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Internal Carotid Artery Dissection Secondary to an Elongated Styloid Process: A Case Report of Major Acute Ischemic Stroke (NIHSS 23)

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Case Report

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Abstract

Context: The internal carotid arteries are major sources of blood supply to the brain. Their dissection can be spontaneous, without a definite cause, or can occur as a result of trauma. One such trauma occurs when an elongated styloid process compresses or dissects the extracranial internal carotid artery, potentially leading to transient ischemic attacks or ischemic strokes. **Case Presentation**: We describe the clinical course of a 54-year-old patient in critical condition, who presented to the emergency department with a severe ischemic stroke following an internal carotid artery dissection secondary to an elongated styloid process. **Conclusions**: The therapeutic strategy should prioritize stabilizing the consequences of the dissection and preventing the recurrence of ischemic events, followed by rehabilitation.

1. Introduction

Carotid artery dissection is responsible for up to 2% of all acute ischemic strokes globally. Depending on the underlying mechanism, internal carotid artery (ICA) dissection can be spontaneous or traumatic, resulting from external mechanical factors that cause arterial shearing. An analysis of data from 1,121 patients across 20 studies reported that the vast majority of dissections are spontaneous (81.7%), while traumatic and iatrogenic mechanisms account for less than 20% of all reported cases [1]. An elongated styloid process or its angulation relative to the artery can cause arterial compression, complete dissection, or pseudoaneurysm formation, leading to a wide range of clinical manifestations [2]. These manifestations can vary from asymptomatic presentations to life-threatening conditions such as transient ischemic attacks or acute ischemic strokes. We present the diagnostic and therapeutic particularities of a rare case of cervical artery dissection, triggered by motor vehicle accident in the presence of an elongated styloid process.

2. Case Presentation

2.1. Background

A 54-year-old male patient, with a history of hypertension, initially presented to a regional hospital with right-sided motor deficit and speech impairment (occurring following a motor vehicle accident). A non-contrast and contrast-enhanced Computed Tomography (CT) scan revealed an occlusion of the left internal carotid artery. Given the eligibility criteria and the absence of absolute contraindications, pharmacological revascularization via intravenous thrombolysis with alteplase was performed 3 hours and 35 minutes after the onset of neurological symptoms; however, no clinical improvement was observed at the end of the procedure. The patient was subsequently transferred to the Neurology Department of the University Emergency Hospital Bucharest for mechanical revascularization.

2.2. Diagnosis and Intervention

Upon admission, the patient was in critical condition, though conscious and hemodynamically stable (blood pressure 155/100 mmHg, heart rate 70 bpm, SpO2 97%). Neurological examination revealed leftward gaze and head deviation, right homonymous hemianopsia, right central facial palsy, right hemiparesis (MRC grade 0/5 in the upper limb and 2/5 in the lower limb), and right-sided tactile hypoesthesia. The National Institutes of Health Stroke Scale (NIHSS) score was 23, with a prior Modified Rankin Scale (mRS) of 0.

During the mechanical revascularization procedure (arterial puncture at 7 hours and 30 minutes post-onset), contrast injection confirmed the occlusion of the left internal carotid artery. The left internal carotid artery was selectively catheterized with a suction catheter, and three passes of thromboaspiration were performed, achieving vessel recanalization.

However, angiographic control revealed an underlying arterial dissection beneath the thrombus, with a dissection flap extending across segments C1-C4 (extracranial), associated with a proximal vascular loop that caused endovascular

navigation difficulties Fig. 1. Multiple serial stenoses were present along the intimal dissection flap. Intracranial injection showed a filling defect in a distal segment of the left middle cerebral artery (MCA).

Endovascular treatment of the dissection was attempted by placing a 5x40 mm stent in the C2 segment, but the aforementioned vascular loop could not be crossed regardless of the technique used. A 7x40 mm stent was then placed at the origin of the left ICA (C1 segment), extending to the vascular loop Fig. 2A, under intravenous cangrelor therapy followed by dual antiplatelet therapy with acetylsalicylic acid and clopidogrel.

Control injection of the right carotid axis showed reduced collateral flow to the left cerebral hemisphere Fig. 2B. Partial cerebral revascularization was achieved with a modified Thrombolysis in Cerebral Infarction (mTICI) of 1. Post-intervention, the NIHSS score remained stable at 23, with no neurological improvement.

