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Endovascular Treatment of Ischemic Stroke due to Acute Carotid Dissection: A Report and Review of the Literature

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Abstract

The efficacy of endovascular therapy in patients with acute ischemic stroke due to tandem occlusion is comparable to that for isolated intracranial occlusion in the anterior circulation. Definitive treatment of carotid dissection-related strokes is currently unproved. The best endovascular technique in this setting remains to be established, but emergency carotid artery stenting (CAS) is frequently considered. We investigated the safety and efficacy of emergency CAS for carotid dissection in patients with acute stroke in current clinical practice.

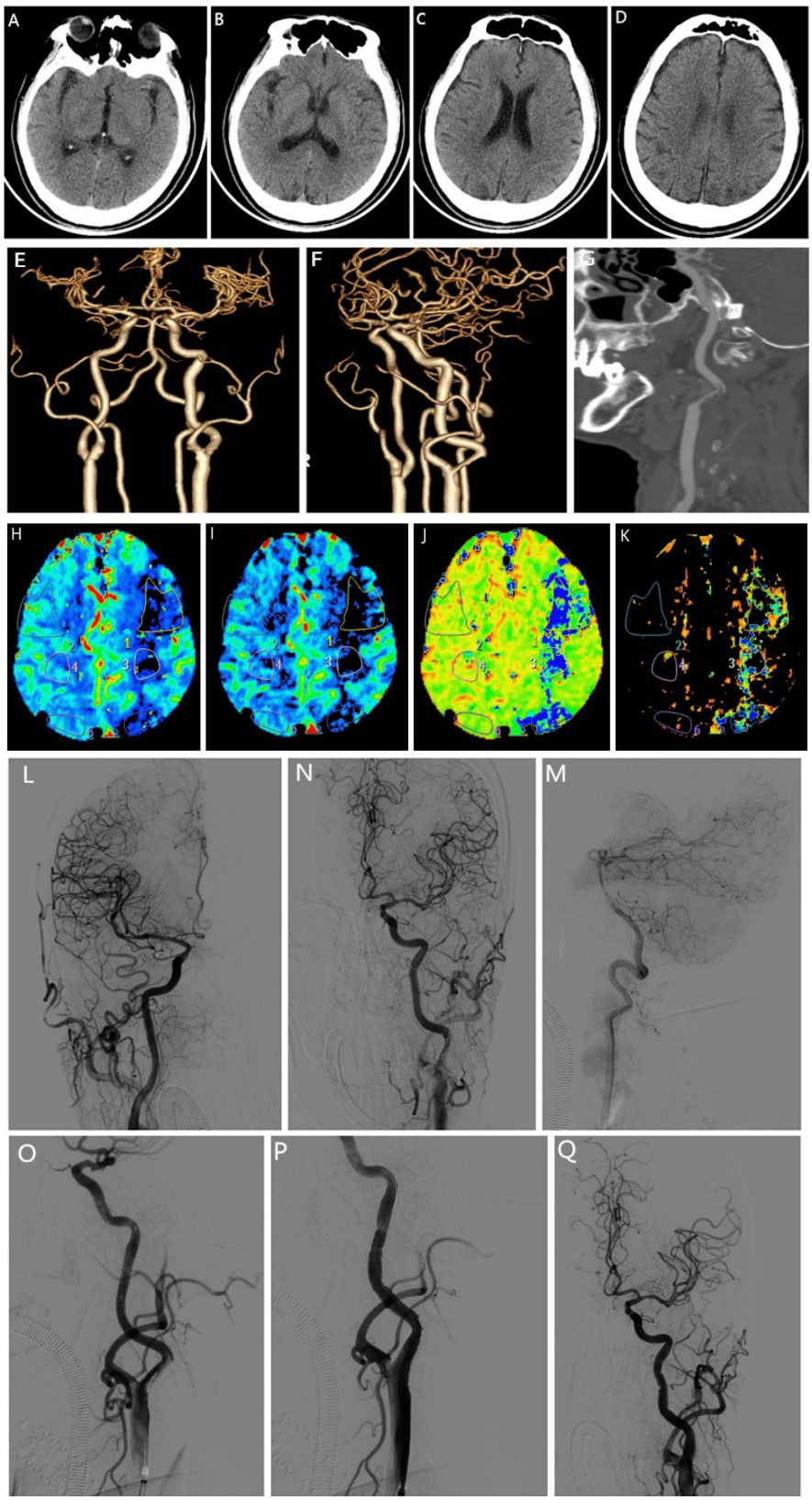
Keywords

Internal Carotid Artery, Dissection, Ischemic Stroke, Endovascular Therapy

1. Case Presentation

The patient, male, 74 years old, chief complaint: sudden right-sided limb movement disorder for about 3 hours; Specialty situation: patient with vague consciousness, nhiss score 17 points; cranial CT before surgery: no obvious intracranial hypodense area, aspects (Alberta stroke program early CT Score) score 10 points (**Figures 1(A)-(D)**). The pupils were 3 mm in diameter bilaterally; light reflexes were present; neck soft, left side muscle strength was normal; right side muscle strength was grade III; Babinski sign was positive in left lower extremity; Babinski sign of right lower extremity was negative. The head CTA examination report before the operation: mixed plaque in the tube wall of C1 segment of the left internal carotid artery, severe stenosis of the lumen (**Figures 1(E)-(G)**); CTP before surgery suggested: multiple patchy areas of relative hy-

poperfusion and some areas of hypoperfusion in the left frontoparietal occipital lobe (Figures 1(H)-(K)).



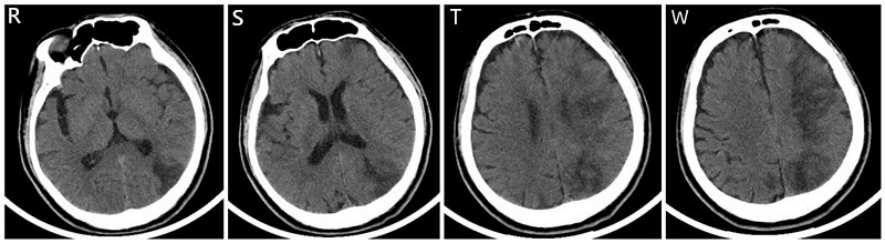


Figure 1. (A~D) no obvious intracranial hypodense area, aspects (Alberta stroke program early CT Score) score 10 points. (E~G) mixed plaque in the tube wall of C1 segment of the left internal carotid artery, severe stenosis of the lumen. (H~K) multiple patchy areas of relative hypoperfusion and some areas of hypoperfusion in the left frontoparietal occipital lobe. (L~M) severe stenosis of the C1 segment of the left internal carotid artery with dissection like changes, intracranial vessel flow was good, the anterior communicating artery was open, and the posterior communicating artery was not open. (O~Q) An Aculink7-9-40 stent was released in the C1 segment of the internal carotid artery to cover the entire diseased vessel, the vessel was well repaired, no new embolization occurred in the distal vessel. (R~W) the head CT was reviewed 2 weeks after the operation for scattered low-density shadow in the left frontotemporal parietal brain lobe.

