Taken together, placement of a bare-metal stent below a proximal stent graft in patients with type B aortic dissection appears to provide favorable short- and midterm outcomes, and treatment should be tailored to each individual patient, although longer-term outcomes are required to better understand the effect of this approach on aortic remodeling and malperfusion. ■

Jonathan Sobocinski, MD, PhD, is with the Aortic Centre, Vascular Surgery, Hôpital Cardiologique, Lille University Hospital, Lille, France. He has disclosed that travel expenses have been covered, and he has received speaker fees, research grants, or consulting fees from Cook Medical. Dr. Sobocinski may be reached at +33 320 445 811; jonathan.sobo@gmail.com.

Nuno V. Dias, MD, PhD, is with the Vascular Center Malmö-Lund, Skåne University Hospital, Malmö, Sweden. He has stated that he has no financial interest related to this article. Dr. Dias may be reached at +46 738 024 139; nunovdias@gmail.com.

Rachel Clough, MD, PhD, is with the Aortic Centre, Vascular Surgery, Hôpital Cardiologique, Lille University Hospital, Lille, France. She has disclosed that she has received speaker fees, research grants, or consulting fees from Cook Medical. Dr. Clough may be reached at +33 320 445 811; rachel.clough@kcl.ac.uk.

Stéphan Haulon, MD, PhD, is with the Aortic Centre, Vascular Surgery, Hôpital Cardiologique, Lille University Hospital, Lille, France. He has disclosed that he has received speaker fees, research grants, or consulting fees from Cook Medical. Dr. Haulon may be reached at stephan.haulon@chru-lille.fr.

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Toward the Uniluminal State

Evolving concepts in the endovascular elimination of aortic dissection.

BY PETER MOSSOP, MD, AND IAN NIXON, MD


BACKGROUND AND RATIONALE

Appreciation of the need to repair the entire dissected aorta in the setting of dissection initiated our evolution of the endovascular approach to aortic dissection in all its forms over a decade ago. Although placement of a proximal endograft to seal the primary entry tear reduces the risk of rupture and leads to shorter-term expansion of the true lumen in the thoracic aorta, the presence of more distal reentries is not addressed. The presence of persistent false lumen flow drives possible true lumen under perfusion and dynamic branch compromise and may increase the likelihood of further distal aortic degeneration in the longer term. This has been demonstrated in a number of studies relating to conventional thoracic endovascular aneurysm repair (TEVAR) for aortic dissection[1] and has been confirmed by computation fluid dynamics modeling.[2]

With this in mind, the aim of stent-assisted reconstruction beyond a proximal endograft to complete aortic dissection repair came about. The primary aims were multiple: to improve short-term outcomes in dissection management by reducing the chance of postoperative malperfusion, particularly for visceral and spinal circulations; to initiate acute and subsequent longer-term remodeling of the true lumen; and in conjunction with ancillary endovascular techniques in a staged fashion, to eliminate false lumen flow. The overarching aim of stent-assisted reconstruction is to achieve improved early and long-term outcomes compared to best medical treatment or TEVAR alone.

STENT-BASED RECONSTRUCTION

Staged total aortic and branch vessel endovascular repair, known as the STABLE concept, was first described in 2005[3] (Figure 1). Although isolated use of stenting for established malperfusion has been reported,[4] STABLE introduced the routine and novel use of an extensive self-expanding, dissection-specific stent scaffold to reexpand the true lumen and reestablish more normal flow dynamics.

Early experience also indicated that endograft and stenting in isolation was insufficient to fully eliminate residual inflows to the false lumen. An additional essential element of this approach involved adjunctive novel techniques for the endovascular elimination of residual false lumen flow. These techniques included closure of branch vessel reentries by focal branch-covered stenting reconnecting the stented aortic true lumen to the branch true lumen (Figure 2), much in the same way that a fenestrated endograft is connected to a native aortic branch. Selective embolization of the false lumen at the site of reentries was also found to be a useful adjunctive tool.

Usually after initial endograft and stent placement, in conjunction with elimination of any major branch perfusion abnormality or reentry, a period of follow-up was performed. Any subsequent persistent major entries or any evidence of false lumen growth were treated with

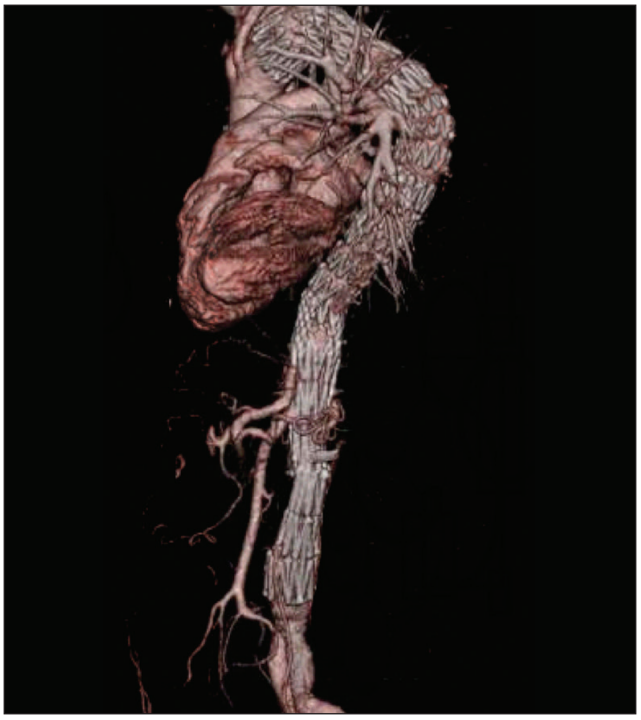

Figure 1. STABLE: Staged aortic and branch vessel endolumenal repair for aortic dissection. Ten-year follow-up computed tomographic angiography. Complicated type B dissection was treated with a TX2 endograft and distal dissection stents, in conjunction with branch vessel-covered stent repair.

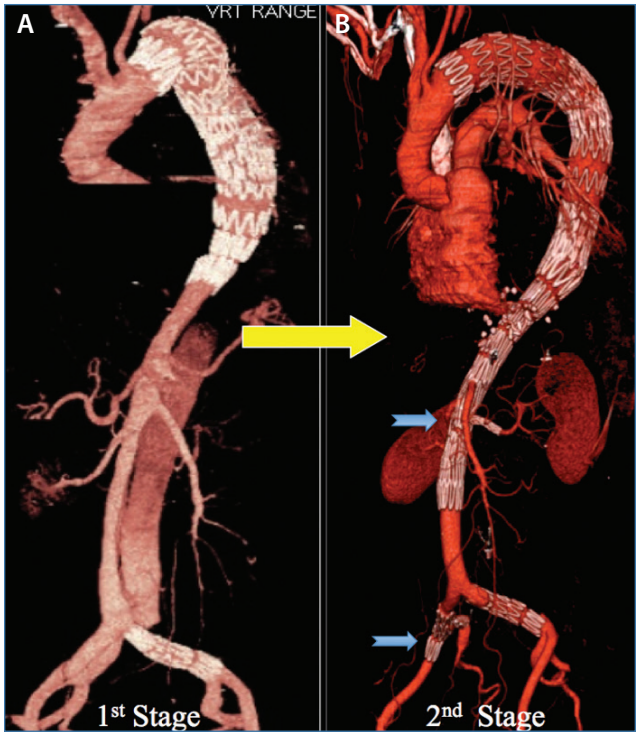

Figure 2. Staged total aortic and branch endolumenal repair. Following initial closure of primary entry tear (first stage), residual renal and iliac reentries are closed by branch-covered stents eliminating false lumen flow (second stage).

a staged intervention. Such procedures were generally preemptive and not indicative of reinterventions for aortic-related complications. When using such techniques to reconstruct the true lumen, it was found that aortic degeneration could be controlled. Hence, STABLE aims to achieve a more robust total aortic repair through application of a staged endovascular approach.

