Ultrasound in Med. & Biol., Vol. ■, No. ■, pp. 1–8, 2016
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0301-5629/$ - see front matter

http://dx.doi.org/10.1016/j.ultrasmedbio.2016.07.027

● *Original Contribution*

TOTAL CEREBRAL BLOOD FLOW IN PATIENTS WITH CARDIOEMBOLIC STROKE: IS IT CLINICALLY MEANINGFUL?

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(*Received* 23 *April* 2016; *revised* 20 *July* 2016; *in final form* 31 *July* 2016)

Abstract—Chronic hypoperfusion may hinder the washout of emboli coming from the heart and facilitate the formation of intra-cavitary thrombi. We investigated whether a decreased total cerebral blood flow (tCBF) resulted in recurrence of stroke and other vascular events in consecutive patients with cardioembolic stroke. We excluded patients with extra-cranial carotid or vertebral stenosis. The recorded tCBF was the sum of blood flow in both the carotid and vertebral extra-cranial arteries as measured with ultrasonography. Patients were followed up to assess stroke recurrence, vascular events and mortality. We also recorded demographic data, vascular risk factors, treatment data, echocardiographic variables and the C congestive heart failure history H Hypertension history A Age D Diabetes S Sex S2 Stroke/TIA/Thromboembolism history Vasc Vascular Disease history (CHA_2DS_2-VASc) score. We studied 79 patients (age 77.9 ± 8.4 y). Mean tCBF was 65.5 ± 15.7 mL/100 g/min. Cox regression analysis found that CHA_2 DS_2-VASc score and ejection fraction were associated with tCBF. After a mean follow-up of 22 ± 8.5 mo, 7.6% of patients experienced a recurrent stroke, 12.7% experienced a vascular event and 21.5% of patients died. Clinical outcomes were not predicted by tCBF. (E-mail: jmarti@santpau.cat) © **2016 World Federation for Ultrasound in Medicine & Biology.**

Key Words: **Acute stroke, Cerebral blood flow measurement, Cardiac embolism, Transcranial Doppler, Ultrasonography, Neurosonology, Atrial fibrillation, Prognosis.**

INTRODUCTION

About 20% of all ischemic strokes are attributable to a cardiac embolism. Atrial fibrillation (AF) is the leading cause of cardiac embolism. It is responsible for at least 50% of all cardioembolic strokes. Oral anti-coagulants (OA) are very effective in reducing the risk of cardioembolic stroke and systemic embolism (Hart et al. 2007; Ruff et al. 2014). However, trials of secondary prevention (Hart et al. 2007) reported an annual stroke recurrence risk of 3.9% in patients receiving OA and 12.3% in those not receiving OA. Studies outside of clinical trials found the rate of recurrent cardioembolic stroke to be 13.7% at 1 y (Petty et al. 2000) and 22% at 2 y (Kolominsky-Rabas et al. 2001) of follow-up.

An important cause of recurrence in cardioembolic stroke is the underuse of OA and the narrow therapeutic index of OA. Several other variables, such as age and previous ischemic stroke, have been identified as predictors of recurrence. The C congestive heart failure history H Hypertension history A Age D Diabetes S Sex S2 Stroke/TIA/Thromboembolism history Vasc Vascular Disease history (CHA_2 DS_2-VASc) score (Lip et al. 2010; Stroke Risk in Atrial Fibrillation Working Group 2008), which combines information from demographic data and vascular risk factors, is one method of determining risk stratification in patients with AF.

The generally accepted mechanism of cardiac embolism is a cardiac thrombus that migrates to the brain. Hypoperfusion secondary to reduced cardiac output or to other causes may also be a mechanism or a complementary mechanism of stroke (Caplan and Hennerici 1998; Caplan et al. 2006). Chronic hypoperfusion may hinder the washout of emboli coming from the heart due to a low-velocity cerebral blood flow (CBF). It may

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also facilitate the formation of intra-cavitary thrombi due to cardiac stasis (Lip and Gibbs 1999). Some studies indirectly support these hypotheses (de Bruijn et al. 2015; Georgiadis et al. 2000; Jung et al. 2010; Porebska et al. 2007; Pullicino et al. 2001). One study (Sabayan et al. 2013) found that low total CBF (tCBF) in older patients was associated with a higher risk of mortality. To our knowledge, however, the clinical relevance of reduced tCBF has not been adequately investigated in patients with cardioembolic stroke. Several studies (Dörfler et al. 2000; Scheel et al. 2000; Schöning et al. 1994) have reported that bedside color-coded duplex sonography provides a reliable method for the evaluation of tCBF and that tCBF values are comparable with those obtained with other techniques.

