Neuroimaging in Cardio – embolic Stroke

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Diagnosis of Cardioembolic Stroke

"The presence of a potential cardioembolic source in the absence of cerebrovascular disease in a patient with a non-lacunar stroke"

Cerebral Embolism Task Force, 1989

Cardioembolic strokes account for 15–20% of all ischemic strokes .

This subtype of stroke tends to have a poor prognosis compared with other ischemic stroke subtypes .

The diagnosis of the cardioembolic origin is an important step in the proper management of patients with a suspected embolic stroke .

Dividing potential cardiac origins of emboli into high or low or uncertain risk categories is clinically useful .

Cardioembolic Sources

High Risk	Medium Risk	Low / Unclear Risk
Atrial fibrillation	LV hypokinesia / aneurysm	Patent foramen ovale
Recent anterior MI	Bioprostetic valve	Atrial septal aneurysm
Mechanical valve	Congestive failure	Spontaneous echo contrast
Rheumatic mitral stenosis	Cardiomyopathy	
Thrombus / tumor	Myxomatous MVP	
Endocarditis		

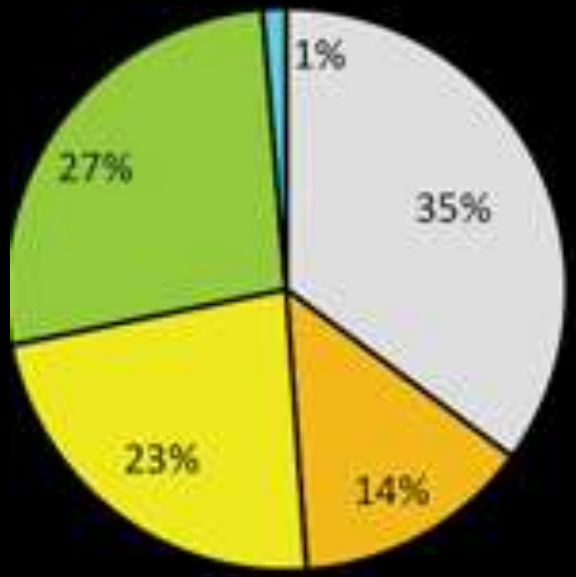
Stroke etiology

Proper diagnosis → Proper treatment

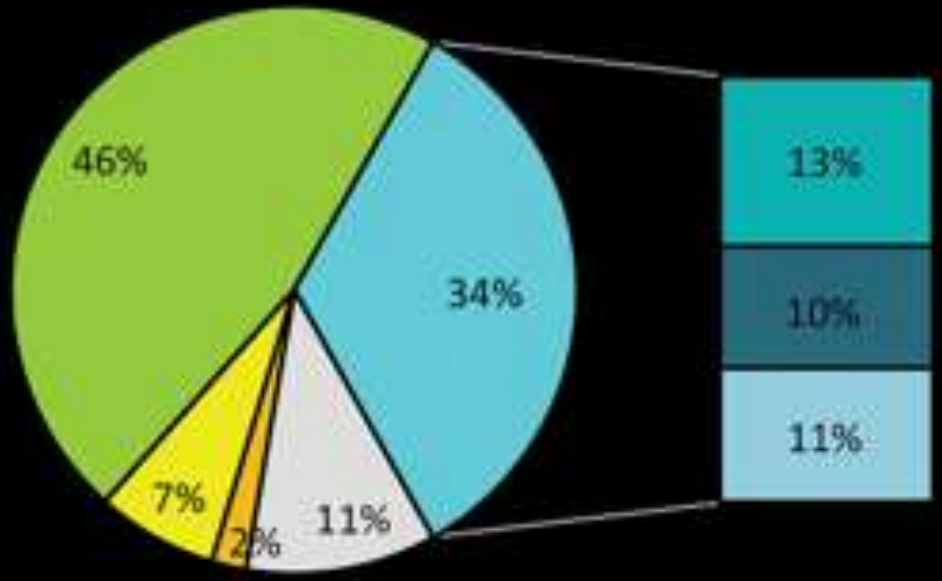
TOAST classification	The Causative Classification of Stroke System (CCS)	The ASCOD classification
Large artery atherosclerosis	**Stroke Mechanism-**	**Phenotype-**
	Large artery atherosclerosis	Atherothrombosis (A)
Cardioembolism	Cardio-aortic embolism	Small-vessel disease (S)
Small vessel occlusion (lacunar)	Small artery occlusion	Cardiac Pathology (C)
Stroke of other etiology	Other causes	Other causes (O)
Stroke of undetermined etiology **a. Two or more causes identified** **b. Negative evaluation** **c. Incomplete evaluation**	Undetermined causes a. Unknown b. Cryptogenic embolism c. Other cryptogenic* d. Incomplete Evaluation e. Unclassified	Dissection (D)
		Grade of Disease-
	Weight of Evidence-	1. If disease is present and potentially causal
	Evident: Sole potential mechanism	2. If disease is present and causal link is uncertain
	Probable: >1 evident stroke mechanisms where one mechanism appears more probable than others	3. If disease is present and causal link is unlikely
	Possible: Absence of any evident causes, mechanisms with a lower or less well defined risk	4. If disease is absent
		9. If the workup is insufficient to grade disease