Subsequently, a number of small case series reported the use of bare-metal stents in the distal thoracoabdominal aorta, with favorable short-term results.[5] In 2008, Melissano and colleagues[6] reported early outcomes using the same Zenith (Cook Medical) dissection stent that was used in our cohort in 11 selected patients with chronic type B dissection. A clinical success rate of 91% was reported at 12 months, with 0% mortality, stroke, or paraplegia.

EARLY OUTCOMES

Evaluation of the early outcomes of the first 31 patients treated using STABLE was encouraging, with 30-day rates of death, stroke, and paraplegia/paresis at 3% (n = 1), 0%, and 0%, respectively.[7]

Malperfusion and Spinal Cord Ischemia

The initial experience of stent deployment beyond a proximal endograft to cover the lower thoracic and abdominal aorta demonstrated reperfusion of the true lumen, thus correcting true lumen collapse and protecting against visceral malperfusion.

In particular, our aim was to limit the extent of aortic coverage by endograft and avoid coverage of the subclavian artery if at all possible (with coverage only after careful assessment of the circle of Willis). After stenting segments of the more distal aorta, follow-up angiographic assessment often indicated improved direct flow from the true lumen to those branches arising from the now much-narrowed or nonexistent adjacent false lumen. In particular, this was frequently seen in relation to intercostal and lumbar branch vessels. This improved direct flow, combined with a reduction of any pressure gradient down the aortic true lumen, may account for the absence of spinal ischemia in this study.

COMPARISON TO TEVAR

STABLE was further validated by the first study comparing composite graft and stent treatment to conventional TEVAR alone. Between 2003 and 2010, 63 patients underwent treatment for acute and chronic dissection. They were divided into two groups: 40 underwent stent-assisted repair (STABLE), and 23 underwent proximal endograft repair alone (TEVAR).[8]

This study demonstrated that the addition of bare-metal scaffolding in the distal thoracoabdominal aorta significantly reduced visceral malperfusion in the acute phase compared with standard endovascular repair (0% vs 17%; $P = .02$). Moreover, this was achieved without increasing periprocedural morbidity or mortality. Spinal ischemia did not occur in the STABLE group (0% vs 4%).

Late follow-up (mean, 49 months) showed that STABLE was superior to TEVAR with fewer late reinterventions (11% vs 43%; $P = .007$), no distal late aortic reintervention (0% vs 19%; $P = .01$), fewer late adverse events (3% vs 10%; $P = .28$), and lower late aortic mortality (3% vs 9%).

Significantly, while bare-stenting of the dissection was at the heart of this approach, it is considered that the ancillary endovascular repairs were also key in achieving the highest level of false lumen exclusion and hence the greatest likelihood of preventing further aortic degeneration.

LONG-TERM OUTCOMES

Remodeling STABLE

Subsequent ongoing follow-up of aortic remodeling data (aortic dimensions measured at mid-descending and celiac) have also reflected aortic survival with thoracic and abdominal aortic dimensions remaining stable (Figure 3) over longer-term follow-up.

Survival

At a mean follow-up of 49 months, a cohort of 40 patients undergoing STABLE repair for acute and chronic dissection had an aortic-specific survival of 90%.[8]

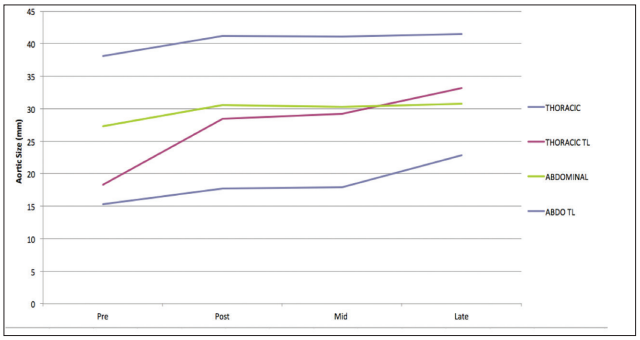

Figure 3. Remodeling after STABLE (mean follow-up of 97 months, range 50–154 months). Thoracic and abdominal diameters were measured at mid-descending thoracic aorta and coeliac levels and at initial (mean, 1 month), mid-term (24 months), and latest follow-up (mean, 97 months), respectively.

More selective follow-up of 33 patients undergoing stent- and endograft-based reconstruction for acute and sub-acute type B dissection demonstrated overall survival of 91% at 97 months (range, 50–154 months), while aortic-specific survival was 94%. Four patients (13%) underwent device-related reintervention. One (3%) late aortic-related death occurred. Survival rates in this group were higher than similar studies in the literature, with survival rates ranging from 56.3% to 87% at 5 years.[9]

INTIMAL DISRUPTION AND RELAMINATION: RECREATION OF THE UNILUMINAL AORTA

Although STABLE has demonstrated the utility of stent-based aortic reconstruction in both acute and chronic dissection, further evolution of the initial approach has occurred over the last decade. The aim was the creation of a more rapid and complete method of repair for acute and chronic dissection, particularly in patients in whom early dilatation of the aorta postdissection had not yet occurred.

In further clinical evaluation of this concept, balloon expansion within the distal endograft at the thoraco-abdominal junction has been demonstrated to seal the upstream false lumen while initiating intimal fenestration, which can be propagated more inferiorly, allowing further stent expansion and intimal relamination. Stent-based reapposition of intima to the aortic wall with creation of a uniluminal aorta resulted in complete elimination of the false lumen space. Hence, mitigation of the significant hemodynamic drivers of false lumen expansion (ie, false lumen shear flow and pressurization) (Figure 4), is achieved. Furthermore, this approach appears feasible in at least 50% of acute dissections currently treated by our group.

Early and Late Outcomes

Early and intermediate results of this investigational study were reported in 2012 in an initial 11 patients having appropriate morphology and undergoing repair of complicated aortic dissection.[10] There were no intraprocedural complications and no early incidence of stroke, spinal, or visceral ischemia. Median follow-up was 18 months (range, 4–54 months). No late adverse events or aortic-related deaths occurred. Complete false lumen obliteration occurred in 90% of patients.

Although limited in scope, this study suggests application in acute dissection may enable elimination of the entire false lumen space in up to 90% of treated cases either through false lumen thrombosis and remodeling of false lumen within the zone treated by endograft or through stent-supported relamination of the intima to aortic wall more distally.

The technique now has (subsequent to our initial report)[10] a maximum follow-up of 60 months (median, 33 months) in 21 patients and is associated with an aortic-specific survival of 95%. Stability or positive remodeling of both the thoracic and abdominal total aortic diameters has occurred in 90% of patients.

