Case Report

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# Atypical Clinical Course in a Patient with Acute Carotid Artery Dissection

Çiğdem DENİZ[1], Tuğçe ÖZDEMİR GÜLTEKİN[2], Gözde ERYİĞİT BARAN[3], Ayşe ARALAŞMAK[1], Ömer GÖKTEKİN[1], Talip ASİL[1]

[1]Bezmialem Vakıf Univesity Faculty of Medicine, Department of Neurology, İstanbul, Turkey
[2]Erzincan Training and Research Hospital, Clinic of Neurology, Erzincan, Turkey
[3]Şişli Training and Research Hospital, Clinic of Neurology, İstanbul, Turkey

## ABSTRACT

Extracranial or intracranial artery dissections are more frequently reported because of cerebrovascular disease occurring in young adults. This condition is more likely to be associated with permanent morbidity and mortality. While anti-coagulant medications are commonly used for its treatment, an increased use of intravenous thromobolytic agents and endovascular treatments has also been reported. In this paper, the case of a patient with an atypical clinical course following thrombolytic treatment for acute extracranial internal carotid artery (ICA) dissection is presented in the context of a discussion regarding the safety and efficacy of aggressive treatment for extracranial arterial dissections.

**Keywords:** Intracranial artery, extracranial artery, dissection, stroke

## Introduction

Carotid or vertebral artery dissections are responsible for approximately 2% of all ischemic strokes (1). In young adults, dissection is an important cause of ischemic stroke, and this rate rises up to 20% under 50 years of age (2, 3).

Dissection is a term that is often used to describe the separation of the intima layer of the artery from the media layer and, more rarely, the separation of the media layer from the adventitia layer. Carotid artery dissection is associated with intramural accumulation of blood. In dissection cases in whom intramural hematoma develops, the mechanism of ischemic stroke is often related to thromboembolism and, more rarely, to hemodynamic failure (4). Intravenous thrombolytic therapy (IVT) is an effective and reliable treatment for acute ischemic stroke.

In this article; a patient who had acute extracranial internal carotid artery (ICA) dissection, who received IVT treatment, and who had an atypical clinical course is reported; the causes of stroke development in extracranial artery dissections, and the efficacy and safety of aggressive treatments in these patients are discussed.

## Case Report

A 31-year-old male patient with no history of any disease or drug use in his history was admitted to an external center with the complaints of weakness in the right half of his body and inability to speak.

It was found out in the neurological examination performed in the external center that his speech was aphasic, right nasolabial groove was faint, and the right upper and lower extremities had 3/5 muscle strength; it was found out in the diffusion weighted magnetic resonance imaging (MRI) taken in the external center that acute infarction was observed in the deep irrigation area of the left middle cerebral artery (MCA), IVT was given after 2.5 hours, the patient completely recovered in the 1st hour

**Address for Correspondence:** Çiğdem DENİZ, Bezmialem Vakıf Univesity Faculty of Medicine, Department of Neurology, İstanbul, Turkey
**E-mail:** teriyaki9@hotmail.com

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of treatment, however, the patient's paresis increased in the 23rd hour. The patient was referred to us for an interventional evaluation upon the detection of a total occlusion in the left ICA in MR angiography.

The general condition of the patient was moderate at the admission, consciousness was lethargic, and the arterial blood pressure was low and tachycardic (fever 36, pulse 140, blood pressure 70/60). In his neurological examination; national institutes of health stroke scale (NIHSS) score was 12 at the admission, he had an aphasic speech, right nasolabial groove was faint, and right upper extremity was observed to have 2/5 muscle strength and lower extremity was observed to have 3/5 muscle strength. The Alberta Stroke Program Early CT Score (ASPECT) was 7 at the admission, and acute infarction area showing diffusion restriction at left caudate nucleus and putamen localization was observed in the diffusion MRI taken at the admission (Figure 1).

The patient was admitted to the intensive care unit. Endovascular procedure was not performed due to impaired general condition and unstable vitals. Dopamine infusion (10 mcg / kg / min) was started due to low blood pressure. When the patient's systolic blood pressure increased to over 130 mmHg, significant improvement was observed in the paresis of the patient. In the follow-up, the patient had motor aphasic speech and the muscle strength was 4/5 in the upper and lower extremities.