*Other cryptogenic: Those not fulfilling the criteria for cryptogenic embolism

Older Adults
>45 years old
583 patients
Kolominsky-Rabas 2001



Young Adults
18-45 years old
215 patients
Ji 2013

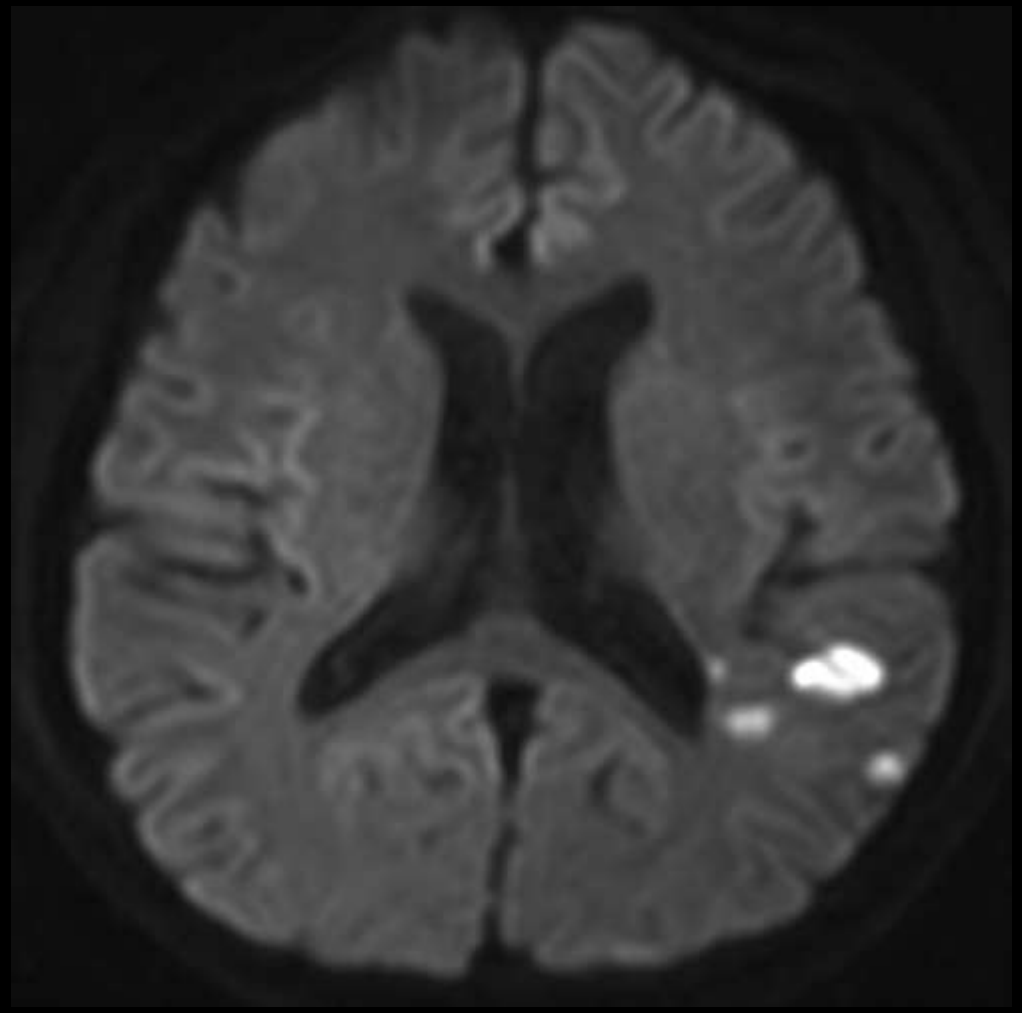


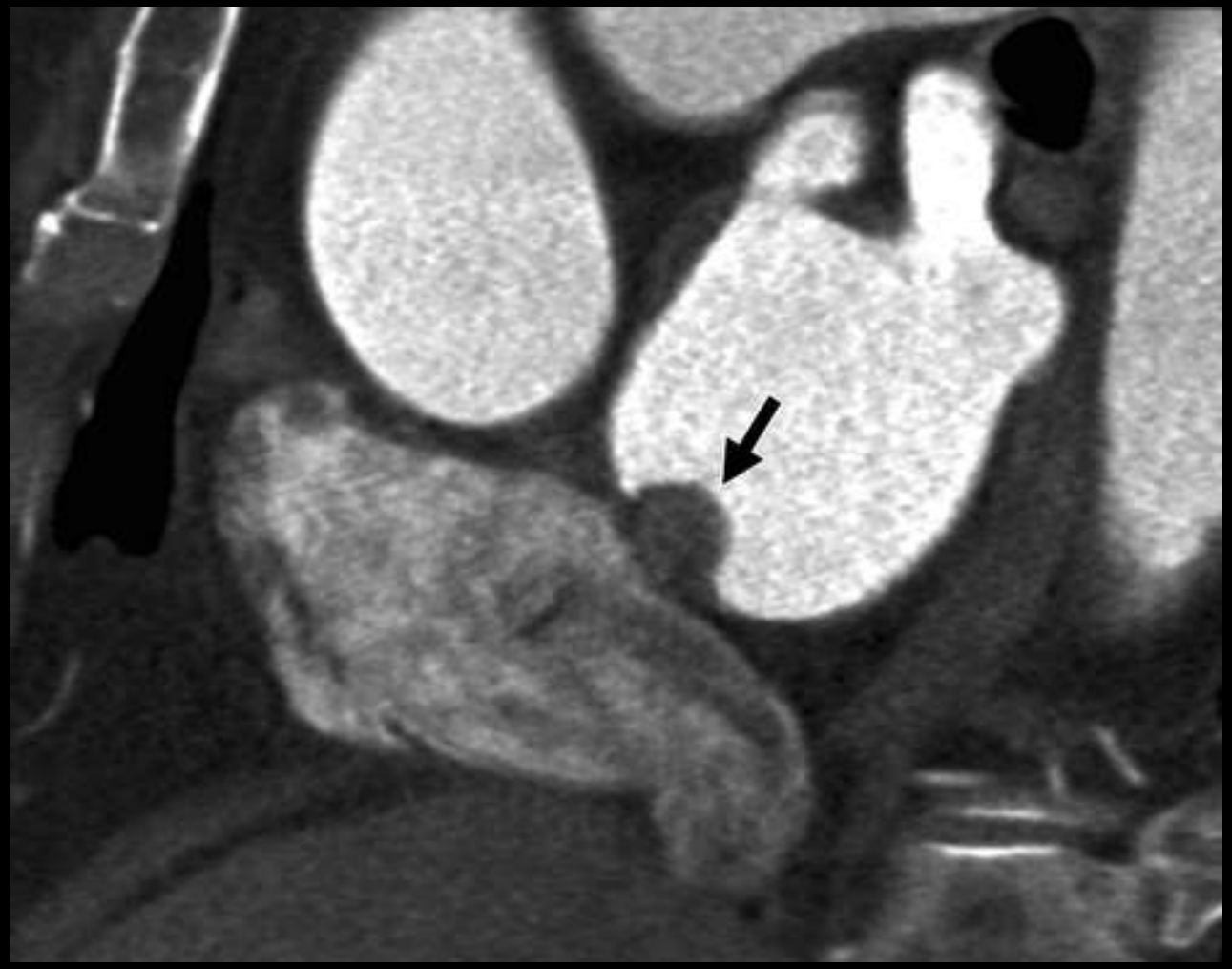
- Undetermined
- Large-artery atherosclerosis
- Small-vessel occlusion
- Cardioembolism
- Other Demonstrated Causes
- Dissection
- Vasculopathy
- Remaining

