Case Report

Literature Review and Case Report of Intravenous Thrombolysis in Acute Cerebral Infarction Attributed to Cervical Arterial Dissection

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Acute cerebral infarction (ACI) caused by cervical arterial dissection (CAD) is a rare clinical disease. Therapeutic approaches include anticoagulant therapy, antiplatelet aggregation, and thrombolytic therapy. Currently, anticoagulant therapy or antiplatelet aggregation is the primary choice, whereas the thrombolytic therapy is still controversial. In this article, we report a patient with ACI caused by right CAD, which led to a compensatory increase in blood supply to the right middle cerebral artery through the anterior communicating artery. After treatment with intravenous thrombolysis, the clinical symptoms of the patient improved, and the National Institutes of Health Stroke Scale (NIHSS) score declined to 2 points from the initial 14 points. In addition, cranial computed tomography scans showed that there were no signs of intracranial or extracranial hemorrhage, but that the vessel occlusion was still uncured. After 17 days of antiplatelet aggregation treatment, a cranial magnetic resonance angiography scan showed complete recanalization of the right internal carotid artery. Furthermore, the NIHSS score was reduced to 1 point when the patient discharged, and for 3 months of follow-up. **Key Words:** Intravenous thrombolysis—acute cerebral infarction—cervical arterial dissection—clinical case.

Cervical arterial dissection (CAD) is a common cause of ischemic stroke in young people although it is rare in the overall population. Management of ischemic stroke caused by CAD remains uncertain, and most studies have focused on the use of antiplatelet drugs or anticoagulation therapy. At present, it is internationally accepted that intravenous (IV) thrombolysis is the preferred treatment in acute cerebral infarction (ACI) within 4.5 hours. However, there are few clinical reports regarding the safety and outcome of thrombolysis treatment in CAD. In this article, the clinical data of a patient with ACI caused by CAD are retrospectively analyzed. Furthermore, some relevant literature in the field is reviewed.

Case Report

The patient was a 56-year-old man, presenting with a headache with paroxysmal inflexibility of left limbs and alalia. The patient started to experience a headache 3.5 hours before being hospitalized, with persistent pain mainly in the right occipitoparietal region. This was accompanied by loss of movement in the left limbs. Moreover, the patient could not walk or hold anything with his left hand and could only express himself verbally although slurring. The symptoms lasted for about 10 minutes and then eased. After half an hour, the symptoms above broke out again. The patient had no history of

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smoking, drinking, or trauma but had a 10-year history of high blood pressure with the highest blood pressure recorded at 185/105 mm Hg and had never received any formal treatment. The patient's first blood pressure reading after hospitalization was 182/103 mm Hg and was accompanied by drowsiness, dysarthria, a shallow left nasolabial groove, reduced muscle tone, level 2 left limb muscle, left hemihypalgesia, and a positive reflex of left pathologica. There were no other obvious positive signs.

An initial craniocerebral computed tomography (CT) scan (Fig 1) showed a low-density shadow of frontal and parietal on the right cerebral hemisphere. The leukocyte concentration in blood was 12.65×10^9 cells/L with a neutrophil and granulocyte content of 83.4%. Four blood coagulation tests were normal, as well as electrocardiogram readings.

The initial National Institutes of Health Stroke Scale (NIHSS) score was 14 points, which conformed to the IV thrombolysis standard. By consent of the patient's family, approximately 4 hours after attack, alteplase IV thrombolysis was carried out; 7 mg of IV injection was first implemented followed by the remaining 63 mg pumped over a 60-minute period. One hour later, the symptoms improved and the NIHSS score declined to 2 points. After that, a series of tests were carried out, including 0.15 PNA urinary kallidinogenase injection test, kallidinogenase, butylbenzene, and medications administered including edaravone, atorvastatin, and aspirin.