A follow-up non-contrast brain CT performed 12 hours post-angioplasty confirmed the presence of an established ischemic infarct, in the middle cerebral artery territory, with areas suggestive of cortical reperfusion Fig. 3A. The imaging findings were consistent with the focal neurological presentation specific to left middle cerebral artery syndrome.

Doppler ultrasonography at 24 hours post-angioplasty revealed the absence of detectable intrastent flow, characteristic of functional occlusion. The re-initiation of intravenous cangrelor treatment was decided. A subsequent CT exam at 48 hours post-angioplasty detected partial recanalization (a subocclusive soft thrombus at the distal end of the stent and filiform flow up to the origin of the M1 segment of the left MCA) Fig. 3B.

2.3. Clinical Course and Outcomes

The clinical course was slightly favorable, with partial improvement of neurological deficits, regarding both motor impairment and speech disorder. Due to dysphagia, the patient required the placement of a nasogastric tube; however, it wasn't tolerated by the patient. Consequently, the patient developed a mild form of aspiration pneumonia, which required antibiotic treatment and showed gradual improvement. Cardiological evaluation revealed findings suggestive of poorly controlled hypertension, including concentric left ventricular hypertrophy. No valvulopathies or other pathological changes with etiologic potential for the acute ischemic event were detected.

To elucidate the etiology of the left ICA dissection, a three-dimensional volume-rendering reconstruction of the cervicocranial region was performed. An elongated styloid process in direct contact with the left ICA was detected, characteristic of the vascular variant of Eagle syndrome. The 3D CT reconstruction of the cranio-cervical region is presented in Fig. 4.

Eleven days after admission, a follow-up non-contrast brain CT scan revealed areas of reperfusion, while Doppler ultrasonography of the cervical vessels demonstrated the absence of intrastent flow. At the express request of the family, the patient was transferred to his home country. At the time of discharge from our unit, the patient's general condition was satisfactory, with improvement in the right-sided motor deficit and swallowing function. The NIHSS score at discharge was 12, and the mRS score was 4.

4. Discussion

Eagle Syndrome is one of the potential causes of carotid artery dissection, occurring through repeated or acute mechanical trauma to the internal carotid artery wall. The triggering factor may be an elongated styloid process, its angulation relative to the artery, or an ossified stylohyoid ligament following cervical trauma or sudden neck movements. The complications associated with Eagle Syndrome are managed medically, endovascularly, or surgically, depending on the clinical symptoms and the risk of further complications [3].

Treatment depends on the patient's age, symptom severity, and the probability of stroke development. The primary goal is to restore cerebral blood flow and prevent recurrent thrombosis. Mechanical thrombectomy in the extended window has significantly advanced emergency neurology. The standard intervention window for carefully selected cases has been extended from 6 to 24 hours from symptom onset. This extension is based on two major clinical trials published in 2018 (DAWN and DEFUSE-3). Currently, international guidelines (AHA/ASA and ESO) recommend mechanical thrombectomy within the 6–24 hour interval for large vessel occlusions in the anterior circulation, provided the patient meets the imaging mismatch criteria established by the DAWN and DEFUSE studies [4,5].

The administration of antiplatelet agents within 24 hours of thrombolytic therapy is contraindicated, as the risk of hemorrhagic transformation of the ischemic infarct is highest during this window [4]. If immediate stenting is required post-thrombolysis, low-dose antiplatelets or thrombectomy without prior thrombolysis may be considered to manage the risk of re-thrombosis. A clinical dilemma arises when a thrombolyzed patient has an ICA occlusion requiring a stent to maintain patency: the stent is a foreign body that undergoes almost instantaneous thrombosis unless potent antiplatelet therapy is administered.

In ICA occlusion, the recanalization rate is a key predictor of favorable clinical outcomes, and success rates vary significantly by therapeutic method. For intravenous thrombolysis, the success rate is relatively low, an average of approximately 13%, due to the large thrombus burden typically found in the ICA [6]. In contrast, endovascular treatment using stent-retrievers or aspiration devices yields much higher success rates, ranging from approximately 75% to 85% [7]. Recanalization success depends on parameters such as occlusion location, type, and time to intervention.

If reperfusion zones are reactivated by restoring blood flow and the patient’s recovery is clinically significant, the intervention is deemed effective. The ischemic penumbra is the peripheral zone where blood flow is reduced, and cells are in a state of functional hibernation. The larger the salvaged penumbra, the more significant the clinical improvement. Reperfusion is measured using the modified Thrombolysis in Cerebral Infarction (mTICI) score: mTICI 0/1 for absent or minimal reperfusion (no clinical improvement), mTICI 2b for partial reperfusion (over 50% of the territory), and mTICI 3 for complete reperfusion.

