Stroke Risk with Transcatheter Aortic Valve Replacement



Jessica Noble, MSN, AGACNP-BC

Objectives

Describe the pathophysiology and clinical impact of severe aortic stenosis

Explain the fundamentals of Transcatheter Aortic Valve Replacement (TAVR) and its role in the management of aortic stenosis

Identify the incidence, timing and clinical consequences of stoke following TAVR

Discuss the primary mechanisms that contribute to stroke after TAVR including embolization and thromboembolism

Recognize patients and procedure-related risk factors associated with post-TAVR stroke

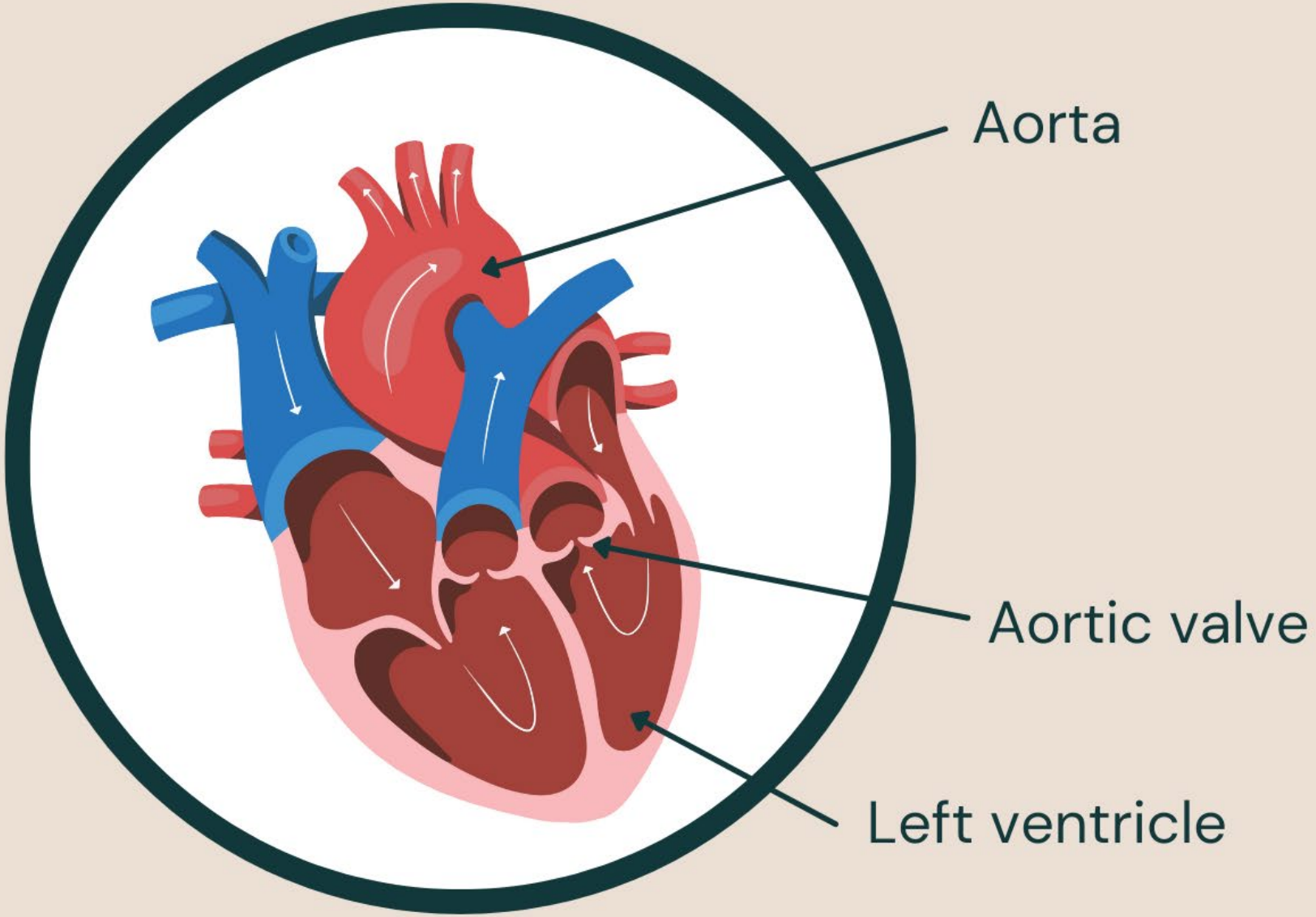
Evaluate current stroke prevention strategies and apply evidence-based approaches to reduce stroke risk and optimize outcomes

Analyze a patient case study to identify stroke risk factors, evaluate clinical decision-making and apply evidence-based stoke prevention following TAVR

The aortic valve

Aortic Valve

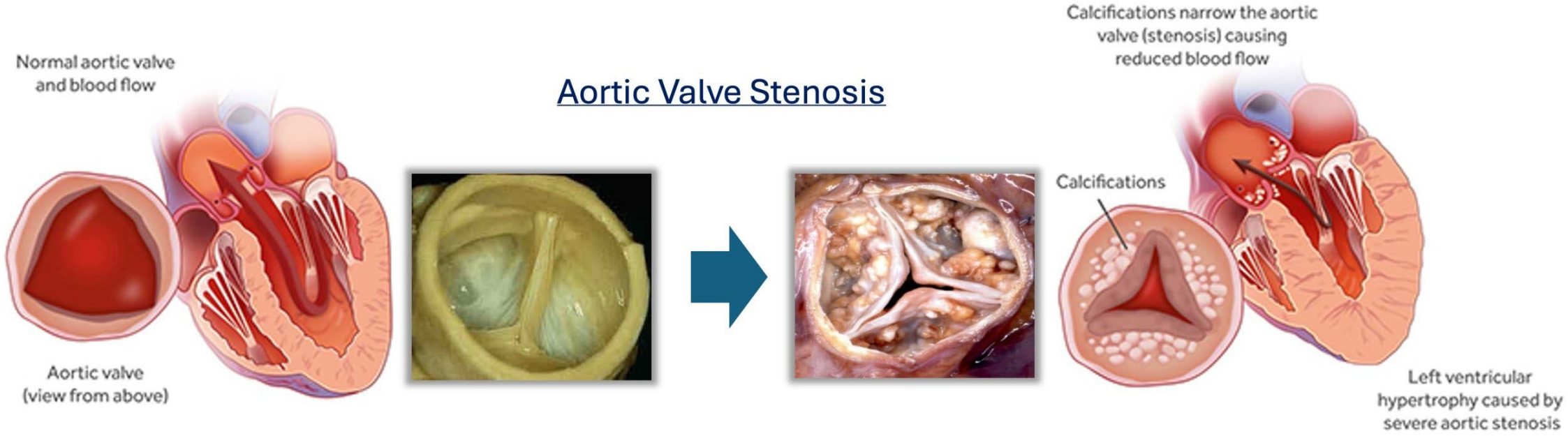
- One way valve between left ventricle and aorta
- Opening during systole to allow the heart to eject oxygenated blood into the aorta and to systemic circulation
- Closing during diastole to prevent blood from leaking back into the ventricle