2. Surgical Procedure

Emergency cerebral digital subtraction angiography suggested severe stenosis of the C1 segment of the left internal carotid artery with dissection like changes, intracranial vessel flow was good, the anterior communicating artery was open, and the posterior communicating artery was not open (**Figures 1(L)-(M)**). An 8F guiding catheter reached the end of the common carotid artery, a micro guidewire microcatheter was passed through the stenotic segment confirmed to be in the distal internal carotid artery, an Aculink7-9-40 stent was released in the C1 segment of the internal carotid artery to cover the entire diseased vessel, the vessel was well repaired, no new embolization occurred in the distal vessel (**Figures 1(O)-(Q)**), and the head CT was reviewed 2 weeks after the operation for scattered low-density shadow in the left frontotemporal parietal brain lobe (**Figures 1(R)-(W)**), and the patient's Mrs score was 2 points at 90 days after the operation.

3. Discussion

Carotid dissection, although an uncommon cause of stroke, is certainly one of the leading causes of ischemic stroke in young patients. Antithrombotic (antiplatelet aggregation or anticoagulation) drugs are the cornerstone of the treatment of carotid dissection, and after antithrombotic treatment, a large proportion of patients can obtain a relatively good prognosis in terms of clinical function and vascular imaging. However, in some special cases, an endovascular interventional approach is required to treat the carotid dissection.

The goal of intervention is to eliminate localized luminal abnormalities due to dissection such as: severe stenosis, dissecting aneurysm, or double lumen sign/intimal sign that normalizes luminal/blood flow at the site of dissection. Because of the continuous progress in materials, therapeutic concepts, and ma-

nipulation techniques, there are more and more reports on the interventional treatment of carotid artery dissection, and the interventional treatment is highly efficient and effective.

Stent implantation is the most commonly used endovascular intervention for carotid artery dissection. Because the incidence is relatively low and it is difficult to form evidence-based studies with large samples, the indications for interventional treatment, the choice of methods, and the timing of treatment are still inconclusive.