Defining Features, Imaging Findings, and Causes	Cardioembolism	Other Demonstrated Causes	Undetermined Cause	Small-Vessel Occlusion (Lacunar)	Large-Artery Atherosclerosis
Defining features and imaging findings	Scattered small or larger confluent infarcts, both superficial and deep, often in multiple vascular territories	Single explained stroke cause Variable appearances at imaging	Two or more potential causes or negative workup results or incomplete workup Variable appearance at imaging	Deep infarct (<1.5 cm) in basal ganglia, internal capsule, thalamus, or brainstem and no concurrent large-artery atherosclerosis in relevant vessel	Cerebral cortex, brainstem, or cerebellar infarct plus extracranial or intracranial large-artery atherosclerosis (ICA origins, carotid siphons, V4 vertebral arteries, basilar artery)
Causes	Septic emboli, bland emboli (from cardiac masses, right-to-left shunts, arrhythmias, and cardiomyopathy), air emboli, and fat emboli	Nonatherosclerotic vasculopathy (dissection, RCVS, moyamoya, vasculitis, drug-related, CADASIL) Prothrombotic (factor V Leiden, antiphospholipid, oral contraceptive or pregnancy related)	Unknown for each case	Lacunar infarction from small end vessel occlusion	Thromboembolic from vulnerable large-vessel plaques Decreased perfusion from parent vessel stenosis or occlusion

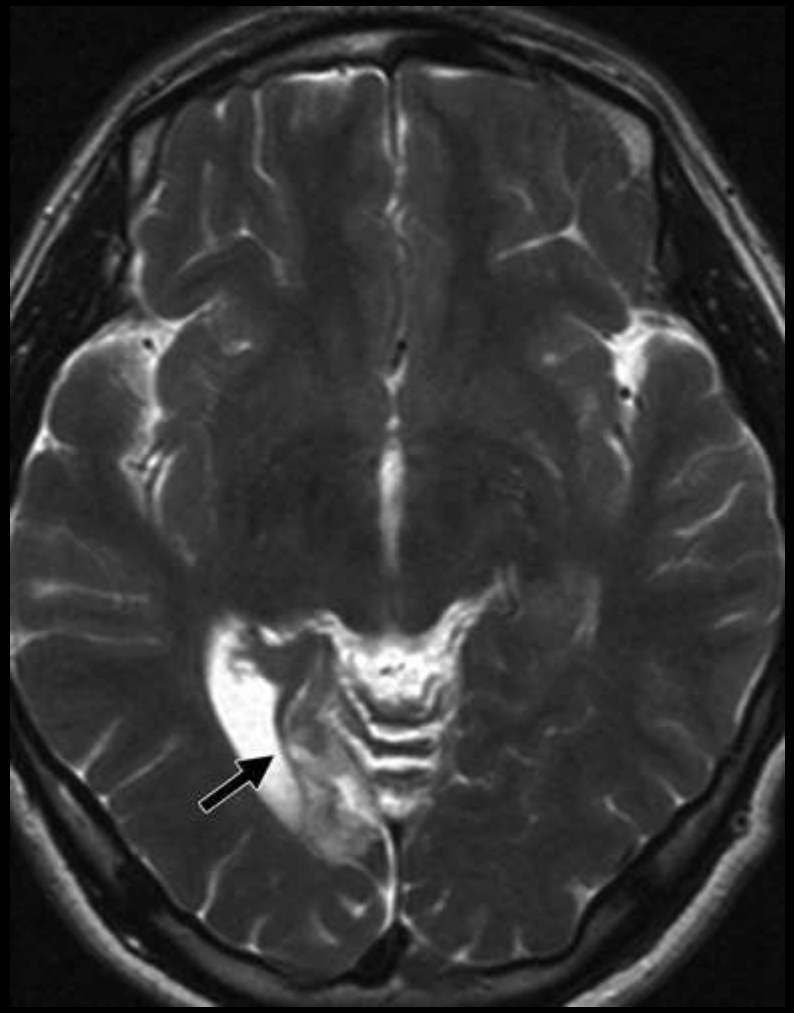
Radiological characteristics of cardio embolic strokes :

