Cryptogenic stroke is a frustrating diagnosis. For the patient who is looking for explanations, for the family members worrying and caring for their loved one, and, perhaps most of all, for the physician trained to localize, identify, and prevent a recurrent stroke. With a large proportion of ischemic strokes (20–30%) still classified as having no definite cause, this diagnostic scenario happens all too often at the bedside. Atrial fibrillation (AF) is a well-established cause of many cardioembolic strokes, but may not always be readily detectable and frequently goes under-recognized by the patient. Occult AF could also explain a proportion of cryptogenic infarcts. However, initiating a treatment before knowing the diagnosis is much like putting the cart ahead of the horse.

The case in point describes a 76-year old woman who suffered a recent embolic-looking occipital stroke with intermittent palpitations and other vascular risk factors. Potential causes of her stroke other than cardioembolic also need to be strongly considered. We are assuming that vertebral artery origins were carefully studied, as they do account for up to 20% of posterior circulation strokes and are often poorly visualized or not imaged during a diagnostic workup. Her clean aortic arch and lack of any intracranial stenosis also helps decrease the probability of atherosclerotic mechanisms. Other rare causes of stroke (vasospasm, hypercoagulable, and autoimmune etiologies) may also need to be considered but are less frequent at this age.

The patient’s intermittent palpitations hint that occult AF could be present, however cannot be construed as making a diagnosis of AF. The majority of intermittent AF is clinically silent, and the unreliability of symptoms in diagnosing AF has been well-documented. In 1 study, fewer than one-third (32%) of the palpitation symptoms corresponded to AF, with a greater percentage (39%) being in sinus rhythm. The patient deserves a careful and thorough cardiac event monitoring. Occult AF has been found more frequently among cryptogenic stroke patients with prolonged ambulatory cardiac monitoring. Longer cardiac monitoring has higher yields, with 60% of AF detected >1 month after the start of monitoring and paroxysmal AF often occurring as rarely as 1 in 10 days. Genomic analysis and biomarker studies have recently identified subpopulations at higher risk for AF, offering a future possibility of screening. Thus, it is reasonable to recommend longer monitoring with an implantable loop recorder, if the initial 30-day period fails to reveal AF, especially if our patient has genetic or serologic susceptibilities indicating a higher likelihood of AF.

We would favor initiating antiplatelet therapy to prevent a recurrence while we gather more definitive information about the presence of AF. Subjecting her to the bleeding risks associated with any oral anticoagulants, new or old, would be risky in the absence of a definitive cardioembolic source. The bleeding risks in this patient with peptic ulcer disease are increased and also need to be addressed. Even antiplatelet therapy will need to be monitored closely, and the proper treatment of her underlying gastrointestinal disease needs to be initiated.

Assuming we later diagnosed AF, we would need to weigh the risks and benefits of oral anticoagulants. Based on her other comorbid features, her CHADS2 (Congestive Heart Failure, Hypertension, Age ≥75, Diabetes and Stroke/TIA) score is 5, which places her in a moderate risk group and corresponds to 12.5% yearly risk of stroke. Using oral anticoagulants would be the most effective way to reduce her chance of a recurrent stroke but also increase the risk of hemorrhage. Warfarin is only within range slightly more than 50% of the time in patients who do manage to take it regularly in a community setting. Moreover, her renal disease also complicates the decision process. Warfarin has been shown to be harder to dose appropriately and to have more hemorrhagic complications in patients with severe kidney disease. Recent clinical trials of dabigatran and rivaroxaban have excluded patients with CrCl <30 because of similar concerns and both drugs (dabigatran only at the 150mg dose) had statistically higher rates of gastrointestinal bleeding compared with warfarin. In our patient with CrCl of 30 and peptic ulcer disease, we would favor instituting a course of lower dose dabigatran or rivaroxaban after the diagnosis of AF was certain.

As stroke clinicians we strive to do our best to prevent a stroke recurrence, but we also must remember the words in our Hippocratic Oath: “I will follow that system of regimen which, according to my ability and judgment, I consider for...”

Received July 19, 2012; final revision received September 23, 2012; accepted September 26, 2012.

The opinions expressed in this article are not necessarily those of the editors or of the American Heart Association. This article is Part 2 of a 3-part article. Parts 1 and 3 appear on pages 298 and 302, respectively.

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(Stroke. 2013;44:300-301.)

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Stroke is available at http://stroke.ahajournals.org

DOI: 10.1161/STROKEAHA.112.658260
the benefit of my patients, and abstain from whatever is del-eterious.” While waiting for a definitive diagnosis of AF in this patient, the safest therapy includes a statin, an antiplatelet agent, and effective control of blood pressure and glucose. Although it may be tempting to attribute a cardiac source as the presumptive cause of an embolic-looking infarct in a patient with cryptogenic stroke, the risks of the therapy calls for a more certain diagnosis.

Disclosures

None.

References


Keywords: atrial fibrillation ■ anticoagulation ■ warfarin
Stroke Patients With Suspected Atrial Fibrillation Should NOT Be Started on Anticoagulation WHILE AWAITING the Results of Long-Term Cardiac Monitoring

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Stroke. 2013;44:300-301; originally published online December 13, 2012; doi: 10.1161/STROKEAHA.112.658260

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