CONCLUSION

In conclusion, it has been our contention that the false lumen should be considered a continuous single

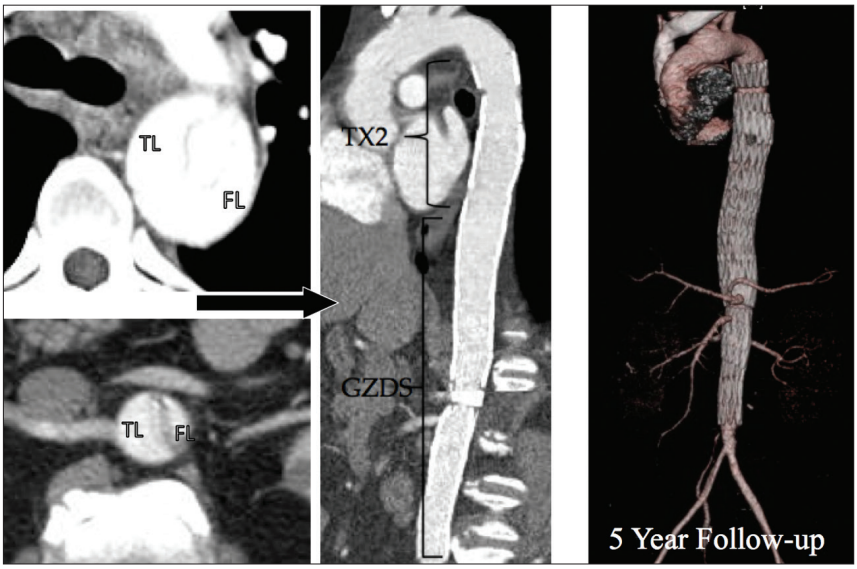

Figure 4. Distal aortic dissection treated with a proximal TX2 graft; more distal intimal disruption and bare stenting (Gianturco Zenith Dissection Stent, Cook Medical) create the uniluminal aorta. Initial and 5-year follow-up CT shows complete elimination of the false lumen.

compartment that communicates with the true lumen via intimal fenestrations throughout the dissected aorta. Hence, proximal endograft treatment alone is incomplete in managing dissection in its totality. Stent-assisted reconstruction and intimal disruption eliminating the false lumen and restoring a uniluminal status give us the ability to address the entire aortic dissection.

Improved short-term clinical outcomes in comparison to conventional endovascular techniques, high rates of aortic stability, and high aortic specific survival suggest the significant potential value of stent-based reconstruction in aortic dissection management.

More significantly, the ability to convert the dissected lumen to a uniluminal state offers the prospect of complete endovascular elimination of aortic dissection. ■

Peter Mossop, MD, is with the Department of Interventional Radiology, St Vincent's Hospital, University of Melbourne School of Medicine in Melbourne, Australia. He has disclosed that he was involved in patenting the Zenith Dissection stent and receives royalties from Cook, Inc. Dr. Mossop may be reached at petermossop@me.com.

Ian Nixon, MD, is with the Department of Cardiothoracic Surgery, St Vincent's Hospital, University of Melbourne School of Medicine in Melbourne, Australia. He has disclosed that he was involved in patenting the Zenith Dissection stent and receives royalties from Cook, Inc. Dr. Nixon may be reached at Ian.nixon@svhm.org.au.

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TEVAR for Chronic Type B Aortic Dissection

A discussion of therapeutic strategies for chronic type B aortic dissection, avoiding expansion of the false lumen, and treatment with endovascular therapy.

BY MASAAKI KATO, MD

It has been 23 years since we started practicing thoracic endovascular aortic repair (TEVAR) for the treatment of Stanford type B aortic dissection. In 1998, we reported that closing the entry site by stent grafts within 6 months from the onset of type B dissection was associated with better clinical outcomes, leading to reduced size of the false lumen.[1] However, in the real-world clinical practice, TEVAR is not always performed in a timely manner, and it is often done after an aneurysm is formed in the false lumen. In such cases of a dissecting aneurysm, TEVAR may not be effective in leading to regression of the false lumen.[2] Open surgery, on the other hand, inevitably requires replacement of the extended thoracoabdominal aorta with a prosthetic graft, because the dissecting aneurysm is often enlarged and straddles the thoracoabdominal segment. The intercostal and lumbar arteries often remain patent, as a result, the steal phenomenon is likely to occur during the open surgery, resulting in increased incidence of paraplegia. Consequently, in the treatment of type B aortic dissection, therapeutic intervention after expansion of the false lumen could yield a poor outcome and encounter greater difficulty. In this article, we first present two clinical cases and then discuss therapeutic strategies for chronic type B aortic dissection, by which expansion of the false lumen can be avoided, and in cases in which it occurs, patients can be treated by endovascular therapy.

CASE 1: A 61-YEAR-OLD MAN

A patient developed acute type B aortic dissection 2 months before referral to our hospital. He had no major complications during the acute phase and received antihypertensive treatment with a β-blocker. We considered that an early intervention by TEVAR was indicated in this case and performed two debranching procedures (right axillary-left carotid/left axillary artery bypass) plus TEVAR (Figure 1). The surgery consisted of the bypass (two debranching procedures) followed

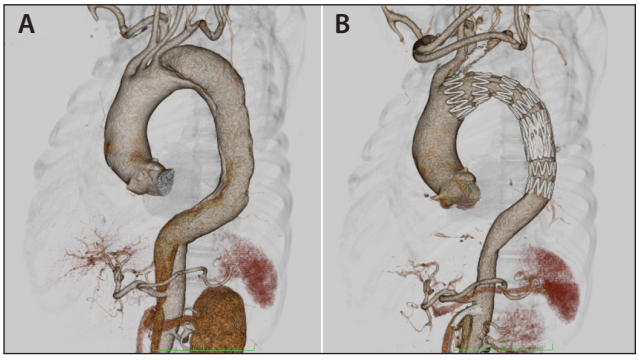

Figure 1. Two debranching procedures plus TEVAR in a patient with a chronic type B dissection 2 months after the onset. Preprocedure (A). Postprocedure (B).

by implantation of a Cook Zenith TX2 distal extension (diameter: 26 mm; length: 80 mm) in the descending aorta through a unilateral femoral artery, subsequent implantation of a Zenith TX2 tapered main body graft (diameter: 36–32 mm; length: 147 mm) in a piled-up manner from zone 1/aortic arch to the descending aorta, and an ensuing entry closure just distal to the left subclavian artery. A contrast-enhanced CT scan performed 4 days after TEVAR showed extensive thrombosis of the false lumen. A CT scan after 6 months showed that the thrombosed false lumen seen at the acute phase of treatment had eventually regressed (Figure 2).

