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Case Report

A Case of Intravascular Treatment for Acute Ischemic Stroke with Internal Carotid Artery Stenosis That Developed While Driving

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Objective: We report a case of acute cerebral infarction that may have been associated with high-energy trauma due to onset while driving.

Case Presentation: A 67-year-old man had a traffic accident. His neurological symptoms were left hemiplegia and contrast CT revealed right middle cerebral artery occlusion. Intravenous thrombolysis with recombinant tissue plasminogen activator (rt-PA) and intravascular treatment were performed. Right carotid artery angiography demonstrated internal carotid artery stenosis. Middle cerebral artery (MCA) revascularization was performed only by percutaneous transluminal angioplasty (PTA) of the internal carotid artery. Thoracic hemorrhage was observed a few hours after surgery, and hemostasis was performed by thoracotomy. Carotid artery stenting (CAS) was performed 8 days after onset. The patient was transferred to a convalescent rehabilitation hospital.

Conclusion: Rt-PA and acute CAS were not recommended for cerebral infarction due to traffic accident.

Keywords ▶ traffic accident, internal carotid stenosis, acute ischemic stroke, intervention

Introduction

The mean annual number of traffic accidents associated with attacks/acute diseases while driving is approximately 200–300. Of these, cerebrovascular disease accounts for approximately 20%–30%, and it is regarded as the most frequent cause of traffic accidents.[1,2] Recently, the number of patients with traffic-accident-associated cerebrovascular disorder was suggested to increase with an increase in the number of stroke patients. One study reported that stroke onset was while driving for 4% of stroke patients, and that it resulted in traffic accidents in 16% of cases.[3] Furthermore, it is difficult to differentiate extrinsic from intrinsic disease in many patients with sudden neurological symptoms while driving.

In acute ischemic stroke patients with trauma, it is hesitate to introduce antithrombotic therapy because of hemorrhage risk. Furthermore, few studies have reported the treatment of acute ischemic stroke that developed while driving.[4] We report a patient with acute ischemic stroke that led to a traffic accident in whom treatment was difficult due to intracranial artery occlusion complicated by internal carotid artery stenosis.

Case Presentation

A 67-year-old male had a traffic accident. Left hemiplegia was noted and a high-energy trauma-related intracranial lesion was suspected. He was brought to our hospital by ambulance 51 minutes after the accident. Physical findings on arrival: The blood pressure, pulse rate, respiratory rate, SpO2, Japan Coma Scale (JCS) score, and Glasgow Coma Scale (GCS) score were 126/78 mmHg, 94 beats/min, 20 times/min, 99%, I-3, and E4V4M6, respectively. The left and right pupillary diameters were both 2.5 mm. The

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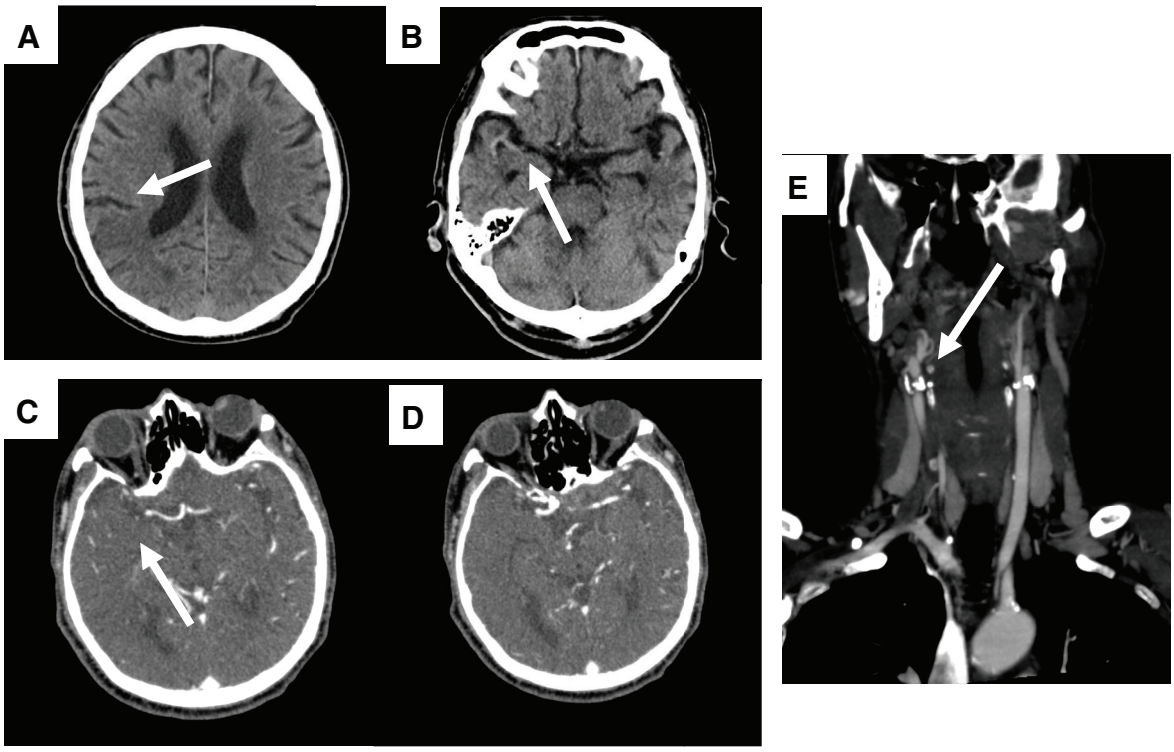

