Endovascular intervention for acute stroke due to infective endocarditis

Case report

HAITHAM DABABNEH, M.D.,1 V. SHUSHRUTHA HEDNA, M.D.,1 JENNA FORD, B.Sc.,1
ZIAD TAIMEH, M.D.,2 KEITH PETERS, M.D.,2 J MOCO, M.D., M.S.,2
AND MICHAEL F. WATERS, M.D., PH.D.1

Departments of 1Neurology, 2Neurosurgery, and 4Radiology, University of Florida/Shands Hospital,
Gainesville, Florida; and 4Department of Medicine, University of Louisville Health Care Center, Louisville,
Kentucky

The overall incidence of neurological complications due to infective endocarditis is as high as 40%, with embolic infarcts more common than hemorrhagic strokes. The standard of care for typical strokes does not apply to infective endocarditis because there is a substantial risk of hemorrhage with thrombolysis. In the last decade there have been multiple case reports of intravenous and intraarterial thrombolysis with successful outcomes for acute strokes with related infective endocarditis, but successful endovascular interventions for acute strokes associated with infective endocarditis are rarely reported. To the authors’ knowledge, this report is the first case in the literature to use a mechanical retrieval device in successful vegetation retrieval in an infective endocarditis acute stroke. Although an interventional approach for treatment of acute stroke related to infective endocarditis is a promising option, it is controversial and a cautious clinical decision should be made on a case-by-case basis. The authors conclude that this approach can be tested in a case series with matched controls, because this condition is rare and a randomized clinical trial is not a realistic option. (http://thejns.org/doi/abs/10.3171/2011.11.FOCUS11263)

KEY WORDS • middle cerebral artery • infective endocarditis • ischemic penumbra • interventional endovascular procedure • penumbra retrieval device

Abbreviations used in this paper: MCA = middle cerebral artery; tPA = tissue plasminogen activator.

Beginning as a subtle feature such as unexplained prolonged fever, untreated infective endocarditis may end up as unstable vegetation producing infective emboli in major blood vessels supplying the brain, kidney, and other vital organs. More than one-third of cases of infective endocarditis are associated with neurological complications such as embolization, hemorrhagic conversion of infarct, cerebral mycotic aneurysm, meningitis, brain abscess, and seizures.14 Death due to stroke that is related to infective endocarditis is substantially higher when compared with other strokes.19 Recently the incidence of infective endocarditis has increased due to the vast number of cardiac procedures performed, and so has the secondary CNS complications.5 The diagnosis of infective endocarditis is based on a constellation of clinical features taking into consideration a detailed medical history, physical examination, blood culture, laboratory results, and imaging.1 The most accepted criteria used for making the diagnosis of infective endocarditis are the modified criteria of Duke.15 Even though neurological deficits are the same, standard stroke treatment guidelines do not apply when treating stroke related to infective endocarditis in view of the high risk of hemorrhagic conversion of infarct, mycotic aneurysm formation, and underlying infection. Thus treatment is based on treating the infection with a prolonged course of antibiotics and supportive care. Intravenous thrombolytics are contraindicated in acute situations, necessitating clinicians find alternate methods to address the embolized clot or vegetation.13,21 In the literature there are multiple case reports of using intravenous tPA, intraarterial tPA, and even endovascular interventions in stroke related to infective endocarditis, which embolden us to attempt interventional options for treating this otherwise devastating condition.

Case Report

History and Examination. This 67-year-old woman had a history significant for a bovine mitral valve replaced 6 months prior, experienced a methicillin-resistant Staphy-
Lococcus aureus line infection in the postoperative period, completed antibiotic treatment with no complications, and experienced intermittent atrial fibrillation on warfarin. She was admitted to the hospital for a 1-day duration of right-sided weakness, loss of appetite, and lethargy for 1 week. On admission she was found to have altered sensorium, thrombocytopenia, fever (38.8°C), atrial fibrillation (ventricular rate 107 beats per minute), and mild renal insufficiency. An examination revealed she was stuporous, had dysarthric speech, right-sided facial droop, right-sided tongue deviation, and right hemiparesis. A sensation and cerebellar system examination was not performed because of the patient’s clinical condition.

