

2023 MID-ATLANTIC CONFERENCE
11th ANNUAL CURRENT CONCEPTS IN
VASCULAR THERAPIES

2023

Hilton Virginia Beach Oceanfront
Virginia Beach, Virginia

APRIL 20-22



2023 MID-ATLANTIC CONFERENCE

11th ANNUAL CURRENT CONCEPTS IN

VASCULAR THERAPIES

2023



Acute Aortic Syndrome: How to Recognize a Surgical Emergency

Hosam F El Sayed, MBBCh, PhD

Sentara Vascular Specialists

April 21, 2023

Acute Aortic Syndrome

- It is a constellation of life-threatening aortic diseases
- They usually have similar presentation in the form of acute chest and back pain.
- They may start as one entity and evolve to another or may coexist.
- They do have distinct demographic, pathologic, clinical and survival characteristics.

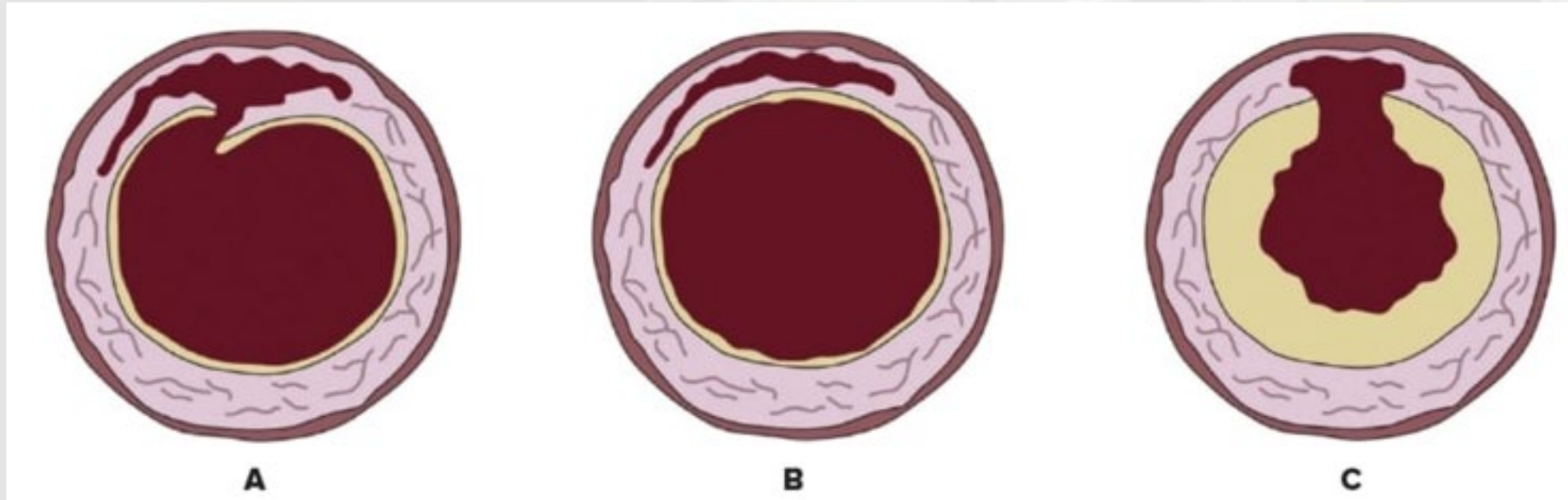


Acute Aortic Syndrome

Aortic Dissection

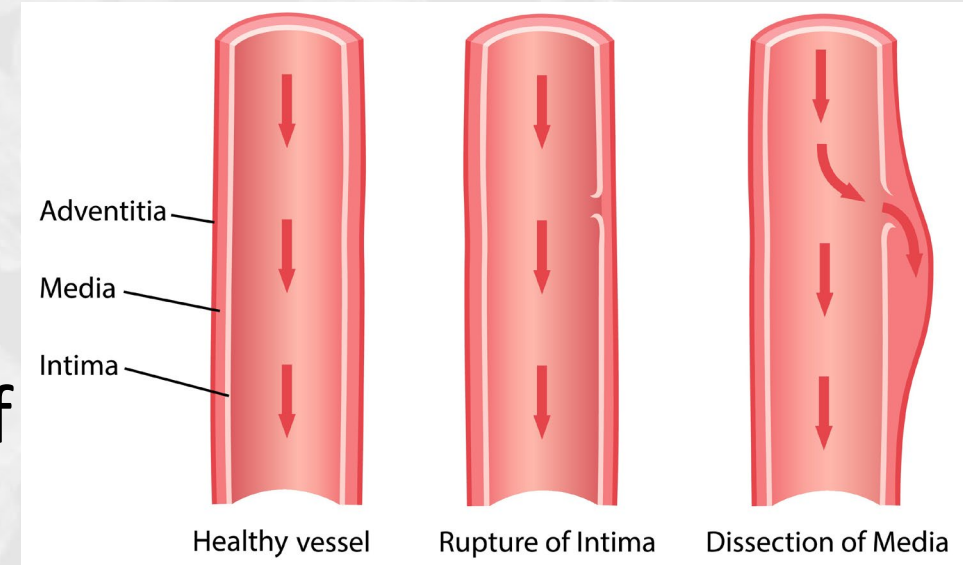
Intramural Hematoma

Penetrating Aortic Ulcer

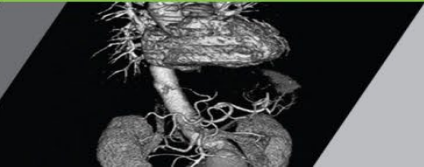
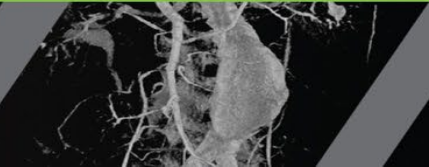
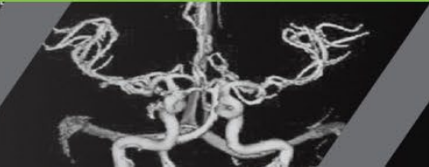


Acute Aortic Dissection

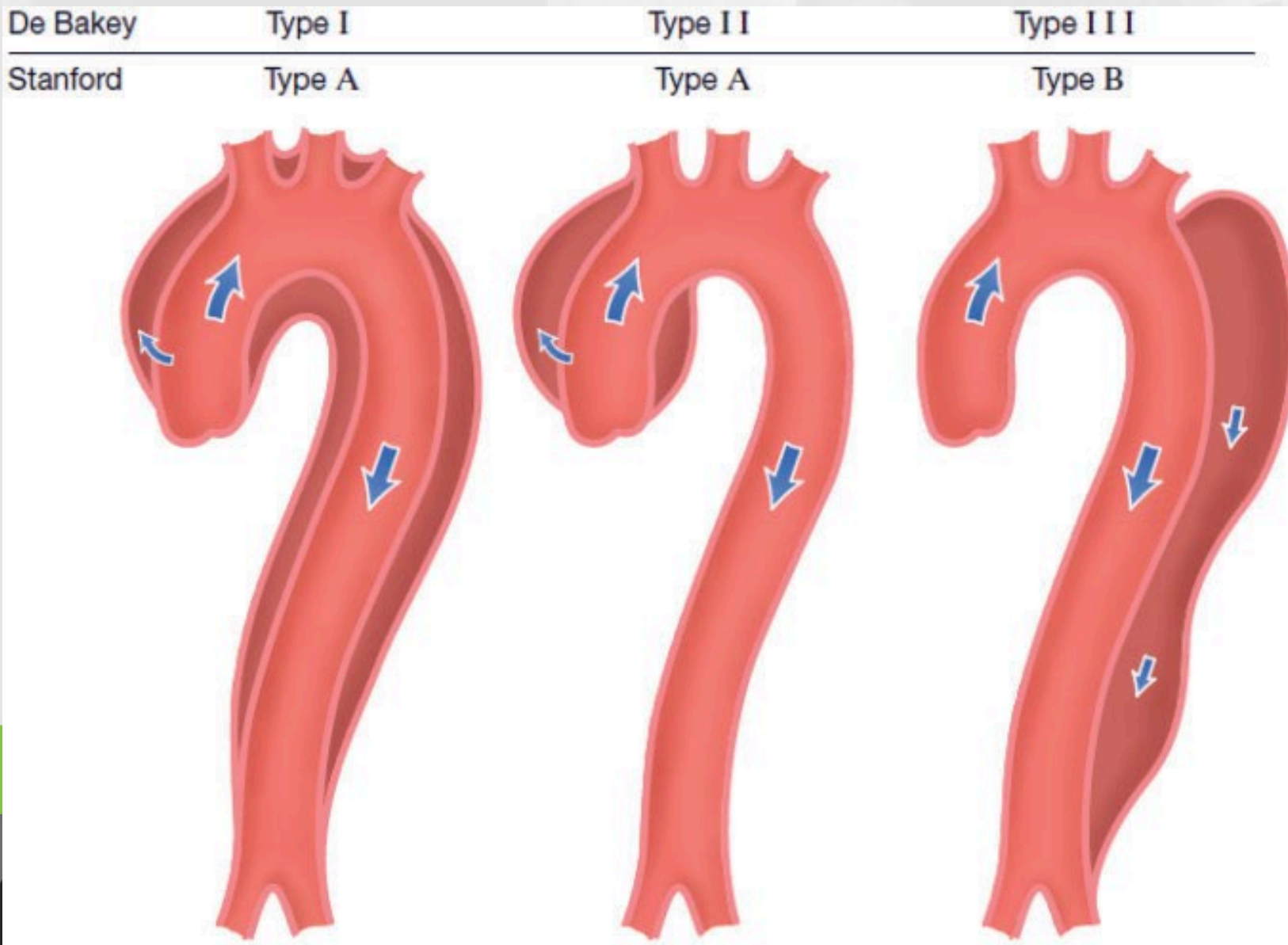
- This is the commonest among the AAS (85-95%)
- Incidence is 3/100,000 person-years.
- 2/3 of the patients are males
- Intimal tear + separation of the layers of the aorta
- False Lumen has higher pressure than True Lumen



