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CASE REPORT

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Internal carotid artery dissection due to direct mechanical damage caused by the ipsilateral elongated styloid process. A 4-year follow-up

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Abstract

Internal carotid artery dissection (ICAD) is a complex clinical problem with a multi-causal etiology, in which the intima of the artery ruptures and is detached by the inflowing blood. Causes include trauma, intense exercise, vomiting, prolonged retroflexion of the neck, and spontaneous dissection. The therapy for this condition consists of treatment with anticoagulants or through endovascular intervention in recurrent cases. The aim of this paper is to present a case of ICAD in a young woman complicated by an ischemic stroke caused by an elongated styloid process. This abnormality was found in 3D computed tomography. In the described case, it was decided to start pharmacotherapy and withdraw from the endovascular treatment due to the technical difficulties of the procedure. The patient had been followed up regularly up to 4 years after the incident.

An elongated styloid process may be the cause of Eagle's syndrome classified into two types: classical type, and the rare carotid artery type, a component of which is ICAD. It is estimated that the styloid process in this form occurs in 4% of the population, which may indicate that the amount of ICAD caused by this variant of the styloid process is underestimated and should be included in the differential diagnosis of ischemic stroke in young people.

Key words: carotid artery dissection (CAD); elongated styloid process (ESP); transient ischemic attack (TIA); Eagle's syndrome

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Introduction

Carotid artery dissection (CAD) is a multicausal condition where the layers of the carotid artery are spontaneously separated [1]. It is often caused by neck injuries, especially extreme neck rotations or extensions [2]. This can lead to recurrent ischemic events such as transient ischemic attack (TIA) [3]. CAD is a major cause of stroke in young (< 50 years old) patients [4]. Therapy of carotid artery dissection is based on anticoagulants which protect patients from peripheral embolism and following cerebral ischemia [2]. This procedure is often sufficient and pathological lesions heal up spontaneously in a six-month time [2].

Case report

A 36-year-old woman, with a history of 3 miscarriages, polycystic ovaries syndrome, and MTHFR gene mutation (heterozygous) diagnosed 3 years earlier

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was admitted to the Department of Neurology due to symptoms of a TIA and severe headache. 3 days prior to admission the patient had suffered a right-sided jolt of the body caused by a German Shepherd.

Neurological examination revealed weakness and stiffness of the left side of the body, central facial palsy, and dysarthria. Computed Tomography (CT) imaging showed a hyperdense middle cerebral artery (MCA) in the M1 segment. Ischemic lesions in basal ganglia on the right side were depicted in the MR scan. 2 hours after the onset of symptoms recombinant tissue plasminogen activator thrombolytic therapy was administered. The total dose was calculated at 59 mg (6 mg with an intravenous bolus and 53 mg by intravenous infusion over 1 hour). At the same time, Angio-CT was performed, which showed occlusion and features of right internal carotid artery (RICA) dissection. Subsequently, the patient underwent the mechanical thrombectomy procedure. In the control CT examination performed after 24 hours, a small hemorrhagic area in the posterior part of the basal ganglia on the right side was seen. After the mechanical thrombectomy, the patient was treated with 5000 IU (International Units) low molecular weight heparin and two antiplatelet drugs — 150 mg acetylsalicylic acid (ASA), 75 mg clopidogrel and 20 mg atorvastatin.

After two weeks of hospitalization, the patient was discharged from the Neurology Department in good general condition and was referred to the Department of Angiology. Duplex Doppler ultrasonography examination of carotid arteries done at an Angiology clinic revealed a turbulent flow with a peak systolic velocity (PSV) over 3 m/s and two lumens with the reversed flow in a false lumen in the RICA. No abnormalities were found in the carotid arteries on the left side as well as in external and common carotid arteries on the right side. A follow-up Angio-CT scan done at that time revealed the cause of the patient's carotid artery dissection. The 3-dimensional reconstruction showed an elongated right styloid process (SP), which abutted with its end to the arterial wall of ICA (Fig. 1, 2). Its length, measured from the temporal bone junction to the tip of the process, was 31 mm (Fig. 3). It was decided to consult the patient with vascular surgeons, who chose not to apply a carotid stent because of the high risk of intraprocedural stroke and the need to use a long stent given that dissection involved extra- and intracranial segments of ICA. Instead, the patient remains on drug treatment consisting of 75 mg ASA and 20 mg atorvastatin. LMWH and clopidogrel were discontinued 6 months after the incident. In addition, she is under constant supervision at follow-up visits held every 3–6 months in the Department of Angiology where the carotid flow in doppler ultrasound is measured.