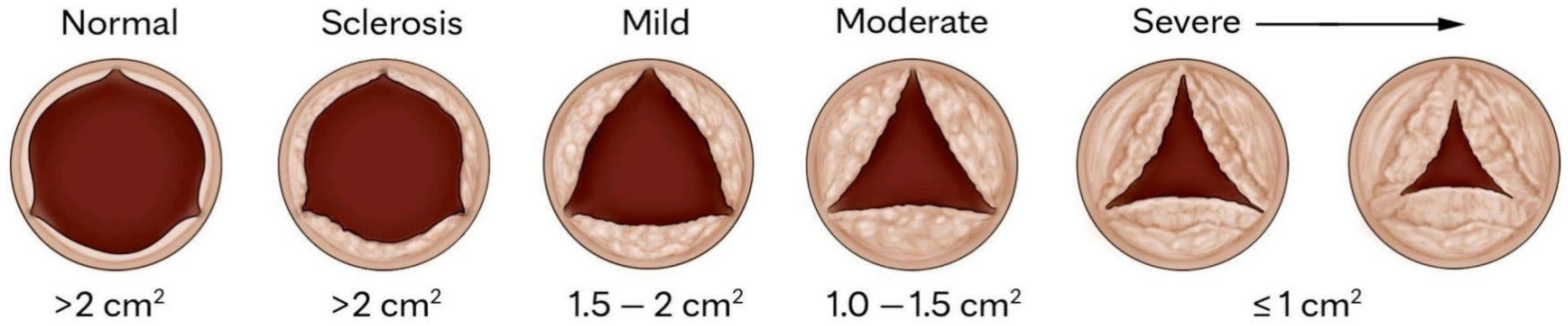


Aortic Stenosis

- Narrowing of the aortic valve
- Leaflets become thickened, fibrosed or calcified
- Leaflets lose pliability and fail to open fully
- Causes of aortic stenosis include calcified degenerative disease, congenital bicuspid valve, rheumatic heart disease
- Common risk factors include older age, male, diabetes, HTN, hyperlipidemia and CKD
- Symptoms include exertional angina, syncope/presyncope, dyspnea, fatigue, heart failure

Aortic Valve Stenosis



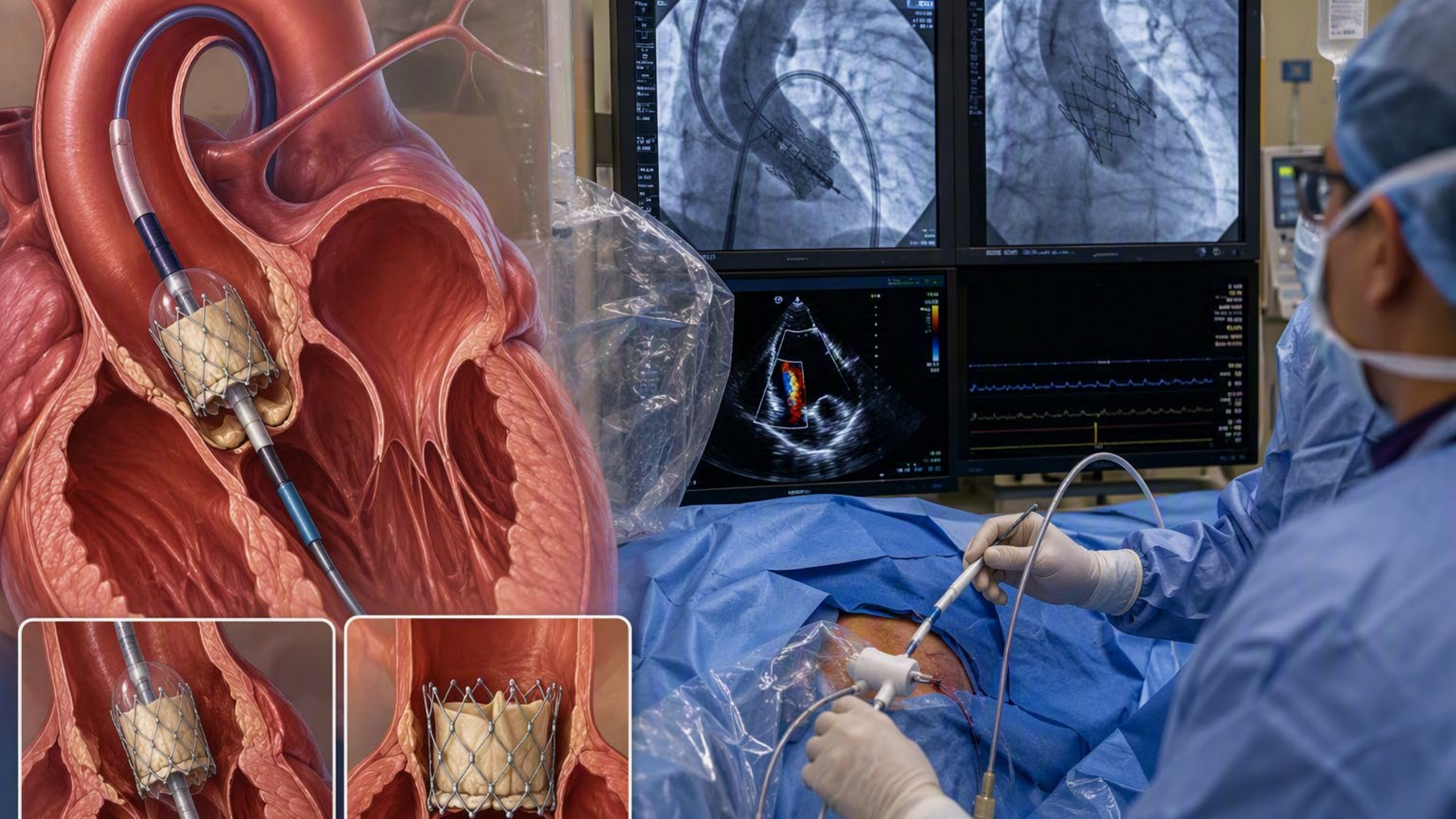


Pre-TAVR CTA

- Pre-TAVR CTA- evaluates aortic valve/root anatomy, device landing zone, vascular access route to guide prosthesis selection and reduce complications, and cardiac calcium score.
- Aortic valve calcium scoring on CTA is a flow independent adjunct to confirm severe AS
- Severe AS likely if calcium score >2000 (men) and >1200(women)

TAVR

- Transcatheter aortic valve replacement
- Minimally invasive, catheter-based procedure that replaces diseased aortic valve without having open heart surgery
- Bioprosthetic valve trileaflet tissue mounted within an expandable metal frame
- Valve is crimped onto a catheter and advanced through the vasculature of the heart, most commonly from transfemoral approach
- Valve crossed with catheter
- Balloon-expandable or self-expanding valve deployed
- New valve displaces diseased leaflets