Fig. 1 CT images on admission. (**A** and **B**) Non-contrast CT showed early signs on the lateral and posterior cortices. (**C** and **D**) Contrast CT showed MCA occlusion. (**E**) Contrast CT showed severe stenosis of the internal carotid artery. MCA: middle cerebral artery

light reflex was brisk on the bilateral sides. The primary survey did not reveal abnormalities in the airway, respiration, or circulation. Regarding the central nervous system, consciousness disorder was noted. A secondary survey revealed conjugate deviation of the right eye, and paralysis of the left upper and lower limbs. CT demonstrated no hemorrhage lesion and the disappearance of the corti-co-medullary junctions of the right parietal and temporal lobes and a hyper-dense middle cerebral artery (MCA) (ASPECTS: 9 points) (**Fig. 1A–1C**). Contrast-enhanced CT of the entire body (trauma pan scan) demonstrated marked stenosis of the right cervical internal carotid artery and occlusion of the right MCA (**Fig. 1D–1G**). In the limbs/trunk, there was no trauma (**Fig. 2A–2C**). A tertiary survey revealed abrasion of the left lower thigh alone. These findings revealed cerebral infarction related to occlusion of the right MCA was made (National Institutes of Health Stroke Scale (NIHSS) score: 12 points). Intravenous thrombolysis with recombinant tissue plasminogen activator (rt-PA) was selected after consultation with the Department of Neurosurgery, Department of Emergency Care, Department of Radiology, and Department of Neurology, and was started 97 minutes after arrival.

Intraoperative findings: Under local anesthesia, a 9-Fr long sheath was inserted through the left femoral artery 99 minutes after arrival. A guiding catheter with a 9-Fr balloon was guided into the right common carotid artery. Ninety-five percent stenosis of the right internal carotid artery (normal vascular diameter: 6.0 mm) with reduced peripheral anterograde blood flow was observed (**Fig. 3A**), and intracranial angiography demonstrated M1 occlusion of the right MCA (**Fig. 3D**). Initially, to perform percutaneous transluminal angioplasty (PTA) for stenosis of the cervical internal carotid artery, pre-dilation was performed using a 3-mm PTA balloon after crossing a 0.014-inch microguidewire over the lesion, and a 4.5-mm PTA balloon was dilated to the RBP (**Fig. 3B**). Using the 0.014-inch microguidewire, a microcatheter was guided to the M2 area of the right MCA and microangiography was performed (**Fig. 2E**). A stent retriever was deployed and recanalization (thrombolysis in cerebral infarction [TICI] 3) was achieved 149 minutes after arrival (**Fig. 3F**). At this time, the end of the balloon guiding catheter was inserted to the lesion of cervical internal carotid artery stenosis, and the flow of the internal carotid artery was blocked with the dilated balloon in the common carotid artery. The stent