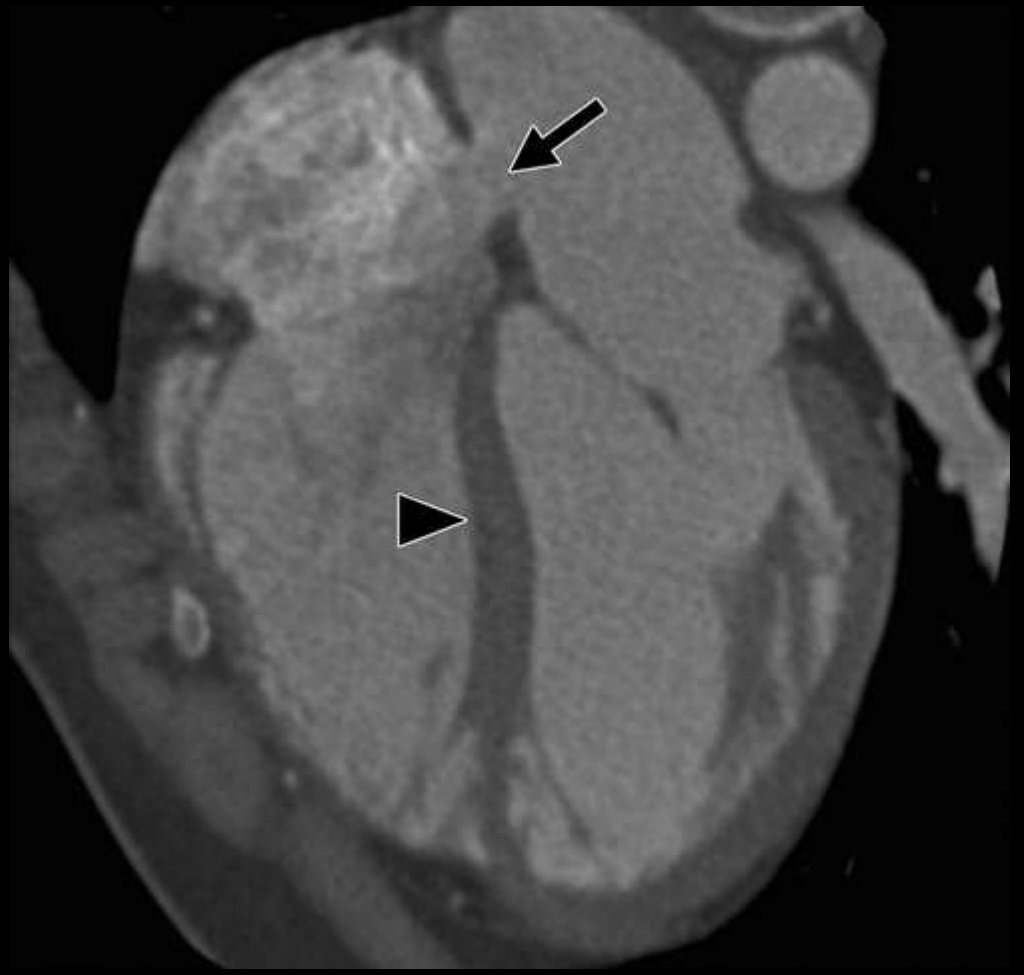
- **Wedge –shaped infarctions based in the cortex**
- **Concurrent acute bilateral infarctions**
- **Concurrent acute infarctions in the anterior and posterior circulations**
- **multiple cortical infarctions in various vascular distributions(even if infarctions in different ages)**
- **Greater tendency to hemorrhagic transformation**