A CT scan performed the next day (Fig 2) showed a flaky low-density shadow in the right-side frontal lobe with an obscure boundary and indistinct sulcus. Brain magnetic resonance imaging (MRI; Figs 3 and 4) revealed ACI in the right-side temporal insula, occipital lobe, and basal ganglia regions. Furthermore, magnetic resonance angiography (MRA; Fig 5) showed that the middle cerebral artery (MCA) on the right side became slim, with a reduced number of branches. Moreover, the right internal carotid artery (ICA) was underdeveloped, and the initiation was broken (Fig 6). After approximately 2 days after arriving at the hospital, the patient started to develop further symptoms including profuse sweating, decreased flexibility in the left limbs, dysarthria, palsy of the left side of the eyes, with the right

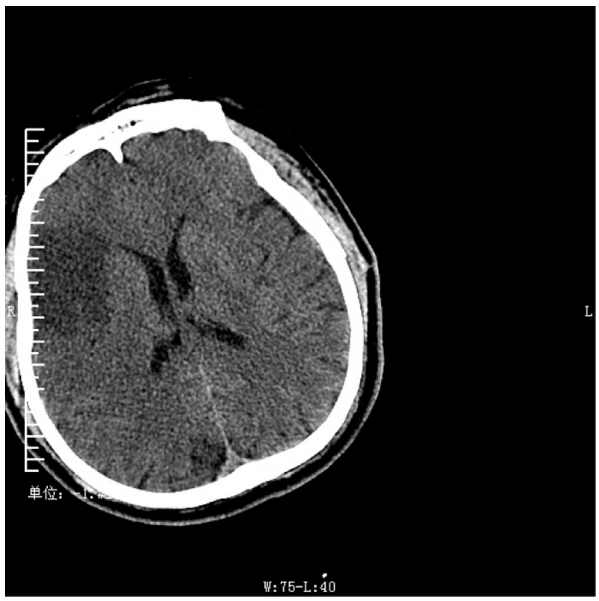

Figure 2. *Cranial computed tomography scans 15 hours after intravenous thrombolysis.*

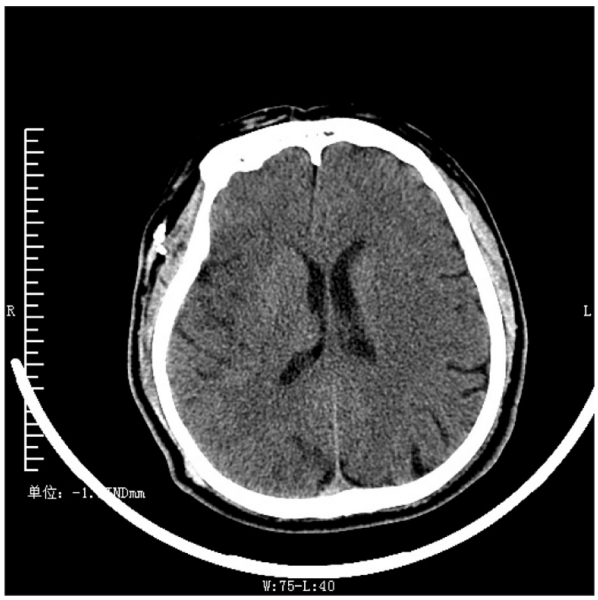

Figure 1. *Cranial computed tomography scans before intravenous thrombolysis.*

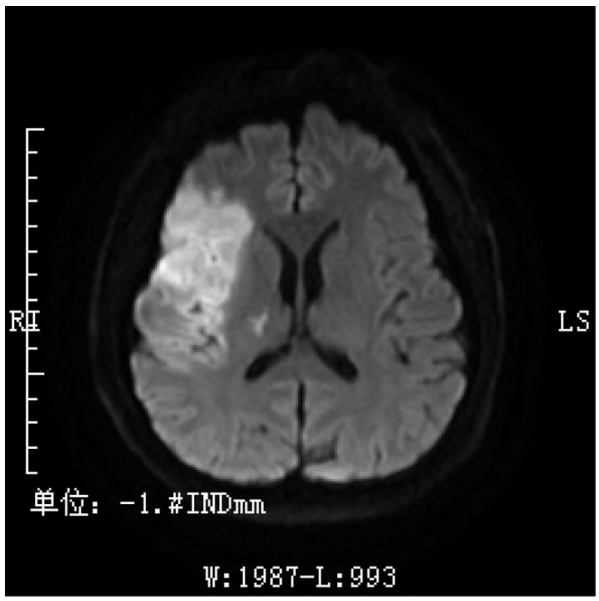

Figure 3. *Diffusion-weighted imaging of cranial MRI 20.5 hours after intravenous thrombolysis, which showed cerebral infarction. Abbreviation: MRI, magnetic resonance imaging.*