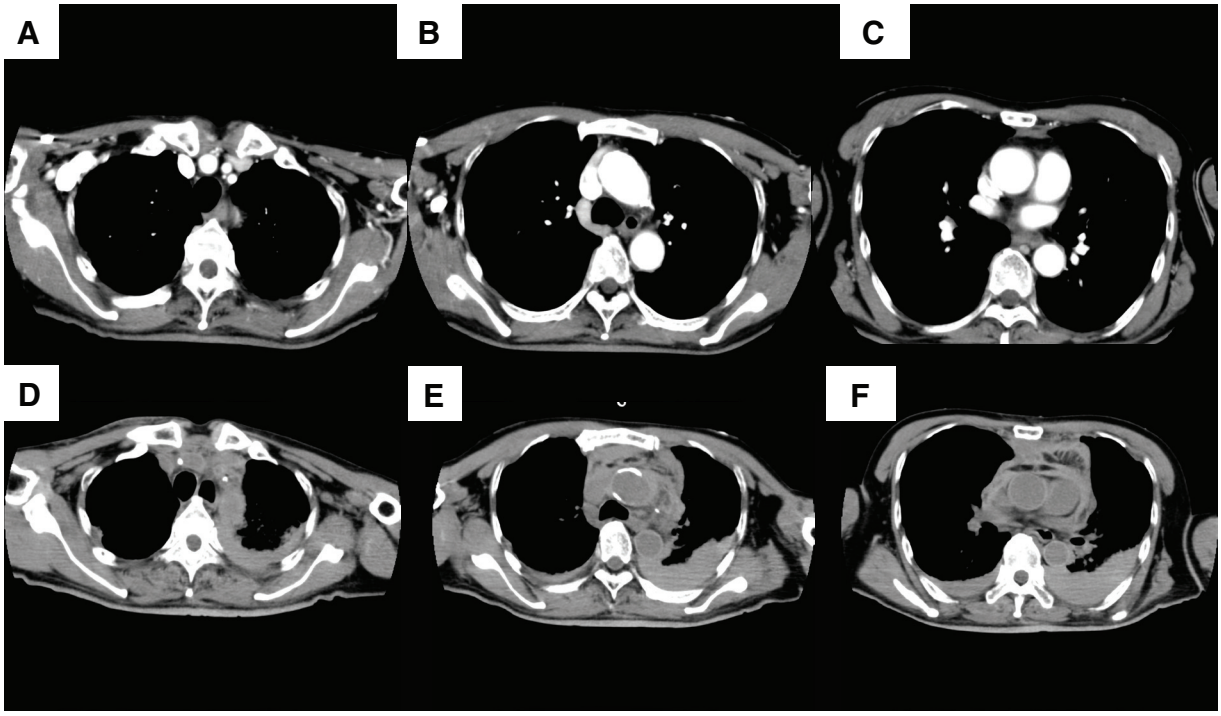

Fig. 2 (**A–C**) Contrast CT on admission did not suggest traumatic changes within the mediastinum. (**D–F**) Non-contrast CT scan after treatment showed hematoma in the mediastinum.