Magnetic resonance imaging results were consistent with an infarct of the left posterior inferior cerebellar artery and multiple small punctate infarcts involving the posterior left temporal lobe, occipital lobe, and left precentral gyrus. A CT angiogram of the head and neck did not reveal intra- or extracranial vessel occlusion. This multiple vascular distribution raised the concern for a thromboembolic source. With the above clinical presentation, medical history, and examination, a suspicion of infective endocarditis was at the top of the differential diagnosis. The patient was not a candidate for tPA treatment given the time between the onset of the symptoms and presentation, possibility of infective endocarditis, and other risk factors. A transthoracic echocardiogram showed an ejection fraction of 30%–35%, moderate left ventricular systolic dysfunction, and mitral valve bioprosthesis with multiple elongated highly mobile masses extending into the left ventricle during diastole, likely vegetations. Blood cultures were growing Gram-negative vancomycin-resistant rods and the patient was started on antibiotics. Her right-sided weakness was improving greatly compared with admission. However, 1 week later while the patient was an inpatient she developed acute right-sided body weakness, global aphasia, and forced left gaze deviation with examination consisting of the flaccid right upper and lower extremity and a brain CT angiogram (Fig. 1), which showed a distal M1 cutoff by either a thrombus or vegetation. After careful review of the case we decided to perform endovascular intervention to prevent poor neurological outcome from this large vessel occlusion and contraindication to use tPA.

Before the endovascular procedure was performed, a head CT angiogram with perfusion (Aquilion ONE Toshiba scanner) showed 2 hypodense lesions involving the left cerebellum and the left higher cerebral convexity involving the MCA, with no early signs of infarction or bleeding. As noted, the CT angiogram showed a left MCA occlusion between the M1 and M2 segments (Fig. 1). The CT perfusion scan showed increased time to peak (Fig. 2A) in the areas supplied by the left MCA, with increased cerebral blood volume (Fig. 2B), a focal area of decreased volume, and an area of increased mean transit time (Fig. 2C), indicating low volume infarct and a large area of tissue at risk; Figure 2D shows cerebral blood flow for comparison. The following day MR imaging showed the area of restriction was significantly decreased compared with the volume of brain tissue loss, due to infarction. The patient was showing some improvement but a week later developed sudden shortness of breath and relative hypoxemia, concerning for a pulmonary embolism. The patient and family requested “do not resuscitate” status and did not want any more interventions, therefore she was transferred to hospice care.

Operation and Postoperative Course. A conventional angiogram-directed Penumbra retrieval device (Penumbra Inc.) was used to gain access by canalizing the femoral artery with a 6 Fr sheath, and a 90-cm guiding catheter was used and advanced via the left internal carotid artery to the M1 segment, where the thrombus was evacuated. The aspiration catheter, along with a separator, was used to debulk and remove the thrombus. Control cerebral angiograms were obtained every 3–4 minutes per manufacturer recommendations to monitor the progression of the aspiration and to reposition the reperfusion catheter to the new thrombus interface. Recanalization was established and Thrombolysis in Cerebral Infarction scores of 2 or 3 of the left MCA and its major branches were recorded. The following day MR imaging showed the area of restriction was significantly decreased compared with the volume of brain tissue loss, due to infarction. The patient was showing some improvement but a week later developed sudden shortness of breath and relative hypoxemia, concerning for a pulmonary embolism. The patient and family requested “do not resuscitate” status and did not want any more interventions, therefore she was transferred to hospice care.

