Intracerebral hemorrhage is the most serious and feared complication of both intravenous and intraarterial thrombolytic therapies. It occurs in about 6 to 10% of patients receiving thrombolytic therapies.1-3 Among the patients having thrombolytic therapies, intracerebral hemorrhage is associated with poor neurological outcomes compared to patients without bleeding. Several parameters such as fibrinogen level, systolic blood pressure, length of time between onset and treatment, and patient’s age are known risk factors for hemorrhagic complication after thrombolysis. We present a patient with complicated hemorrhage after thrombolytic therapies at the site of a prior microbleed, contralateral to the acute ischemic event.

CASE REPORT

A 60-year-old right-handed man was admitted with sudden left hemiparesis and dysarthria. His symptoms occurred 1 hour before arrival. He has been on anti-hypertensive and antidiabetic medication for 6 years. On admission, his blood pressure was 160/80 mmHg, heart beat was 72 /min, and blood glucose level was 72 mg/dL. Neurologic examination showed a slightly drowsy mental status with dysarthria, left hemiparesis with pathologic reflexes, central left facial nerve paresis, eyeball deviation to the right side and sensory extinction. National Institutes of Health Stroke Scale (NIHSS) score was 10 points. Brain computed tomography (CT) did not show any evidence of intracerebral hemorrhage and ischemia. He was treated with intravenous recombinant tissue plasminogen activator (tPA) according to the previously published protocol (0.9 mg/kg, 10% of bolus and 90% of continuous infusion over 1 hour).1 Brain magnetic resonance imaging (MRI) showed acute ischemic injury in the right middle cerebral artery (MCA) territory with diffusion–perfusion mismatch and multiple micro–
bleeds in both basal ganglia (Fig. 1–A, B), MRA revealed an occluded trunk of the right MCA. As the patient did not show any neurological improvement after intravenous tPA administration for 1 hour, we performed intraarterial thrombolysis 4 hours after the symptom onset. The right internal carotid angiogram showed occlusion in the MCA, a microcatheter (2.7F, Tracker–18, Target CMI) was placed immediately proximal to the occlusion site and 350,000 IU of urokinase was subsequently infused over a period of 1 hour. Complete recanalization was achieved 5 hours after the symptom onset, after that the patient showed 3 points reduction in NIHSS score and improved the consciousness. The highest blood pressure during intravenous/intra-arterial thrombolytic therapies was 170/90 mmHg and it was usually lower than 170/90 mmHg.

Thirty minutes after recanalization, patient had right hemiparesis (Medical Research Council grade IV) and drowsy mental status. Brain CT showed an intracerebral hemorrhage in the left basal ganglia confirmed by serial brain CT work up (initially checked after onset of right hemiparesis and followed 24 hours after intra–arterial thrombolysis) (Fig. 1–C). He showed partial recovery after stereotactic evacuation of hematoma.

**DISCUSSION**

We describe a case of intracranial hemorrhage (ICH) at the site of a prior microbleed, contralateral to the acute ischemic site in a patient treated with intravenous and intra-arterial thrombolytic therapies.

Currently known risk factors of ICH are inadequate to predict the individual risk for subsequent intracerebral hemorrhage among patients on thrombolytic therapies. Gradiant echo T2-weighted MRI is able to detect small deposits of hemosiderin in the brain, which is regarded as evidence of previous rupture of small vessels. These microbleeds are most commonly associated with microangiopathy due to hypertension, cerebral amyloid angiopathy, or prior ischemic injury. Despite the uncertainty of the underlying pathology, evidence of previous microbleeds may identify patients with a high risk of further hemorrhage from a diseased vessel. Especially, 20% of all symptomatic hemorrhages occurred outside of the vascular distribution of the presenting ischemic stroke. In our case, symptomatic hemorrhage occurred in the prior microbleed site after thrombolytic therapy, and is important as a risk factor of ICH. So, if these lesions do represent markers of bleeding-prone angiopathy and
increase risk of ICH after thrombolytic therapy, it may have clinical implications when treating stroke patients. However, there are few studies about the relationship between old microbleeds and ICH after thrombolytic therapy. We believe larger-scale studies are needed to evaluate if the presence of microbleeds is useful in predicting patients who are at a higher risk for intracerebral hemorrhage from thrombolysis or long-term anticoagulation therapy.

REFERENCES