Why TAVR Has Become Popular

- Less invasive procedure- avoids sternotomy and cardiopulmonary bypass
- Shorter/faster recovery
- Compared to SAVR, TAVR has shown lower rates of major bleeding, acute kidney injury, new Afib, and better early quality of life recovery
- Expanded from high-risk to lower-risk populations
- Excellent outcomes in appropriately selected patients

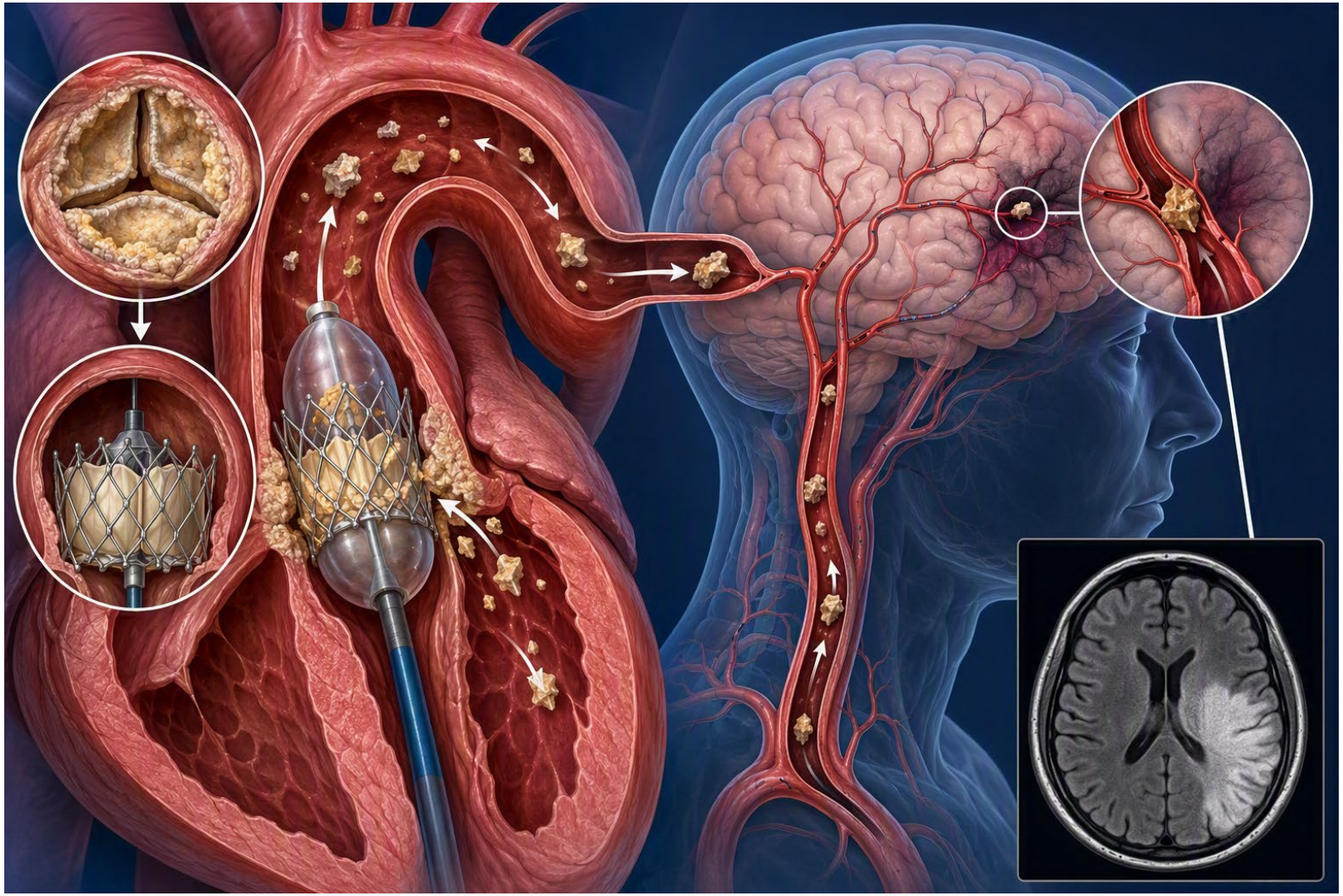
Stroke After TAVR: Why It Matters

- Stroke is one of the most feared complications
- Clinically evident strokes happen in roughly 1-3% of low-risk TAVR patients and 3-5% in high-risk patients
- Associated with increased mortality and disability
- Can occur during procedure, post procedure or even after discharge
- Most events occur within 48 hours of the procedure



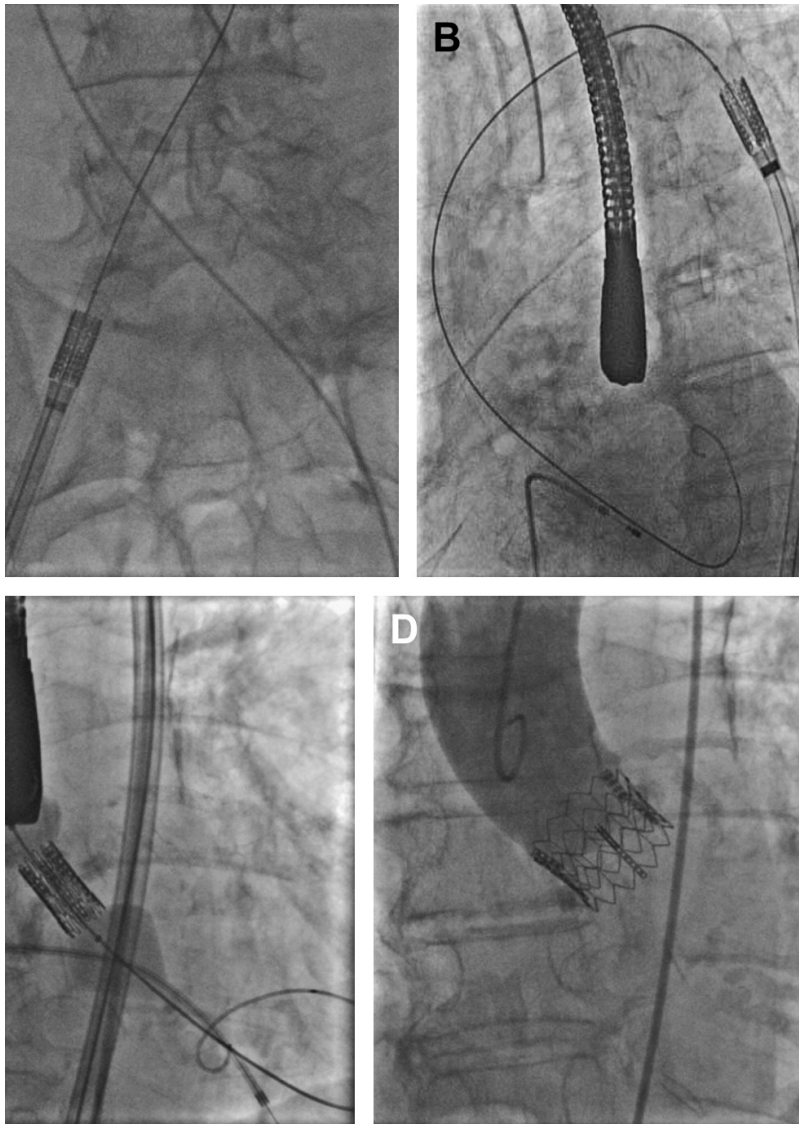
Mechanism #1: Embolization (dominant)