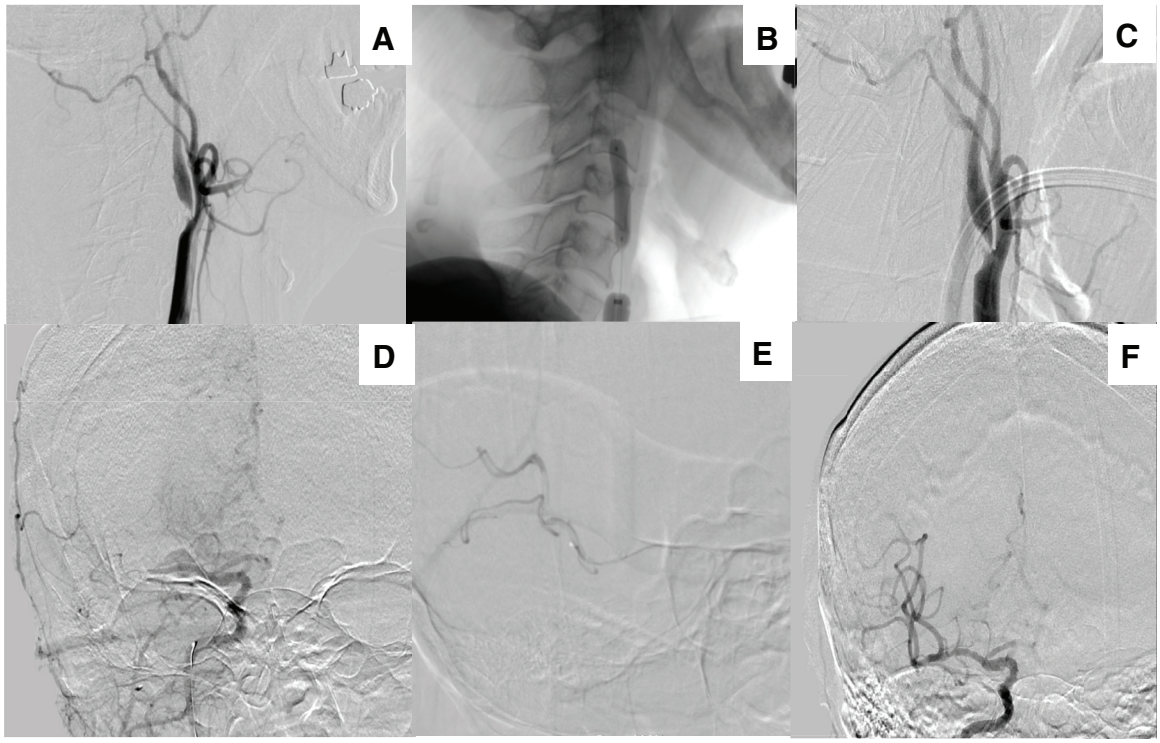

Fig. 3 (**A**) Right common carotid angiography revealed severe stenosis of the ICA lesion. (**B**) PTA was performed using a PTA balloon with proximal balloon protection. (**C**) The right internal carotid artery was slightly opened after PTA. (**D**) Pre-treatment right common carotid angiography demonstrated middle cerebral artery occlusion. (**E**) The micro-catheter passed through the thrombus in MCA. (**F**) Recanalization of the right MCA was achieved after treatment. Right common carotid angiography revealed severe recurrent stenosis of the ICA lesion after PTA. ICA: internal carotid artery; MCA: middle carotid artery; PTA: percutaneous transluminal angioplasty

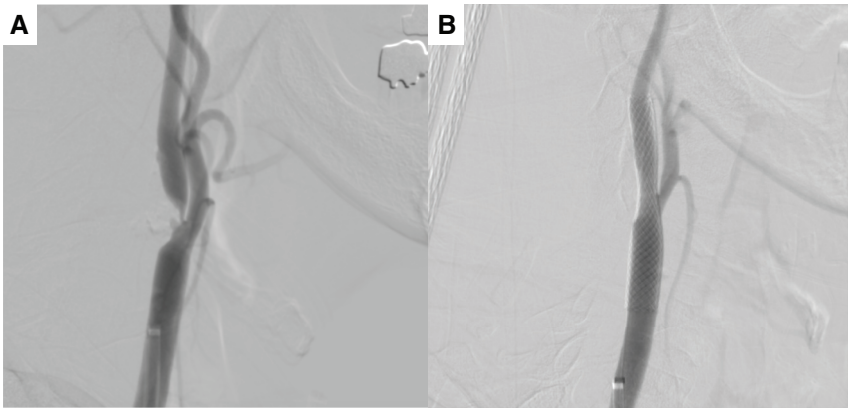

Fig. 4 (on the 8th hospital day) (**A**) Right CCA angiography showed the remaining severe stenosis before re-treatment. (**B**) The right ICA was opened by carotid artery stenting without stenosis. CCA: common carotid artery; ICA: internal carotid artery

retriever was pulled with syringe aspiration. Recanalization of the intracranial MCA was achieved, but restenosis of the cervical internal carotid artery was observed over time. Therefore, this artery was additionally dilated to the RBP using a 5.5-mm PTA balloon. This procedure was completed without performing carotid artery stenting (CAS), allowing marked residual stenosis because of favorable intracranial blood flow (**Fig. 3C**).