Classic Teaching



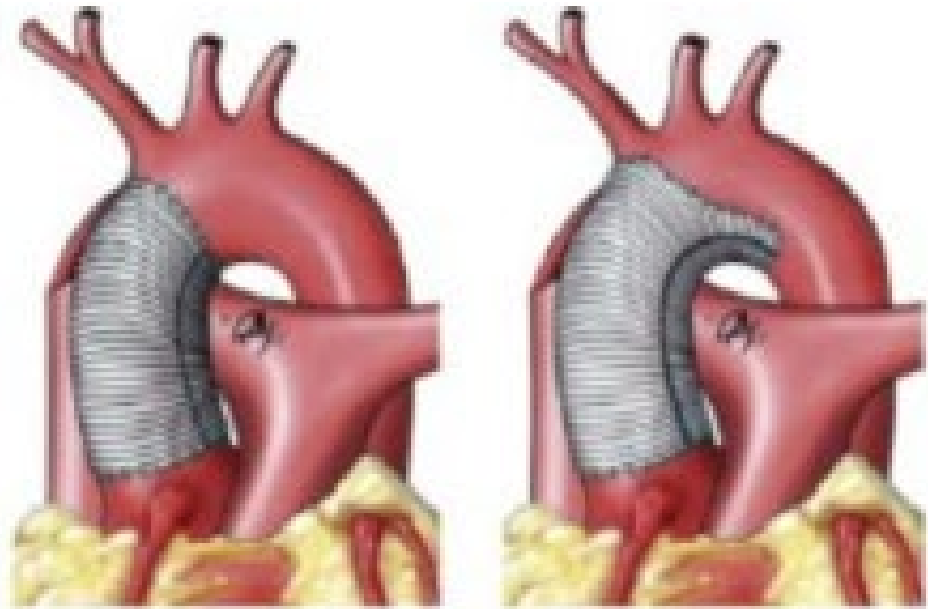
Classification



Acute Type A Dissection

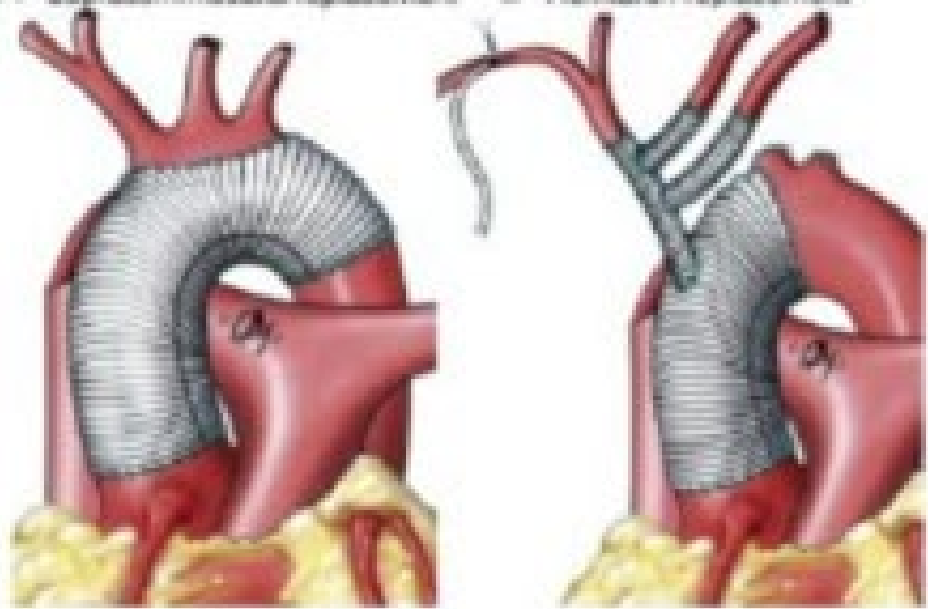
- More common than type B aortic dissection (2/3 of cases)
- High mortality (1%/hour) (Rupture, pericardial tamponade, Acute AI, Coronary dissection)
- It is a surgical emergency
- Different levels of repair based on the pathology.
- All require ascending aortic replacement.
- May require aortic root replacement, coronary reimplantation, aortic valve replacement





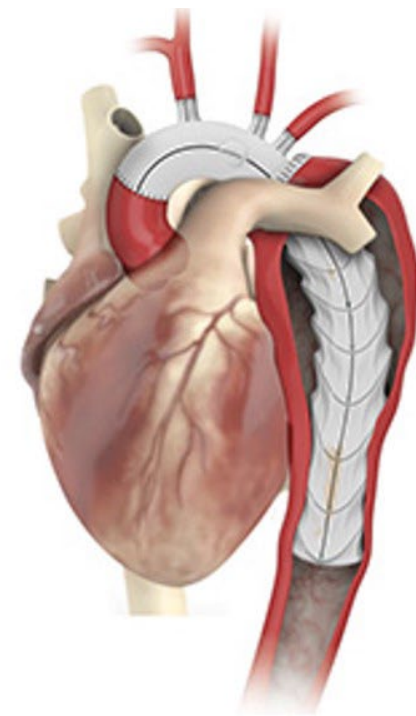
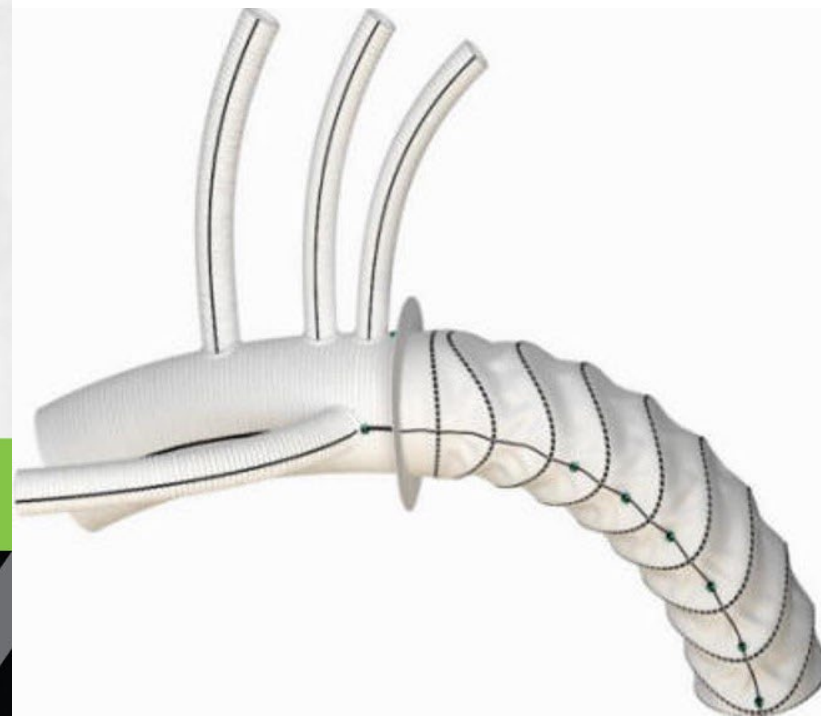
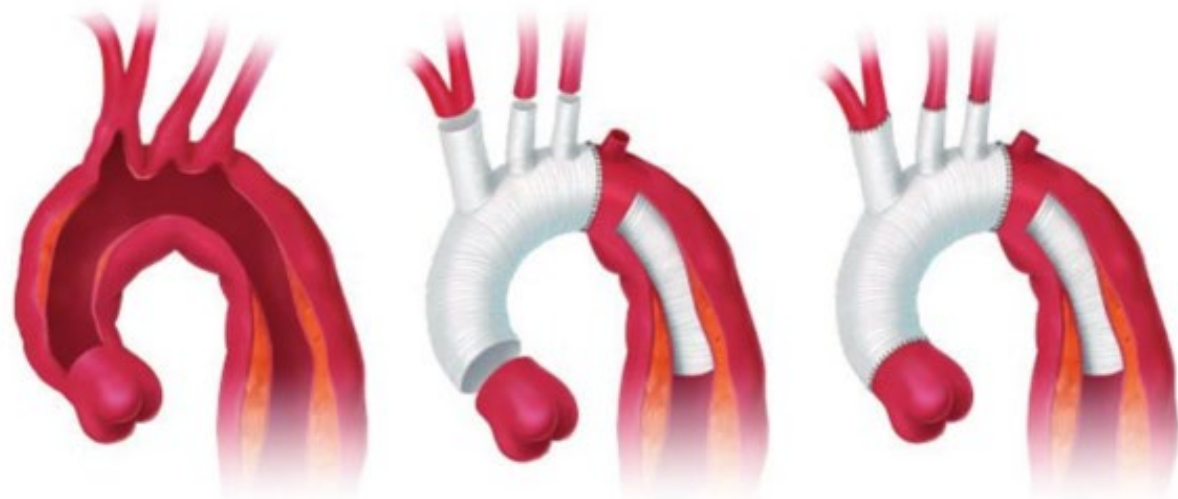
A Supracommissural replacement

B Hemiarch replacement



C Total arch replacement

D Trifurcated graft



Acute TBAD

- Intimal tear is distal to the LSC artery.
- Constitute 1/3 of all dissection patients.
- Patients present as uncomplicated or complicated cases.



Uncomplicated TBAD

- The more common presentation of TBAD (2/3 of cases)
- Treatment is always medical therapy in ICU setting (OMT)

Aggressive blood pressure control. Goal of Systolic < 120 mmHg

Impulse control

Combination of Esmolol & Cardene

Pain control with opiates

Repeat CTA if intractable pain

CTA before discharge

Carotid & Renal Duplex Studies



Complicated TBAD

- Acute TBAD with:
 - Rupture or impending rupture.
 - Clinical end organ malperfusion
 - Mesenteric malperfusion
 - Renal malperfusion
 - LE malperfusion
- Extremely high mortality rate
- Treatment is emergency surgical intervention



Open Surgical Repair

- Open Fenestration
- Aortic Replacement
- Fem-fem bypass



(Panneton JM, et al, JVS, 2000)



Two Important Changes

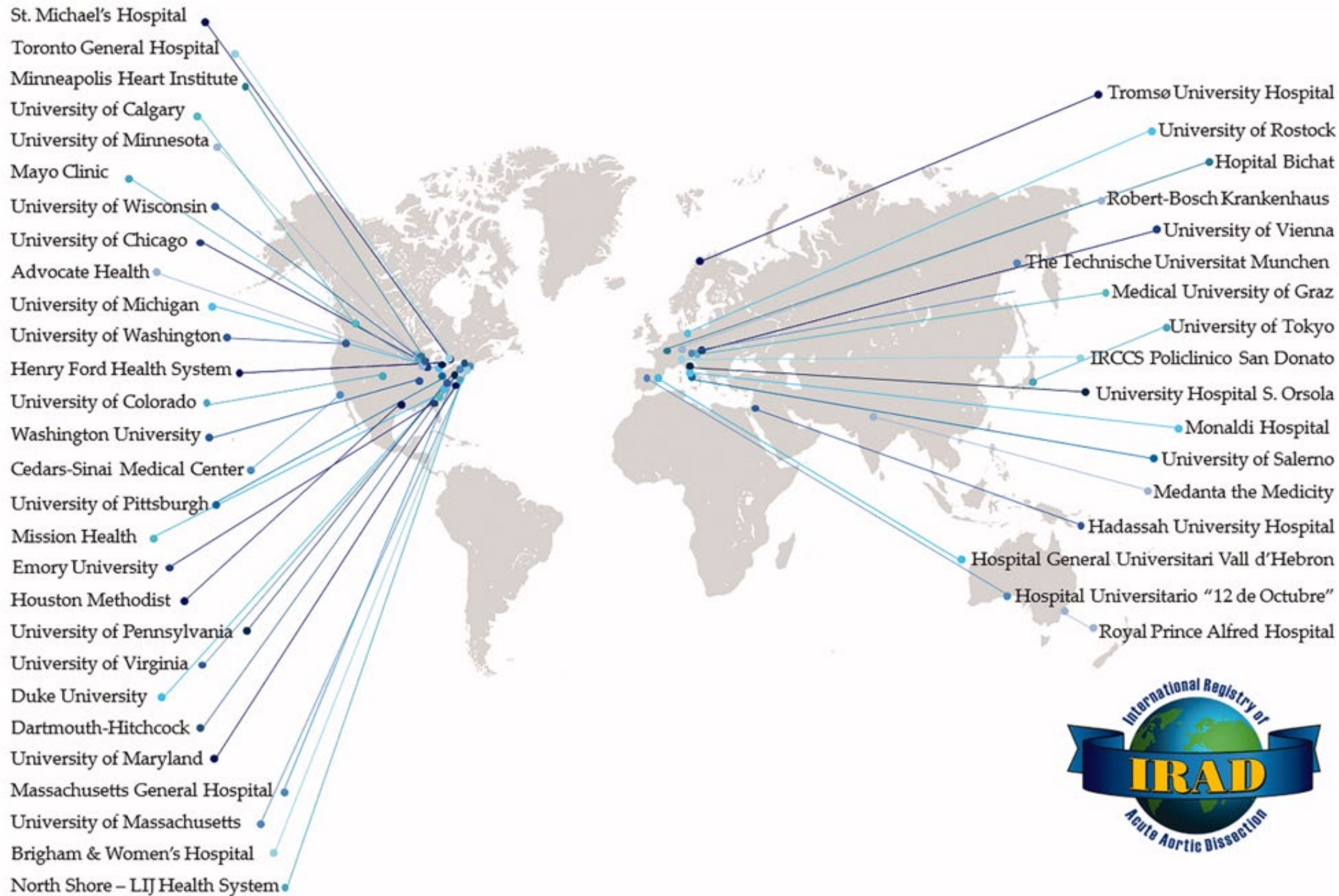
- The development of IRAD (International Registry of Acute Aortic Dissection) and evaluation of the TBAD outcomes
- The introduction of TEVAR