The patient's routine biochemistry values were within normal limits. No features were found in young age stroke etiology in terms of infectious, vasculitic and coagulation parameters.

Since no proximal artery occlusion was considered when the patient was admitted to the external center, no angiographic images were obtained before IVT. However; when progression was observed in his clinic, MR angiography was performed and occlusion was detected in the left ICA; subsequently, he was referred to us for endovascular intervention considering proximal artery occlusion. Because the clinic of the patient had a fluctuating course and the occlusion was proximal in the MR angiography, digital subtraction angiography (DSA) was performed on the next day of hospitalization for endovascular procedure. It was seen in the angiography that the left ICA was 100% occluded, and the left MCA and the anterior cerebral

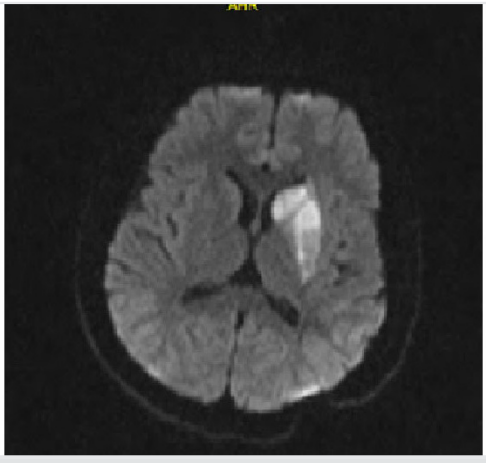

**Figure 1.** Acute infarction in the left caudate nucleus and putamen in MR diffusion imaging

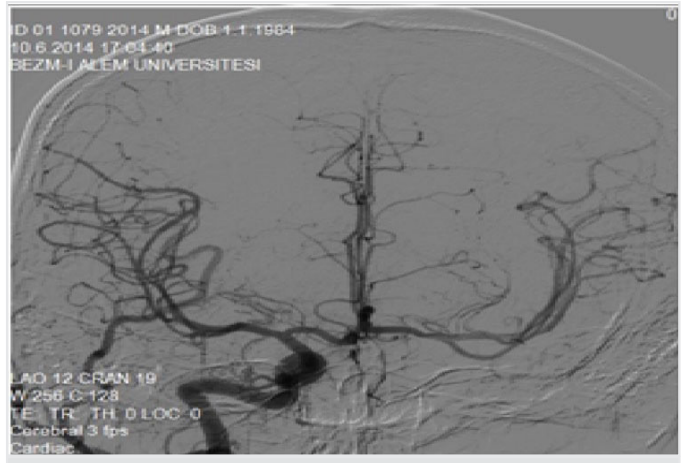


**Figure 3.** Retrograde filling of the left MCA and ACA in cerebral angiography

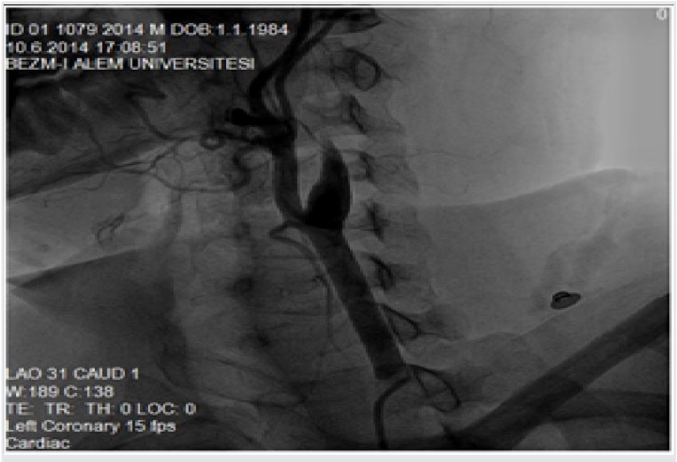


**Figure 2.** Dissection in the left ICA in cerebral angiography

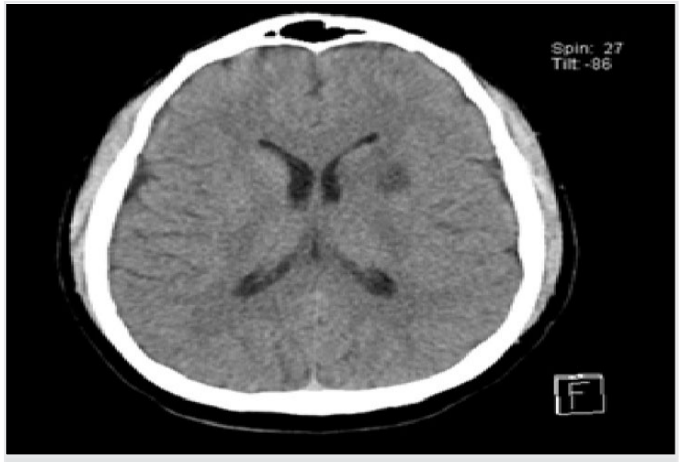