The significant correlation between reperfusion zones and clinical improvement is the cornerstone of interventional neurology. However, cases exist where reperfusion does not lead to clinical benefit, a phenomenon where blood cannot circulate at the capillary level due to edema or micro-thrombi (no-reflow phenomenon). Clinical improvement is correlated with a decrease in the NIHSS score and is maximized by factors such as a valid therapeutic window, robust collateral circulation, and a low baseline risk of disability.

Data on internal carotid artery dissection secondary to an elongated styloid process are limited, predominantly originating from case reports and retrospective studies. The actual incidence of such clinical events is rare and poorly documented, as evidenced by literature analysis. This rarity precludes large-scale studies regarding management and prognosis.

In our case, the literature search was conducted for the 2012–2026 period using the following keyword combination: ("Eagle Syndrome" OR "elongated styloid process" OR "stylohyoid complex") AND ("Carotid Artery, Internal, Dissection" OR "carotid dissection"), with a "Case Reports" filter. Thirty-three cases of carotid artery dissection secondary to an elongated styloid process were identified.

To further refine the search, the term "trauma" was added: AND ("Trauma" OR "Injuries" OR "Fracture" OR "traumatic"), aiming to identify case studies similar to ours. This returned five cases, one of which featured a mechanism similar to Eagle Syndrome. For analysis, data such as patient characteristics, triggering factors, symptoms, and treatment strategies were extracted and compiled in Table 1. The 2012–2026 timeframe was selected because the first trauma-related case returned by the PubMed database following this query was published on March 1, 2012.

Table 1

Cases from literature review (2012–2026) of internal carotid artery dissection secondary to compression by the stylohyoid complex and adjacent cervical structures associated with cervical trauma.

Authors (Year)	Age/ Sex	Triggering Factors	Symptoms at Presentation	In-hospital Findings/Symptoms	Therapeutic Strategy	Observations
Yamamoto et al. (2013) [8]	51/M	Transient ICA compression by the styloid process induced by cervical rotation.	Right orbital pain and Horner syndrome onset three weeks prior to presentation.	Intramural hematoma at the level of the right cervical internal carotid artery.	Conservative treatment with Aspirin; gradual symptomatic remission.	Follow-up MRI at 6 months: resolution of ICA dissection.
Subedi et al. (2017) [9]	47/F	Sudden hyperextension of the cervical spine (looking at the ceiling) while using a phone.	Dysarthria, paresthesia, and motor deficit in the left upper limb.	Left cervical and periocular pain during leftward head rotation, persisting for 4 months.	Balloon angioplasty and self-expanding stent placement (Rebel 4×20 mm). Dual antiplatelet therapy (ASA 325 mg, Clopidogrel 75 mg) and Atorvastatin 20 mg/day.	Patient scheduled for surgical resection of the styloid process due to recurrent symptoms.
Shindo et al. (2019) [10]	49/M	Hyperextension of the cervical spine during deglutition (swallowing).	Sudden motor deficit in the left extremities and dysarthria immediately following neck hyperextension; neck pain.	Dysarthria and left hemiparesis (NIHSS score 4).	Argatroban administration (first week), followed by oral antiplatelet therapy, physical therapy, occupational therapy, and speech therapy.	Complete luminal recanalization at 6 months; no dissection recurrence. Subsequent styloidectomy performed without adverse events.
Vicente Pascual et al. (2019) [11]	53/M	Vigorous and repeated head rotations (auto mechanic); "Eagle-like" compression mechanism given proximity to the cervical vascular bundle.	Sudden onset of motor deficit in the left upper limb during occupational activity.	CT Angiography: Intimal flap with absent flow in the right internal carotid artery.	Placement of two carotid stents and mechanical thrombectomy for intracranial embolism. Post-procedural dual antiplatelet therapy; nearly complete motor recovery.	Established ischemic lesions; absent blood flow in the right jugular vein.
Pfaff et al. (2023) [12]	40/M	Unspecified physical exercise.	Palpebral ptosis and photopsia in the left eye.	After 7 days: transient paresthesia in the right hand (minor stroke); NIHSS 1, mRS 1.	Implantation of a double-layer flow diverter device (5.5 mm × 32/26 mm).	Follow-up angiography at 8 months: occlusion of the flow diverter and the left extracranial

Authors (Year)	Age/ Sex	Triggering Factors	Symptoms at Presentation	In-hospital Findings/Symptoms	Therapeutic Strategy	Observations
						ICA, without associated neurological symptoms.

