Successful Treatment of Basilar Artery Thrombosis with Both Heparin and Tissue Plasminogen Activator in the Setting of Traumatic Vertebral Artery Dissection

Victor M. Erlich, PhD, MD, and David W. Newell, MD

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Tissue plasminogen activator (tPA), effective in the treatment of acute ischemic stroke when administered within 3 hours of the onset of symptoms,[1] has not been well-studied in strokes associated with serious trauma (including arterial dissection) and/or seizure. Patients with these problems were specifically excluded from the controlled, randomized studies that demonstrated the efficacy of tPA. An uncontrolled study of 11 patients whose internal carotid artery dissections were treated with tPA produced no serious complications,[2] but experience in this realm remains limited. Furthermore, standard use of tPA for acute stroke excludes the concomitant use of heparin. We report a case of severe basilar artery thrombosis from a traumatic vertebral artery dissection, accompanied by seizure, successfully treated with both heparin and tPA.

CASE REPORT

A 27-year-old man with a remote history of peptic ulcer disease fell while skiing, suffering persistent headache and neck pain radiating into the right arm. Four days later, he developed dysarthria and ataxia at 8:30 AM. As his wife drove him to the hospital, he suffered a convulsion. After recovering consciousness, he arrived in the emergency room with quadriparesis and dysarthria. An unenhanced computed tomographic scan of the brain showed a dense basilar artery (Fig. 1*A*). The patient abruptly became mute and quadriplegic. At 11:15 AM, the patient was unresponsive, with spontaneous decerebrate posturing. Pupils were fixed at 4 mm, and oculovestibular responses were absent. The patient was immediately given 5,600 units of heparin for presumed traumatic vertebral artery dissection with consequent basilar artery thrombosis. The patient awoke but was unable to move his limbs or mouth. To command, his right eye tracked right as the left eye bobbed slightly up and down, and his attempt at left gaze produced only bobbing in the left eye. At 11:30 AM, the patient was given intravenous tPA (0.9 mm/kg 10% as a bolus, and the remainder over 1 hour). After the bolus and 15 minutes of infusion, the patient sat up, virtually normal, neurologically. At the end of the infusion, he developed a left internuclear ophthalmoplegia and was given 2,000 additional units of heparin, with resolution of the dysconjugate gaze. At 1:30 PM, a magnetic resonance angiogram (MRA) showed a right vertebral artery dissection (Fig. 1*B*) and persistent, severe obstruction of flow in the basilar artery, mainly proximally (Fig. 1*C*). The patient was placed on a heparin drip and warfarin was started. For the next 24 hours, the patient experienced loss of the right visual field when he sat up. An MRA 3 months later showed a normal vertebrobasilar system and no infarct. The patient is now working full time and has made a full recovery.

DISCUSSION

Basilar artery thrombosis with ominous long-tract and brain stem signs carries a high morbidity regardless of what action is chosen. In this case, stopping treatment after the administration of heparin appeared likely to leave the patient locked-in. Intra-arterial thrombolysis would have required at least an hour to set up and would have entailed cannulating the only good vertebral artery. Once heparin was tried (given precisely because tPA is not accepted treatment for stroke in this setting and because heparin is the standard treatment for dissection), tPA became all the more problematic. However, features of this case made the use of tPA seem less a risk than might first appear. The patient's injured vertebral artery had low flow in it and thus seemed less likely to rupture, his basilar artery was intrinsically healthy and it seemed that it might tolerate extreme anticoagulation, the circulation beyond the basilar could be presumed normal, and the patient's positive response to the preadministration of heparin suggested that a channel had been opened to allow tPA to work on the broad face of the clot rather than on its small proximal end.

Of course, consideration of these speculations could not eliminate the substantial risk of hemorrhagic complication, especially in the ischemic brain stem. The combination of heparin with tPA during intra-arterial fibrinolysis for basilar

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From the Department of Neurology, University of Washington, Stroke Unit, Northwest Hospital (V.M.E.), and Department of Neurological Surgery, Harborview Medical Center, University of Washington School of Medicine (D.W.N.), Seattle, Washington.

Address for reprints: David W. Newell, MD, Department of Neurological Surgery, 700 Ninth Avenue, Seattle, WA 98104; email: dwn@u.washington.edu.

