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Review article:

Cardio-embolic stroke: Pathophysiology –Review

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Abstract:

The presence of a venous source of embolus, most commonly a deep venous thrombus, may provide confirmation of the importance of a PFO with an accompanying right-to-left shunt in a particular case. Three randomized trials of PFO occlusion for secondary prevention of ischemic stroke were negative, although each lacked sufficient power to be conclusive. 7At present, there is no supportive evidence to offer percutaneous PFO closure for stroke prevention. Bacterial endocarditis can be a source of valvular vegetations that give rise to septic emboli. The appearance of multifocal symptoms and signs in a patient with stroke makes bacterial endocarditis more likely. Infarcts of microscopic size occur, and large septic infarcts may evolve into brain abscesses or cause haemorrhage into the infarct, which generally precludes use of anticoagulation or thrombolytics. Mycotic aneurysms caused by septic emboli may also present as SAH or intracerebral haemorrhage.

Keywords: stroke, emboli , cardiovascular disease

Introduction:

Cardio embolism is responsible for ~20% of all ischemic strokes. Stroke caused by heart disease is primarily due to embolism of thrombotic material forming on the atrial or ventricular wall or the left heart valves. These thrombi then detach and embolize into the arterial circulation. The thrombus may fragment or lyse quickly, producing only a TIA. Alternatively, the arterial occlusion may last longer, producing stroke. Embolic strokes tend to occur suddenly with maximum neurologic deficit present at onset.[1]

With reperfusion following more prolonged ischemia, petechial haemorrhages can occur within the ischemic territory. These are usually of no clinical significance and should be distinguished from frank intracranial haemorrhage into a region of ischemic stroke where the mass effect from the haemorrhage can cause a significant decline in neurologic function. Emboli from the heart most often lodge in the intracranial internal carotid artery, the MCA, the posterior cerebral artery (PCA), or one of their branches; infrequently, the anterior cerebral artery (ACA) is involved. Emboli large enough to occlude the stem of the MCA (3–4 mm) lead to large infarcts that involve both deep grey and white matter and some portions of the cortical surface and its underlying white matter. [2]

Pathophysiology:

A smaller embolus may occlude a small cortical or penetrating arterial branch. The location and size of an infarct within a vascular territory depend on the extent of the collateral circulation.[3]

The most significant causes of cardioembolic stroke in most of the world are nonrheumatic (often called nonvalvular) atrial fibrillation, MI, prosthetic valves, rheumatic heart disease, and ischemic cardiomyopathy. Nonrheumatic atrial fibrillation is the most common cause of cerebral embolism overall. The presumed stroke mechanism is thrombus formation in the fibrillating atrium or atrial appendage, with subsequent embolization. Patients with atrial fibrillation have an average annual risk of stroke of ~5%. The risk of stroke can be estimated by calculating the CHADS2 score. [4]

Left atrial enlargement is an additional risk factor for formation of atrial thrombi. Rheumatic heart disease usually causes ischemic stroke when there is prominent mitral stenosis or atrial fibrillation. Recent MI may be a source of emboli, especially when transmural and involving the anteroapical ventricular wall, and prophylactic anticoagulation following MI has been shown to reduce stroke risk.

Mitral valve prolapse is not usually a source of emboli unless the prolapse is severe. Paradoxical embolization occurs when venous thrombi migrate to the arterial circulation, usually via a patent foramen ovale or atrial septal defect. Bubble-contrast echocardiography (IV injection of agitated saline coupled with either transthoracic or transoesophageal echocardiography) can demonstrate a right-to-left cardiac shunt, revealing the conduit for paradoxical embolization. Alternatively, a right-to-left shunt is implied if immediately following IV injection of agitated saline, the ultrasound signature of bubbles is observed during transcranial Doppler insonation of the MCA; pulmonary arteriovenous malformations should be considered if this test is positive yet an echocardiogram fails to reveal an intracardiac shunt. [5]

Both techniques are highly sensitive for detection of right-to-left shunts. Besides venous clot, fat and tumour emboli, bacterial endocarditis, IV air, and amniotic fluid emboli at childbirth may occasionally be responsible for paradoxical embolization. The importance of a patent foramen ovale (PFO) as a cause of stroke is debated, particularly because they are present in ~15% of the general population. Some studies have suggested that the risk is only elevated in the presence of a coexisting atrial septal aneurysm. [6]

Conclusion:

The presence of a venous source of embolus, most commonly a deep venous thrombus, may provide confirmation of the importance of a PFO with an accompanying right-to-left shunt in a particular case. Three randomized trials of PFO occlusion for secondary prevention of ischemic stroke were negative, although each lacked sufficient power to be conclusive. [7]At present, there is no supportive evidence to offer percutaneous PFO closure for stroke prevention. Bacterial endocarditis can be a source of valvular vegetations that give rise to septic emboli. The appearance of multifocal symptoms and signs in a patient with stroke makes bacterial endocarditis more likely. Infarcts of microscopic size occur, and large septic infarcts may evolve into brain abscesses or cause haemorrhage into the infarct, which generally precludes use of anticoagulation or thrombolytics. Mycotic aneurysms caused by septic emboli may also present as SAH or intracerebral haemorrhage. [8]

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