- Manipulation of heavily calcified valve and or aortic arch
- Calcium and tissue debris released
- Debris travels through aortic arch
- Cerebral artery occlusion causes ischemic stroke



Mechanism #2: Thromboembolism

- Catheters and prosthetic material can promote thrombus formation
- Periprocedural clot embolization
- New or existing atrial fibrillation increases risk- blood thinners held prior to procedure
- Valve thrombosis is uncommon but possible

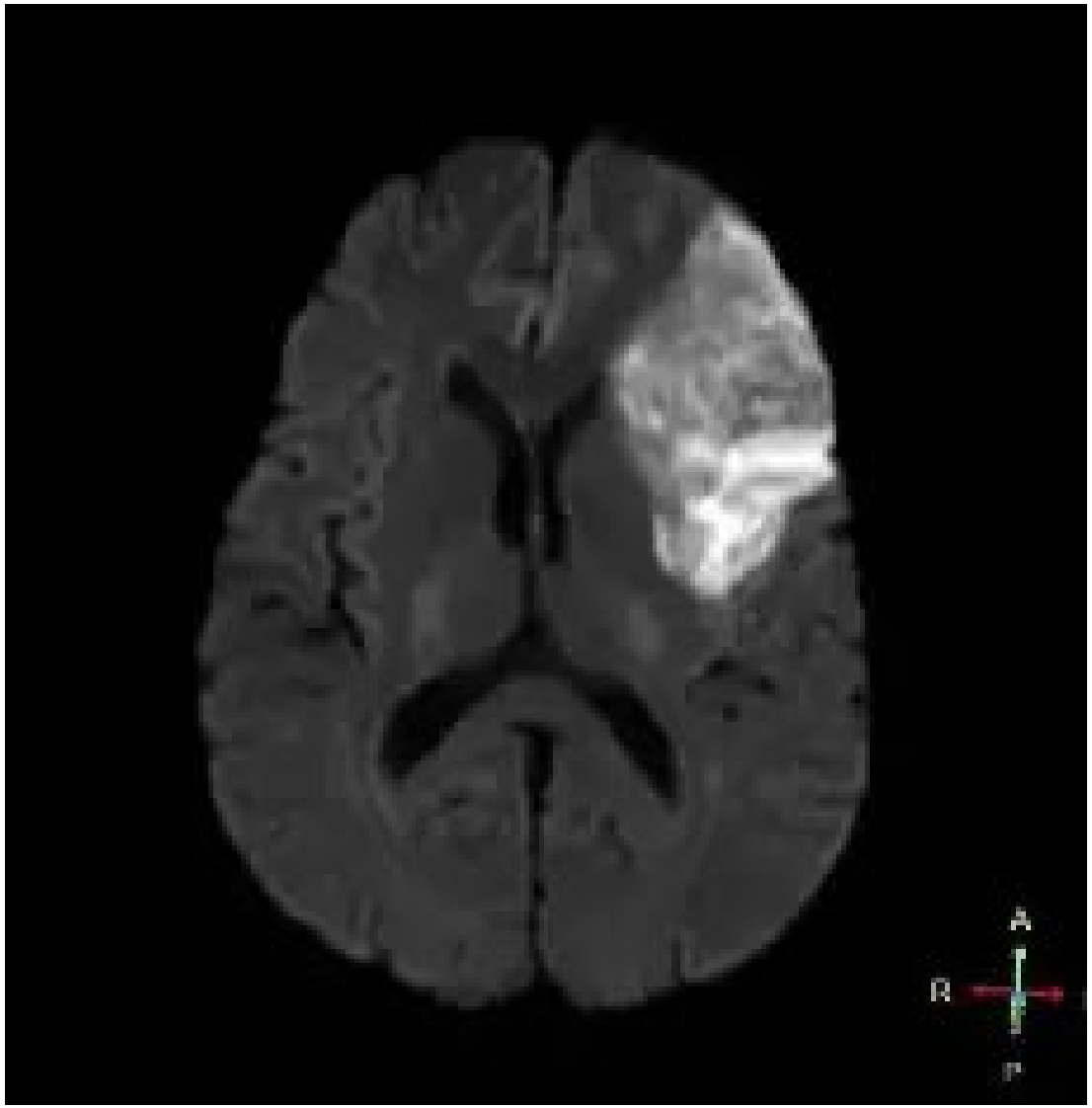


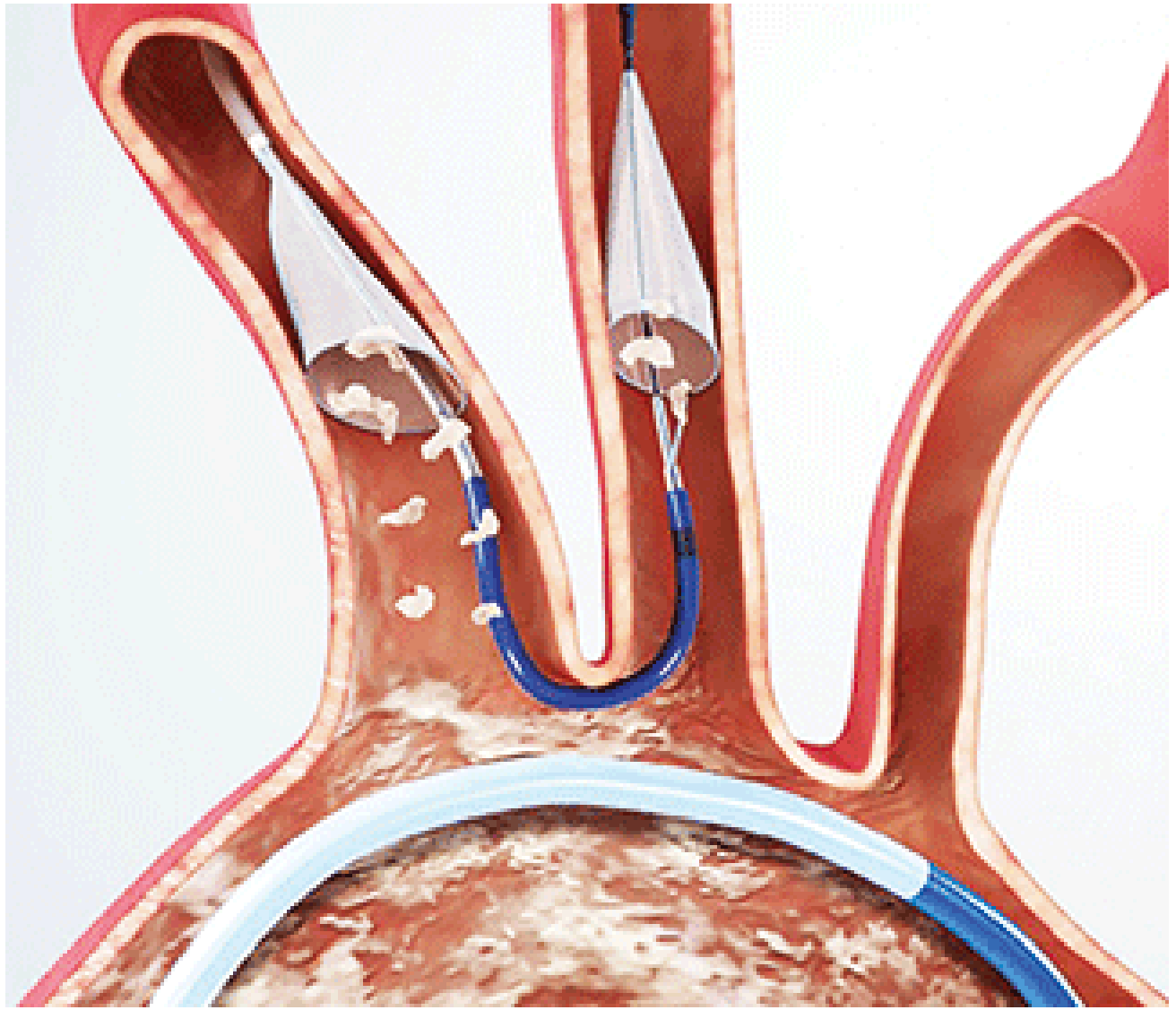
Stroke after TAVR risk factors

- Early stroke- new onset Afib, balloon post-dilation, valve migration or reposition, smaller aortic valve area, heavily calcified valve, low EF
- Late stroke- prior stroke or TIA, advanced age, hx of Afib, CKD, diabetes, PVD, valve in valve procedure, lack of anticoagulation at discharge

