

Neurointerventional Therapies for Stroke in Atrial Fibrillation Illustrated Cases

Amit B. Sharma, MD^a, Enoch B. Lule, MD^a,
Anmar Razak, MD^b, Syed I. Hussain, MD^b,
Shalini Sharma, MD^c, Peerawut Deeprasertkul, MD^a,
Ranjan K. Thakur, MD, MPH, MBA, FHRS^{a,*}

KEYWORDS

• Stroke • Atrial fibrillation • Ischemic stroke • Intracranial hemorrhage • Thromboembolism

KEY POINTS

- Stroke is the leading cause of disability in adults and the most serious complication of atrial fibrillation (AF). Strokes may be ischemic (80%) or hemorrhagic (20%). AF is the leading cause of cardioembolic strokes; these strokes are caused by large thromboemboli and are more disabling.
- Distinction between ischemic and hemorrhagic strokes at the time of presentation is critical because approaches to treatment are different. This distinction can be made rapidly with a noncontrast computed tomography scan.
- The goal of treatment of acute ischemic stroke is reperfusion of ischemic brain tissue, whereas the treatment of hemorrhagic stroke is supportive therapy and correction of the underlying condition that may have led to bleeding.
- The treatment of acute ischemic strokes is similar to treatment of acute myocardial infarction, which requires timely reperfusion for optimal results. The main reperfusion strategies include intravenous and/or intra-arterial thrombolysis, mechanical thrombectomy, and (rarely) angioplasty and/or stent.

INTRODUCTION

Approximately 800,000 strokes occur in the United States every year, resulting in 200,000 deaths, making stroke the leading cause of disability in adults.^{1,2} In general, ischemia accounts for 80% of acute strokes and the remainder are hemorrhagic events.^{1,2} Early distinction between these types of strokes is critical because the approaches to treatment are different. The goal for acute ischemic stroke is reperfusion of ischemic brain

tissue, whereas the treatment of hemorrhagic stroke is supportive therapy and correction of the underlying conditions that may have led to bleeding, such as uncontrolled hypertension, reversal of anticoagulation, ruptured aneurysms, and bleeding into an ischemic stroke. Treatment may be complicated by concomitant occurrence of ischemia and hemorrhage; an initial ischemic event may lead to secondary hemorrhage and vice versa. In these cases, treatment is prioritized

Disclosures: None.

^a Sparrow Thoracic and Cardiovascular Institute, Michigan State University, 1200 East Michigan Avenue, Lansing, MI 48912, USA; ^b Department of Neurology and Ophthalmology, Michigan State University, Lansing, MI, USA;

^c Department of Radiology, Michigan State University, Lansing, MI, USA

* Corresponding author. Sparrow Thoracic and Cardiovascular Institute, 1200 East Michigan Avenue, Suite 580, Lansing, MI 48912.

E-mail address: thakur@msu.edu

Card Electrophysiol Clin 6 (2014) 169–180

<http://dx.doi.org/10.1016/j.ccep.2013.10.004>

1877-9182/14/\$ – see front matter © 2014 Elsevier Inc. All rights reserved.

to the management of hemorrhage and supportive therapy for the ischemic component.

Acute ischemic strokes may be thrombotic or embolic. Acute embolic strokes are usually caused by thromboembolism from the heart or atheroembolism from the aorta, carotid or vertebral arteries. Emboli from the heart (cardioembolism), account for 10% to 30% of all strokes.³ Strokes caused by atrial fibrillation (AF) are generally ischemic events (ischemia may be complicated by secondary hemorrhage) caused by cardiogenic thromboembolism and are the most serious complication of AF, in part because these strokes are larger and more disabling.^{4,5} The treatment of acute ischemic strokes is similar to treatment of acute myocardial infarction (AMI), which requires timely reperfusion for optimal results. Like AMI, reperfusion strategies include intravenous (IV) and/or intra-arterial thrombolysis and catheter-based therapies. However, unlike AMI, not many patients with stroke receive reperfusion therapy because of late presentation (>4.5 hours from symptom onset), few qualified hospitals and specialists, and unavailability of rapidly accessible advanced neuroimaging facilities in most hospitals. Even if an intervention is possible, reasons for not intervening include small vessel occlusion (lacunar stroke), transient ischemic attacks (TIA), end-of-life strokes, and mild strokes outweighing the risks of intervention. As a result of these factors, few neurointerventional procedures are performed for acute strokes.