IRAD

- Founded in 1996
- Collaborative efforts of 12 aortic centers in 6 countries.
- IRAD contains patients with both acute type A and B dissections, and patients are included if they have a symptom onset of less than two weeks, i.e. present to the centers in the acute phase of the dissection.
- Today IRAD receives data from thousands of cases from 43 centers worldwide



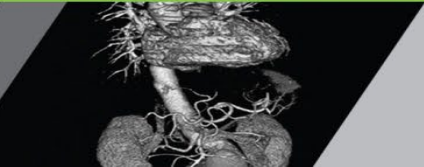
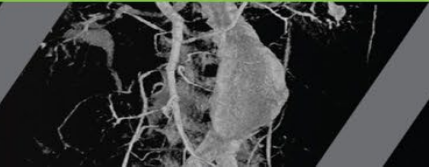
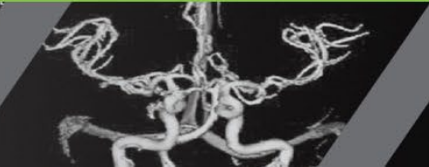


On Follow Up (IRAD)

- Aortic remodeling (healing), on OMT is unlikely
- Less than half of the patients are adequately treated medically to achieve the desired clinical end-points
 - HR < 60
 - BP < 120
- Over half of the patients on long term follow up will develop late aortic aneurysmal degeneration

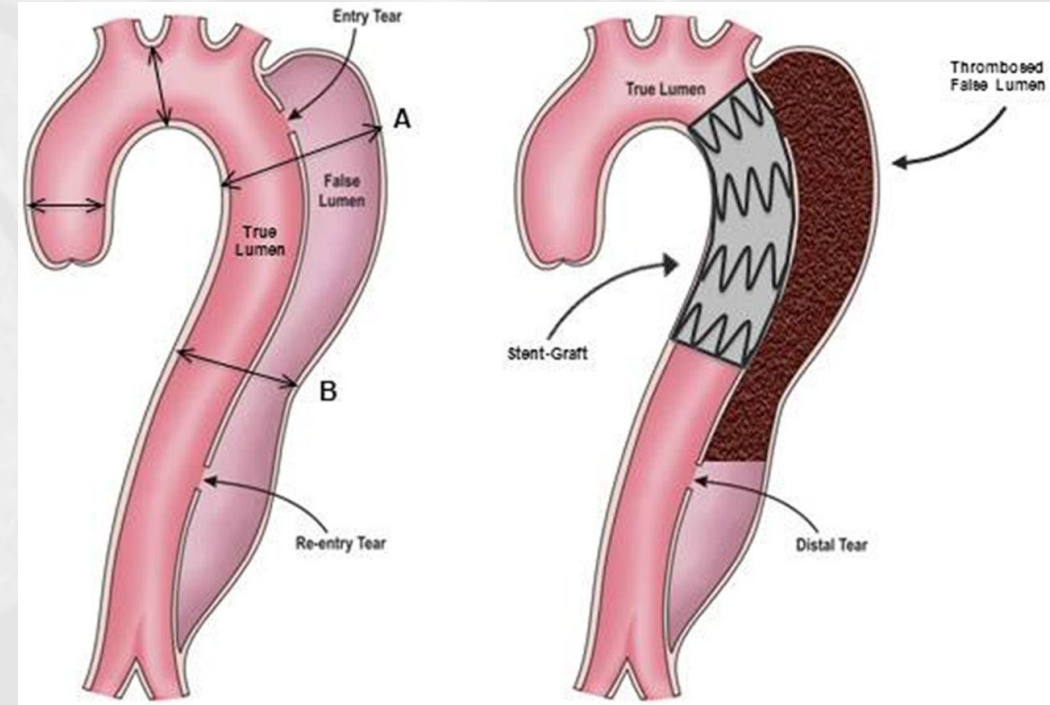


Paradigm Shift in Therapy



Routine TEVAR for uTBAD

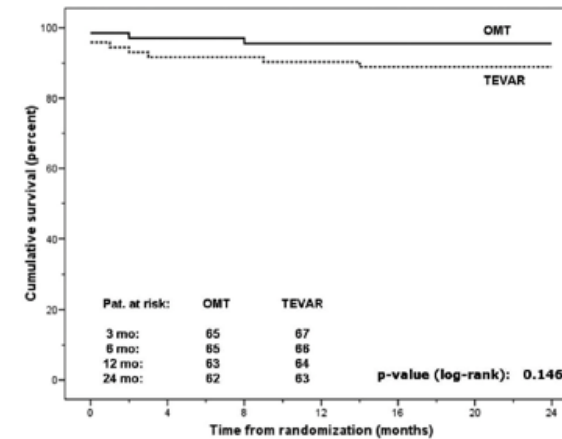
- INSTEAD trial: (Investigation of Stent Grafts in Aortic Dissection)
 - Randomized trial for TEVAR and OMT vs OMT alone for uTBAD
 - 2 year follow up
- INSTEAD-XL trial:
 - 5 year follow up for the INSTEAD patients
- VQI data
 - Results of TEVAR in uTBAD



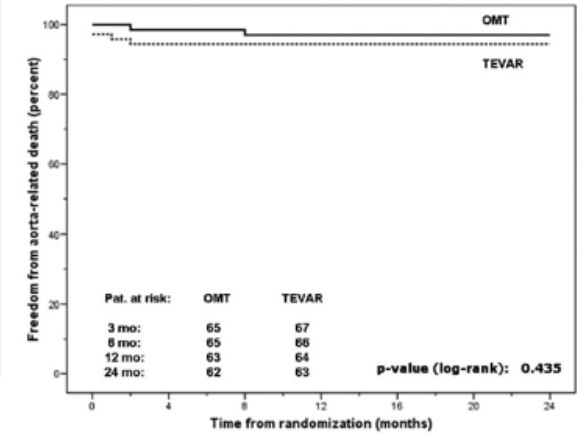
INSTEAD Trial

- No survival benefit for routine TEVAR vs OMT for uTBAD over 2 years (89 vs 96%)
- Improved aortic remodeling in the TEVAR group vs OMT (91 vs 19%)

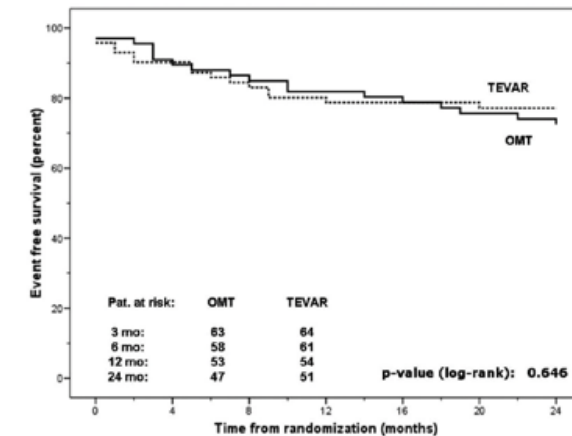
A Cumulative survival within 24 months after randomization



B Freedom from aorta-related mortality within 24 months after randomization



C INSTEAD: Freedom from progressive aortic disease



INSTEAD-XL Trial

- Continued aortic remodeling in the TEVAR group
- 5 year all cause mortality was not statistically significant (11 vs 19%)
- Aorta specific mortality was significant (6 vs 19%)

Table 5. Aortic Morphology at 5 Years

	OMT	OMT+TEVAR	P Value
FL thrombosis	11/50 (22.0%)	48/53 (90.6%)	<0.0001
Partial FL/no FL thrombosis	39/50 (78.0%)	5/53 (9.4%)	<0.0001
Remodeling of thoracic aorta*	5/50 (10.0%)	42/53 (79.2%)	<0.0001
Critical expansion of thoracic aorta†	33/50 (66.0%)	11/53 (20.8%)	<0.0001

Outcome	1-14 days (n = 446 [64.8%])	15-30 days (n = 242 [35.2%])	P value
30-day mortality	26 (7.5)	5 (2.7)	.021
1-year mortality	39 (13.3)	14 (8.2)	.129
Any complication	103 (23.1)	41 (16.9)	.063
Any hematoma	6 (1.6)	1 (0.5)	.431
Any site occlusion	1 (0.2)	1 (0.4)	1
MI (troponin or ECG)	8 (1.8)	3 (1.2)	.755
Dysrhythmia	32 (7.2)	15 (6.2)	.752
Congestive heart failure	7 (1.6)	2 (0.8)	.505
Cerebrovascular	16 (3.6)	5 (2.1)	.355
Respiratory	48 (10.8)	12 (5.0)	.010
New dialysis	8 (1.8)	2 (0.9)	.507
Spinal cord ischemia	11 (2.5)	10 (4.1)	.249
Upper extremity ischemia	5 (1.1)	1 (0.4)	.671
Lower extremity ischemia	3 (0.7)	2 (0.8)	1
Compartment syndrome	1 (0.2)	1 (0.4)	1
Intestinal ischemia	6 (1.3)	1 (0.4)	.431
Renal ischemia	9 (2.0)	2 (0.8)	.344
Surgical site infection	1 (0.2)	0 (0)	1
In-hospital reintervention	34 (7.7)	12 (5.0)	.203
Reintervention within 30 days	28 (11.4)	10 (6.8)	.160
Reintervention within 1 year	45 (26.5)	19 (17.3)	.081
Dissection extension at 30 days	3 (2.0)	1 (1.0)	.426
Dissection extension at 1 year	6 (6.6)	6 (9.0)	.763

Outcome	1-14 days (n = 446 [64.8%])	15-30 days (n = 242 [35.2%])	P value
30-day mortality	26 (7.5)	5 (2.7)	.021
1-year mortality	39 (13.3)	14 (8.2)	.129
Any complication	103 (23.1)	41 (16.9)	.063
Any hematoma	6 (1.6)	1 (0.5)	.431
Any site occlusion	1 (0.2)	1 (0.4)	1
MI (troponin or ECG)	8 (1.8)	3 (1.2)	.755
Dysrhythmia	32 (7.2)	15 (6.2)	.752
Congestive heart failure	7 (1.6)	2 (0.8)	.505
Cerebrovascular	16 (3.6)	5 (2.1)	.355
Respiratory	48 (10.8)	12 (5.0)	.010
New dialysis	8 (1.8)	2 (0.9)	.507
Spinal cord ischemia	11 (2.5)	10 (4.1)	.249
Upper extremity ischemia	5 (1.1)	1 (0.4)	.671
Lower extremity ischemia	3 (0.7)	2 (0.8)	1
Compartment syndrome	1 (0.2)	1 (0.4)	1
Intestinal ischemia	6 (1.3)	1 (0.4)	.431
Renal ischemia	9 (2.0)	2 (0.8)	.344
Surgical site infection	1 (0.2)	0 (0)	1
In-hospital reintervention	34 (7.7)	12 (5.0)	.203
Reintervention within 30 days	28 (11.4)	10 (6.8)	.160
Reintervention within 1 year	45 (26.5)	19 (17.3)	.081
Dissection extension at 30 days	3 (2.0)	1 (1.0)	.426
Dissection extension at 1 year	6 (6.6)	6 (9.0)	.763

Routine TEVAR for uTBAD

- No survival benefit for up to 5 years of follow up
- There is significant morbidity and mortality associated with routine TEVAR for a disease that is deemed uncomplicated (up to 15% stroke, SCI and death)



- Multiple studies looked at high risk patient factors and anatomic factors associated with progression of TBAD.
- This is an ongoing research and right now is a moving target.