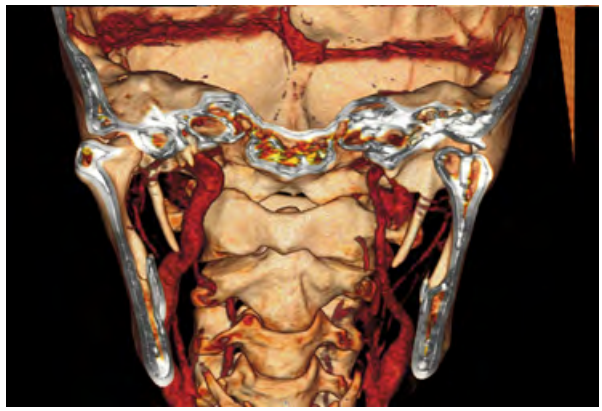

Figure 1. 3D reconstruction of angiography CT scan showing the site of right internal carotid artery damage caused by the ipsilateral styloid process

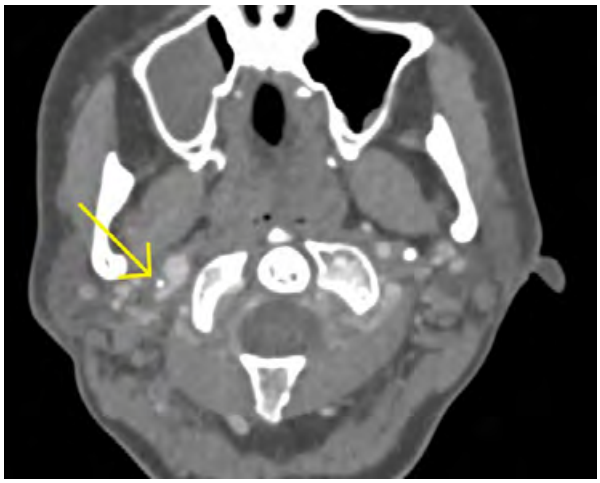

Figure 2. Axial reconstruction of angiography CT scan showing the tip of the styloid process compressing the right internal carotid artery

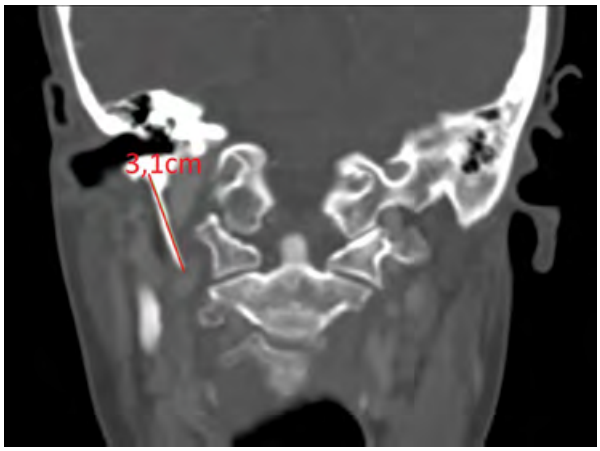

Figure 3. Coronal reconstruction of angiography CT scan showing the length of the right styloid process

Currently, the dissection is stabilized, the carotid flow gradually decreases and totals 2 m/s. Figure 4 presents the most recent USG (January 2023) duplex-doppler examination of RICA in our patient.