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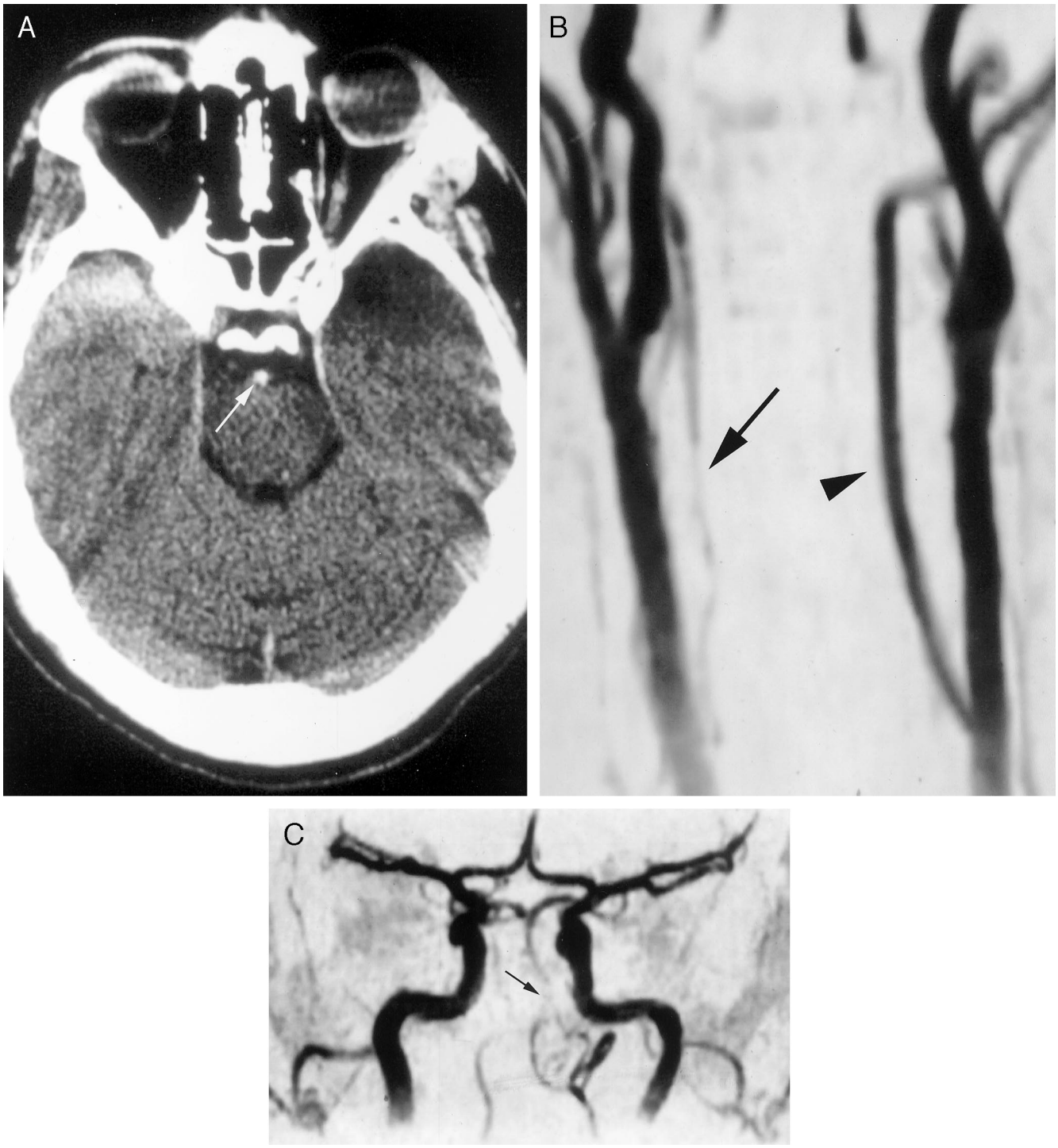

Fig. 1. *(*A*) CT scan showing a dense basilar artery (*arrow*). (*B*) MRA showing dissected right vertebral artery (*arrow*) and normal left vertebral artery (*arrowhead*). (*C*) MRA showing sluggish flow in the basilar artery, especially proximally (*arrow*).*

artery thrombosis can produce a high rate of hemorrhage,[3] and under certain (clinically nonapparent) concentrations of clotting factors, heparin can cause massive stimulation of tPA activity.[4] In contrast, the combination of heparin and tPA has shown promising results in the treatment of cerebral venous thrombosis.[5]

Although this single case cannot be taken as a general recommendation for the concomitant use of heparin and tPA or for the broad use of thrombolytics in cases of arterial dissection and seizure, it does suggest that in carefully chosen cases, aggressive treatment can be safe and effective in this setting. It also suggests that if the clinical picture is clear,

aggressive treatment need not be withheld until all possible tests are performed. In this case, the history, the physical examination, and the CT scan sufficed to make the diagnosis. A delay for an MRA might have led to the patient's demise.

This case also raises the difficult question of what the correct treatment should be for the patient with uncomplicated basilar artery thrombosis (i.e., no dissection) who is successfully treated with tPA under the standard protocol (i.e., no heparin) and then deteriorates. Should heparin be added in these "simpler" cases, given the possibility that heparin may help in basilar artery thrombosis if not in stroke in general? Also, in cases like the current one, what should be done in the presence of vertebral artery dissection, assuming tPA was used at the outset? With the patient suffering recurrent symptoms after his initial recovery, with his MRA showing substantial clot in the basilar artery an hour after the completion of tPA, and with his dissected vertebral presumably harboring more emboli, should the clinician depend on the ongoing activity of tPA over 8 hours and await the marginally improved results obtained 3 months after the use of tPA? Or should worsening symptoms prompt further treatment with heparin, assuming no hemorrhagic complication? Further experience is needed to answer these questions.

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