High Risk Criteria TBAD

- Refractory pain
- Refractory hypertension
- Initial maximal aortic diameter > 40 mm
- Intimal tear > 1 cm
- Intimal tear on the lesser curve
- Radiographic only malperfusion
- False lumen diameter > 22 mm
- Readmission
- Bloody pleural effusion



New Classification (SVS/STS Classification of Aortic Dissection)

- Attempt to create a unified consensus on reporting, nomenclature, and classification of TBAD
- The purpose is to provide structure to the reporting of TBAD, with particular attention to those attributes of TBAD that, based on the best available evidence to date, would appear to have an impact on outcomes

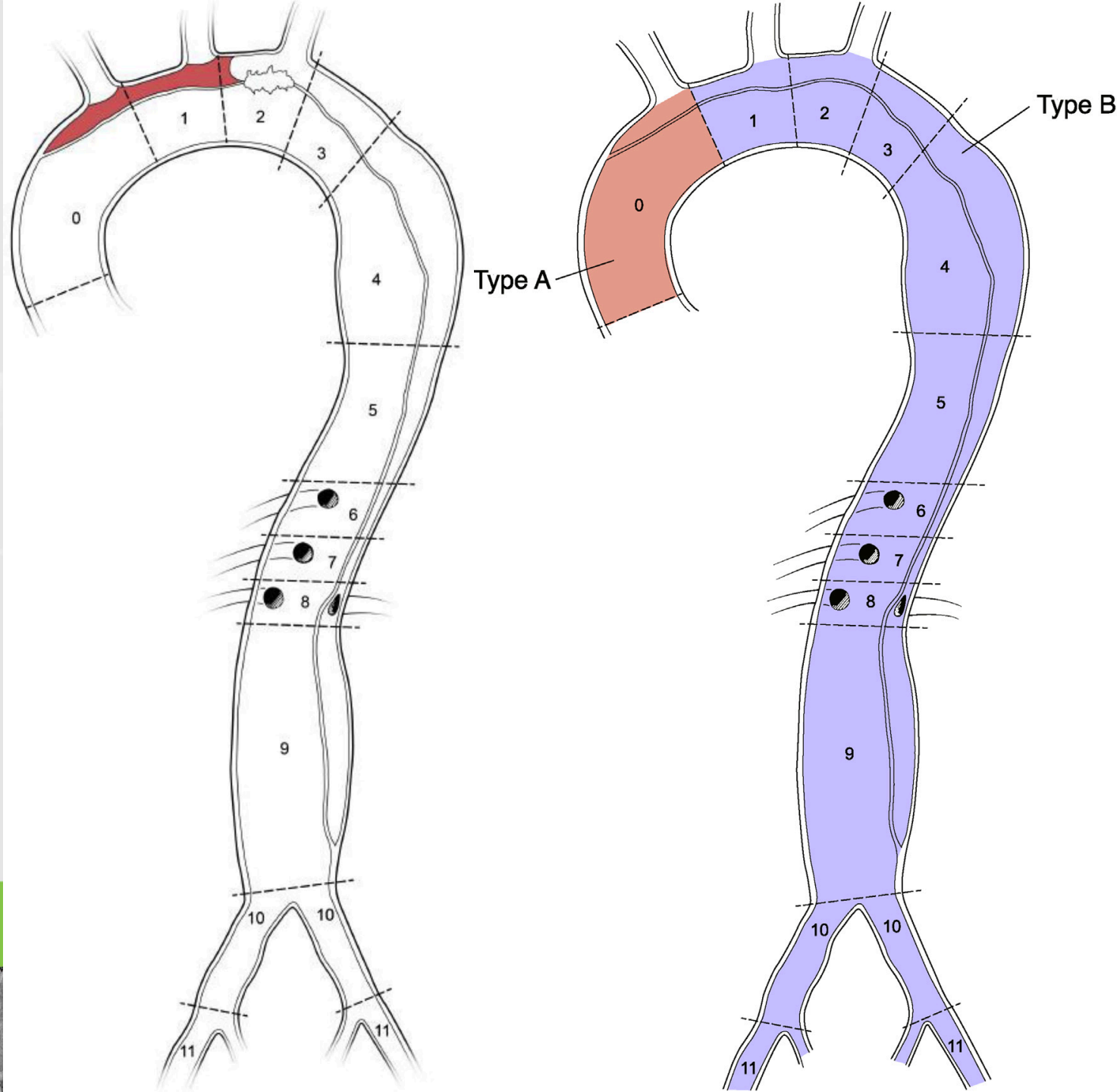
(Lombardi, et al, JVS, 2020)



SVS/STS Classification

- Type (A or B)
- Proximal extent
- Distal extent
- Example: B0,9

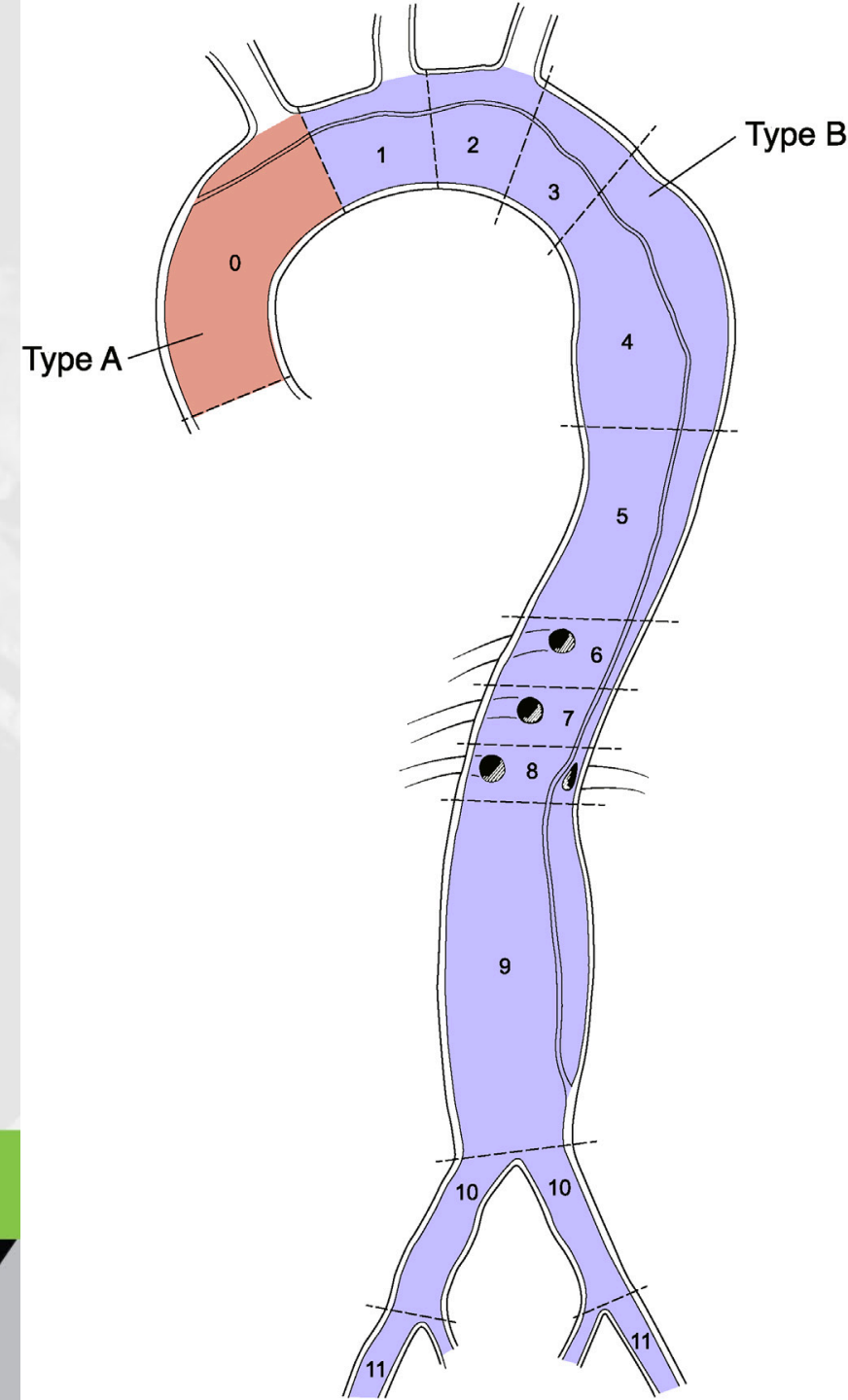
(Lombardi, et al, JVS, 2020)



SVS/STS Classification

- Anatomic Classification: Type A or B
- Acuity Classification:
 - Uncomplicated
 - Complicated
 - High risk features
- Timing:
 - Hyperacute: first 24 hours
 - Acute: 2-14 days
 - Subacute: 2 w: 90 days
 - Chronic: > 90 days

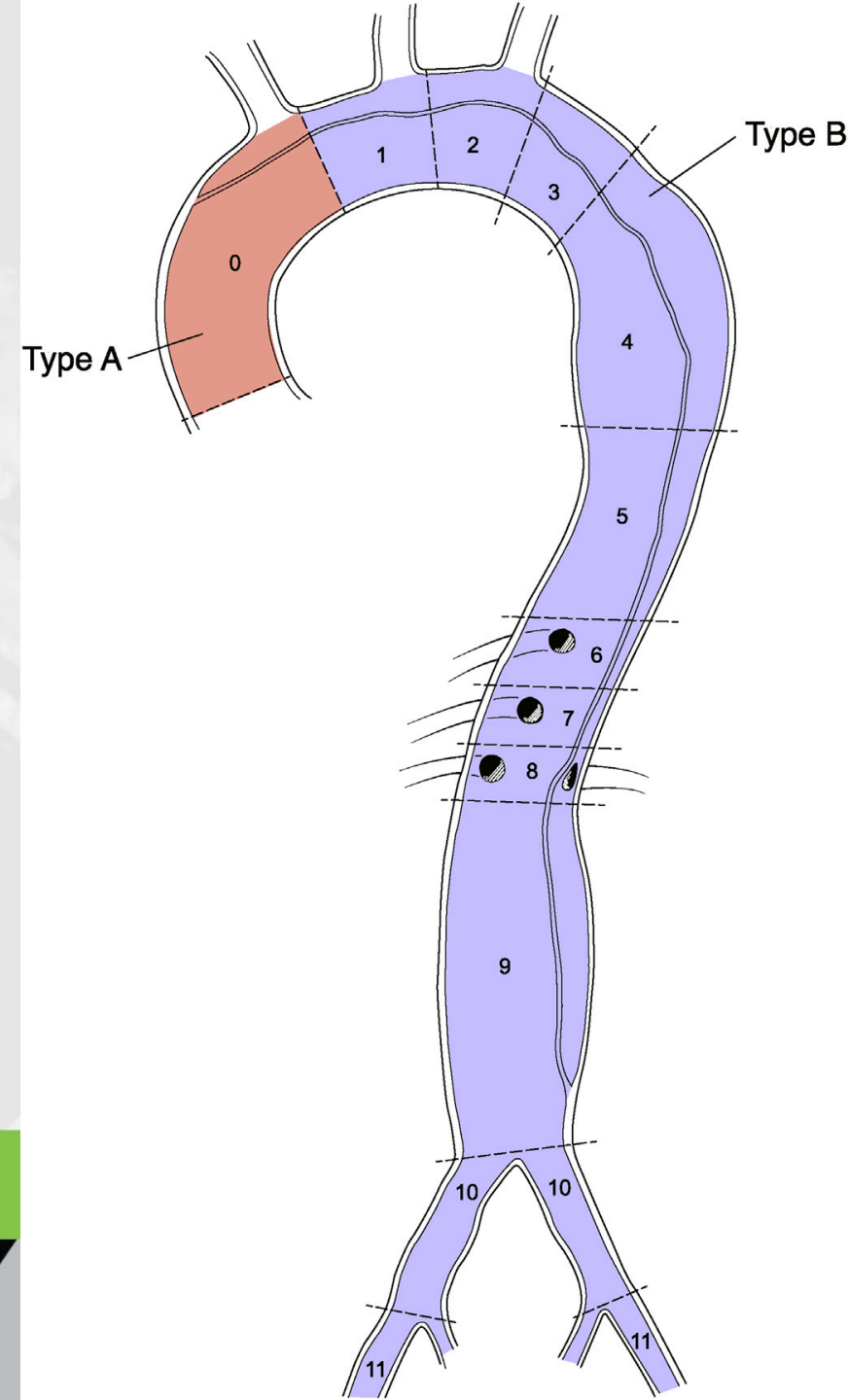
(Lombardi, et al, JVS, 2020)