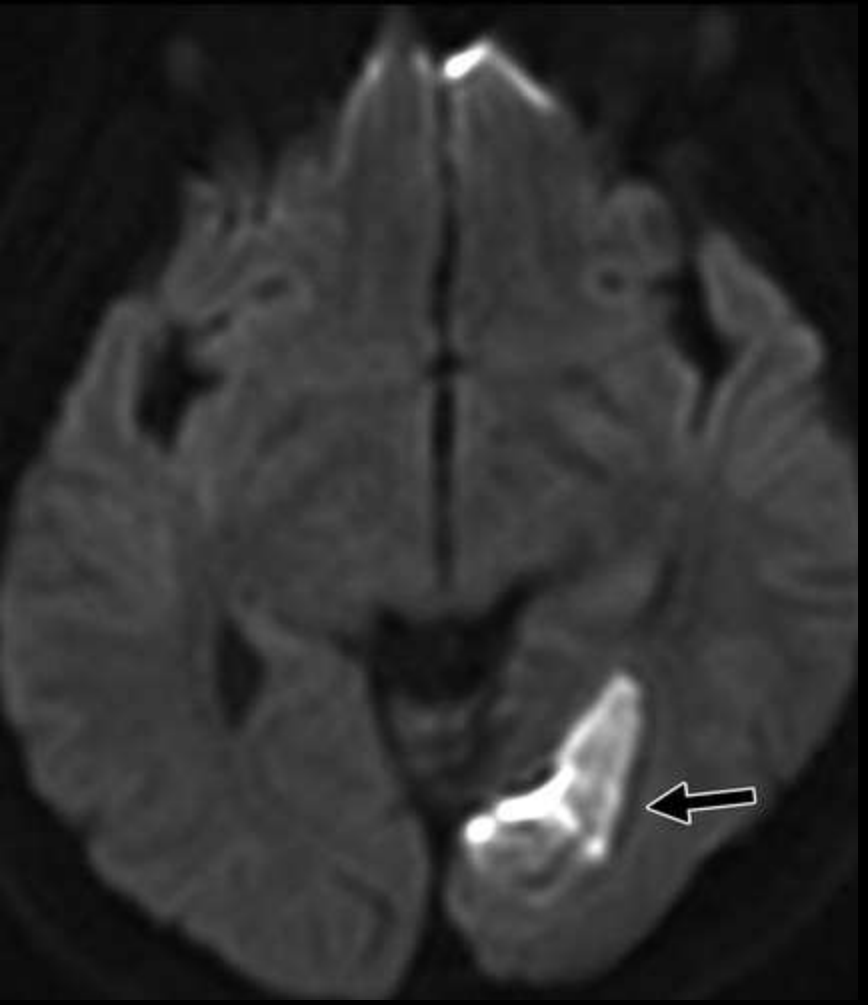


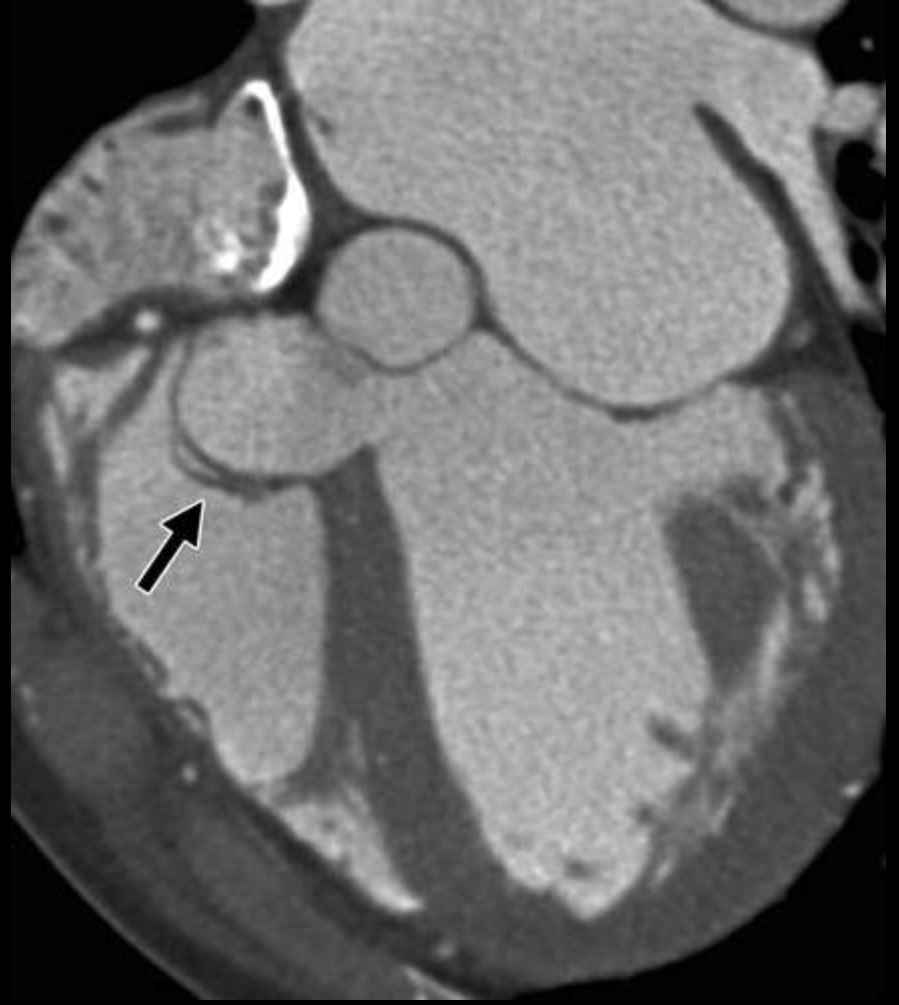
71-year-old woman with visual disturbance. Diffusion MR image shows multiple small embolic infarctions in left temporal lobe and parietal lobe.





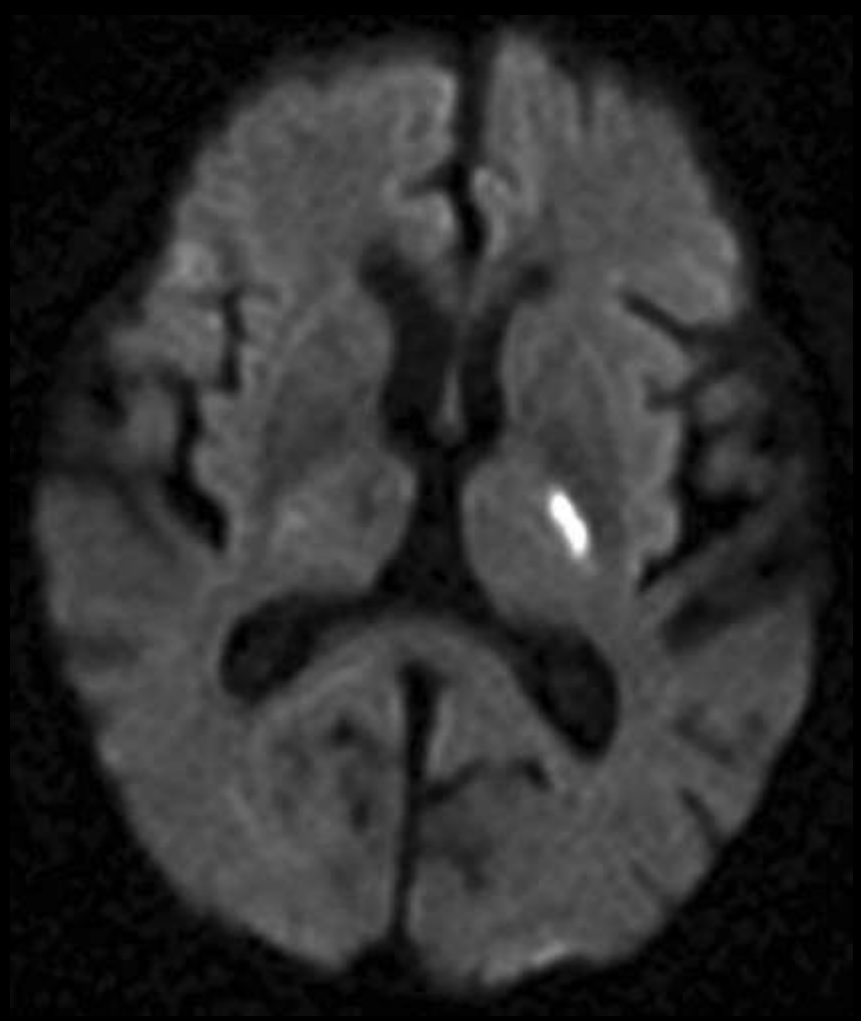
21-year-old woman with no vision bilaterally and paradoxical embolism. Atrial septal defect (*arrow*) is easily visualized on MDCT image. Note associated finding of bowing of ventricular septum to left (*arrowhead*) and dilatation of right atrium and right ventricle due to atrial septal defect.