Postoperative course: Considering the risk of postoperative occlusion of carotid artery stenosis, heparinization was continued during/after surgery. After postoperative admission to the intensive care unit (ICU), there was a gradual decrease in blood pressure. The systolic blood pressure reached ≥70 mmHg and catecholamine administration was started. Although the hemoglobin (Hb) level on arrival was 12.6 mg/dL, it was 7.9 mg/dL at this point. Under a diagnosis of hemorrhagic shock, blood transfusion was initially started without reversing heparin considering the risk of carotid artery occlusion. Contrast-enhanced thoracic CT revealed mediastinal hemorrhage (**Fig. 2D–2F**), suggesting venous hemorrhage from the thymus. Under thoracotomy, hematoma removal and hemostasis were performed. The volume of intraoperative blood loss was 1310 mL. Concerning the volume of blood transfusion from shock until the completion of surgery, 10 units of an erythrocyte preparation, 10 units of a platelet preparation, and 10 units of a plasma preparation were administered. After surgery, the Hb level was 10.4 mg/dL; there was no progression of anemia, but an antiplatelet-drug- and anticoagulant-free period was required. The blood pressure was stable from 2 days after surgery and catecholamine was not required. The continuous administration of heparin at 10000 units was started to treat the carotid artery lesion after consultation with the Department of Cardiovascular Surgery. After extubation, his consciousness improved and amelioration of left hemiplegia was noted, with an NIHSS score of 4. The administration of antiplatelet drugs (cilostazol at 200 mg and aspirin at 100 mg) was started 5 days after onset. CAS for carotid artery stenosis was performed 8 days after onset (**Fig. 4A** and **4B**). After surgery, MRI revealed no new lesion (**Fig. 5A–5F**). The patient was referred to recovery-phase rehabilitation hospital 40 days after onset, with a modified Rankin Scale (mRS) score of 4.

Discussion

We treated a patient with acute ischemic stroke, cervical internal carotid artery stenosis, and suspected high-energy trauma. In our hospital, thrombectomy is performed through CT angiography alone considering the time without MRI. In the present case, contrast-enhanced CT for investigating the presence of trauma involving the entire body revealed occlusion of the MCA. Therefore, MRI was not performed and endovascular treatment was conducted after rt-PA administration. For primary care of trauma patients, focused assessment with sonography for trauma (FAST) is repeatedly performed, but if possible, echographic assessment of carotid artery lesions may also be useful. During endovascular treatment, angiography demonstrated marked cervical carotid artery stenosis and it is hard to obtain the dilation of carotid artery using PTA. However, PTA was completed without performing CAS considering the risk of hemorrhage related to antiplatelet drugs after stenting. As a result, mediastinal hemorrhage was observed after surgery, requiring the discontinuation of antithrombotic therapy. As etiological factors for hemorrhage,

