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ORIGINAL ARTICLE

Evaluation of cerebral blood flow in acute ischemic stroke patients with atrial fibrillation: A sonographic study

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transcranial color-coded duplex sonography

Background/purpose: Although cerebral emboli are a frequent cause of cardiogenic stroke, the possibility of a reduction in cerebral perfusion consequent to arrhythmia or impaired cardiac function should be considered in patients with atrial fibrillation (AF).
Methods: We reviewed sonographic studies and clinical features of patients with acute ischemic stroke. A total of 144 patients with AF and 144 age- and sex-matched patients with small vessel occlusion but without AF were included.
Results: Patients with AF had significantly lower peak systolic velocity (PSV), mean velocity, flow volume ($p < 0.001$), and end-diastolic velocity ($p = 0.035$) of the internal carotid artery (ICA); significantly lower cerebral blood flow ($p < 0.001$); and lower flow velocities of the middle cerebral artery ($p < 0.01$) than patients with small vessel occlusion but without AF. In patients with AF, there was an inverse linear correlation between ICA end-diastolic velocity, mean velocity ($p < 0.001$), flow volume ($p = 0.025$), middle cerebral artery flow velocities ($p < 0.05$), and age. Cardiac ejection fraction had a positive linear correlation with ICA PSV ($p = 0.016$) but an inverse correlation with the heart rate ($p = 0.009$). There was a significant decline in PSV ($p = 0.002$), resistance index ($p < 0.001$), and flow volume ($p = 0.0121$) of the ICA as well as cerebral blood flow ($p = 0.009$) as the heart rate increased.

Conflicts of interest: The authors have no conflicts of interest relevant to this article.

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Conclusion: Cerebral blood flow is markedly reduced in ischemic stroke patients with AF as compared with that in patients with small vessel disease but without AF.

Introduction

Atrial fibrillation (AF) is the most frequent cardiac disease associated with stroke.[1] Although the formation of thrombi within the auricle is the predominating factor in the genesis of cerebral ischemia in patients with AF, a number of other factors contribute to unstable cerebral hemodynamics. Carotid artery occlusive disease, hypertension, diabetes, and hyperlipidemia have been reported to be important comorbidities in patients with AF.[2] Reduced cerebral blood flow (CBF) as a result of arrhythmia or impaired cardiac function due to AF may also play a role in the genesis of cerebral ischemia.

Acute cardiac arrhythmias can reduce cardiac output to a degree where the cerebral circulation is compromised and neurological manifestations may appear.[3–5] Widely used methods for the assessment of CBF include xenon-enhanced computed tomography (CT), positron emission tomography, single-photon emission CT, and magnetic resonance imaging.[6,7] However, each of these methods has disadvantages, and not all are available in all hospitals. Recent studies have described the use of sonographic techniques to estimate CBF by measuring flow volume and velocities.[8–10] Color-coded carotid duplex sonography (CCD) demonstrates the diameter of the extracranial cervical arteries, allowing for the measurement of blood flow velocities and flow volumes (FVs) of the cervical arteries. Transcranial color-coded duplex sonography (TCCS) provides real-time two-dimensional depiction of cerebral parenchyma, vascular structures, and flow velocities of intracranial basal cerebral arteries. In this study, we aimed to use CCD and TCCS to evaluate the possible differences of cerebral hemodynamics between patients with AF and those with small vessel disease but without AF during acute ischemic stroke.

Patients and methods

Patients who were treated for stroke in the neurological ward during the period January 2011 to December 2014 were retrospectively selected from the stroke registry database. Inclusion criteria included a diagnosis of acute ischemic stroke and electrocardiographic evidence of AF with further echocardiographic study. To avoid variable factors that might influence cerebral hemodynamics, patients in whom CCD revealed $\geq$50% extracranial carotid stenosis, and patients with severe cerebral infarction with a National Institute of Health Stroke Scale (NIHSS) score of $\geq$15, increased intracranial pressure from a large area of brain edema, and clinical evidence of shock (systolic blood pressure $\leq$90 mmHg) were excluded. The clinical features, CCD, TCCS, brain CT, transthoracic two-dimensional echocardiographic (2D ECHO) images, electrocardiograms, and functional outcomes (Barthel index and modified Rankin Scale) were reviewed. The comparison group comprised age- and sex-matched patients with small vessel occlusion but without AF (lacunar stroke) who were classified according to the classification of Trial of ORG 10172 in Acute Stroke Treatment.[11]

All patients received CCD, TCCS, and 2D ECHO scans within 1 week of admission. All the CCD and TCCS images were obtained with a Philips IU22 duplex ultrasound system (Philips Healthcare, Bothell, WA, USA), with a 3–9 MHz transducer combining real-time B-mode imaging and a pulsed Doppler for CCD and a 2 MHz transducer for TCCS studies. We measured the peak systolic velocity (PSV), end-diastolic velocity (EDV), mean velocity (MV), resistance index (RI; RI = (PSV−EDV)/PSV), and FV (FV = time-averaged velocity $\times$ area $\times$ 60) of the middle or distal portion of the common carotid artery, internal carotid artery (ICA), and external carotid artery at about 2 cm distal to common carotid artery bifurcation, and the transverse portion of the vertebral artery of both sides, with the Doppler angle adjusted at or below 60°. The diameter of the vessel was measured by setting the calipers perpendicular to its course as the distance between the parallel walls of the vessel contrasted by color Doppler. At the same site, a sample volume was positioned to cover at least two-thirds of the entire luminal width. CBF was determined as the sum of the FVs of the ICA and the vertebral artery (VA) of both sides.[8,9] PSV, EDV, MV, and RI of bilateral middle cerebral arteries (MCAs) were measured in a TCCS study. A cardiac cycle with the highest peak and EDVs, which represents a more synchronized cardiac contraction and hence better cardiac output, was selected when measuring the velocities of arteries in patients with AF.[12] RI was used to evaluate flow resistance instead of pulsatility index to minimize the effect of a prolonged pause between cardiac contractions. Heart rate was recorded simultaneously during CCD and TCCS studies through the ultrasound system.

All patients received at least one brain CT during hospitalization. Ejection fraction (EF) was determined with cardiac 2D ECHO to clarify the relationship between cardiac function and CBF. The clinical features and risk factors were obtained from a detailed review of the medical records.

Differences in means of continuous variables were tested by the two-sample *t* test. Linear regression was performed to evaluate the potential influence of age, EF, and heart rate on CBF. The regression plot was constructed using STATVIEW software (SAS institute Inc., NC, USA). A p value of $<$0.05 was considered to indicate statistical significance. All statistical analyses were computed using the