The mean age of onset in the reported cases is 48 years, with a sex distribution of four males and one female. Neurological events were triggered by head rotation or vertical movements in four out of the five cases. The fifth case could also be suspected of involving sudden head movements, although physical exercise was the only explicitly mentioned trigger. The PubMed query yielded no cases triggered by a motor vehicle accident, although our case likely falls into the category of cervical rotation-induced trauma.

Our case describes a 54-year-old patient with a severe ischemic stroke (NIHSS score of 23) specific to middle cerebral artery syndrome, with imaging revealing internal carotid artery occlusion. Symptoms debuted following a motor vehicle accident. Intravenous fibrinolysis performed at a local facility 3 hours and 35 minutes post-onset did not lead to neurological improvement; consequently, the patient was transferred to the Neurology Department for mechanical revascularization.

Aspiration thrombectomy removed the initial blockage, but control angiography revealed the underlying cause: extensive ICA dissection with an intimal flap extending across the extracranial segments (C1-C4). Stenting of the dissection was attempted, but a proximal vascular loop hindered endovascular navigation. Successful stent placement was achieved proximal to the loop (C1). Partial revascularization was obtained with a mTICI score of 1, indicating absent or minimal reperfusion (without clinical improvement).

The subsequent clinical course was characterized by progressive partial resolution of neurological symptoms. CT imaging revealed areas of cortical reperfusion and identified the cause of the dissection: an elongated styloid process, characteristic of the vascular form of Eagle Syndrome. During hospitalization, the reintroduction of antiplatelet therapy with intravenous cangrelor was necessary due to recurrent stent occlusion. Favorable progress was achieved, with the patient reaching an NIHSS score of 13 at the time of transfer. At the express request of the family, the patient was transferred to his home country.

5. Conclusions

When a patient presents to the emergency department with an ischemic stroke, most management strategies for carotid artery dissection focus on treating the acute stroke and preventing recurrence. Carotid artery dissection has a specific treatment protocol as long as ischemic symptoms are absent; however, therapeutic decisions must be individualized and managed by a multidisciplinary team (neurologists, interventional radiologists, and surgeons). Surgical resection of the elongated styloid process becomes a necessary option when standard medical treatment fails and the patient experiences recurrent strokes.

Declarations

Institutional Review Board Statement

Ethical review and approval were waived for this study because single-patient case reports are exempt from institutional review board approval under our institution's regulations.

Informed Consent

Statement: Written informed consent has been obtained from the patient to publish this paper.

Conflicts of Interest:

The authors declare no conflicts of interest.

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Conceptualization, Ș.-A.A and C.T.; methodology, Ș.-A.A and C.T.; validation, Ș.-A.A. and C.T.; formal analysis, Ș.-A.A. and A.-I.M.; investigation, Ș.-A.A. and A.-I.M.; resources, A.-I.M. and C.T.; data curation, C.T.; writing—original draft preparation, Ș.-A.A., writing—review and editing, Ș.-A.A., A.-I.M. and C.T.; visualization, Ș.-A.A., A.-I.M. and C.T.; supervision, C.T. All authors have read and agreed to the published version of the manuscript.

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Data Availability Statement:

The data supporting the findings of this study are derived from the patient's medical record. Due to privacy and ethical restrictions, these data are not publicly available.

Abbreviations

ICA	Internal Carotid Artery
CT	Computed Tomography
NIHSS	National Institutes of Health Stroke Scale
mRS	Modified Rankin Scale
MCA	Middle Cerebral Artery
mTICI	Modified Thrombolysis in Cerebral Infarction

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Figures



Figure 1

Dynamic digital subtraction angiography evaluation. (**A**), (**B**), (**C**) Extracranial angiograms demonstrating the course of the left ICA. A proximal vascular loop is observed at the base of the neck, associated with filiform narrowing and extensive serial stenoses across the extracranial C1–C4 segments, features pathognomonic for an intimal dissection flap underlying a thrombus. (**D**) Intracranial angiogram revealing a focal filling defect (contrast medium cutoff) in a distal segment of the left MCA, of thromboembolic etiology.



Figure 2

Post-procedural digital subtraction angiography. (**A**) A 7x40 mm stent deployed at the origin of the left internal carotid artery (C1 segment), extending distally to the level of the cervical vascular loop, under antiplatelet therapy. (**B**) Selective control injection of the right carotid axis reveals reduced compensatory collateral circulation via the circle of Willis to the affected left cerebral hemisphere.