Interventional therapies for acute stroke include IV and/or intra-arterial thrombolysis, mechanical thrombectomy, angioplasty, and stent. The following cases are examples of the use of these therapies in patients with strokes caused by AF.

CASE 1

A 76-year-old woman with a history of hypertension and hyperlipidemia (CHADS₂, 2; CHA₂DS₂-Vasc, 4) was brought to the hospital with slurred speech, left facial droop, and right-sided weakness 5 hours after onset of symptoms. Initial head computed tomography (CT) was normal (Fig. 1). Electrocardiogram (ECG) showed AF with rapid ventricular rate at 160/min. Duration of AF was unknown and she was asymptomatic with respect to AF. She was not on any anticoagulation therapy. Rate control was achieved with IV diltiazem.

A diagnosis of acute right middle cerebral artery (MCA) ischemic stroke was made. On the National Institutes of Health Stroke Scale (NIHSS), the stroke severity was graded to be 15. CT

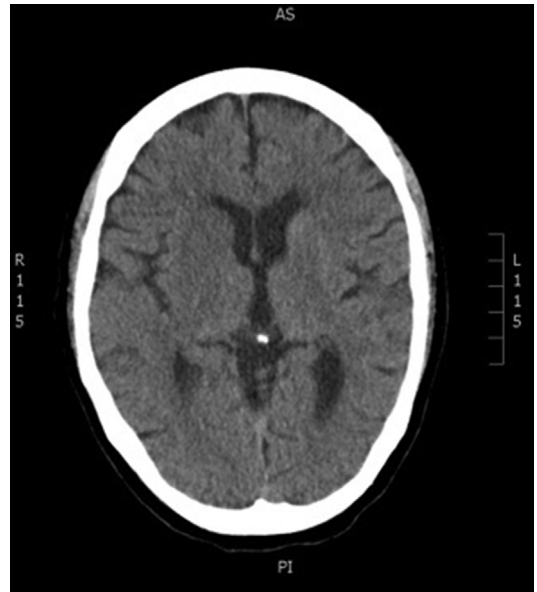


Fig. 1. CT of the head on presentation of the patient in case 1 shows no abnormalities.

angiography (CTA) of head and neck showed acute occlusion in the right MCA. CT perfusion showed mismatch between mean transit time and cerebral blood volume, indicating penumbra in the right MCA area.

She was out of the window of the IV thrombolysis and it was decided to take her to the catheterization laboratory for mechanical thrombectomy. Right MCA occlusion was shown with Thrombolysis in Cerebral Infarction (TICI) flow graded as 0 (see Fig. 2); this was treated with Trevo stent retriever along with aspiration using a Penumbra System, Neuron 5MAX aspiration catheter (Fig. 3). After 1 pass, the clot was retrieved. End result with angiography showed TICI 2a perfusion with persistent small M3 superior division branch occlusion (Fig. 4). Follow-up CT showed hemorrhagic transformation of infarct in the right basal ganglia including the head of the right caudate nucleus with moderate mass effect on the right lateral ventricle (Fig. 5).

She continued to have left-sided neglect, right gaze preference, and weakness in upper and lower extremities, although primarily in the left upper extremity. Oral intake was also poor, so a percutaneous endoscopic gastrostomy tube was placed and she continued to receive oral diltiazem. On the fourth day, she spontaneously converted to sinus rhythm.

With physical therapy, she was able to stand with assistance but not ambulate. She had a high blood lactate dehydrogenase level and a renal

Download English Version:

<https://daneshyari.com/en/article/2897138>

Download Persian Version:

<https://daneshyari.com/article/2897138>

[Daneshyari.com](https://daneshyari.com)