**Figure 4.** Intra-infarction hemorrhagic transformation in the left caudate nucleus and putamen in CT

artery (ACA) were observed to be filled by collaterals through the right carotid artery (Figure 2, 3). Thereupon, endovascular intervention was not considered.

In the diffusion MRI taken on the fifth day of his hospitalization; signaling changes of the subacute period infarction in MCA irrigation area were observed in the insular cortex in the deep gray matter and in frontal, parietal opercular region. At the same time, intra-infarction hemorrhagic transformation (grade 1) was observed in the cranial computed tomography (CT) (Figure 4). In the neurological examination performed on the 9th day of his admission; he had aphasia, his right nasolabial groove was faint, and he had first hemiparesis on the right. The patient in whom dopamine infusion (3 mcg / kg / min) was continued and whose general condition was good was admitted to the service.

Upon the observation of hemodynamic deterioration on the 11th day of his admission, he was admitted to the intensive care unit and DSA (Figure 5-8) was repeated. Dissection starting from the left ICA and continuing up to M1 was observed in DSA. Because complete perfusion, in other words, TIMI III (thrombolysis in myocardial infarction) was observed, medical treatment was decided.

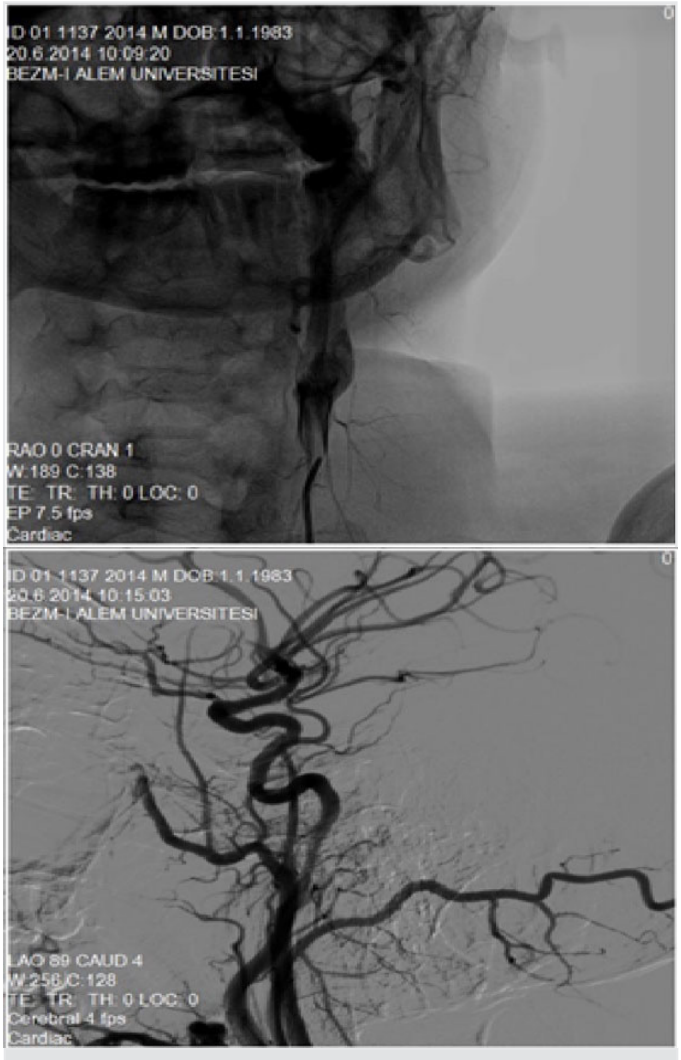


**Figure 5a, b.** (a) Dissection appearance starting from the left ICA in cerebral angiography. (b) Dissection appearance starting from the left ICA and extending to M1 in cerebral angiography