SVS/STS Classification

- Anatomic Classification: Type A or B
- Acuity Classification:
 - Uncomplicated
 - Complicated
 - High risk features
- Timing:
 - Hyperacute: first 24 hours
 - Acute: 2-14 days
 - Subacute: 2 w: 90 days
 - Chronic: > 90 days

(Lombardi, et al, JVS, 2020)



Current Status



Optimum Medical Therapy (OMT)

- Indicated in all cases of aortic dissection
- Can be the only modality (Uncomplicated TBAD)
- Is a bridge towards surgical therapy (Uncomplicated with high risk features)
- Post-operatively in cases that needed surgical therapy



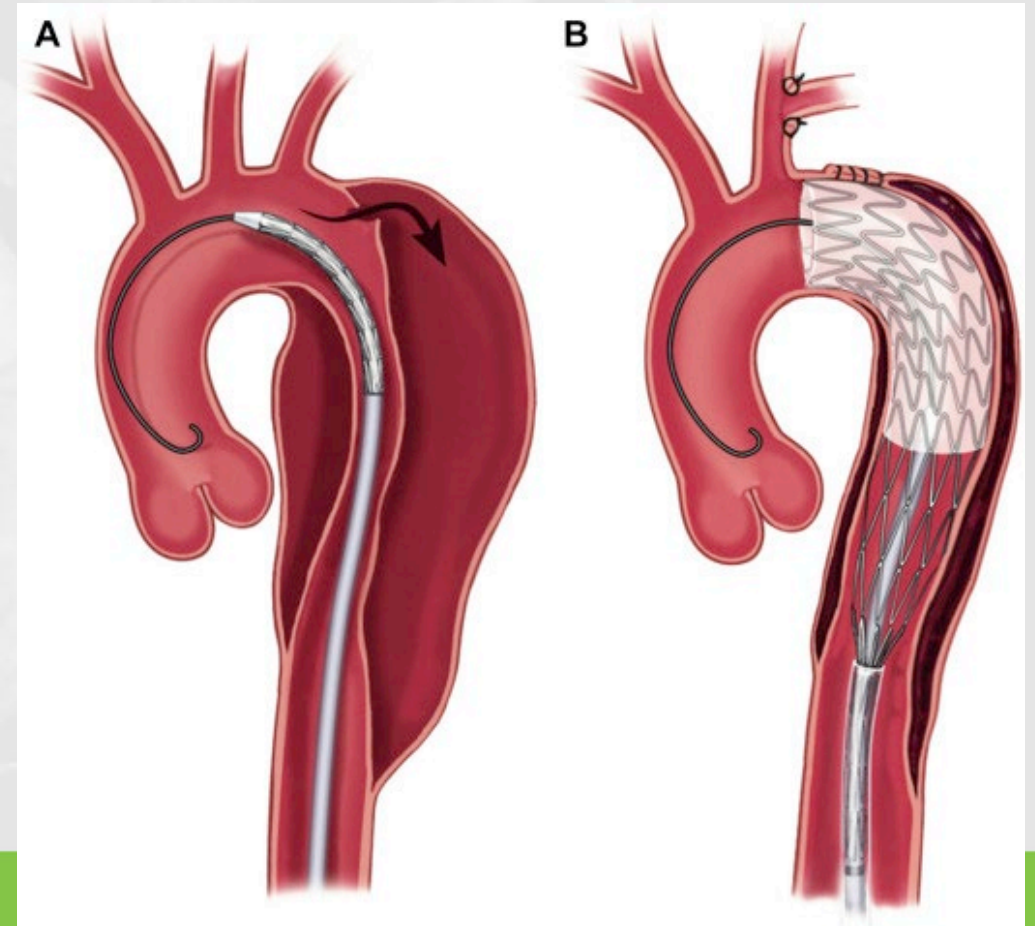
Surgical Management (Mostly Endovascular)

- Emergently implemented in cases of complicated TBAD.
- After cooling down for high risk cases, preferably during the subacute phase.
- For chronic TBAD with aneurysmal degeneration



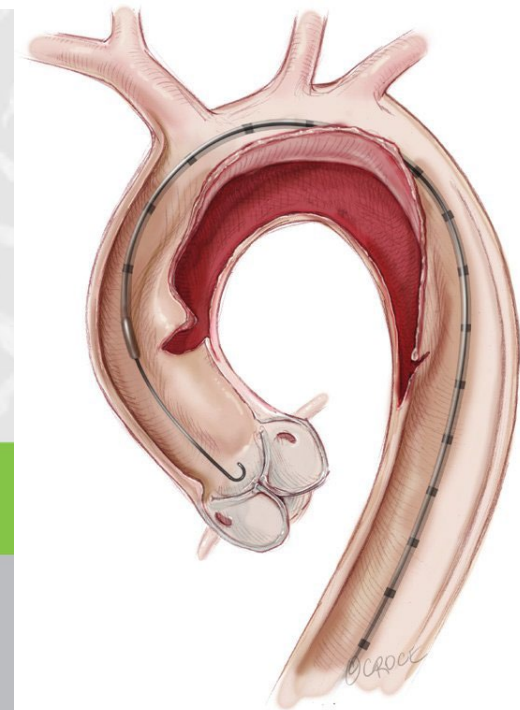
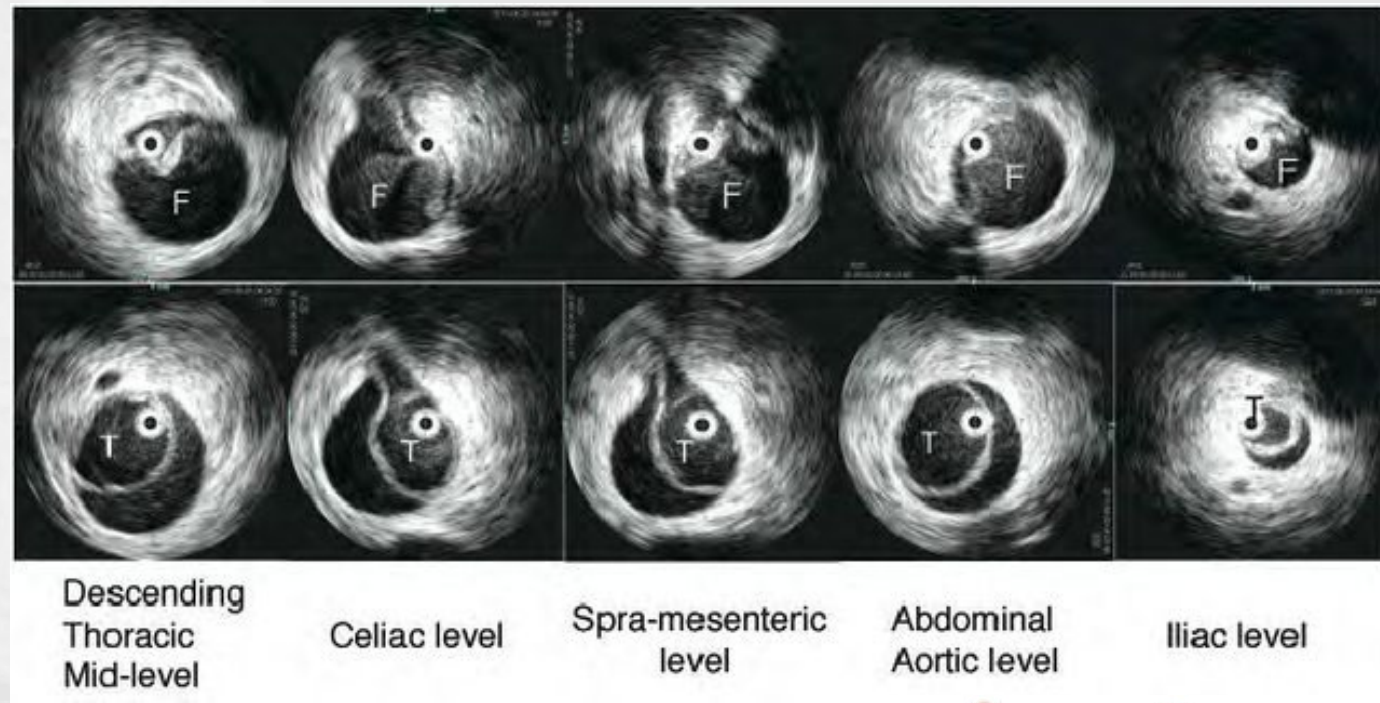
TEVAR

- Covering the entry tear to exclude the FL and induce thrombosis
- Always land in a healthy proximal aorta
- Distally to cover all fenestrations, usually down to celiac artery
- No oversizing and no ballooning
- Always use IVUS
- Highest complication rate in the hyperacute phase of TBAD

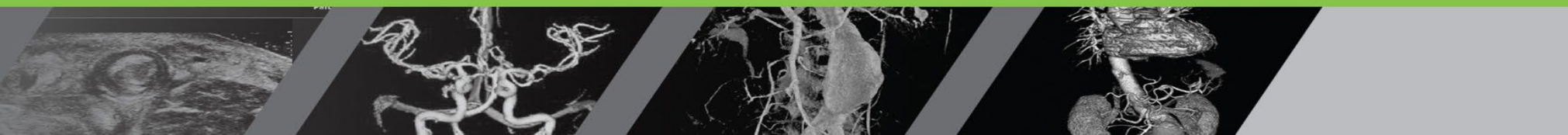


IVUS

- There should be no TEVAR without IVUS for TBAD
- Important to make sure we remain in the true lumen. False lumen TEVAR is usually a disaster.
- Evaluate accurately diameter measurement.
- Assist in evaluating branch vessels and type of malperfusion
- Assists with branch vessel stenting.



(Kurimoto et al, 2011)



