Recurrent Transient Ischemic Attacks as the Initial Presenting Manifestation of Type A Aortic Dissection

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A case is reported of an 84-year-old woman with recurrent episodes of aphasia and right-sided weakness with spontaneous complete resolution associated with hypotension. She subsequently developed lower gastrointestinal bleeding, ischemic toes, and anuria without associated chest pain. An emergent transesophageal echocardiography showed a type A aortic dissection with aortic valve insufficiency and tamponade. Emergent thoracotomy was performed, which confirmed a severe acute type A aortic dissection. This is a reported case of acute painless aortic dissection presenting initially as recurrent transient ischemic attacks. The etiology of focal neurologic deficits should be crucially determined by an experienced clinician prior to thrombolytic administration. This case illustrates the importance of aortic disease in the etiology of acute stroke syndromes.

Aortic artery diseases involving the ascending and the arch portion have been implicated in stroke, most commonly due to atherosclerotic disease and subsequent thromboembolization. Rarely has aortic dissection been implicated in the initial evaluation of cerebrovascular disease victims. Stroke occurs in about 3% of type A aortic dissection\(^1\) and less commonly as the initial presentation.\(^2,3\) Documented cases are accompanied by chest pain or hemodynamic instability, and neurological presentations include cerebral or spinal cord infarction, hypoxic encephalopathy, and ischemic neuropathy.\(^4-6\) A case is presented of a patient with recurrent transient ischemic attacks as an initial presenting manifestation of type A aortic artery dissection. This case stresses the importance of considering proximal aortic disease as an etiology of acute ischemic neurologic deficits.

CASE REPORT

We present an 84-year-old right handed woman with a history of rheumatic heart disease with aortic valve stenosis, osteoarthritis, mild chronic renal insufficiency, and diverticulosis who experienced three episodes of transient aphasia and right-sided weakness in 2 hr. She denied any acute chest or back pain. Axial computerized tomography (CT) scan of the head was normal. Thrombolytics and anticoagulation were not administered, given her improving neurologic status.

Physical examination revealed a blood pressure of 76/40 mmHg, heart rate of 80/min, a loud aortic end-diastolic murmur, and positive hemocult stools without palpable rectal lesions. Neurological evaluation during an episode showed nonfluent aphasia and right hemiparesis involving mainly the face and arm that resolved completely within 10 min. Hemodynamic parameters were maintained and the patient was transferred to the neu-
DISCUSSION

With the introduction of tissue plasminogen activator in acute stroke care, timely and accurate diagnosis of focal neurologic deficits is invaluable to avoid inadvertent use with possible lethal consequences. Painless aortic dissection has been reported in less than 10% of cases, and can create a diagnostic dilemma. Cerebral ischemia may be the presenting feature of aortic dissection, but the presence of other clinical signs and symptoms would direct clinicians to the diagnosis. Unusual aspects of this case include the lack of chest pain and the recurrent transient ischemic attacks in the anterior left middle cerebral artery distribution.

Transient ischemic attacks have been rarely reported as the initial presenting manifestation of aortic dissections, so its relationship to outcome is unknown. Different possible mechanisms have been entertained. Cerebral hypoperfusion due to hemodynamic compromise and subsequent watershed infarcts, diffuse generalized ischemia, or focal infarct can occur if stenotic vascular disease is present intra- or extracranially. Acute occlusion or stenosis of common carotid artery by dissection extension, and artery-to-artery or cardiac embolization have also been implicated as a possible mechanisms. In our patient, on the basis of the preoperative evaluation and intraoperative findings, we believe that the most likely etiology is recurrent embolization, either cardiac or artery-to-artery.

Standard conventional angiography and, more recently, magnetic resonance angiography imaging are considered the gold standard for diagnosis, provided the patient is stable enough to undergo those procedures. Transthoracic echocardiography has an advantage of easy accessibility, can be done at the bedside even on critically ill patients, and allows for rapid cardiac function evaluation. Moreover, it provides accurate diagnosis with a sensitivity of 100% and specificity of 98%. Conventional angiography was not performed in our patient because of the acute renal failure and hemodynamic instability. Noninvasive diagnostic tools were utilized rapidly to provide a diagnosis and aid in decision making in this case.

The poor outcome in this case was probably not related to the initial neurologic presentation, but the severity of the type A aortic dissection. The mortality rate is variable, but as a general rule, it increases with extent and severity of the dissection, and with time. The mortality rate may range between an average of 13% within the first 12 hr to 74% in the first 2 weeks, and may be reduced by