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Review article – *Special issue: Acute Ischemic Stroke*

The role of echocardiography in patients after ischemic stroke

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ARTICLE INFO

Article history:
Received 2 December 2015
Received in revised form
6 February 2016
Accepted 9 February 2016
Available online 10 March 2016

Keywords:
Ischemic stroke
Echocardiography
Computed tomography
Magnetic resonance imaging
Cardioembolic sources
Prophylaxis

ABSTRACT

Therapy of acute phase of ischemic central vascular accident is focused on immediate actions to suppress the severity of damage with the earliest possible initiation of reperfusion strategy together with initiation and maintenance of adequate therapy to prevent further cerebral reinfarctions. The key factor for adequate and effective secondary prevention is elucidation of the etiology of ischemic central vascular accident because the risk of brain reinfarction is the highest in the first weeks after the primary event. The exclusion of potential cardiac or vascular sources of embolization into the cerebrovascular system is essential in choosing adequate secondary prevention. Origins of embolization are important to identify because they represent different thromboembolic risks. Transesophageal echocardiography is a frequently used diagnostic method after ischemic stroke. However, because of the variability in the frequency of cardiac findings between studies of similar populations, the lack of correlation between cardiac abnormalities thought to be associated with each other and cryptogenic stroke, it is unclear if routine use of echocardiography in patients with cryptogenic stroke should be recommended. Its routine use to elucidate the causes of stroke has a role in some patient groups, especially in young patients who present with cryptogenic stroke and no cardiovascular risk factors, as well as in the setting of a deep venous thrombosis, and older patients with a suspicion for structural heart disease or left ventricular or left atrial thrombus.

Contents

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http://dx.doi.org/10.1016/j.crvasa.2016.02.003

The essential prerequisite for adequate and effective secondary prevention is recognition of a cause of ischemic cerebrovascular accident (CVA) because the highest risk of repetition of cerebral ischemic event is during the first few weeks after the primary CVA.

Therapy of an acute phase of CVA is focused on immediate actions to limit further brain damage with the earliest possible initiation of reperfusion strategy. The next immediate step is to initiate secondary prevention. It is to establish an adequate pharmacotherapy to prevent further cerebrovascular accidents.

A search for possible origins of thromboembolism is an integral part of management of ischemic stroke [1,2]. Current published data suggest the need for basic categorization of possible origins of thromboembolism according to the presumed risk of a tromboembolic event. There are high risk origins as well as the intermediate and low risk origins (Table 1) [3,4].

Left atrium thrombi

(***incidence**: high*, ***thromboembolic risk**: high*, ***prophylaxis**: pharmacotherapy*)

Atrial fibrillation (AF) is the most frequent arrhythmia with exponentially increasing prevalence with age and in individuals with structurally damaged heart. Prevalence of atrial fibrillation in non-selected population is estimated in rather broad estimated range of 1–6%. In the seventh decennium aged patients, the AF prevalence reaches 5%. The octogenarians' population carries 10% AF prevalence. More than two thirds of all atrial cases represent patients between 65 and 85 year of age and the median of age of a patient with AF is set at age of 75. AF is more frequent in men than in women, though men represent roughly a half of all atrial fibrillation patients due to their shorter life span. Atrial flutter is the second most common supraventricular arrhythmia and also has age-dependent incidence which oscillates from 0.005% in patients aged 50 years or younger to 0.59% in patients above the age of 80. Atrial flutter is 2.5 times more frequent in men than in women and again occurs more frequently in patients with structural heart disease or in patients suffering from chronic obstructive pulmonary disease. Of importance, there is no difference in risk of thromboembolic event in between permanent and paroxysmal forms of both AF and atrial flutter. The risk of formation of thrombi in left atrium or left atrial appendage is virtually identical (Figs. 1 and 2).

Table 1 – Cardio-embolic sources.

Prophylaxis/ incidence	Medium-high	Low
Anticoagulation	Atrial flutter/ fibrillation	Thrombi in the left ventricle and on left-sided heart valves Spontaneous echocontrast Prosthetic mechanical valves
Non-pharmacological	Patent foramen ovale, septal defects	Persistent left superior vena cava Intracardiac tumors Infective endiocarditis Pulmonary arteriovenous malformations

There is well established relation between AF and ischemic cerebrovascular accidents which represent about 85% of all thromboembolic events in patients with AF. Vice versa, AF is considered to be a cause of 16% of all ischemic cerebrovascular accidents. Ten percent of ischemic cerebrovascular accident sufferers have left atrium thrombi found in the left atrium. It represents the two thirds of all patients suffering ischemic stroke due to AF. AF is a cause of almost 80% of all thromboembolic cerebrovascular accidents (CVA). AF is associated with five times higher risk of CVA, two times higher risk of death (due to CVA or heart failure) and finally it is associated with higher risk of vascular cognitive deficit in comparison to healthy controls. Ischemic cerebrovascular accidents due to AF are associated with the worse functional outcomes and higher risk of death in comparison to other causes of ischemic stroke. It can be explained by more frequent closures of major brain vessels together with older age and higher burden of other major comorbidities in this cohort. The risk of cerebrovascular accident in patients with AF progressively increases with age. There is 1–5% risk of CVA in 50–59 year old cohort and on the other hand up to 23–25% risk in cohort of 80 year old and above. The annual risk of stroke in patients suffering of AF is 2% but in the presence of other risk factors the risk climbs above 10%. The most significant risk factor is a prior cerebrovascular accident or transient ischemic attack (relative risk [RR] 2.5) followed by older age (RR 1.4, increasing by 1.3–1.6 with a decade), arterial hypertension (RR 1.9), systolic BP $>$ 160 mmHg (RR 1.4) and diabetes (RR 1.7).

The annual incidence of thromboembolic complications in patients with AF without the prophylaxis is 6% of average (span of 1.9–12.3%/year). Metaanalysis of studies focusing on