Stroke Prevention Strategies

- Meticulous procedural technique
- Appropriate anticoagulation and antiplatelet therapy
- Management of atrial fibrillation
- Careful patient selection

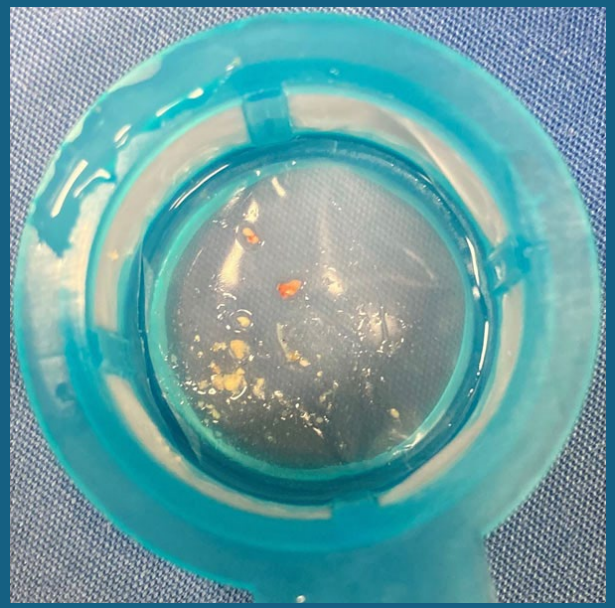




Cerebral Embolic Protection Devices

- Sentinel device placed in the brachiocephalic artery and left common carotid artery
- It captures embolic material in up to 99% of patients
- Pivotal FDA trial confirmed high debris capture
- However, reduction in new cerebral lesion volume was not significant
- Protect TAVR study (n=3000) endpoint of clinic stroke within 72hr or before discharge was not significantly reduced (2.3 % CEP vs 2.9% control)

Why debris capture doesn't equal stroke prevention



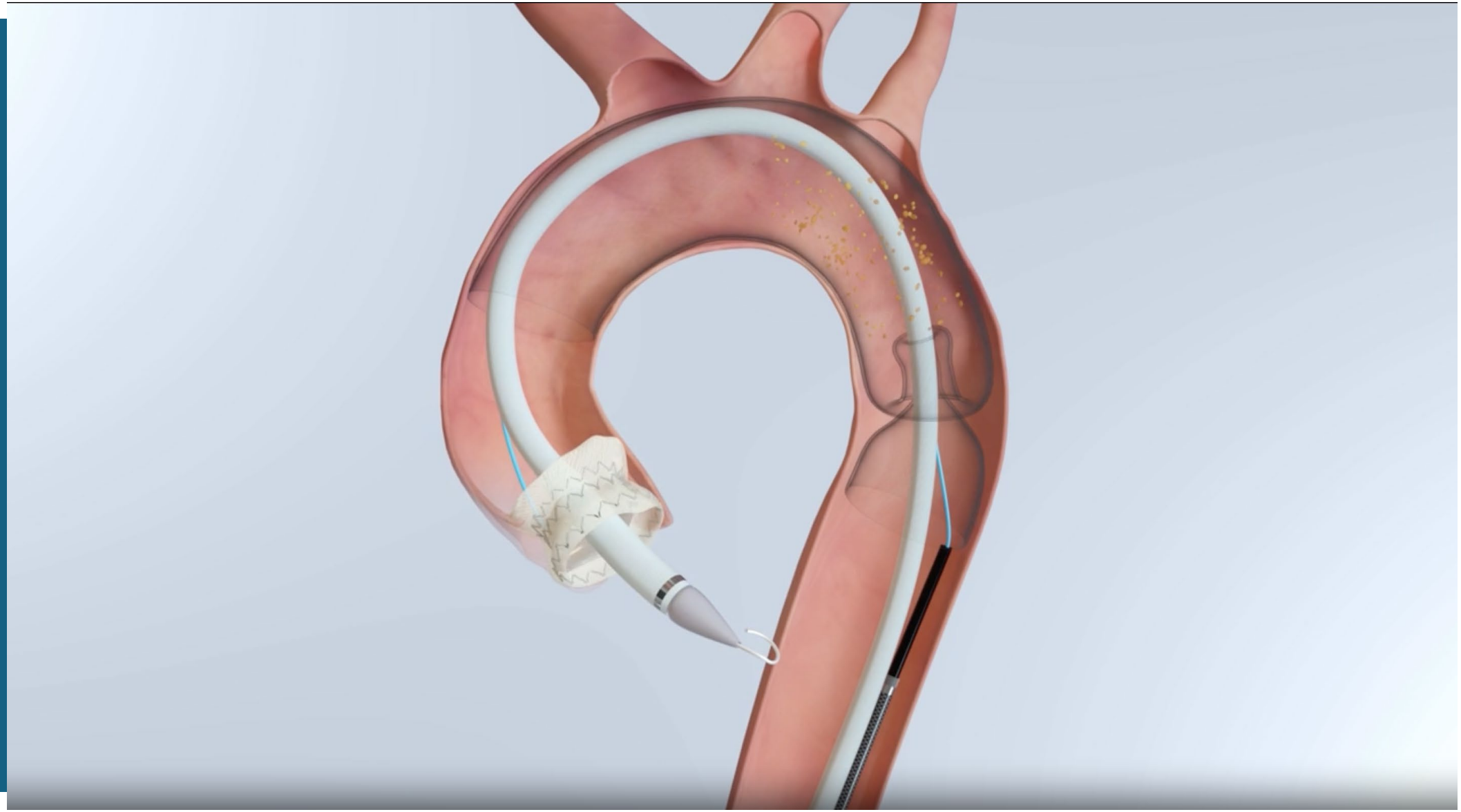
Device does not protect all cerebral territories- left vertebral artery/posterior circulation is typically unprotected