Because this case was referred to our hospital 2 months after the onset of dissection, and considering the patient's age and anatomical features (maximum aortic diameter of 45 mm), we assumed that expansion of the false lumen would likely occur in the chronic phase. Certainly, the standard initial treatment options for acute type B aortic dissection are antihypertensive treatment and bed rest. Patients who have overcome the acute phase with optimal medical treatment are usually followed in ambulatory care for any expansion of the false lumen. In order to avoid such clinical progress, we previously explored predictive factors at the onset of dissection that may be associated with chronic-phase

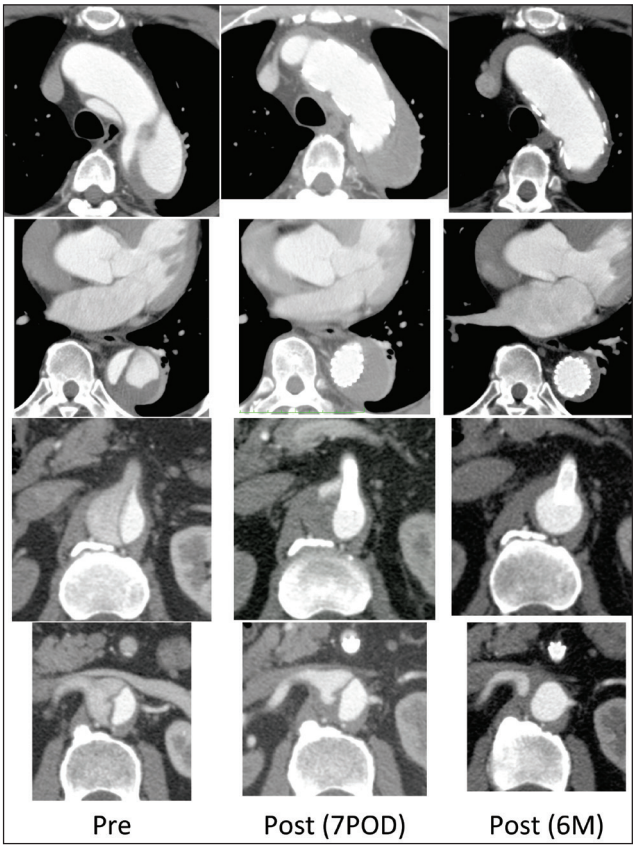

Figure 2. CT at preprocedure, postoperative day (POD) 4, and 6 months after the TEVAR for chronic type B dissection. Note that the false lumen eventually shrunk 6 months after the TEVAR.

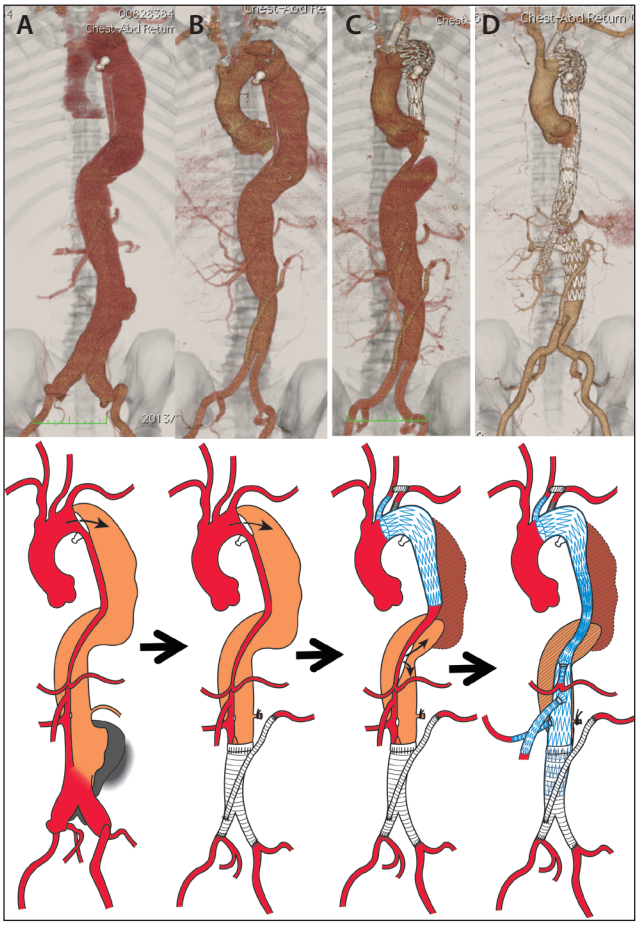

Figure 3. Total endovascular treatment in a patient with a chronic type B dissecting aneurysm 2 years after the onset.

expansion of the false lumen. We found that chronic-phase expansion of the false lumen is likely to occur when: (1) the maximum aortic diameter is ≥ 40 mm at the acute phase of aortic dissection and (2) the entry site of dissection is patent in the thoracic region.[3] Using these two predictors, it is possible to identify patients with type B aortic dissection who can benefit from TEVAR at an early stage in terms of prevention of false lumen expansion. Taking into account a patient's age at the onset, the etiology of the dissection, and the previously mentioned predictors for false lumen expansion, we developed the following criteria to perform early TEVAR intervention in patients with uncomplicated chronic type B aortic dissection (CBAD):

1. The maximum aortic diameter at the onset is ≥ 40 mm, and an entry site is patent in the thoracic region,
2. The age at onset is ≤ 65 years, the maximum aortic diameter is ≥ 35 mm, and an entry site is patent in the thoracic region, or
3. The patient has hereditary or possibly hereditary aortic dissection, and an entry site is patent in the thoracic region.

CASE 2: A 45-YEAR-OLD MAN

A patient with CBAD in the second year after the onset of dissection was referred to our hospital for emergency care because of a rupture of an infrarenal abdominal aortic aneurysm at the distal end of a false lumen (Figure 3A). As false lumens of the descending and thoracoabdominal aorta were already enlarged, we judged that all dissected aortas should be treated. The primary entry tear was located in the distal arch. The other entries were also patent, one at the thoracic descending aorta, one immediately superior to the celiac artery, and one at the left renal artery bifurcation. The reentry was found in the thrombosed infrarenal abdominal aorta, which constituted the distal end of the false lumen. At the visceral arteries level, the celiac artery, the superior mesenteric artery, and the right renal artery originated from the true lumen, whereas the left renal artery branched from the false lumen. Because the abdominal aortic aneurysm in the distal end of the false lumen had ruptured, emergency replacement of the abdominal aorta with a prosthetic graft was carried out.

For the left renal artery branching from the false lumen, subsequent reconstruction was deemed difficult, so a bypass surgery from the prosthetic graft was performed (Figure 3B). The proximal part of the aortic dissection was anastomosed so that a double-barrel aorta was maintained. Twenty-one days after this emergency abdominal surgery, the patient underwent a debranching procedure (left carotid-left subclavian artery bypass) plus TEVAR (implantation of a Zenith TX2 distal extension [diameter: 22 mm] in the descending aorta and a subsequent insertion of a Zenith TX2 tapered main body graft [diameter: 34–38 mm] in a piled-up manner from zone 2 to the descending aorta) in order to close the primary entry at the distal arch (Figure 3C). Fifty-five days later, fenestrated endovascular aortic repair (FEVAR) was carried out on the thoracoabdominal aorta using a Cook custom-made device (Figure 3D). Consequently, all entry and reentry tears in this case were successfully closed, and the false lumen in the entire region of the dissected aorta was thrombosed. CT scans 1 year after the thoracoabdominal endovascular repair showed shrinkage of the thrombosed false lumen (Figure 4).

We previously investigated changes in the false lumen diameter and aortic diameter by following patients who had false lumen enlargement ≥ 6 months after the onset of dissection and underwent closure of the primary entry tear by TEVAR alone.[2] We found that the percentage of patients who had shrinkage of the false lumen was small (38.9% had ≥ 5-mm shrinkage of the aortic diameter at the maximum diameter site in the thoracic aorta). Moreover, when the lesion was extended into the thoracoabdominal aorta, patients were more likely to have enlargement of the false lumen (46% had ≥ 5-mm expansion of the aortic diameter at the site immediately superior to the celiac artery). Therefore, in patients such as the present case, who had enlargement of a false lumen involving the thoracoabdominal aorta with several entries and reentries, we considered that closure of the primary entry tear alone would not lead to regression of the false lumen, and thus, closure of all entry and reentry tears should be undertaken.