In a proof-of-concept prospective study, we have non-invasively measured tCBF in patients with cardioembolic stroke using ultrasonography. Our aim was to evaluate the variables associated with tCBF and to test the hypothesis that a decreased tCBF increases the risk of stroke recurrence and other vascular events during follow-up.

MATERIAL AND METHODS

The ethics committee approved our study, and patients or their legal representatives signed a written consent form to participate.

Patients

We studied patients with acute ischemic stroke who were admitted to the hospital's neurology department prospectively and consecutively. The inclusion criteria required (i) a cardioembolic etiology with a demonstrable high-risk cardiac source of embolism according to the Stop Stroke Study - The Trial of Org 10172 in Acute Stroke Treatment (SSS-TOAST) criteria (Ay et al. 2005); and (ii) a tCBF measure during admission. Exclusion criteria were as follows:

1. Extracranial stenosis of ≥50% in any internal carotid or vertebral artery (which may make tCBF measurement unreliable). This criterion was fulfilled when peak systolic velocity was >120 cm/s, and it was confirmed by computed tomography angiography or magnetic resonance angiography. If an intra-cranial stenosis was detected in addition to the cardioembolic source, the patient was not excluded.
2. Technical difficulties occurred in the tCBF measurements (high carotid bifurcation, vessel tortuosity).
3 The patient was non-cooperative (*eg,* agitation, dyspnea).
4. Follow-up was not possible.
5. Etiology was other than cardioembolic stroke.

Measurement of tCBF

The measurement of tCBF was performed during admission according to the methodology published in previous studies (Dörfler et al. 2000; Scheel et al. 2000; Schöning et al. 1994). The measurement was taken while the patient was in the supine position after at least 10 min of rest and with the head not elevated. We used a 7.0 MHz linear transducer connected to a color duplex ultrasonography apparatus (Aplio Toshiba; Toshiba America Medical Systems, Inc., Tustin, CA, USA).

To measure flow from one internal carotid artery (ICA), the head of the patient was slightly rotated to the opposite side. The flow was measured at least 1.5 cm distal to the carotid bifurcation in a longitudinal examination. Flow from both vertebral arteries (VA) was measured in the V2 (foraminal) segment with the head in a neutral position or rotated <10 degrees.

The volume sample was placed to cover the entire width of the lumen of the arterial segment with velocity measurement adjusted to the angle of insonation, which must be $\leq 60^{\circ}$. The blood flow of each artery was calculated from the time-averaged velocity and the diameter-based estimation of the cross-sectional area of the artery. Time-averaged velocities were measured over a minimum of four cardiac cycles. CBF in each artery was calculated three times, and the final value was the mean of the three values for each of the four arteries (left and right ICA and VA). The tCBF was the sum of the 4 measured flows, and the value was divided by 1300 (average adult brain weight) and then multiplied by 100 to express the value in mL/100 g/min. In a sample of patients (n = 10), we evaluated the reproducibility of the results by comparing the tCBF obtained by two experienced neurologists. Figure 1 shows an example of the results of the tCBF measurement. In patients with a good temporal acoustic windows, we also measured the velocity of both middle cerebral arteries (MCA) at a depth of 50 mm.

Follow-up

A neurologist unaware of the results of the tCBF measurement obtained the follow-up data. Follow-ups were scheduled at 1, 6, 12, 18 and 24 mo after the index stroke. In patients unable to attend a face-to-face visit, the required information was obtained by telephone from the patient or a caregiver. The clinical events assessed were (i) ischemic stroke recurrence (defined as the sudden onset of a focal neurologic deficit in a location consistent with the territory of a major cerebral artery and either a neuroimaging confirmation or a reliable clinical history assessed by a neurologist), (ii) vascular event (any diagnosis of ischemic or hemorrhagic stroke, myocardial infarction, aortic dissection, acute peripheral limb ischemia, acute mesenteric ischemia and sudden death), (iii) vascular death (sudden death or death after any vascular event), (iv) death from a

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