Most periprocedural neurologic injury is subclinical and reducing lesion burden has not been proven superior

Stroke rates without CEP are already low (approx. 2%) which leave little room for benefit and rendering trials underpowered

The Emboliner

- Newly FDA approved (9/10/2026)
- A novel total or whole body cerebral/systemic embolic protection catheter designed for use during TAVR
- Filter based catheter positioned in the aorta to trap embolic particles generated during TAVR
- Deployed via same femoral access used for TAVR procedure
- All filters that were placed during the initial study contained debris and histopathology showed predominantly acute thrombus and valve/arterial tissue, with lesser amounts of calcified tissue



Key Takeaways

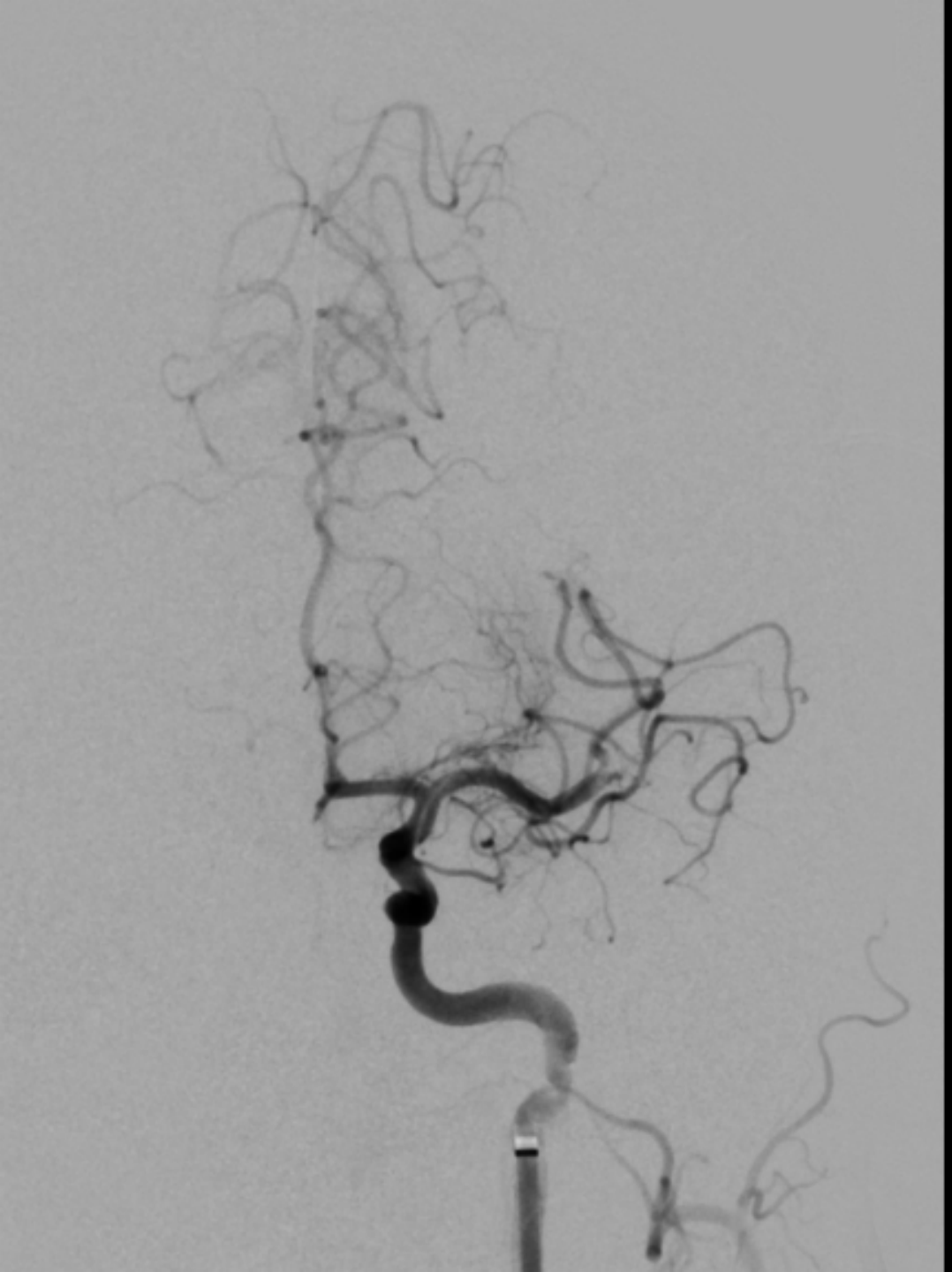
- TAVR is a safe and effective treatment for severe symptomatic AS
- Stroke remains one of the most serious complications of TAVR
- The primary mechanism of stroke is embolization
- Patient- specific risk factors increase stroke risk in those undergoing TAVR
- Careful procedural technique and appropriate antithrombotic therapy and management of Afib
- Current cerebral embolic protection devices are effective at capturing debris however have not shown significant stroke reduction
- Ongoing innovations may further reduce neurologic complications in the future
- Early recognition and management of neurologic symptoms after TAVR are critical to improving patient outcomes