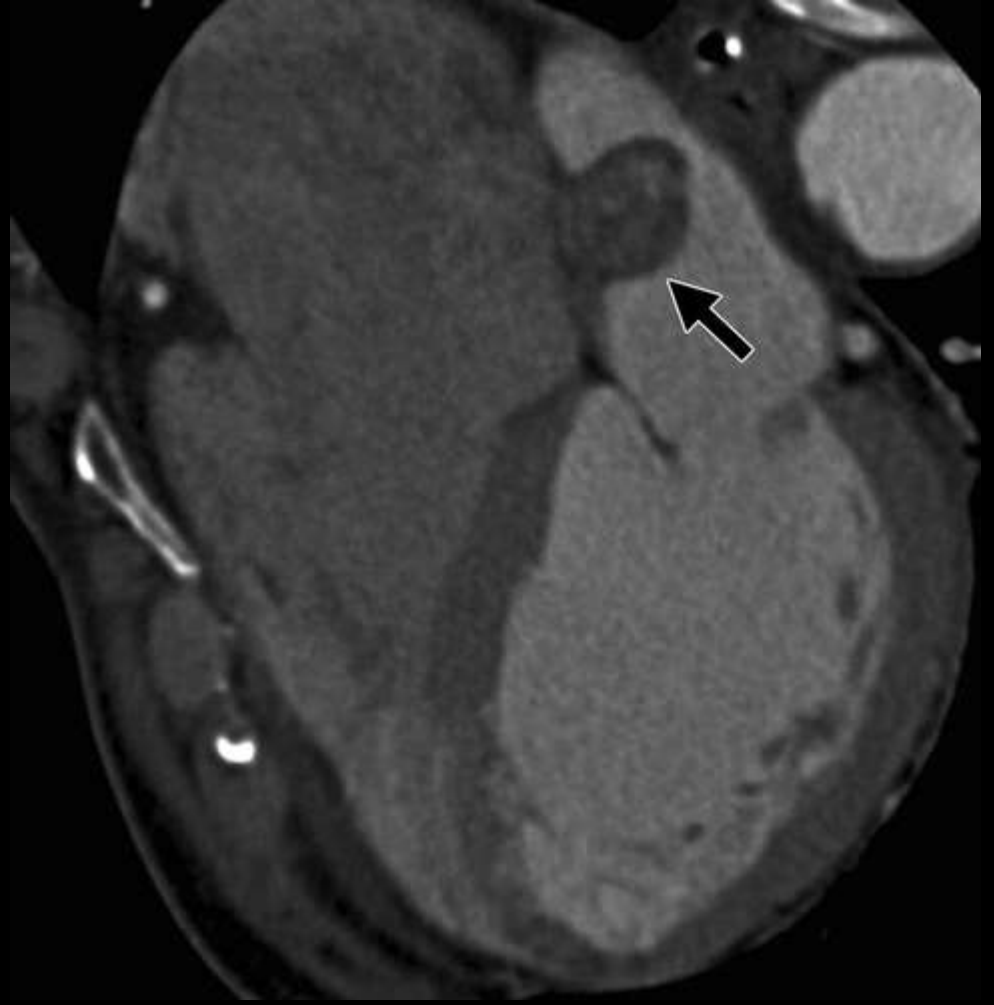




28-year-old man with acute onset of headache and paradoxical embolism. Diffusion MR image shows infarction (*arrow*) at left occipital lobe.

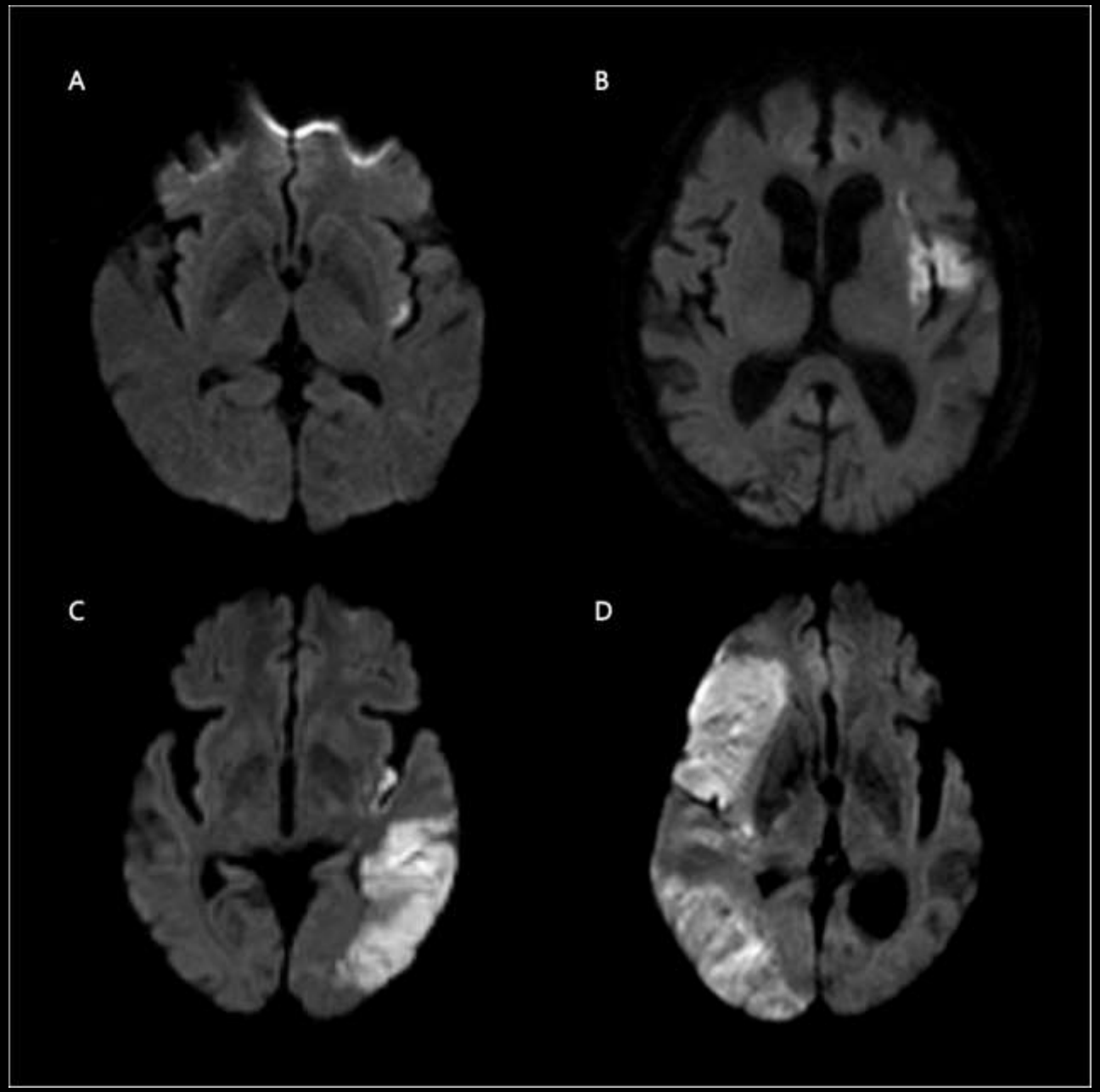
MDCT image shows ventricular septal defect