Eventually, stent grafts need to be placed extensively from the distal arch down to the abdominal aorta and the iliac artery level. Major problems associated with such endovascular treatment include how to treat visceral arteries and the risk of spinal cord injury (SCI). We have overcome these problems by adopting hybrid thoracoabdominal endovascular aortic repair (hybrid TAEVAR) since 1997[4] and FEVAR since 2010[5] for revascularization of visceral arteries. In hybrid TAEVAR, reverse bypass surgery is performed from the abdominal aorta, iliac artery, or prosthetic abdominal aortic graft to visceral arteries (celiac, superior mesenteric artery, and bilateral renal arteries). After the revascularization, a stent graft is inserted from the thoracic descending aorta into the true lumen of the abdominal aorta/iliac arteries so that entry and reentry tears can be closed in the entire region of dissection. This operative procedure involves a major open abdominal surgery and a long retrograde bypass, so there is a concern that a sudden occlusion, intestinal adhesion, fistel formation, and infection may occur. FEVAR, on the other hand, is a procedure to reconstruct visceral arteries using Cook's custom-made device; a small-caliber stent graft such as Atrium Advanta (Maquet Vascular Systems) or Fluency (Bard Peripheral Vascular, Inc.) is inserted into visceral arteries via a fenestration that is designed according to the anatomy of individual patients' arteries.

Compared to a total endovascular repair for the

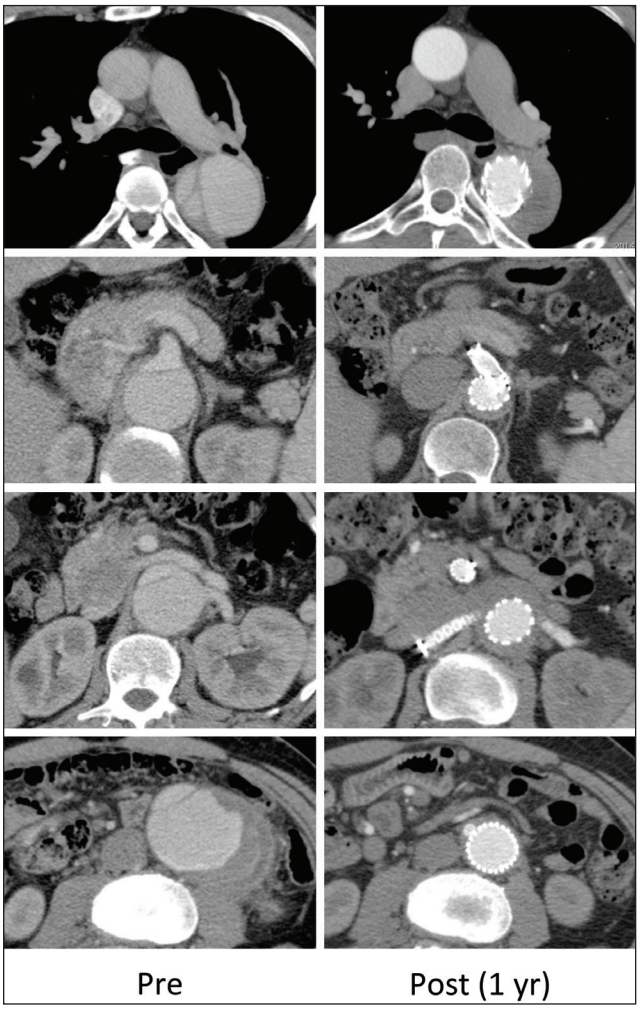

Figure 4. CT at preprocedure and 1 year after the total endovascular treatment for a chronic type B dissecting aneurysm. Note that the false lumen was thrombosed in the entire region of the dissected thoracoabdominal aorta and shrunk eventually 1 year after the thoracoabdominal endovascular treatment.

treatment of a true thoracoabdominal aortic aneurysm, this procedure for aortic dissection has the disadvantage of being extremely technically difficult in that the compressed true lumen allows only a narrow working space for branch reconstruction and in that branching of a false lumen and dissection of branches per se may occasionally be involved.

A possible increase in the incidence of paraplegia and paraparesis was initially of concern in the practice of endovascular treatment of these thoracoabdominal aortic regions, because the procedure entails occlusion of the intercostal and lumbar arteries in the entire region of stent graft implantation. However, the apprehension has lessened with the increasing number of patients treated with these procedures. According to our experience, the incidence of SCI is kept low by securing collateral circulation to the spinal cord and maintaining an adequate spinal cord perfusion pressure.[4,5] To prevent spinal nerve complications, we have developed the criteria and treatment regimen in high-risk patients for SCI as follows:

SPINAL CORD PROTECTION

We established and strictly adhere to the following principles to prevent SCI in patients undergoing treatment for thoracoabdominal aortic dissection:

1. During planning, the aim is to minimize the extent of the intercostal and lumbar arteries covered with the endovascular graft.
2. Cerebrospinal fluid drainage is established the day before the procedure for patients with the following required conditions:
   a. The treatment region covers ≥ 4 segments of the intercostal and/or lumbar artery between thoracic vertebra (T) 8 and lumbar vertebra (L) 1.
   b. The treatment region covers 3 segments of the intercostal and/or lumbar artery between T8 and L1, and the patient has had previous aortic surgery and/or malperfusion in the subclavian artery or internal iliac artery.
3. Preoperative or intraoperative reconstruction of the internal iliac artery and/or subclavian artery is to be performed, if possible, for any potential stenosis.
4. A statin is administered preoperatively for ≥ 1 week (since 2011).
5. Opioid use is avoided on the day of the TEVAR and in the early postoperative period.
6. Mean systemic blood pressure in the intraoperative and immediate postoperative period is strictly maintained at ≥ 80 mm Hg.
7. The time required for intraoperative malperfusion of the iliac, femoral, and subclavian arteries, particularly for the internal iliac arteries, is minimized.
8. Bypass surgery and TEVAR, as well as each TEVAR are done separately, as much as possible.

With introduction of hybrid TAEVAR, FEVAR, and prevention measures against SCI, it is now possible to close all entry and reentry tears in patients with CBAD who have had expansion of the false lumen. Consequently, complete thrombosis and subsequent regression of the false lumen can be achieved. In other words, use of those measures is invaluable in the treatment of CBAD with an expanded false lumen.

CONCLUSION

In the treatment of CBAD, it is important to perform TEVAR to close the primary entry tear within 6 months from the onset of dissection when there is a possibility that a false lumen may be expanding. In patients who were not indicated for early TEVAR and consequently had false lumen expansion, all entry and reentry tears should be closed individually. The percentage of patients with CBAD indicated for TEVAR in the total aortic dissection, including treatment for postoperative type A dissection, is estimated to be approximately 30% to 40%, constituting a growing field of TEVAR. Establishment of clearly defined therapeutic indications and treatment goals is essential to reduce futile medical interventions and the incidence of complications. ■

Masaaki Kato, MD, is with the Department of Cardiovascular Surgery, Morinomiya Hospital, in Osaka, Japan. *He has disclosed that he receives research grants from Medtronic, Japan Lifeline Co., Ltd.; serves as an advisor and has received speaker fees, and/or consulting fees from Cook Japan, Inc.; and serves as an advisor to Japan Gore Inc., Junken Medical Co., Ltd.*

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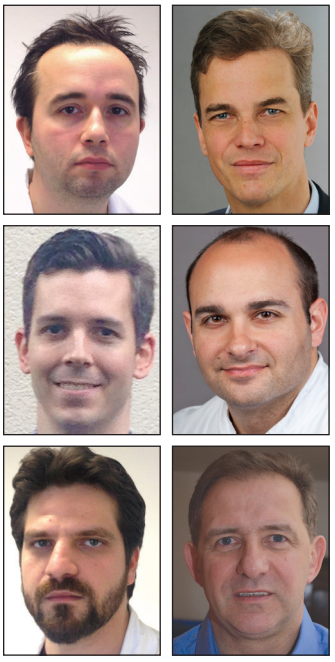
Postdissection Aneurysms

Devices and techniques for treating this condition.