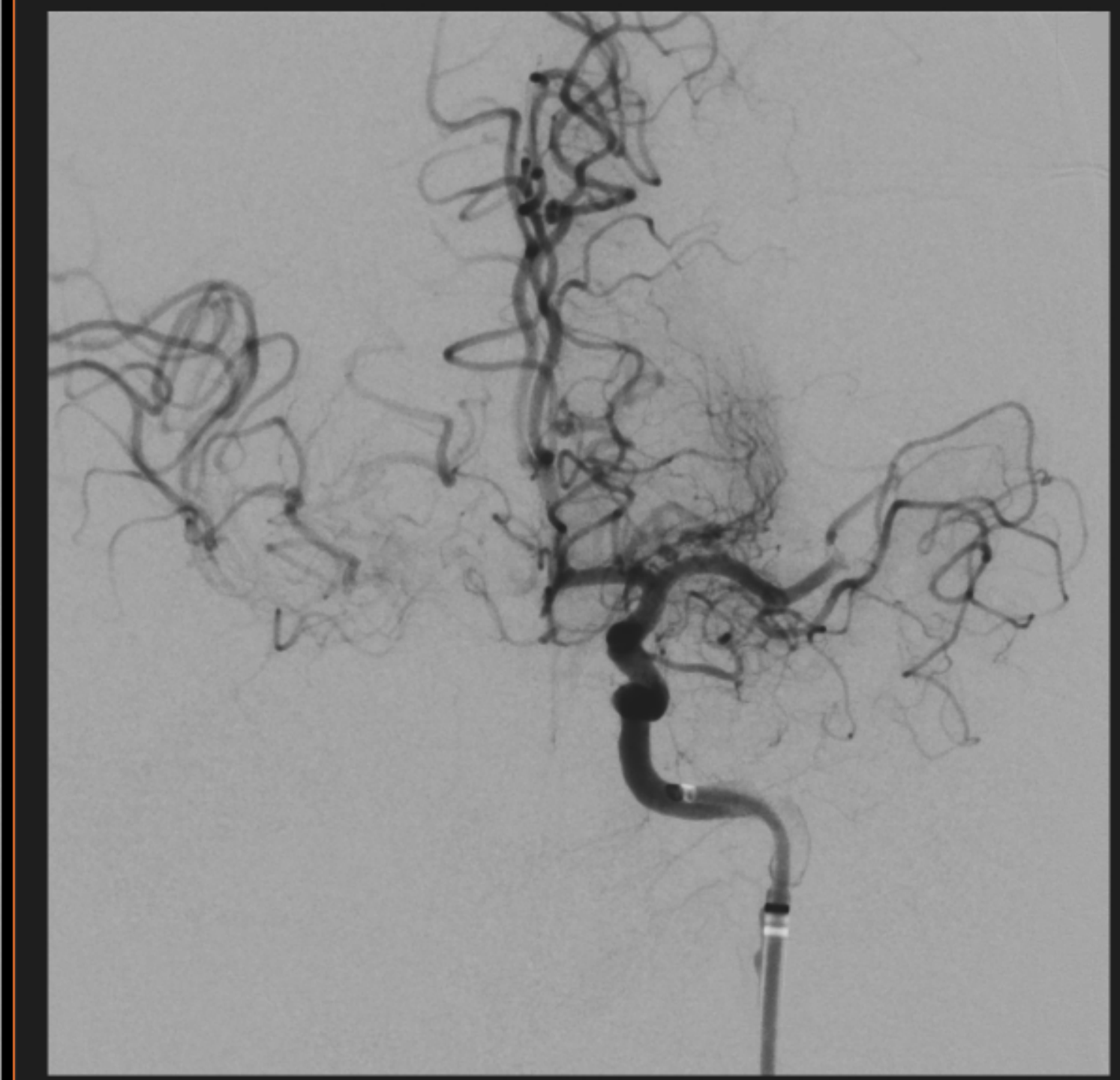
Case Study

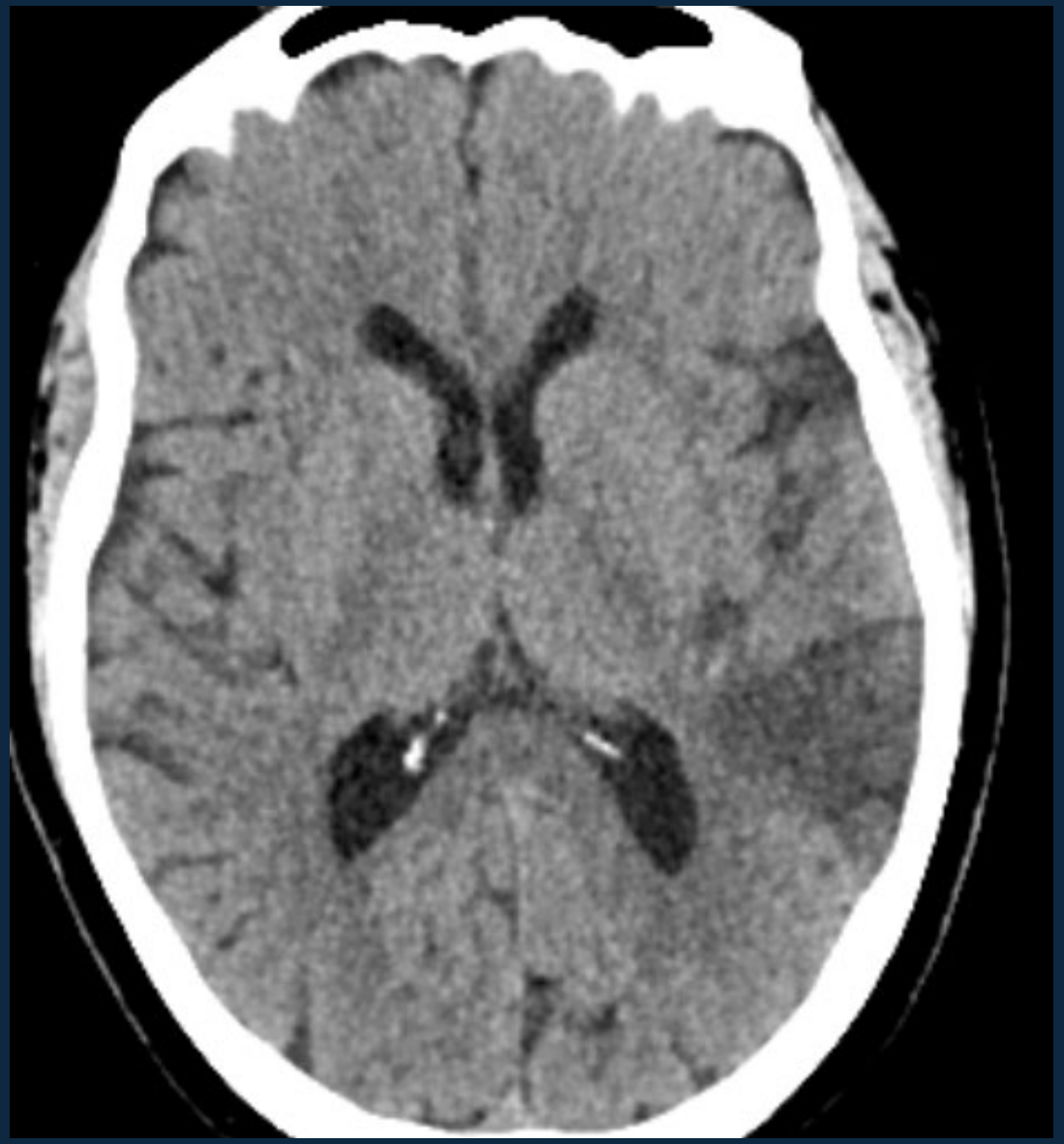
- 67 yr old female with PMH HFpEF, PAF, HTN, HLD, carotid artery disease, sinus node dysfunction s/p PPM, aortic stenosis, obesity
- Pre-procedure echo showed EF 60-65%, aortic valve area 0.68 cm2, mean gradient 44 mmHg and peak velocity 410 cm/s
- Her symptoms of AS included- increasing SOB and fatigue, stated her chores were starting to take twice as long as before, winded with a flight of stairs
- CT angiography TAVR- Calcium score 2043, conventional aortic arch anatomy, mild calcifications of thoracic aorta, severe atherosclerotic disease of the abdominal aorta with calcified and noncalcified plaque
- She was deemed high risk for SAVR and underwent TAVR