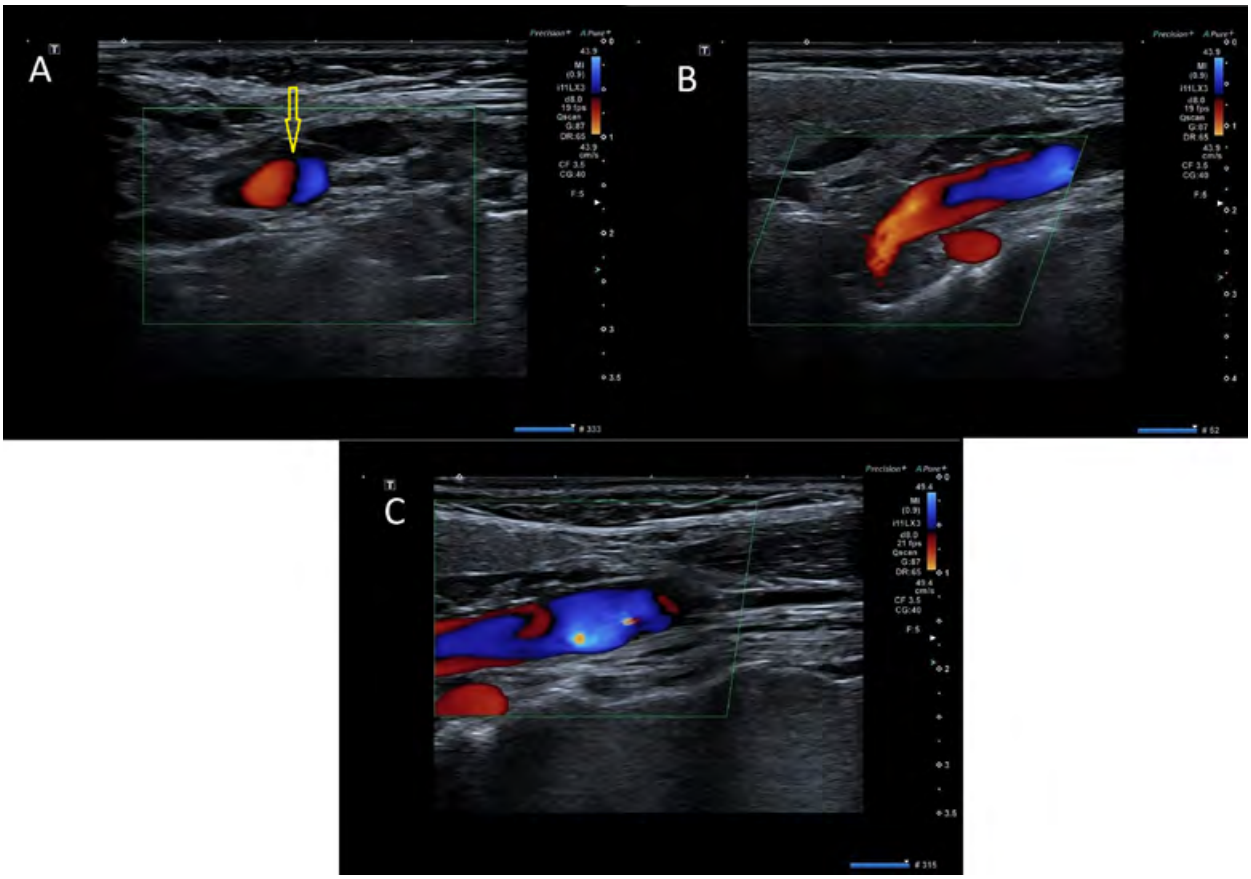

Figure 4. Duplex-doppler ultrasonography examination of RICA shows a dissection flap (yellow arrow [**A**]) separating the true and the false lumen. Retrograde flow is visible in the latter (**B**, **C**). The measured PSV value was 173 cm/s

The neurological condition of the patient was reassessed during the follow-up visit in February 2023 — the patient presented a mild weakness in the left limbs.

Discussion

The annual prevalence of internal carotid artery dissection is 2.5–3 cases per 100,000 [5, 6]. CAD is responsible for almost 20% of strokes in young adults and for about 2,5% among older patients [5, 7]. However, this incidence could be undiagnosed due to slight clinical symptoms [8]. Moreover, according to Paciaroni et al. CAD is more likely to occur during winter than in summer [9].

CAD can be divided based on the segment of the artery which was involved. It can be classified as the dissection of the vertebral or carotid segment and as the dissection of the intracranial or extracranial segment [8]. The most common, with 75% prevalence, are dissections of extracranial segments [10].