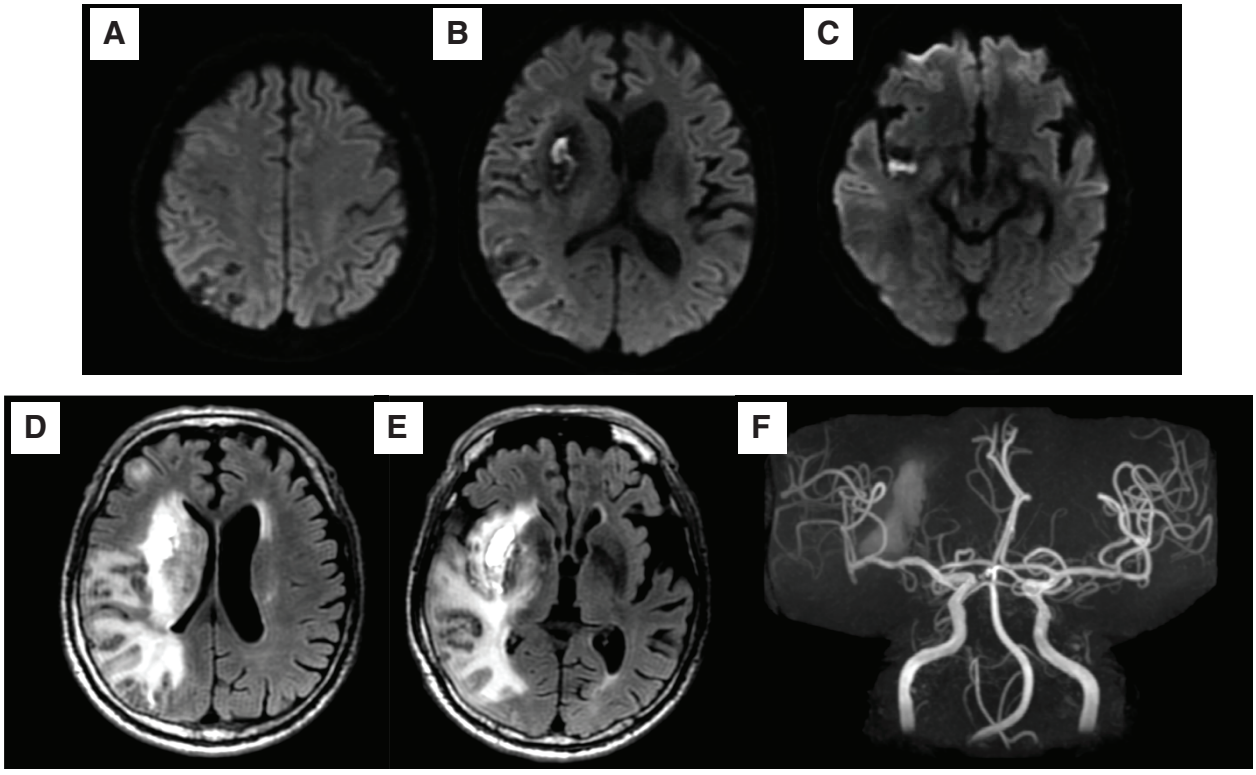

Fig. 5 (on the 12th hospital day) (**A–C:** DWI; **D** and **E**: FLAIR): Postoperative MRI demonstrated hemorrhagic infarction in right deep white matter. (**F**) Postoperative MRA demonstrated recanalization of the right MCA. DWI: diffusion weighted image; MCA: middle carotid artery

rt-PA and heparin administration during/after endovascular treatment were suggested. In the treatment of stroke with trauma, therapeutic strategies in consideration of the risk of antithrombotic drug discontinuation may be necessary. Few studies have reported the association between traffic trauma or falls at the onset of cerebral infarction and t-PA. To our knowledge, no study has reported this issue involving endovascular treatment. In this method, items for careful t-PA administration include trauma within 10 days, but there is no concrete definition about trauma; it depends on clinical evaluation. In the present case, traffic-accident-related high-energy trauma was suspected, although it was not clear. In the future, endovascular treatment alone in the absence of rt-PA administration should be considered in such cases.

Regarding acute ischemic stroke complicated by cervical carotid artery stenosis, which of two options, PTA alone and PTA + CAS, should be selected remains controversial. A previous study reported that the mortality rate after PTA was higher than that after CAS,[5] whereas another study suggested that the outcome was favorable.[6] However, after CAS, the oral administration of antiplatelet drugs is necessary, and the risk of cerebral hemorrhage related to treatment itself is approximately 0%–39%.[7–10] The outcomes were reported to be similar between the two procedures,[11] but several studies suggested that stenting should not be performed if peripheral cerebral blood flow is maintained by PTA alone in the acute phase considering the risk of hemorrhage related to oral antiplatelet drugs after treatment, and found that elective CAS or carotid endarterectomy (CEA) led to a favorable prognosis. However, these studies remain controversial.[8,9,12,13] In the present case, mediastinal hemorrhage developed after surgery, and the administration of antithrombotic drugs was discontinued. A stent was not inserted and stent occlusion was avoided. If a latent factor for trauma, such as a traffic accident, is present, CAS should be avoided considering the risk of antithrombotic drug discontinuation. If stenting is considered necessary despite the above circumstances, PTA alone should be performed for revascularization and stenting should be conducted after confirming the absence of trauma-related hemorrhage during a few hours.

Concerning CAS, which was performed 8 days after onset, CEA should also be considered for blood vessels that are not dilated on PTA. However, in our hospital, CAS with a closed cell stent is indicated for lesions other than circumferential calcification. In the present case, CAS was

also performed because relatively favorable results had been maintained.

Conclusion

We treated a patient who developed embolism of the right MCA with cervical internal carotid artery stenosis while driving. In acute ischemic stroke patients with high-energy trauma, such as traffic-accident-related trauma, antithrombotic therapy, including rt-PA administration, may cause unexpected complications even if there are no findings of trauma on arrival. In those with cervical carotid artery stenosis, the possibility that antithrombotic therapy is impossible after surgery must be considered.

Disclosure Statement

We declare no conflicts of interest regarding the content of this article.

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