Héloïse Ifergan *et al.* results suggests that Early ischemic recurrence following the diagnosis of acute spontaneous cervical artery dissection is more frequent than previously reported, and that its risk might be stratified on admission with a standard workup. In particular, the presence of a poor circle of Willis, intracranial extension (other than just V4), cervical occlusion, or cervical intraluminal thrombus are associated with high risk of EIR, for which specific management should be further evaluated [1].

A Japanese Nationwide Survey reveal [2]: 35 neurosurgical centers reported patients within 2 weeks after ischemic onset due to intracranial carotid artery dissection causing cerebral ischemia treated between January 2015 and December 2020. Data on clinical and radiological findings were statistically analyzed. Twenty-eight patients met the inclusion criteria. The median age was 36 years (range, 7 - 59 years), without sex differences. Headache at onset was documented in 60.7% of the patients. Dissection findings were categorized into stenosis (71.4%) or occlusion (28.6%). Initial treatments, including various antithrombotic agent combinations in 23 (82.1%) patients, effectively improved or prevented aggravation in half of the patients. The patients with stenotic dissection were significantly more likely to experience aggravation during the initial treatment than did those with occlusive dissection ($P = 0.03$). In addition, the patients with moderate to severe neurological deficits on admission had poorer outcomes at discharge more frequently than did those with mild neurological deficits on admission. Eight patients undergoing endovascular therapy had no procedural complications or further aggravation after intervention. In conclusion, patients with intracranial carotid dissection causing cerebral ischemia who had a stenotic dissection were at risk of further aggravation, and endovascular therapy effectively improved or prevented aggravation.

Farouk M, *et al.* [3], found that endovascular therapy for internal carotid artery dissection-related strokes has high rates of reperfusion and favorable outcomes. Stent-assisted angioplasty of carotid dissection is thought to be safe and effective.

Marnat G, *et al.* pointed out that Emergency stenting of the dissected cervical carotid artery during endovascular therapy for tandem occlusions seems safe, whatever the quality of the intracranial reperfusion.

Perry BC, Al-Ali F. found that Vertebral artery dissections were predominately type I (11/14, 79%), while ICA dissections were predominately type II (28/38, 74%) ($p < 0.001$). Type I dissections had statistically higher chances to present

with ischemic symptoms (14/21, 67%) when compared to type II dissections (7/31, 22.5%) ($p = 0.001$), while Type II dissections had higher chances to present with local symptoms (15/31, 48%) than type I dissections (5/21, 24%), almost reaching a statistical significance ($p = 0.057$). Type I, however, had a statistically higher chance of healing than type II (75 vs. 15%, respectively) ($p < 0.001$) [4] [5].

Borgess classification of sCAD, based on the presence or absence of intimal tear as depicted on imaging studies and effect on blood flow. In the Borgess classification, type I dissections have intact intima and type II dissections have an intimal tear. Type I dissections show an intact intima. Type IA has persistent antegrade flow. Type IB is completely occluded. Type II dissections show intimal disruption. Type IIA has a small disruption of the intima with a small side-wall aneurysm. Type IIB shows a clear intimal flap and aneurysmal dilation [5].

In this patient with acute onset and severe clinical symptoms with an nhiss score of 17, DSA suggested that the neck artery dissection resulted in severe stenosis of the vessel's lumen, and the dissection thrombosis caused embolism to the brain, but DSA suggested that the intracranial blood flow was not affected, and the question was whether to immediately release stents in the carotid artery to repair the vessel, We took into account that the patient had severe carotid stenosis and instability of acute carotid dissection follow-up may lead to carotid occlusion, new thrombus caused by carotid dissection leads to new embolism in brain may, the patient has completed cerebral angiography, CAS technique is simple, and it is decided to immediate repair vessel; The patient recovered well after surgery.

4. Conclusions

Antithrombotic therapy is difficult to heal double lumen signs/intimal signs resulting from tearing of the carotid intima and aggressive vascular intervention is reasonable in patients with Borgess classification type II.

Carotid dissection selection of the timely machine for endovascular intervention and its pathological outcome are closely related, most of the intramural hematomas can be partially or completely absorbed in the repair period (within 1 - 3 months), and the local lumen returns to normal or basically normal. However, in a few cases, the intramural hematoma is poorly resorbed or the intramural hematoma continues to enlarge, with aggravation of luminal stenosis or occlusion; or bulging out of a thin canal wall to form an aneurysm with intramural hematoma resorption. Endovascular intervention may be considered in the above cases to restore normal lumen/blood flow.

Conflicts of Interest

The author declares no conflicts of interest regarding the publication of this paper.

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