CAD mostly occurs spontaneously or after neck or head trauma which leads to a tear in the intimal layer of the carotid artery which creates an intramural hematoma [11]. The hematoma can result in stenosis and thrombus formation which results in cerebral ischemia. The risk factors of CAD are excessive physical exercise, emesis, long-lasting retroflection, or bronchoscopy [2]. The genetic factors are Ehlers-Danlos syndrome, Marfan's syndrome, Osteogenesis Imperfecta, or Behcet's disease [2].

The course of carotid artery dissection can be asymptomatic and that is why this condition is often undiagnosed [8]. Headache occurs among 2/3 patients and is often described as severe, acute, stabbing, or pounding and is localized on the side of the dissection [2, 6]. Other clinical manifestations of CAD are cranial nerve palsies, ipsilateral Horner's syndrome, and retinal or cerebral ischemia [8]. The symptoms of cerebral ischemia are episodes of *amaurosis fugax*, TIA, and brain stroke [2].

The most common, easily accessible, and noninvasive examination to diagnose carotid artery dissection is doppler ultrasound [2]. This procedure also allows monitoring of the course of CAD in later periods [2]. Doppler ultrasound should be always verified by neuroimaging techniques including magnetic resonance imaging (MRI), computerized tomographic angiography (CT), and conventional angiography [2, 8]. Angiography

is an invasive examination, which is associated with some mild complications, but allows one to implement endovascular treatment or to plan surgical procedures [2].

In the case described here, a three-dimensional Angio-CT scan revealed the cause of the ICAD. According to the literature, the styloid process is classified as elongated when it exceeds 30 mm in length [12]. About 4% of styloid processes are found to be elongated, but of these, only about 4% present with symptoms [13, 14]. Eagle syndrome is a rare condition caused by an ESP which interferes with the neighboring structures leading to various symptoms [13]. The typical Eagle syndrome is associated with sore throat, otalgia, and foreign body sensation in the throat due to compression of the ESP on the glossopharyngeal nerve, while the rare carotid type is caused by compression of the carotid artery in its extracranial segment, causing cerebrovascular symptoms such as syncope, dizziness, or transient ischemic attacks. There are a handful of reports on mechanical injury to the internal carotid artery caused directly by the styloid process [15–17]. Nevertheless, an ESP is considered a risk factor for CAD that can lead to ischemic stroke. Given the prevalence of ESP, the differential diagnosis of ischemic stroke in young individuals should include this abnormality of the styloid process once other causes have been ruled out.

Drug treatment of carotid artery dissection primarily consists of antithrombotic therapy: anticoagulant and antiplatelet therapies [18]. According to Misztal et al. who reviewed the literature of the treatment of CAD, there is no significant difference in the survival rate of patients who used anticoagulants or antiplatelet drugs, which means that both of them can be successfully used [2, 19]. However, for patients with recurrent ischemic incidents, an endovascular intervention should be considered [2]. Surgical procedures are indicated only in special cases such as enlarging of dissection range, presence of critical hemodynamically arterial stenosis, and presence of aneurysm which is bigger than the twofold dimension of the normal internal carotid artery, but for the majority of patients, endovascular therapy is preferred [2].

The prognosis of carotid artery dissection is good if no ischemic stroke or irreversible eye lesions occurred [2]. Most cases of carotid artery dissection can be conservatively treated with anticoagulation or antiplatelet drugs and have good clinical outcomes, with anatomic resolutions and low rates of recurrence [20].

Conflict of interest

None.