Discussion
Infective endocarditis, the infection of the inner layer of the heart valves, is most commonly caused by the Streptococcus viridans group and S. aureus. In advanced cases when antibiotic treatment is not initiated in a timely fashion, unstable infective vegetations frequently develop, which exhibit a high propensity to embolize into the blood stream causing ischemic events in the brain,
Endovascular treatment for stroke due to infective endocarditis

heart, lungs, and intestines. Intracranial hemorrhage occurs in 5% of patients with infective endocarditis, either due to hemorrhagic conversion of acute ischemic strokes or the formation of mycotic aneurysms. Generally, treatment with intravenous thrombolysis up to 4.5 hours from symptom onset has become the standard of care in acute ischemic strokes. To date, there has been much debate on how to treat ischemic embolic strokes from infective endocarditis. Administration of tPA to patients with acute ischemic stroke and infective endocarditis is not well documented and therefore not considered safe by the American Heart Association, Stroke Council, and American Stroke Association. For instance, although the use of thrombolytic therapy in patients with infective endocarditis has been reported without hemorrhagic complications, many cases show the use of tPA for acute ischemic stroke significantly increases the risk of bleeding. In a report by Bhuvu et al., 3 patients suffering from acute ischemic stroke due to infective endocarditis developed multifocal intracranial hemorrhages. Intracranial hemorrhage has also been reported in patients receiving intravenous tPA for myocardial infarction due to infective endocarditis. Another option is administration of recombinant prourokinase up to 6 hours after the onset of symptoms, but this treatment has not been approved by the US FDA. Therefore, a more conservative approach is treatment of the blood infection and the complications thereafter once they develop, as has been the policy used in infective endocarditis-associated acute ischemic stroke.

In the past few years, the use of interventional endovascular methods in treating infective endocarditis–associated acute ischemic stroke has not been extensively reported nor implemented. Several cases initially addressed the use of endovascular intervention to treat mycotic aneurysms resulting from infective endocarditis, but only a few cases have been reported using interventional endovascular methods in infective endocarditis–associated ischemic strokes, including catheter-guided intraarterial tPA. In this article we present a potential option in the treatment of infective endocarditis-associated acute ischemic stroke: the successful use of an endovascular method in a case in which thrombolytic therapy was not an option. Using a Penumbra retrieval device, we were able to remove the clot and decrease the volume of brain tissue loss due to infarction, without significantly increasing the patient’s risk of hemorrhage. From these results, it appears that endovascular intervention can provide a promising treatment for stroke patients who are not eligible for thrombolytic therapy. These results need further validation for safety and effectiveness, but this initial study is promising.

Disclosure

Dr. Peters serves on the speakers bureau for Toshiba America Medical Systems. Dr. Mocco received research support from ev3; has served as a consultant to Concentric and Actelion; serves on the advisory board of Lazarus Effect, Edge Therapeutics, and NFocus; and has ownership in Codman Neurovascular.

Author contributions to the study and manuscript preparation include the following. Conception and design: Waters, Dababneh, Ford, Taimeh, Mocco. Acquisition of data: Waters, Dababneh, Ford, Taimeh. Analysis and interpretation of data: Waters, Dababneh, Ford, Taimeh, Mocco. Drafting the article: Waters, Dababneh, Ford. Critically revising the article: Dababneh, Hedna, Ford, Peters, Mocco. Reviewed submitted version of manuscript: Waters, Dababneh, Ford, Taimeh, Peters, Mocco. Approved the final version of the manuscript on behalf of all authors: Waters. Statistical analysis: Waters, Dababneh, Ford, Mocco. Administrative/technical/material support: Waters, Dababneh, Ford, Mocco. Study supervision: Waters, Dababneh, Hedna, Ford, Peters, Mocco.

References


H. Dababneh et al.

Manuscript submitted September 27, 2011. Accepted November 8, 2011. Please include this information when citing this paper: DOI: 10.3171/2011.11.FOCUS11263.
Address correspondence to: Michael F. Waters, M.D., Ph.D., Department of Neurology, University of Florida, College of Medicine, HSC Box 100236, Gainesville, Florida 32610. email: mwaters@neurology.ufl.edu.