BY GEORGE N. KOUVELOS, MD; TILO KÖLBEL, MD, PHD; ATHANASIOS KATSARGYRIS, MD; NIKOLAOS TSILIMPARIS, MD, PHD; KYRIAKOS OIKONOMOU, MD; AND ERIC L.G. VERHOEVEN, MD, PHD



Chronic type B aortic dissection (indication to treat: postdissection aneurysm) constitutes a unique condition that needs a different treatment strategy than applied in the treatment of acute or subacute type B aortic dissection. In acute type B dissection, the main goal is to close the entry tear and redirect blood flow to the true lumen, or to correct organ ischemia. In chronic type B dissection, the challenge is to prevent aneurysmal degeneration. As a postdissection aneurysm usually involves the thoracoabdominal aorta, the repair involves taking care of the visceral branches of the aorta in most cases. Open repair is very technically demanding and is associated with high mortality and morbidity, and the role of endovascular techniques in the treatment of chronic dissection is not yet defined.[1-3]

INDICATION

Diagnosis and management of acute aortic dissection have improved significantly during the last decades. Nevertheless, a number of surviving patients will develop a postdissection aneurysm. Ongoing aortic dilatation is usually the one indication in chronic type B dissection, while malperfusion rarely occurs years after the acute dissection. The extensive remodeling of the aorta with a small true lumen and the increasing fibrotic stiffness of the intimal flap represent an anatomy with specific technical challenges.

TREATMENT OPTIONS FOR POSTDISSECTION ANEURYSM

Open Repair

Due to many patients being unfit for such a major procedure, open surgical repair of postdissection thoracoabdominal aortic aneurysms (TAAAs) has been associated with significant risks. Current data focusing exclusively on open surgical repair of secondary postdissection aneurysms are limited to high-volume centers with significant experience in complex open aortic surgery.[4] Tian et al conducted a systematic review of literature on open surgical repair for chronic type B dissection and found 19 studies, which included 970 patients.[5] Overall, pooled short-term mortality was 11.1%, while stroke, spinal cord ischemia (SCI), renal dysfunction, and reoperation for bleeding occurred in 5.9%, 4.9%, 8.1%, and 8.1% of the patients, respectively. Late reintervention was needed in 13.3% of the patients, and aggregated survival at 3, 5, and 10 years was 74.1%, 66.3%, and 50.8%, respectively. Although these poor outcomes were partially attributed to patient selection and the extent of open surgery, because most centers selectively reserved open repair for patients with extensive diseases, it is clear that open surgical repair is an extensive procedure and should only be considered for patients in reasonably good condition.

Standard TEVAR

Thoracic endovascular aneurysm repair (TEVAR) is an established treatment in acute and subacute dissections that seems to promote false lumen thrombosis and aortic remodeling;[6] however, the role of TEVAR in treating patients with chronic type B dissection is not well defined. In a systematic review of midterm outcomes of TEVAR for chronic type B aortic dissection, Thrumurthy et al reported a nearly 90% technical success rate and 3.2% 30-day mortality rate.[7] However, the midterm reintervention rate of up to 60% was detailed, while nearly 10% of the patients developed aneurysmal progression of the aorta. In a small study of 76 patients, TEVAR resulted in a significantly decreased aortic diameter along the stent-grafted segment, but not in the distal untreated segments.[8] A recent study by Mani et al showed that total false lumen thrombosis occurred in only one-third of patients after TEVAR and was more common in dissections confined to the thoracic aorta (83%)

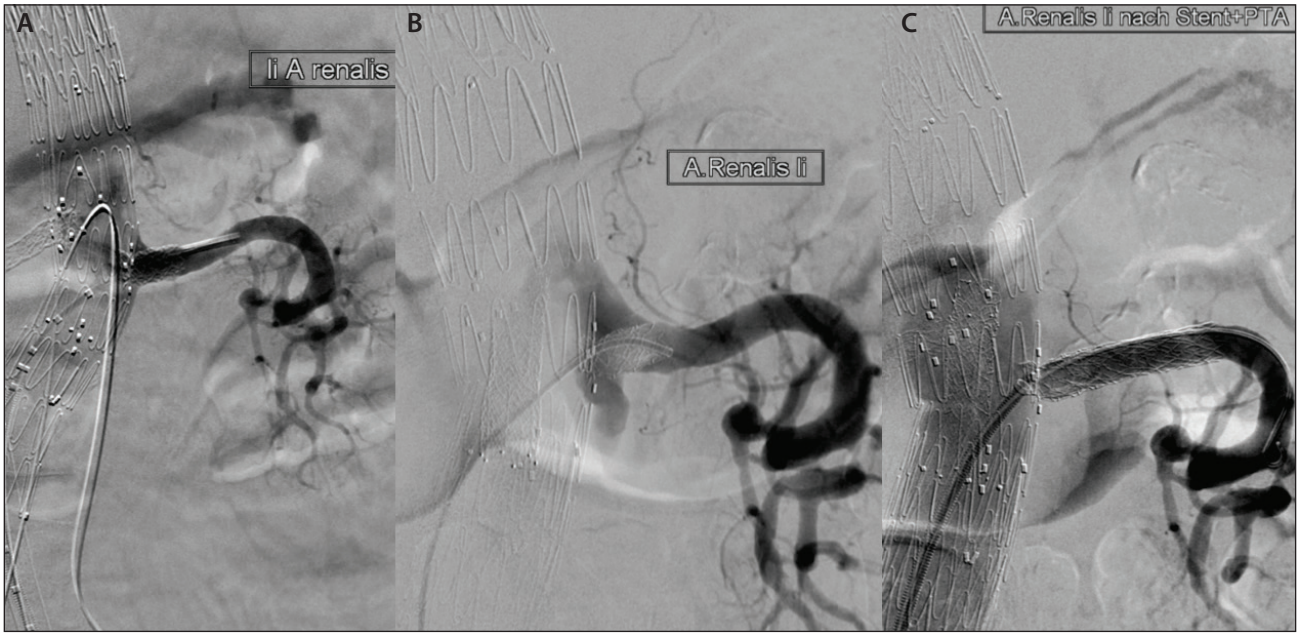


Figure 1. Completion angiogram after endovascular treatment of a postdissection aneurysm showing left renal artery patency with no sign of an endoleak (A). Selective angiography during follow-up showing a type Ib endoleak originating from the left renal artery (B). The endoleak was treated with distal extension of the covered renal bridging stent with an Atrium Advanta V12 stent graft (Maquet Vascular Systems) (C).

than those extended to the abdominal aorta (23%).[9] Therefore, standard TEVAR seems to play a minor role in the treatment of chronic type B dissection, and only for postdissection aneurysms limited to the thoracic aorta. In patients with extensive postdissection aneurysms, TEVAR plays no role.[10]