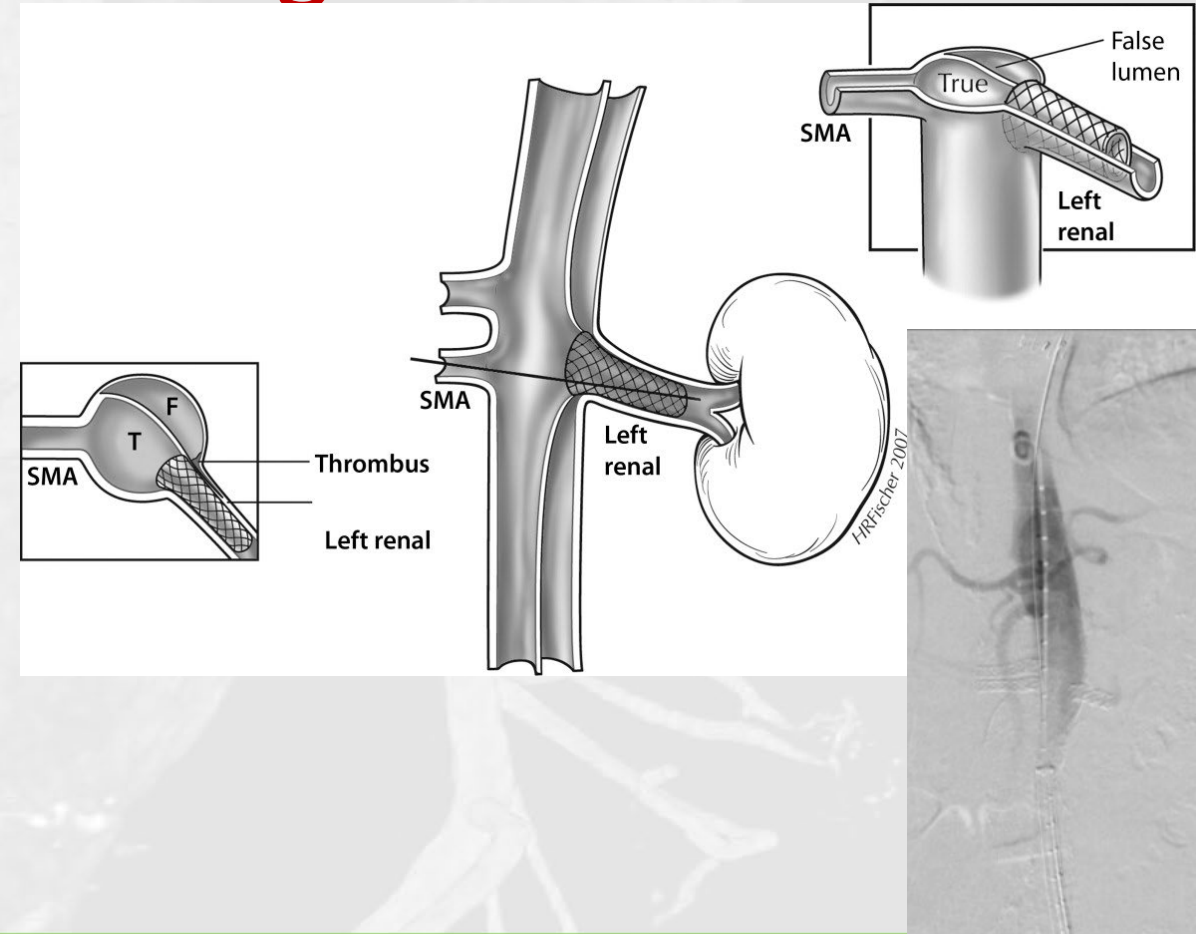
TEVAR Adjuncts

- TEVAR is usually all that is needed for uncomplicated acute TBAD and in 80-90% of complicated acute TBAD.
- Other adjuncts may be needed in complicated TBAD
 - Branch vessel management: branch vessel stenting with and without fenestration or septotomy
 - False lumen management, especially in acute complicated cases with rupture (coil embolization, candy plug, etc)
 - True Lumen management: Zenith dissection endovascular system (Petticoat and Stabilize Techniques)

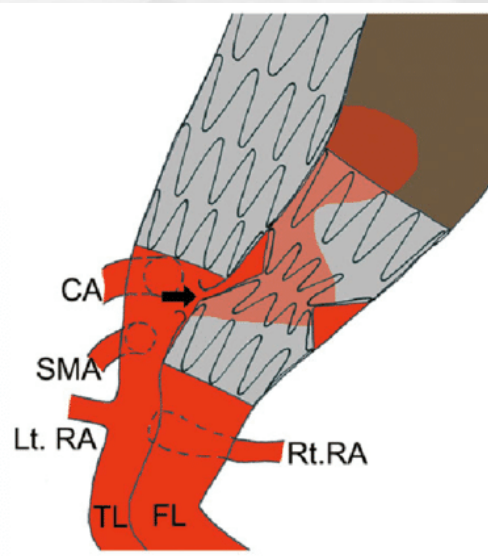
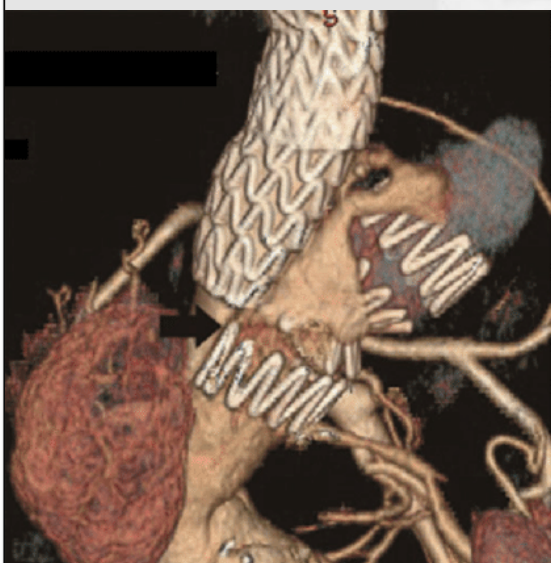
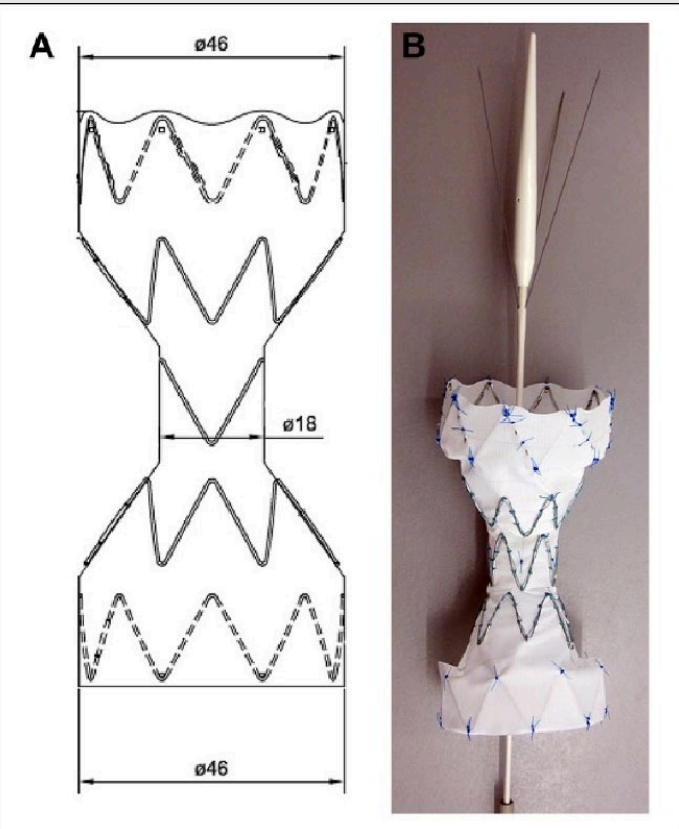


I. Branch Vessel Management

- Endovascular fenestration
- Branch vessel stenting
- Combination of the two



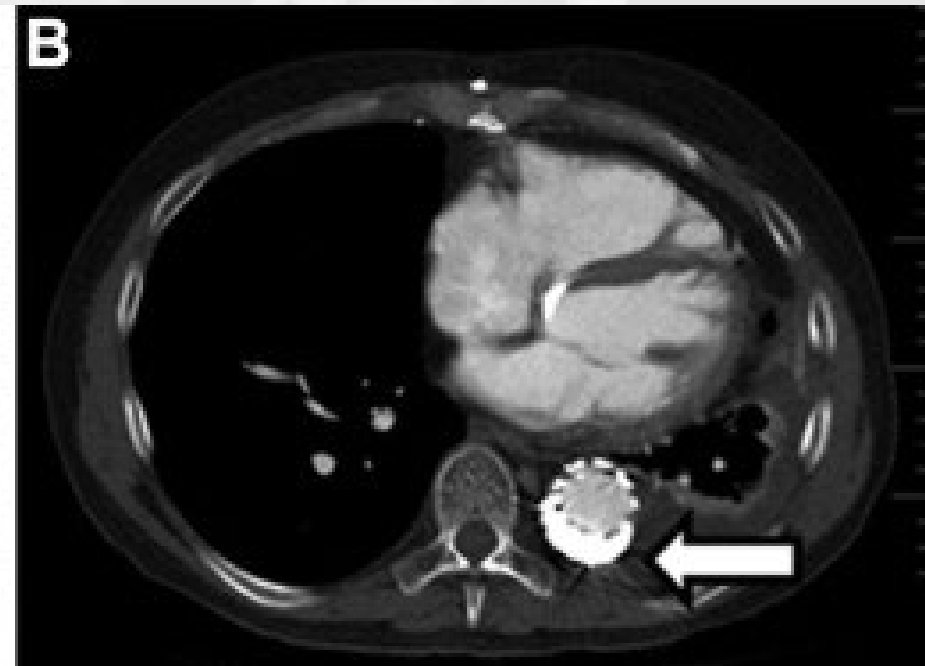
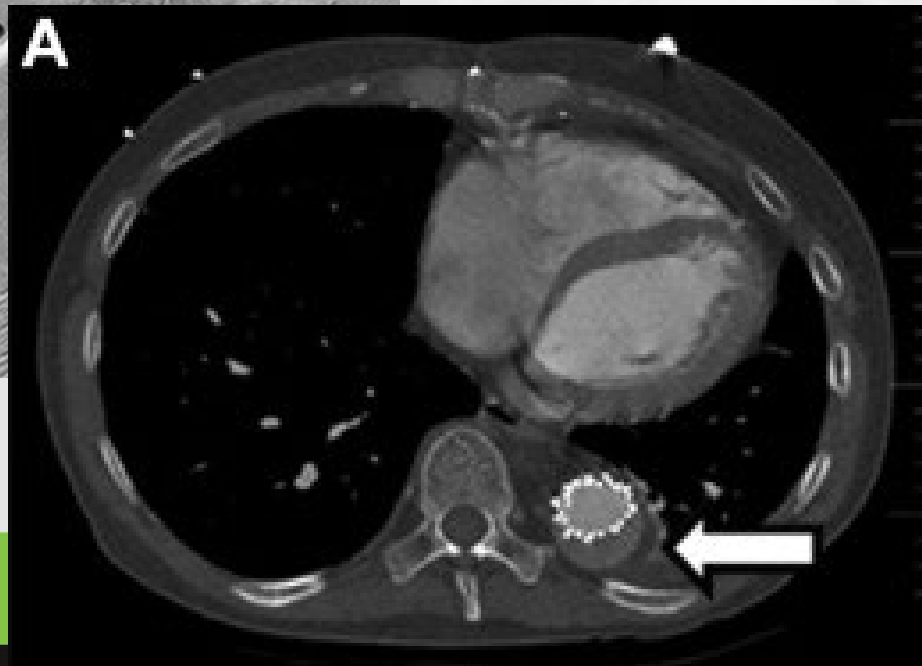
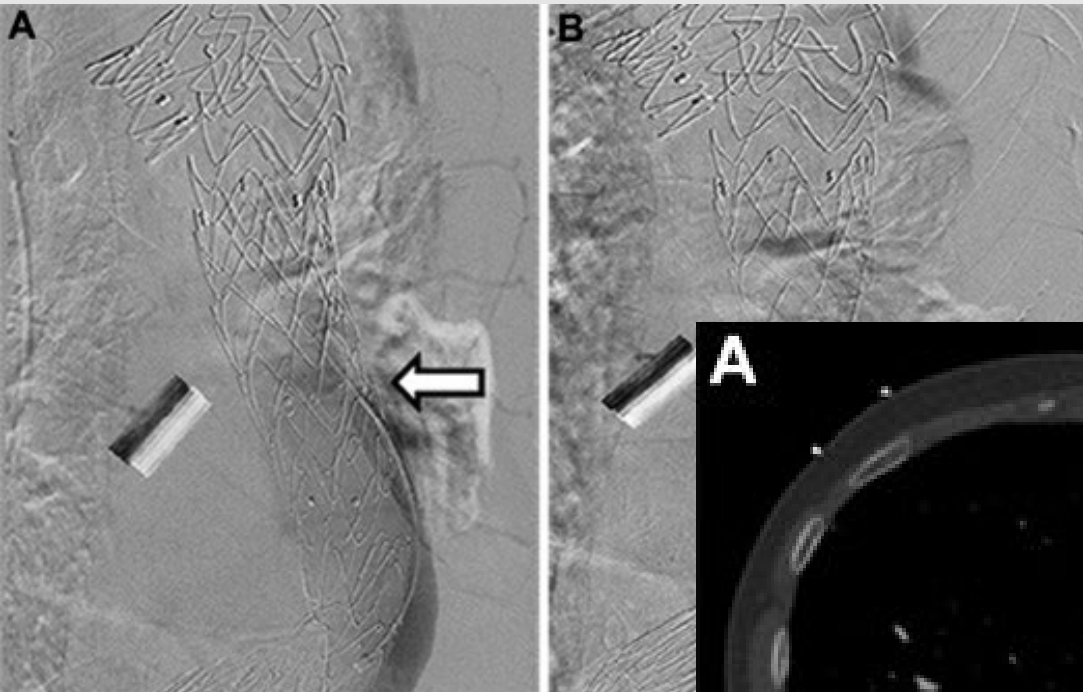
II. False Lumen Management (Candy Plug Technique)