In the CT angiography performed on the 17th day of his admission; there was flow in the left ICA, and an eccentric diameter increase in the proximal of the intracranial distal segment and a dissection-induced thrombus reaching a thickness of 5 mm in the lumen were observed. Secondary to this, there was a decrease in calibration in the lumen, and the rate of stenosis was observed to be around 40% according to the North American Symptomatic Carotid Endarterectomy Trial (NASCET) scoring. While no flow was observed in the petrous and cavernous segments of the left ICA in the previous examination, there was flow in this examination and the left ICA was recanalized. Normal flow was observed in the left MCA and its branches.

On the 23rd day of hospitalization, the patient's general condition improved and dopamine was stopped; subsequently, he was admitted to the service, and oral warfarin and subcutaneous enoxaparin treatment was started. Warfarin was given as anticoagulant therapy to prevent thrombus formation developing

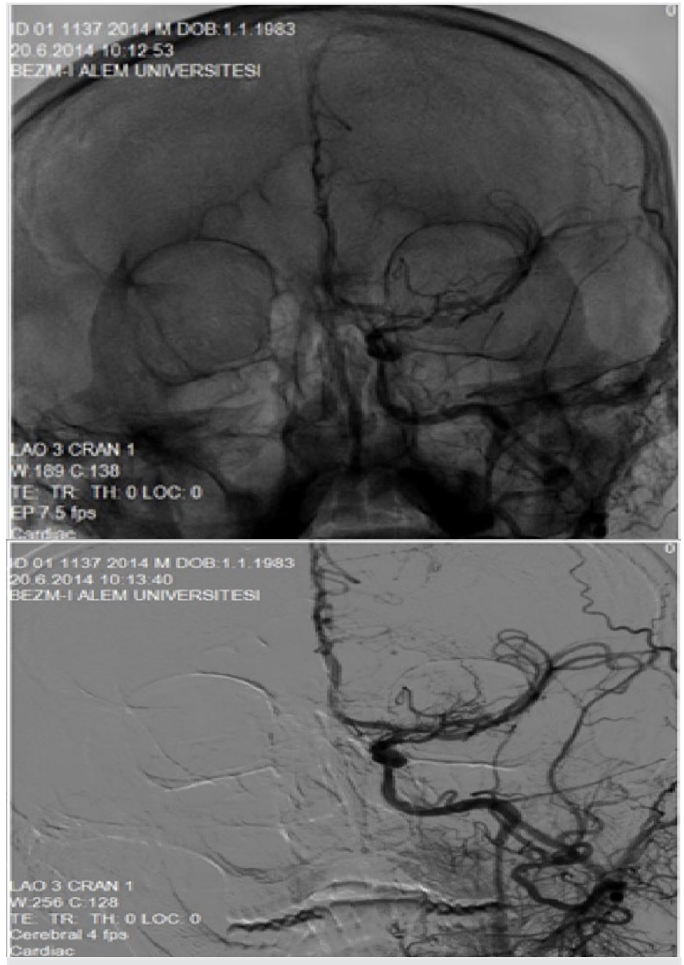


**Figure 6a, b.** (a) Dissection view of the left ICA to M1 at cerebral angiography. (b) Dissection appearance extending from the left ICA and to M1 and TIMI III flow

secondary to lumen obstruction caused by dissection. The patient was discharged two days later.

## Discussion

Internal carotid artery dissection occurs spontaneously or due to trauma. Spontaneous ICA dissection constitutes 5-20% of ischemic cerebrovascular disease in young and middle ages.

An intramural hematoma in the vascular wall causing congestion or obstruction of the arterial lumen and disrupting cerebral hemodynamics, or thromboembolism, in other words, artery-to-artery embolism are the underlying causes of the dissection-induced ischemic damage.