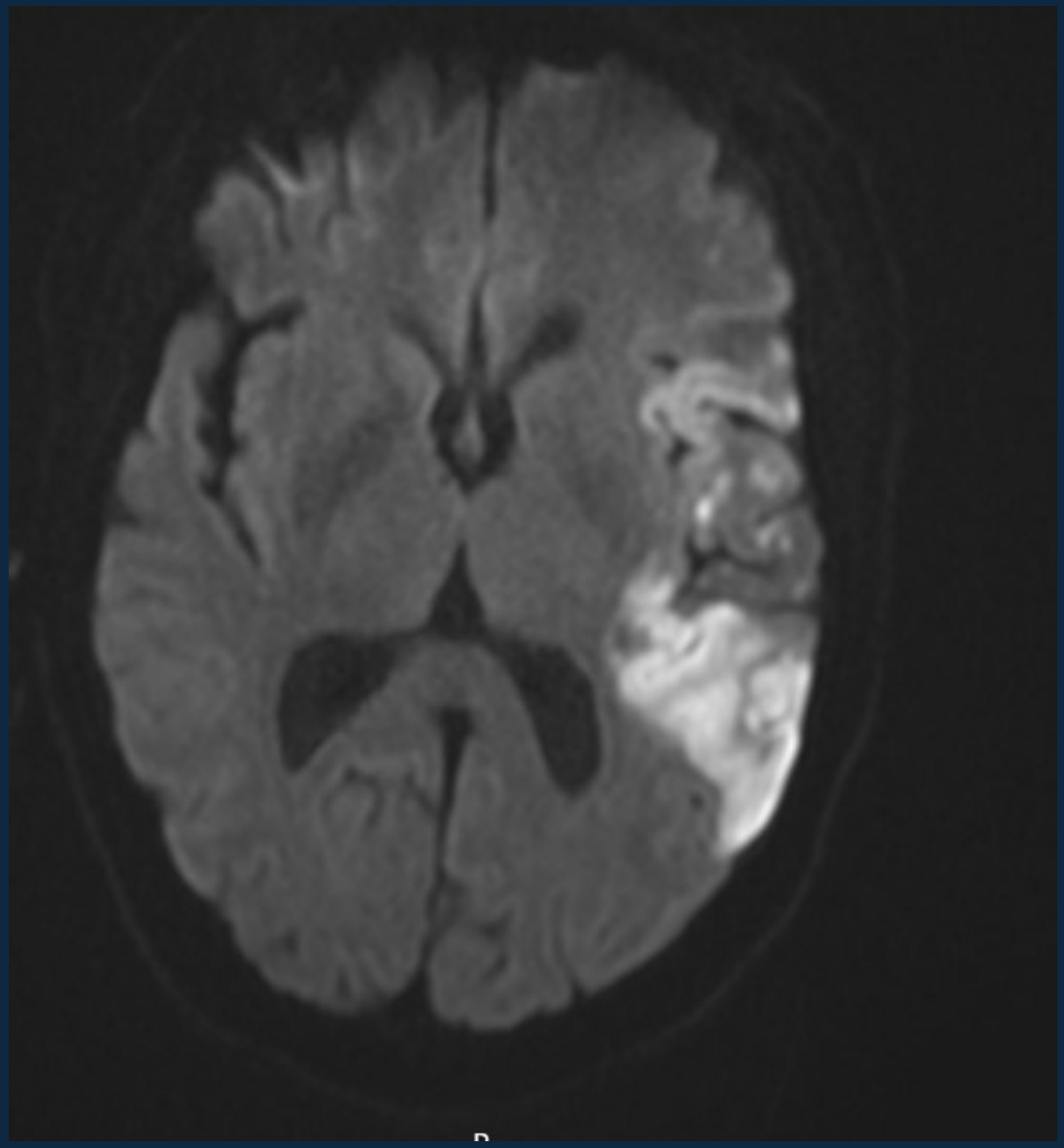
Case study

- Patient arrived to PACU hemodynamically stable, however upon assessment patient had right sided weakness, right side facial droop and expressive aphasia, she was able to shake her head yes/no
- Code Stroke was called and patient taken to CT for imaging
- CT head showed-no acute intracranial abnormalities
- CTA head showed- occlusion at distal left M1/M2 branch point with diminished left-sided M2 branches flow
- NIH score 16- for failure to answer orientation questions, partial gaze palsy, possible R partial visual field deficit, R facial droop, R side extremities some effort against gravity, aphasia
- She was given TNK and taken for thrombectomy









Case study recovery

Thrombectomy impression- occlusion of distal M1 segment of L MCA with reperfusion score TICI 2a (only partial filling of the entire vascularized territory is visualized)

Patient was discharged to rehab facility, and at her 1 month follow up she was still having difficulty walking and trouble with expressive word finding

At 3 month follow up she was walking with a cane, but still have speech issues

By her one year from TAVR follow up with was no longer walking with a cane and was able to carry a conversation with mild word finding issues

References

- Otto CM, Nishimura RA, Bonow RO, et al. 2020 ACC/AHA Guideline for the Management of Patients With Valvular Heart Disease: A Report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines. *Circulation.* 2021;143(5):e72-e227. doi:10.1161/CIR.0000000000000923.
- Kumar S, Elison D, Pershad A, Falluji N. Stroke risk after TAVR: Balloon-expandable vs self-expanding valves in community practice. JACC Advances. 2026;5(5):102727. doi:10.1016/j.jacadv.2026.102727
- Bjursten H, Fagman E, Koul S, et al. Calcium load in the aortic valve, aortic root, and left ventricular outflow tract and the risk for a periprocedural stroke. Structural Heart. 2022;6:100070. doi:10.1016/j.shj.2022.100070
- Foley M, Hall K, Howard JP, et al. Aortic valve calcium score is associated with acute stroke in transcatheter aortic valve replacement patients. J Soc Cardiovasc Angiogr Interv. 2022;1:100349. doi:10.1016/j.jscai.2022.100349.
- Patel KP, Rathod KS, Lansky AJ, et al. Cerebrovascular events in patients undergoing transcatheter aortic valve replacement: A review. Stroke. 2024;55(11):2754-2764. doi:10.1161/STROKEAHA.124.047149.
- Stortecky S, Lanz J, O'Sullivan CJ, et al. Long-term risk of stroke after transcatheter aortic valve replacement: Insights from the SwissTAVI Registry. Stroke. 2024;55(2):432-441. doi:10.1161/STROKEAHA.123.043678.
- Greenbaum AB, Nazif T, et al. PROTECT H2H: Emboliner vs Sentinel Embolic Protection Devices in Transcatheter Aortic Valve Replacement. Presented at the American College of Cardiology Annual Scientific Session (ACC.26); March 2026; New Orleans, LA.
- Kapadia SR, Makkar R, Leon MB, et al. Cerebral embolic protection during transcatheter aortic-valve replacement. N Engl J Med. 2022;387(14):1253-1263. doi:10.1056/NEJMoa2204961.