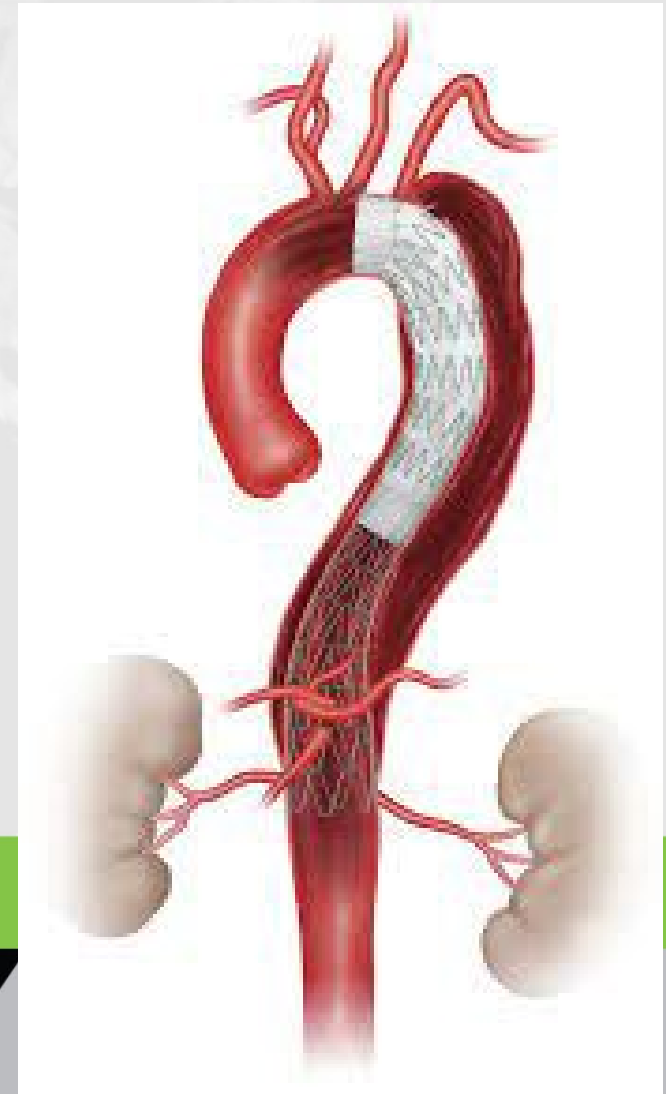
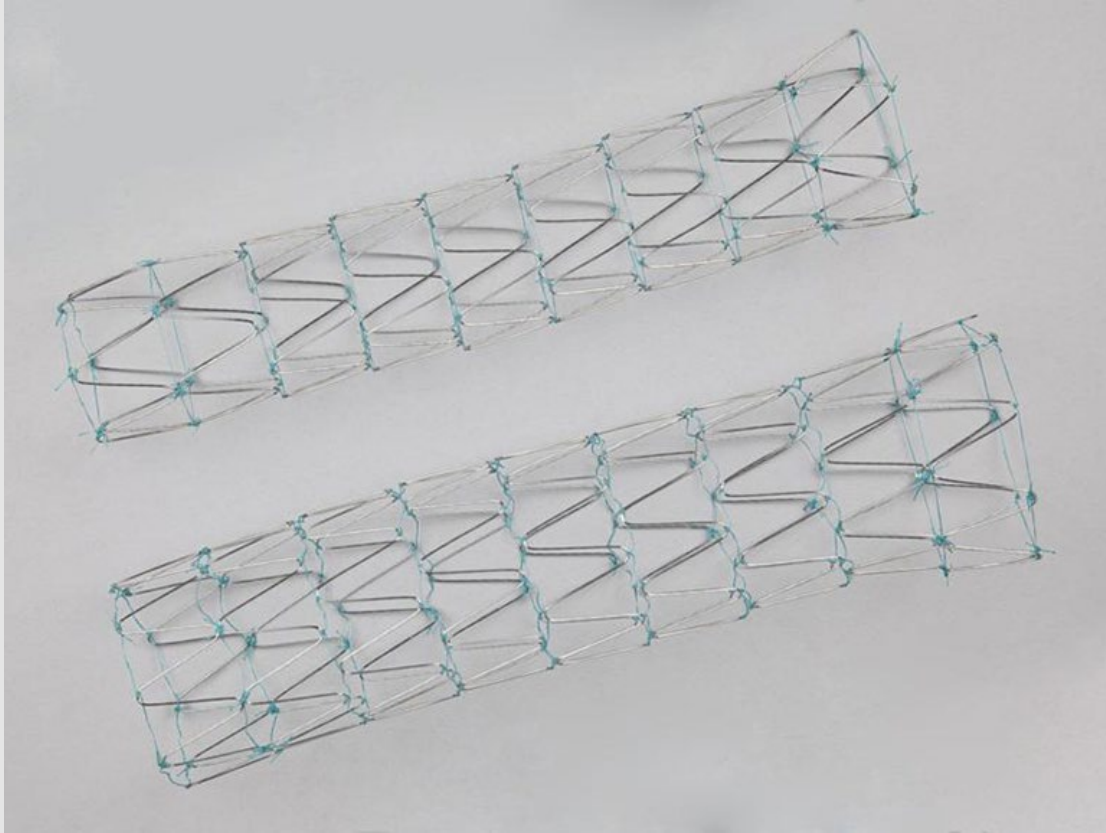
II. False Lumen Management (Knickerbocker Technique)



II. False Lumen Management (Coil Embolization)

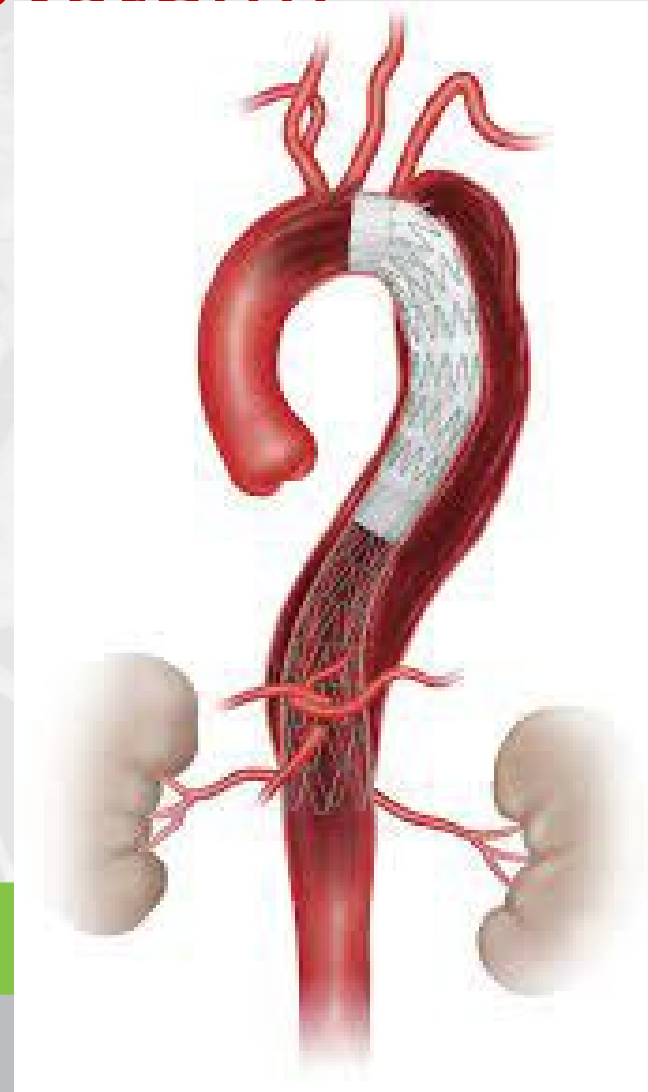


III. True Lumen Management (Zenith Dissection Endovascular System)



III. True Lumen Management (Zenith Dissection Endovascular System)

- PETTICOAT Technique (provisional extension to induce complete attachment). To enhance TL perfusion, bare stents may be distally deployed in the TL to the covered stent graft to increase TL size, thus treating dynamic malperfusion and stabilizing the intimal lamella
- STABILIZE Technique (stent-assisted balloon-induced intimal disruption and relamination in aortic dissection repair), involves ballooning the TL inside the stent graft and the distally deployed bare stents to intentionally rupture the lamella and allow full expansion of the stent in a single-channeled aorta



Current Unresolved Questions

- Better defining the role of TEVAR in uTBAD
- Optimum time of intervention
- Optimum extension of TEVAR for acute TBAD
- Better defining the role of PETTICOAT & STABILIZE techniques
- Better defining patients with high risk features that will need to have routine TEVAR to avoid future aortic related adverse events and improve mortality.
- What is the acceptable bench-mark for morbidity and mortality of TEVAR for uncomplicated TBAD



IMH vs Acute Aortic Dissection

- A hematoma confined to the media in the aortic wall without identifiable intimal tear. May represent an early stage of aortic dissection, or aortic dissection with thrombosed FL
- Patients with IMH are older (mean 68 vs 61 yrs)
- More distal aortic involvement (60% type B vs 35%)
- Less malperfusion syndrome
- As lethal with same hospital mortality
- 25% mortality at 1 yr for hospital survivors
- Has worse prognosis if associated with PAU

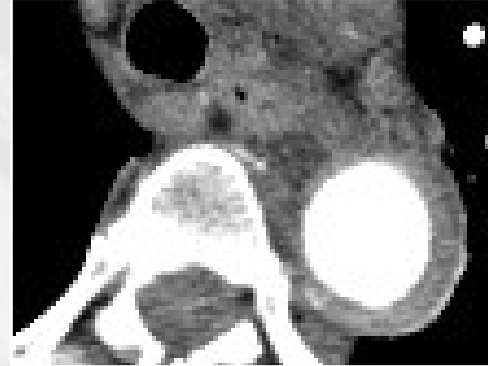


IMH

Initial Management Protocol

- Admission to Vascular Intensive Care Unit
 - Aggressive blood pressure control
 - Goal of Systolic < 120 mmHg
 - Combination of Esmolol & Cardene
 - Pain control with opiates
 - Repeat CTA if intractable pain
 - CTA before discharge
 - Carotid & Renal Duplex Studies

Thin IMH
Limited
involvement



Thick IMH with
circumferential
involvement



IMH

EVMS Experience: Outcomes of Medical Therapy

1. Determine outcomes of patients treated with medical therapy (AREM)
2. Identify risk factors for medical therapy failure

OUR TREATMENT ALGORITHM PROTOCOL

EMERGENT TEVAR	DELAYED TEVAR
Aortic rupture (includes contained)	Intractable pain > 24-48 hours with good blood pressure control
Impending rupture	Uncontrollable hypertension
Severe pain, large aortic diameter (> 55 mm)	Changes on repeat imaging: IMH Thickness or new/worse CFEs
Malperfusion	Aortic enlargement

From the Southern Association for Vascular Surgery

Medical therapy in type B aortic intramural hematoma is associated with a high failure rate 

Tomaz Mesar, MD, Maggie J. Lin, MD, Ishraq Kabir, MD, David J. Dexter, MD, Animesh Rathore, MBBS, and Jean M. Panneton, MD, Norfolk, Va