References

1. Goodfriend SD, Tadi P, Koury R. Carotid artery dissection. In: StatPearls. Treasure Island (FL): StatPearls Publishing 2022, indexed in Pubmed: 28613585.
2. Misztal M, Kwiatkowska W, Ohly P, et al. Internal carotid artery dissection-symptomatology, diagnosis and treatment. Kardiol Pol. 2011; 69(9): 958–962, indexed in Pubmed: 21928211.
3. Brown SC, Falcone GJ, Hebert RM, et al. Stenting for acute carotid artery dissection. Stroke. 2020; 51(1): e3–e6, doi: 10.1161/STROKEAHA.119.027827, indexed in Pubmed: 31795908.
4. Engelter ST, Traenka C, Gensicke H, et al. TREAT-CAD investigators. Aspirin versus anticoagulation in cervical artery dissection (TREAT-CAD): an open-label, randomised, non-inferiority trial. Lancet Neurol. 2021; 20(5): 341–350, doi: 10.1016/S1474-4422(21)00044-2, indexed in Pubmed: 33765420.
5. Lee VH, Brown RD, Mandrekar JN, et al. Incidence and outcome of cervical artery dissection: a population-based study. Neurology. 2006; 67(10): 1809–1812, doi: 10.1212/01.wnl.0000244486.30455.71, indexed in Pubmed: 17130413.
6. Schievink W, Roiter V. Epidemiology of cervical artery dissection. Front Neurol Neurosci. 2005; 20: 12–15, doi: 10.1159/000088125, indexed in Pubmed: 17290107.
7. Bogousslavsky J, Regli F. Ischemic stroke in adults younger than 30 years of age. Cause and prognosis. Arch Neurol. 1987; 44(5): 479–482, doi: 10.1001/archneur.1987.00520170009012, indexed in Pubmed: 3579657.
8. Blum CA, Yaghi S. Cervical artery dissection: a review of the epidemiology, pathophysiology, treatment, and outcome. Arch Neurosci. 2015; 2(4), doi: 10.5812/archneurosci.26670, indexed in Pubmed: 26478890.
9. Paciaroni M, Georgiadis D, Arnold M, et al. Seasonal variability in spontaneous cervical artery dissection. J Neurol Neurosurg Psychiatry. 2006; 77(5): 677–679, doi: 10.1136/jnnp.2005.077073, indexed in Pubmed: 16614034.
10. Kidwell CS, Burgess RE. Dissection syndromes eMedicine; 25 May 2010.
11. Goodfriend SD, Tadi P, Koury R. Carotid artery dissection. In: StatPearls. Treasure Island (FL): StatPearls Publishing; December 24, 2021; 28613585, indexed in Pubmed: 28613585.
12. Sokler K, Sandev S. New classification of the styloid process length-clinical application on the biological base. Coll Antropol. 2001;25(2):627-632, indexed in Pubmed: 11811294.
13. Bokhari MR, Graham C, Mohseni M. Eagle Syndrome. In: StatPearls. Treasure Island (FL): StatPearls Publishing; July 10, 2022, indexed in Pubmed: 28613540.
14. EAGLE WW. Elongated styloid process; symptoms and treatment. AMA Arch Otolaryngol. 1958; 67(2): 172–176, doi: 10.1001/archotol.1958.00730010178007, indexed in Pubmed: 13497389.
15. Subedi R, Dean R, Baronos S, et al. Carotid artery dissection: a rare complication of Eagle syndrome. BMJ Case Rep. 2017; 2017, doi: 10.1136/bcr-2016-218184, indexed in Pubmed: 28288997.
16. Razak A, Short JL, Hussain SI. Carotid artery dissection due to elongated styloid process: a self-stabbing phenomenon. J Neuroimaging. 2014; 24(3): 298–301, doi: 10.1111/j.1552-6569.2012.00759.x, indexed in Pubmed: 23163559.

17. Sveinsson O, Kostulas N, Herrman L. Internal carotid dissection caused by an elongated styloid process (Eagle syndrome). BMJ Case Rep. 2013; 2013, doi: 10.1136/bcr-2013-009878, indexed in Pubmed: 23761567.
18. Peng J, Liu Z, Luo C, et al. Treatment of cervical artery dissection: antithrombotics, thrombolysis, and endovascular therapy. Biomed Res Int. 2017; 2017: 3072098, doi: 10.1155/2017/3072098, indexed in Pubmed: 28607929.
19. Engelter ST, Brandt T, Debette S, et al. Cervical Artery Dissection in Ischemic Stroke Patients (CADISP) Study Group. Antiplatelets versus anticoagulation in cervical artery dissection. Stroke. 2007; 38(9): 2605–2611, doi: 10.1161/STROKEAHA.107.489666, indexed in Pubmed: 17656656.
20. Rao AS, Makaroun MS, Marone LK, et al. Long-term outcomes of internal carotid artery dissection. J Vasc Surg. 2011; 54(2): 370–4; discussion 375, doi: 10.1016/j.jvs.2011.02.059, indexed in Pubmed: 21620626.