Treatment With Fenestrated and Branched Grafts

To achieve a complete exclusion of the thoracoabdominal aneurysmal degeneration, a more extensive endovascular approach with fenestrated and branched (F/Br) stent grafts was attempted; however, only in recent years and only by a few expert centers.[11-13] Early on, the specific anatomy in postdissection aneurysms discouraged even expert centers to address this pathology with F/Br grafts. A narrow aortic true lumen characterizes most of the cases and complicates planning. Fenestrations require less true lumen space for deployment, although the planning of the orientation of the fenestrations is more tedious than that for the treatment of standard TAAA. Branches are easier to plan, while the cannulation of target vessels may be simpler with a sharp take-off over a transaxillary access. Nevertheless, branches require more working space that is usually lacking in the narrow true lumen. Visceral branches originating from the false lumen create technical difficulties for catheterization. Different techniques have been used to perforate the dissection flap: wires with tips that can be stiffened, the back of a wire,

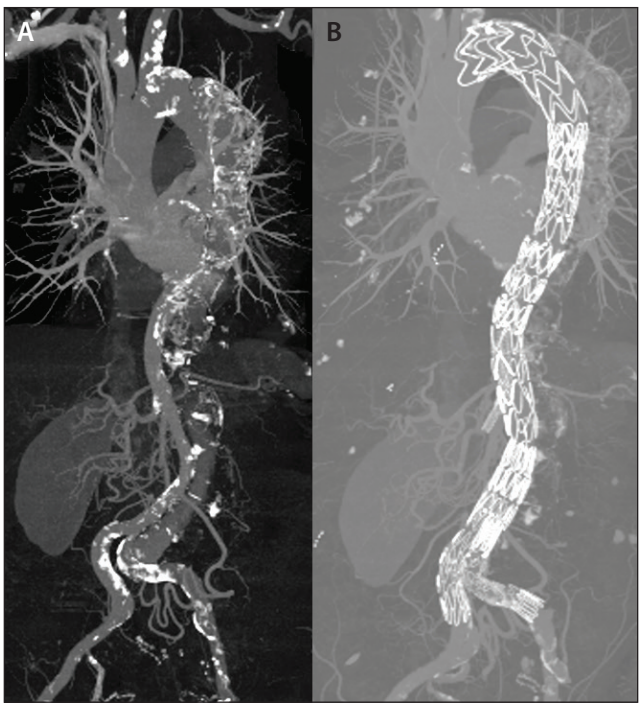


Figure 2. CT angiography (CTA) of a patient with a postdissection thoracoabdominal aortic aneurysm (A). CTA 2 years after endovascular treatment with fenestrated and branched stent grafting showing expansion of the true lumen and shrinkage of the false lumen (B).

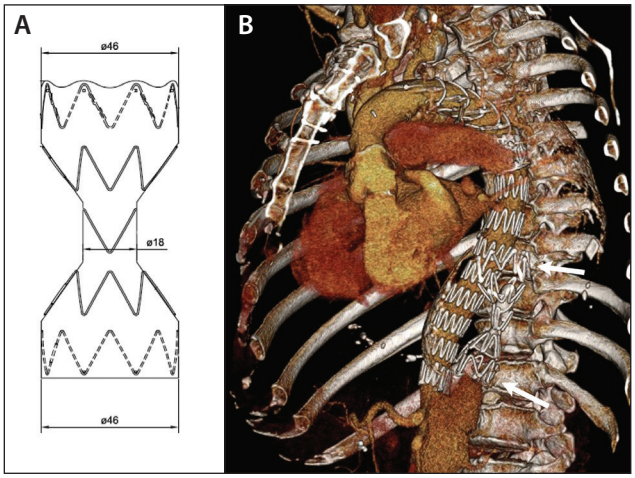


Figure 3. Technical drawing of a 46-mm Candy-Plug (A). 3D volume rendering of the postoperative CTA showing a patient after TEVAR extension to the celiac artery after previous frozen elephant trunk repair and Candy-Plug occlusion of the false lumen at the distal descending thoracic aorta (arrow) (B).

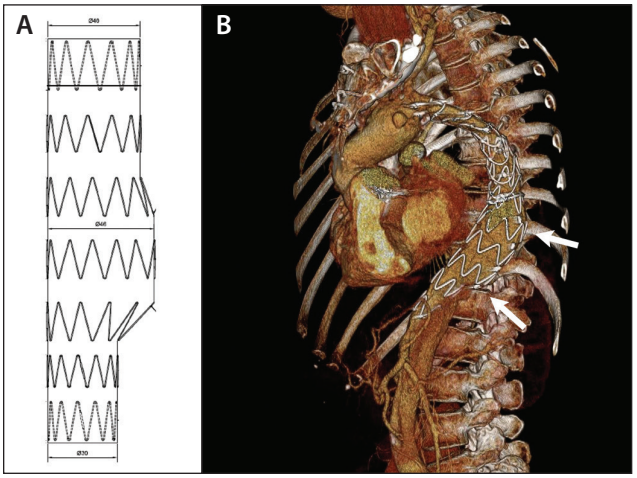


Figure 4. Technical drawing of a Knickerbocker graft with a 46-mm midsection (A). 3D volume rendering of the postoperative CTA showing a patient after TEVAR extension to the celiac artery after a previous frozen elephant trunk repair using a Knickerbocker graft for occlusion of the false lumen at the distal descending thoracic aorta. Arrows point at the bulbous midsection (B).

a wire with support of a guiding sheath, or even a TIPPS needle. Also, the distal landing zone can be compromised when the dissection extends to the common iliac arteries. Hypogastric artery flow should be preserved to reduce the risk of paraplegia. This can be achieved by incorporating an iliac branched device (IBD) to the repair plan, or by landing in a dissected common iliac artery and hoping for adequate sealing.

Data from the literature are still scarce and lack longer follow-up. Kitagawa et al reported the Cleveland Clinic experience on 30 patients with chronic dissection (15 focal and 15 with thoracoabdominal extent) treated with F/Br-EVAR.[13] Technical success was achieved in all patients, and no perioperative deaths occurred. One aortic-related death occurred at 3 months due to progression of a pre-existing untreated arch dissection. No ruptures, cardiac, renal, pulmonary, or SCI complications occurred. Also, no graft compression was noted, despite the initially narrow true lumen dimensions. During a mean follow-up period of 1.7 years, aneurysm sac growth was noted in two patients, related to type II endoleaks, which were treated with translumbar glue embolization. Eight (26.7%) patients (five with type I and three with type III endoleaks) underwent reintervention, with four patients requiring multiple endovascular procedures.

The Nuremberg experience in the treatment of postdissection thoracoabdominal aneurysm with F/Br-EVAR includes 31 patients (26 male, mean age 65 ± 9.6 years) treated between October 2010 and August 2015. Part of this experience was published before.[11-12] All cases were technically successful, but in one case, a retroperitoneal approach was needed for renal artery catheterization.