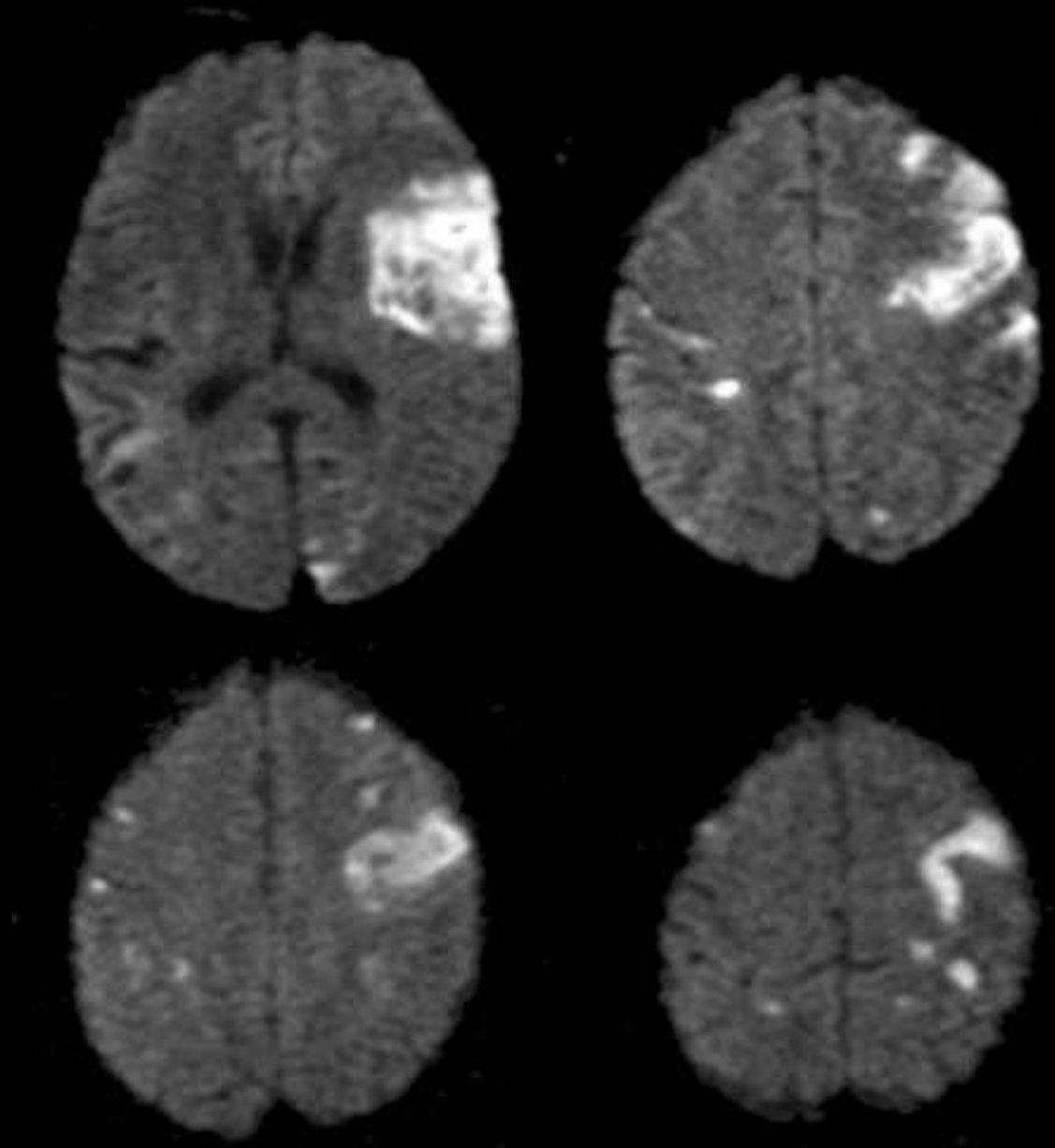
85-year-old man with weakness and dysarthria of right side. Diffusion MR image shows acute infarction at left internal capsule.

Two-chamber CT image shows 1.8-cm nonhomogeneous polypoid mass (*arrow*) in left atrium that is attached to interatrial septum by broad pedicle. Mass was confirmed to be left atrial myxoma.



Cardioembolism and Involvement of the Insular Cortex in Patients with Ischemic Stroke

A 55 year-old woman presented with confusion, right sided



Shower of Emboli: Diffusion weighted MRI axial images. Note the multiple areas of diffusion abnormalities that indicate acute infarction. These areas are present in the bilateral middle cerebral and left posterior cerebral artery distributions. The large infarct in the left frontal lobe resulted in the patient's symptoms of aphasia and right sided weakness. This pattern of multiple, acute cortical strokes in different vascular territories usually signifies a shower of emboli, typically from a cardiac source. In this patient, the atrial fibrillation was the likely cardiac etiology.

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Cardioembolic stroke

- Patients with an arterial occlusion, presumably due to an embolus arising in the heart
- Sources divided into high-risk and medium-risk groups
  - Propensity for embolism
- Large artery atherosclerotic sources of embolism should be eliminated

TOAST classification of high and medium risk sources of cardioembolism

High-risk sources

- Mechanical prosthetic valve
- Mitral stenosis with atrial fibrillation
- Atrial fibrillation
- Left atrial/atrial appendage thrombus
- Sick sinus syndrome
- Recent myocardial infarction (<4 weeks)
- Left ventricular thrombus
- Dilated cardiomyopathy
- Akinetic left ventricular segment
- Atrial myxoma
- Infective endocarditis

Medium-risk sources

- Mitral valve prolapse
- Mitral annulus calcification
- Mitral stenosis without atrial fibrillation
- Left atrial turbulence (smoke)
- Atrial septal aneurysm
- Patent foramen ovale
- Atrial flutter
- Lone atrial fibrillation
- Bioprosthetic cardiac valve
- Nonbacterial thrombotic endocarditis
- Congestive heart failure
- Hypokinetic left ventricular segment