Anticoagulant therapy is generally used in treatment (3-5). Anticoagulant therapy is recommended to prevent thrombus formation developing secondary to lumen congestion caused by dissection (6).

A large randomized trial giving a definite conclusion about the efficacy and feasibility of intravenous thrombolytic therapy could not be performed in patients with ischemic stroke developing secondary to carotid artery dissection. In our patient, acute signs caused by artery to artery embolism initially improved rapidly with intravenous thrombolytic therapy; however, due to the occlusion of the ICA, findings related to hemodynamic deficiency occurred.

In a study in which bleeding risk and functionality were measured in patients with ischemic stroke secondary to cervical artery dissection; no significant difference was observed between those in whom thrombolytic therapy was and was not administered; however, major hemorrhage was observed more commonly in the group in which thrombolytic therapy was applied (1). In parallel to this literature, hemorrhage was also observed in our case on the 5$^{th}$ day; however, it was asymptomatic.

In a meta-analysis in which 22 cases and 180 patients from 14 retrospective studies were examined, positive evidence was presented for the safety of thrombolytic therapy (1).

Although it was observed that dissection extended from the extracranial segment to the intracranial M1 segment, there was a significant improvement in the patient's clinic on the 9$^{th}$ day of hospitalization, and Modified Rankin Scale (mRS) was calculated as 3. The mRS was calculated as 2 in the third month. The patient gave much better results in terms of functionality.

When we consider the dissections theoretically, IVT treatment can increase and expand the intramural hematoma in the dissection. It may increase luminal stenosis and occlusion and cause distal embolism (1). As a matter of fact, the dissection extended from the extracranial area to the intracranial M1 segment in our case, and this condition obstructed the lumen of the artery and disrupted cerebral hemodynamics, causing neurological deficits. In a similar publication, a functional recovery was defined despite the expansion of the intramural hematoma in a patient (1).

However; the fact that the patient did not have thromboembolism, that MCA was fed retrograde in the first angiography, that 40% stenosis was observed in ICA in CT angiography, that the middle cerebral artery was open, and that there was no distal embolism could have prepared a functional recovery in the patient. The presence of an occlusion in MCA in carotid artery dissections negatively affects the prognosis of dissection (7).

The main aim in cervical carotid artery dissections is to prevent the ischemic complications. Though many dissection patients may recover spontaneously, a treatment method should be identified for thromboembolic and hemodynamic complications that may develop. Although anticoagulants and antiplatelet agents are the classical methods of treatment, no superiority among each other has been found in the studies. For thrombolytic therapy, which is another method, can be given if the patient comes with stroke-like symptoms and has the thrombolytic therapy indications, but these are limited on a case-by-case basis. Surgical and endovascular interventions considered to be the last available treatment modality are recommended in dissections that do not respond to medical treatment and in recurrent dissections (8).

## Conclusion

Anticoagulant and antiaggregant treatment is given to prevent thrombus formation developing secondary to lumen obstruction caused by dissection, however, thrombus occurs simultaneously with dissection. This may raise the use of thrombolytic therapy.

It is not known whether or not the absence of thromboembolism in our patient can be attributed to IVT. However, it is also a fact that although the IVT treatment in our patient caused the dissection to extend from extracranial area to the intracranial area by increasing the intramural hematoma, the patient functionally improved on the 9$^{th}$ day.

Therefore, if hemodynamics is kept in balance, we believe that IVT may decrease thrombus formation in the early period and consequently a functional improvement may occur in the flow continuing in the lumen.

**Informed Consent:** Written informed consent was obtained from patient who participated in this study.

**Peer-review:** Externally peer-reviewed.

**Author Contributions**

Concept - Ç.D., T.A.; Design - Ç.D., T.A.; Supervision - Ç.D., T.A.; Resources - Ç.D., T.A., Ö.G., A.A.; Materials - Ç.D., T.A.; Data Collection and/or Processing - Ç.D., T.Ö.G.; Analysis and/or Interpretation - Ç.D., T.Ö.G.; Literature Search -Ç.D., G.E.B.; Writing Manuscript - Ç.D.; Critical Review - T.A., Ö.G., A.A.

**Conflict of Interest:** The authors have no conflicts of interest to declare.

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