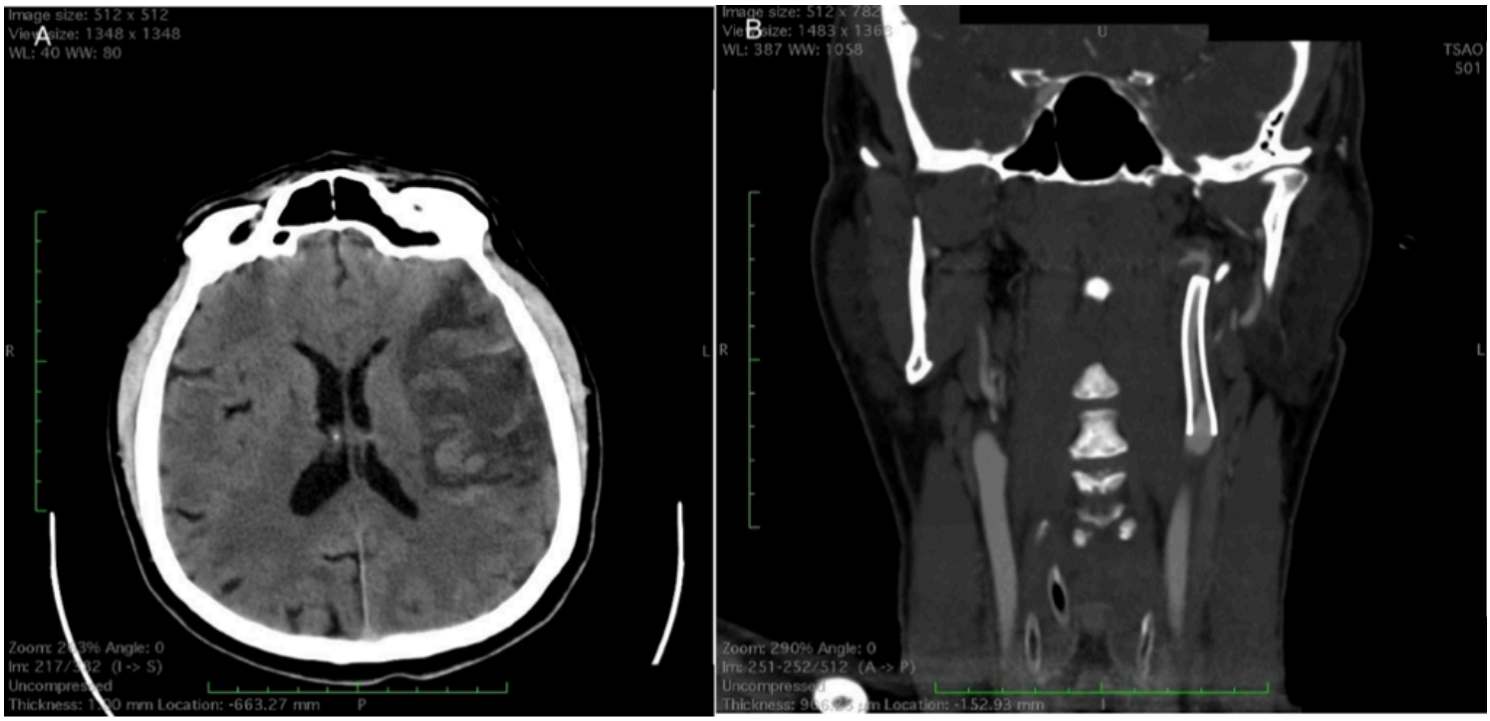


Figure 3

Follow-up CT examination. (**A**) At 12 hours post-angioplasty, a non-contrast axial CT scan demonstrates an established ischemic infarct within the left middle cerebral artery territory, associated with hyperdense cortical areas suggestive of reperfusion hemorrhagic transformation. (**B**) Coronal CT angiography reconstruction at 48 hours post-procedure reveals partial recanalization of the vascular axis. It highlights the presence of a subocclusive soft thrombus at the distal end of the left carotid stent and an extended filiform residual flow reaching the origin of the M1 segment of the middle cerebral artery.



Figure 4

Three-dimensional volume rendering reconstruction of the cranio-cervical CT. Marked elongation of the left styloid process is demonstrated, extending distally to establish a direct contact relationship with the course of the internal carotid artery, a pathognomonic feature of the vascular variant of Eagle syndrome.