Cardioembolic stroke

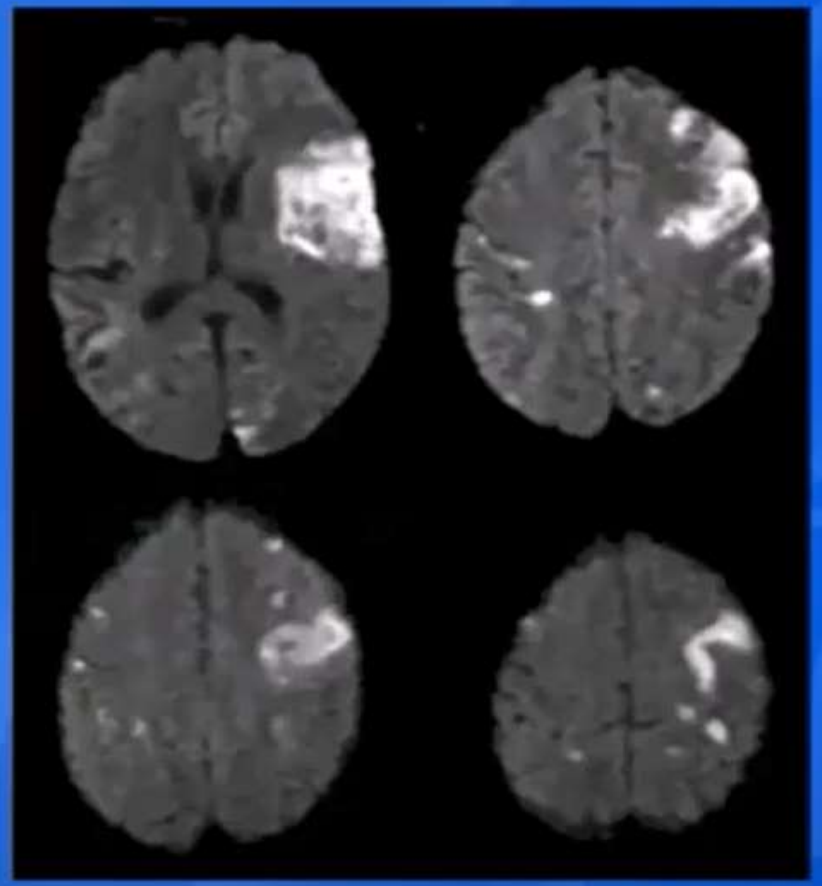
- More severe than other stroke subtypes
  - 5-year mortality rate for CS has been reported as high as 80%
- Higher risk of recurrence
- Typical presentation:
  - maximal onset
  - sudden onset
  - large artery occlusion or multiple vascular territories

Most commonly cited clinical features

- Isolated focal deficit
- Seizure at onset
- Loss of consciousness at onset
- Peak of deficit at onset
- Symptoms suggesting involvement of >1 vascular territory
- Evidence or suggestion of systemic embolism

Findings on Imaging

- Multiple infarcts in more than one territory
- Deep and superficial infarcts
- Hemorrhagic conversion
- Absence of large-artery stenosis or occlusion in parent vessels
- Rapid recanalization



Potential clinical evaluation for embolic stroke

- Physical examination: Temperature, cardiac auscultation, assessment for peripheral edema, lung auscultation, retinal examination, skin examination
- Imaging studies: CT/MRI of brain, vascular imaging, CXR
- Cardiac monitoring: serial ECG, cardiac telemetry, extended cardiac monitoring
- Cardiac evaluation: Echocardiography→ Transthoracic, transesophageal

Atrial Fibrillation: Mechanism

- Responsible for nearly half of all cardioembolic strokes
- Increases with age
- Short, asymptomatic periods of AF can be sufficient to form a thrombus
- Believed to be caused by embolization from the left atrium (LA) and atrial appendage
- Stasis→ clot formation→ subsequent embolization

Why is anticoagulation recommended?

- Antiplatelet agents prevent formation of **white thrombus**
  - platelet aggregates that form in the setting of fast flow, in arteries and perhaps on heart valves
- **Red Thrombus**: Stasis, such as in the left atrial appendage in atrial fibrillation, in ventricular dyskinesia and in deep veins (leading to paradoxical embolism)
  - It results from polymerization of fibrin→ formation of a mesh of long fibrin strands → entrapped red blood cells
- To prevent formation of red thrombus it is necessary to use anticoagulants
- This is why antiplatelet agents are much less effective than anticoagulants in preventing stroke from atrial fibrillation

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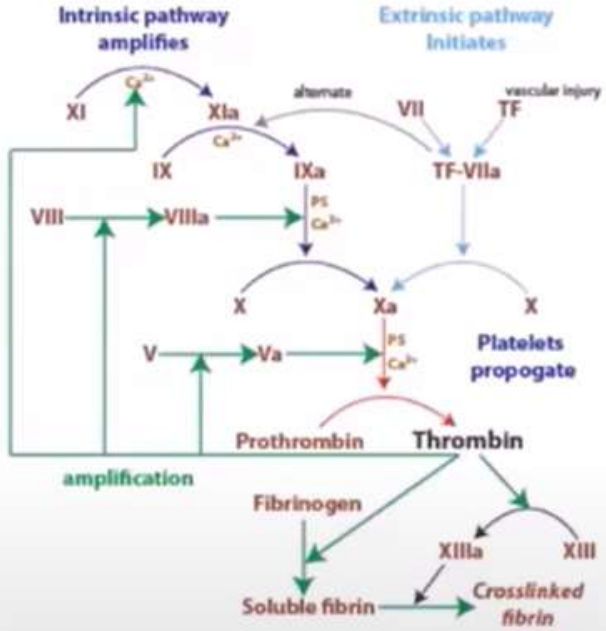
Anticoagulation for Stroke Prevention

Pharmacologic Profiles of Approved and Investigational Oral Anticoagulants

	Warfarin	Dabigatran	Rivaroxaban	Apixaban	Edoxaban
Target	Vitamin K epaxide	Thrombin	Xa	Xa	Xa
Administration	Once daily	Twice daily	Once daily	Twice daily	Once daily
Prodrug	No	Yes	No	No	No
Half-life	40 hrs	12-14 hours	9-13 hours	8-15 hours	6-11 hours
Bioavailability	>95%	6.5%	80%	~66%	50%
Renal excretion	Minor	80%	30-60%	25%	35-39%
Time to Peak Plasma	72-96 hours	2 hours	2.5-4 hours	3 hours	1-2 hours
Metabolized by CYP3A4	Yes	No	Yes	Yes	Yes

Uchiyama S, et al. *J Stroke Cerebrovasc Dis*. 2012;21:165-173; Eriksson BI, et al. *Annu Rev Med*. 2011;62:41-57



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