Two (6.4%) patients died within 30 days postoperatively, one due to multiple organ failure and one due to cardiac failure. Renal function impairment occurred in one (3.2%) patient. Perioperative SCI occurred in five (16.1%) patients. One (3.2%) patient suffered paraplegia with significant improvement prior to discharge, and four (12.9%) patients suffered transient paraparesis with complete recovery prior to discharge. One (3.2%) patient developed late (6 months) SCI with urinary incontinence and lower limb weakness due to regression of a type II endoleak. Mean follow-up was 17.6 months (range, 1–54 months). There was one death due to an aortoesophageal fistula 26 months postoperatively. Four target vessel occlusions were reported (three renal arteries, one celiac trunk) during follow-up. In one case, an iliac-renal bypass was carried out. One patient had a known occlusion of the left renal artery and became dialysis dependent after occlusion of the right renal artery stent graft. The remaining two cases were asymptomatic and did not require treatment. Endoleak was diagnosed in 13 (41.9%) patients during follow-up. These included five (16.1%) type Ib endoleaks (renal artery, n = 4; renal artery and superior mesenteric artery, n = 1), two (6.4%) distal type Ib endoleaks from dissected iliac arteries, five (16.1%) type II endoleaks, and one (3.2%) type III endoleak from a renal artery bridging stent graft. In four of the five patients with type Ib endoleak, a stent graft extension placed deeper into the target vessel resolved the problem (Figure 1). In the fifth patient with a type Ib endoleak, the renal artery stent graft was successfully reflared. In one patient, a type II endoleak from a lumbar artery was treated with embolization. In one patient with

bilateral type Ib endoleak from iliac arteries, iliac branched devices were implanted bilaterally. Finally, the one type III endoleak was treated with a bridging stent in the renal artery. Aneurysm sac regression during follow-up was significant from 67.4 ± 6.4 mm to 59.1 ± 7.5 mm (P = .007), with a false lumen thrombosis rate of 77.7% for patients that completed 12-month follow-up (Figure 2).

Endovascular Techniques for Occlusion of the False Lumen

Continued false lumen perfusion limits the response to endovascular treatment in patients with chronic type B aortic dissection by retrograde flow arising from distal entry tears within the abdominal aorta and iliac arteries. Occlusion of these connections between the true and false lumens can be achieved by stent graft coverage using fenestrated and branched endografts into the iliac arteries. This strategy allows for complete false lumen thrombosis throughout the dissected thoracoabdominal aorta, but as described above, these interventions are challenging and carry significant risks, especially for spinal cord ischemia because all segmental arteries may be covered during the treatment.

A proportion of patients with postdissection aneurysms develop aneurysmal dilatation in the distal aortic arch and the proximal and mid-descending thoracic aorta alone, while the abdominal segment remains relatively normal, not requiring treatment. This subset of patients may be treated with a less complex procedure, with a lower risk for SCI. This can be achieved by using standard TEVAR covering the thoracic aorta down to the celiac artery in combination with techniques to occlude the false lumen. Three options are described below:

Direct false lumen occlusion. Embolization of the false lumen channel at the level of the distal descending thoracic aorta can be achieved by a variety of embolizing agents. This method was first described by Loubert et al as the "cork-in-the-bottle neck" strategy that places cava filters, detachable balloons, thrombin, and Talent occluders (Medtronic) into the false lumen to successfully achieve occlusion.[14] Hofferberth et al described an extension of this method with the additional use of coils and cyanoacrylate glue in a study including 31 patients.[15] Recently, Idrees et al reported on 21 patients with chronic thoracoabdominal aortic dissection who underwent iliac occluders to embolize the false lumen. A 100% technical success rate and false lumen thrombosis in all patients at a median follow-up of 25 months were reported.[16] In patients with large false lumen diameters, embolization can be challenging, as commercially available materials for arterial embolization are not suitable for the large diameters required. To address the problem, two new techniques for direct false lumen occlusion at the level of the distal descending thoracic aorta were recently introduced, as described in the information to follow.

The Candy-Plug technique. Initially, this technique was introduced by using a 42-mm thoracic stent graft that was modified into a large candy-shaped plug by adding a diameter-restricting suture in the middle of the graft (Figure 3).[17] The plug was positioned in the false lumen opposing the distal end of the endograft in the true lumen. The restricted mid-section of the stent graft requires occlusion by a large Amplatzer vascular plug (St. Jude Medical, Inc.). More recently, the Candy-Plug has been produced as a custom-made implant with a maximum diameter of 50 mm and a mid-section of 18 mm, allowing for retraction of the dilator tip of the introduction system. To date, at the University Heart Center Hamburg, Candy-Plugs have been used successfully in 10 patients with large false lumen diameters.

The Knickerbocker technique. With this technique, the false lumen is occluded by expanding a large-diameter stent graft placed in the true lumen, which ruptures the dissection membrane into the false lumen on a limited segment of aorta.[18] After an initial experience with using oversized standard tubular stent grafts, custom-made double-tapered stent grafts with a bulbous section are used today. Gold markers direct the bulbous section toward the false lumen. The Knickerbocker graft is deployed within the intended segment of the aorta with a sufficient overlap to the proximal stent graft and ending proximal to the celiac artery (Figure 4). After orienting the gold markers toward the false lumen and deploying the graft, a compliant balloon is used to dilate the bulbous section of the stent graft until the dissection membrane ruptures in the intended segment of the aorta and the oversized stent graft expands into the false lumen, sealing off false lumen backflow. The resulting shape of the stent graft is similar to knickerbocker trousers, hence the denomination. At the University Heart Center Hamburg, Knickerbocker grafts have been used successfully in nine patients with large false lumen diameters.

CONCLUSION

Postdissection patients need treatment in case of aneurysmal degeneration. This aneurysmal degeneration usually involves the thoracoabdominal aorta. Open surgery represents a challenging procedure for both patients and physicians who are involved. Standard TEVAR plays virtually no role, as it cannot exclude the distal aneurysm. Fenestrated and branched endografts have been used with success, but longer follow-up is needed to demonstrate effectiveness and durability. Special endovascular techniques that aim at occluding the false lumen distally, using custom-made grafts to perforate the dissection flap or vascular plugs to embolize the false lumen, have demonstrated technical feasibility and could play a role in the subset of patients where the abdominal aorta is not involved. ■

George N. Kouvelos, MD, is with the Department of Vascular and Endovascular Surgery, Klinikum Nuernberg in Nuernberg, Germany. He has stated that he has no financial interests related to this article.

Tilo Kölbel, MD, PhD, is with the Department of Vascular Medicine, University Heart Center in Hamburg, Germany. He has disclosed that he is an intellectual property holder of Cook Medical, and he has received speaker fees, research grants, or consulting fees from Cook Medical. Prof. Kölbel may be reached at t.koelbel@uke.de.

Athanasios Katsargyris, MD, is with the Department of Vascular and Endovascular Surgery, Klinikum Nuernberg in Nuernberg, Germany. He has stated that he has no financial interests related to this article.

Nikolaos Tsilimparis, MD, PhD, is with the Division of Vascular Surgery and Endovascular Therapy, Emory University School of Medicine in Atlanta, Georgia. He has disclosed that he has no financial interests related to this article.

Kyriakos Oikonomou, MD, is with the Department of Vascular and Endovascular Surgery, Klinikum Nuernberg in Nuernberg, Germany. He has stated that he has no financial interests related to this article.

Eric L.G. Verhoeven, MD, PhD, is Chief, Department of Vascular and Endovascular Surgery, Klinikum Nuernberg, Paracelsus Medical University Nuernberg in Nuernberg, Germany. He has disclosed that he has received educational grants and is a consultant for Cook Medical, Gore & Associates, Siemens, AtriumMaquet, and Medtronic. He also provides educational speaker services to TriVascular, Inc.

Prof. Verhoeven may be reached at +49 9113982651; eric.verhoeven@klinikum-nuernberg.de.

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