ABSTRACT
Objective: Intramural hematoma (IMH) is on the spectrum of acute aortic syndrome, but optimal management is poorly understood. The aim of this study was to evaluate outcomes of patients with type B IMH (TBIMH) after best medical therapy (BMT) and to assess for risk factors associated with failure of BMT.
Methods: This is a single-institution retrospective chart review of all patients with TBIMH between January 2008 and December 2017. Failure of BMT was defined as any of the following end points: aortic rupture, aorta-related death, aortic enlargement to at least 55 mm or growth of >10 mm within 12 months, or need for surgical aortic intervention for failed BMT.
Results: We identified 92 patients, of whom 25 received emergent thoracic endovascular aortic repair; 67 patients were initially managed with BMT, and of these, 32 underwent thoracic endovascular aortic repair within 14 days for early BMT failure. Two additional patients had early BMT failure; one died of aortic rupture due to retrograde type A dissection, and one patient was advised to undergo repair but did not comply and was lost to follow-up. Fourteen patients (20.9%) received endovascular therapy for late failure of BMT after the initial hospitalization. Medical management was successful in 19 patients (28.4%), although 5 patients had aortic enlargement but below the threshold for elective repair (maximal aortic diameter of 55 mm). On univariate analysis, presenting IMH thickness and growth of IMH thickness were risk factors for BMT failure. On multivariate analysis, presenting IMH thickness was the sole predictive risk factor for medical therapy failure (odds ratio, 1.083; 95% confidence interval, 1.021-1.149; $P = .008$), with an odds ratio of 6.810 (95% confidence interval, 1.921-24.146; $P = .002$) with a presenting IMH thickness of ≥ 8.0 mm, which was the calculated IMH thickness cutoff value with highest sensitivity and specificity to predict failure of BMT (area under the receiver operating characteristic curve = 0.795; $P = .001$; $J = 0.62$).
Conclusions: BMT for TBIMH is associated with a high failure rate and need for interventions. IMH thickness on admission is the most reliable factor to predict failure of BMT. (J Vasc Surg 2020;71:1088-96.)
Keywords: Type B aortic intramural hematoma; TEVAR; Medical therapy

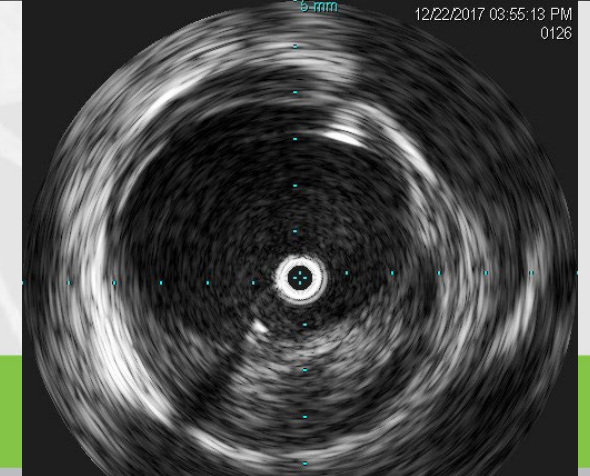
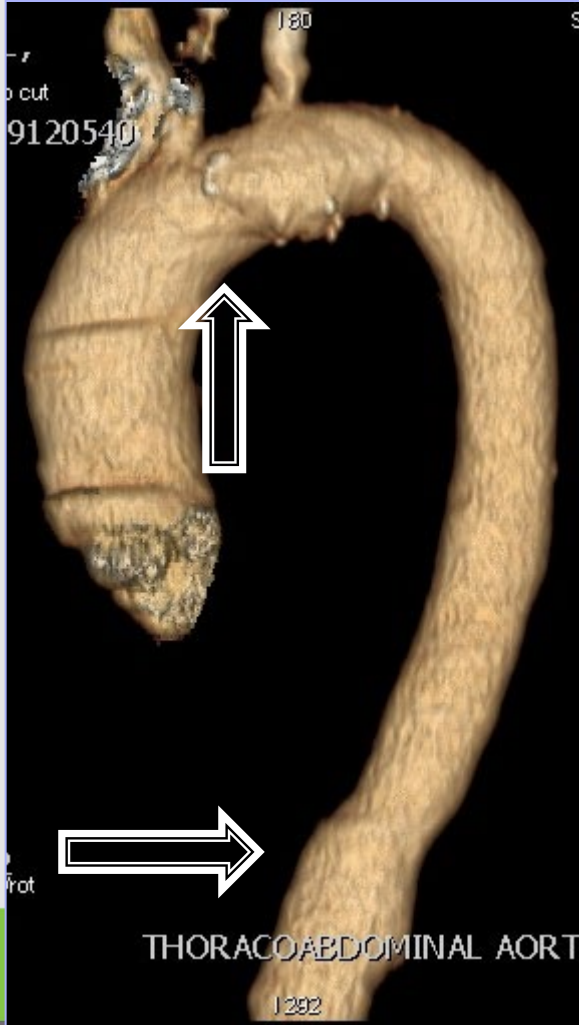
J Vasc Surg 2020;71:1088-96.



IMH

Operative principles

- Use IVUS for sizing and extent
- Keep the proximal edge of the endograft in healthy aorta
- Cover the PAU or presumptive entry tear
- Extend distally to where the IMH is thinner
- No oversizing, no ballooning



PAU

- Penetrating aortic ulcers (PAUs), a subset of aortic disease that are generally believed to be closely linked to aortic dissection and intramural hematoma.
- They are defined as an ulcer-like projection into the medial lining of an artery originating at the site of a soft plaque.



PAU: Outcomes

From the Society for Vascular Surgery

The natural history and outcomes for thoracic and abdominal penetrating aortic ulcers

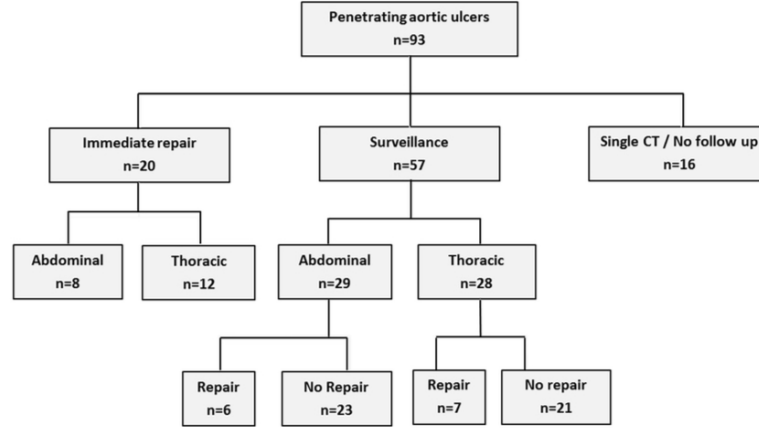
Shaun M. Gifford, MD, Audra A. Duncan, MD, Lawrence E. Greiten, MD, Peter Glovicki, MD, Gustavo S. Oderich, MD, Manju Kalra, MBBS, Mark D. Fleming, MD, and Thomas C. Bower, MD, Rochester, Minn

Objective: The objective of this report was to define the natural history of penetrating aortic ulcers (PAUs) in the descending thoracic and abdominal aorta.

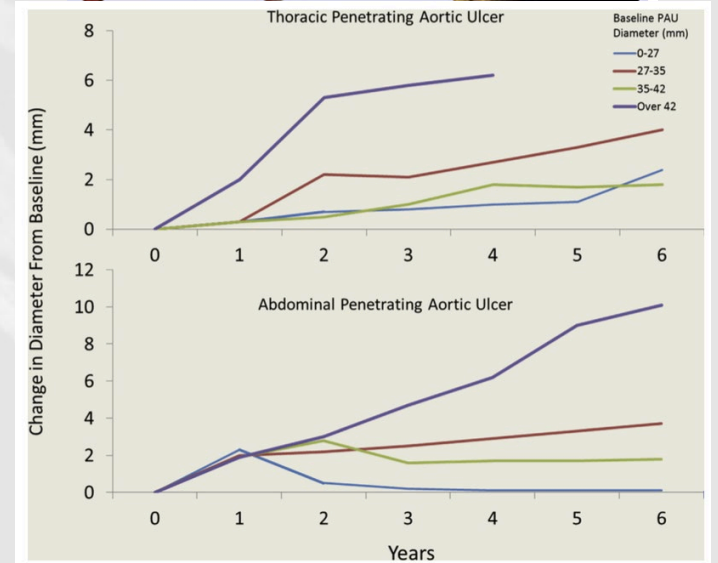
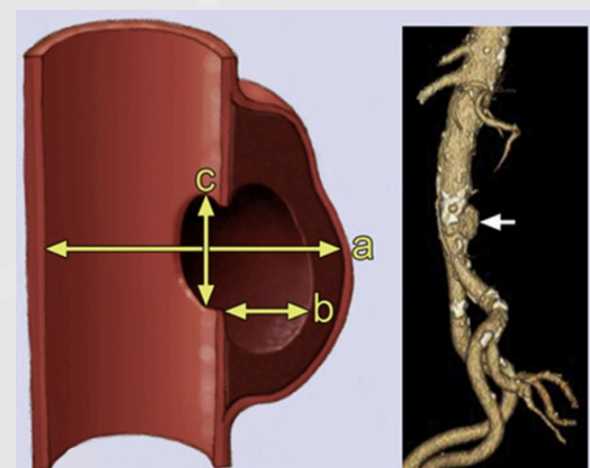
Methods: Data from consecutive patients with PAU from January 1, 1998 to December 31, 2012 were retrospectively reviewed. Computed tomography (CT) scans were analyzed for anatomic changes. End points analyzed were changes in size, development of symptoms or signs of rupture, morbidity, and mortality.

Results: Ninety-three patients were identified; 57 were followed up with two or more CT studies 3 months apart (group 1), and 20 had immediate repair (group 2). Sixteen had one CT scan and no intervention or follow-up and were excluded from analysis. In group 1, mean age was 75 years (29 men, 28 women), with 28 descending thoracic aorta and 29 abdominal aorta PAUs. Fifty patients were asymptomatic, whereas five had pain and two had emboli. Mean follow-up was 38 months (range, 3-108 months). Ulcer growth rate was as follows: length, 2.0 mm/y; depth, 1.2 mm/y; and aortic diameter, 2.2 mm/y. Thirteen (23%) went on to repair at a mean of 37 months after diagnosis because of size (54%; 7/13), rapid growth (31%; 4/13), and high-risk morphology requiring bypass after descending thoracic aorta PAU unrelated causes, and 1 of rupture after refusing repair. All repairs in group 1 were endovascular. The 30-day surgical mortality was 0%. One patient had an access site complication requiring bypass after descending thoracic aorta PAU repair. At a mean follow-up of 32 months, all ulcers were excluded on CT; one (8%) had a type II endoleak. Group 2 included 13 men and seven women with a mean age of 70 years, with 12 descending thoracic and eight abdominal aorta PAUs. Repair indications were rupture (n = 3), symptoms (n = 10), or size (n = 7) and included one open and 19 endovascular repairs with 0% 30-day mortality. Major complications (3/20; 15%) included myocardial infarction, access site disruption, and hematoma; four of 20 patients had type II endoleaks.

Conclusions: PAU growth rate and risk of rupture are low. Endovascular repair of symptomatic, ruptured, and large PAUs is safe and effective with excellent long-term results. For asymptomatic PAUs, serial CT surveillance is associated with a low rate of rupture or complications. (J Vasc Surg 2016;63:1182-8.)



21.5% immediate repair
22.8% delayed repair



“PAU growth rate and risk of rupture are low. Endovascular repair of symptomatic, ruptured, and large PAUs is safe and effective with excellent long-term results. For asymptomatic PAUs, serial CT surveillance is associated with a low rate of rupture or complications”

Summary

- AAS are a group of closely related acute aortic pathologies
- They have distinct morbidity and mortality rates
- Management is currently evolving
- TEVAR has significantly changed and will continue to change the treatment paradigm.





Thank You

