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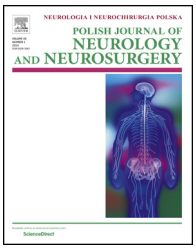

Case report

Traumatic bilateral dissection of cervical internal carotid artery in the wake of a car accident: A case report

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ABSTRACT

Background: Bilateral carotid artery dissection secondary to severe trauma is rare and can be potentially life -threatening if not diagnosed and treated properly.

Case Presentation: We report a 29-year-old female who was admitted to the emergency department after a car accident. The patient was conscious at the time of admission and presented with an initial Glasgow Coma Scale (GCS) of 15 presenting normal vital signs. The patient developed motor dysphasia with right upper limb paresis a few hours after the admission. Magnetic resonance imaging (MRI) revealed a bilateral cervical internal carotid artery (ICA) occlusion in addition to left frontal lobe infarct in a subacute phase. Medical management was successful and the patient was discharged from the hospital two weeks after the admission.

Discussion: Noninvasive vascular imagining modalities are merging as the gold standard in the early detection of carotid artery dissection (CAD). Typical pathognomonic findings on MRI include double lumen and intimal flap. The management with systemic anticoagulation or antiplatelet therapy is aimed to prevent the development of ischemic stroke. In case of medical therapy being ineffective or in case of complication or any disorders suffered by a patient, endovascular treatment is performed.

Conclusion: With early detection and proper management, traumatic dissection of cervical carotid artery can have a benign outcome. As for the current patient, medical treatment with anticoagulation was sufficient and surgical management was therefore not required. Improvement in the patients' speech was observed; nevertheless the continuation of speech therapy was indicated.

1. Background

Carotid artery dissection (CAD) is a serious fatal condition accounting for up to 25% of cerebral ischemia in young and middle-aged patients [1,2]. Permanent neurological deficit rate ranges from 12.5% to 80% with long-term mortality occurring in up to 40% in all cases [3–5]. CAD is rare and uncommon where the annual incidence is estimated to be around $2.6/10^5$ [3]. The majority of the dissections are considered spontaneously without antecedent injuries where the remainders occur as a result of severe trauma, with the latter accounting for 0.86% of all cases [5,6]. Motor vehicle accidents are the most

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common cause of blunt traumatic injury to the carotid arteries with a rate of 69% of all traumatic CAD [6]. Sports accidents, fights and falls are also indicated as possible causes of traumatic CAD [5]. Interestingly, CAD may occasionally be observed bilaterally with an incidence of 2–10% of all CADs [3,5]. The present report describes a rare case where a young patient develops a traumatic bilateral carotid artery dissection in the wake of a car accident.

2. Case presentation

A previously healthy 29-year-old woman was admitted to the emergency department after a car accident. The patient was conscious and responsive at the time of admission and had an initial Glasgow Coma Scale (GCS) of 15. No neurological deficits were detected. No posttraumatic changes were found in chest or abdomen. Hypokalemia was the only abnormal finding revealed during routine laboratory studies. As part of trauma examination a chest X-ray was obtained and revealed normal lung tissue without any sign of fluid accumulation or fractures. An axial noncontrast computed tomography (CT) of the head and neck were performed with slice thickness of 2.5 mm (Fig. 1). The brain tissue showed no focal lesions with unremarkable ventricles, although the shortness of brain sulci of the left hemisphere was present. Moreover, bones of both the cranium and cervical column had no posttraumatic changes except a slight disc herniation with spinal compression at the level of C4–C5.

About 8 h after the admission, the patient developed motor dysphasia with right upper limb paresis assessed as 4 points in the National Institutes of Health Stroke Scale (NIHSS), which raised suspicion related to focal changes of the left hemisphere. MRI without contrast revealed ischemia of the brain parenchyma in the left pole of the frontal lobe (25 × 40 mm) and in the posterior part of the left frontal lobe (86 × 26 mm). This finding corresponds with foci of infarct in a subacute phase. MRI with contrast was performed with the use of 1.5 T Avanto system (Siemens). Techniques of Turbo Spin Echo with Fat Saturation (TSE FATSAT) and DIXON sequences were used to obtain T1 and T2-weighted images in three views; coronal, sagittal, and axial. The fat-saturated T1-weighted images disclosed a bilateral occlusion of the carotid arteries. On the right side there was observed, the dissection 45 mm above the bifurcation of the common carotid artery (CCA) where an intramural thrombus almost completely filled the vessel lumen leaving only a 2 mm patent canal of blood flow (Figs. 2 and 3). This finding is described as a string sign or rat-tail in literature. The dissection of the right internal carotid artery (RICA) encompasses the carotid siphon and terminates just below its intracranial division. Similar findings were present on the left side where both, the dissection and the thrombus were observed in the extracranial division up to carotid siphon. These changes are hyperintensive in T2 and FLAIR MR images, due to the restriction of diffusion, without contrast enhancement. The remaining brain structures are shown without any focal changes. The ventricle system showed no dislocation or enlargement and no signs of increased intracranial pressure.

Medical management of the patient started immediately. Intravenous (IV) infusion of fluids was administered in order to maintain the blood pressure while mannitol, an osmotic diuretic, was given to decrease the intracranial pressure.

According to Biffl and Colleagues scale, the presented patient is categorized into grade IV [7]. Thus, anticoagulation therapy was immediately started.

Enoxaparinum natrium (Clexane), a low-molecular weight heparin (LWMH), was started to stop the thrombotic process and to prevent further emboli formation. Let us note that,

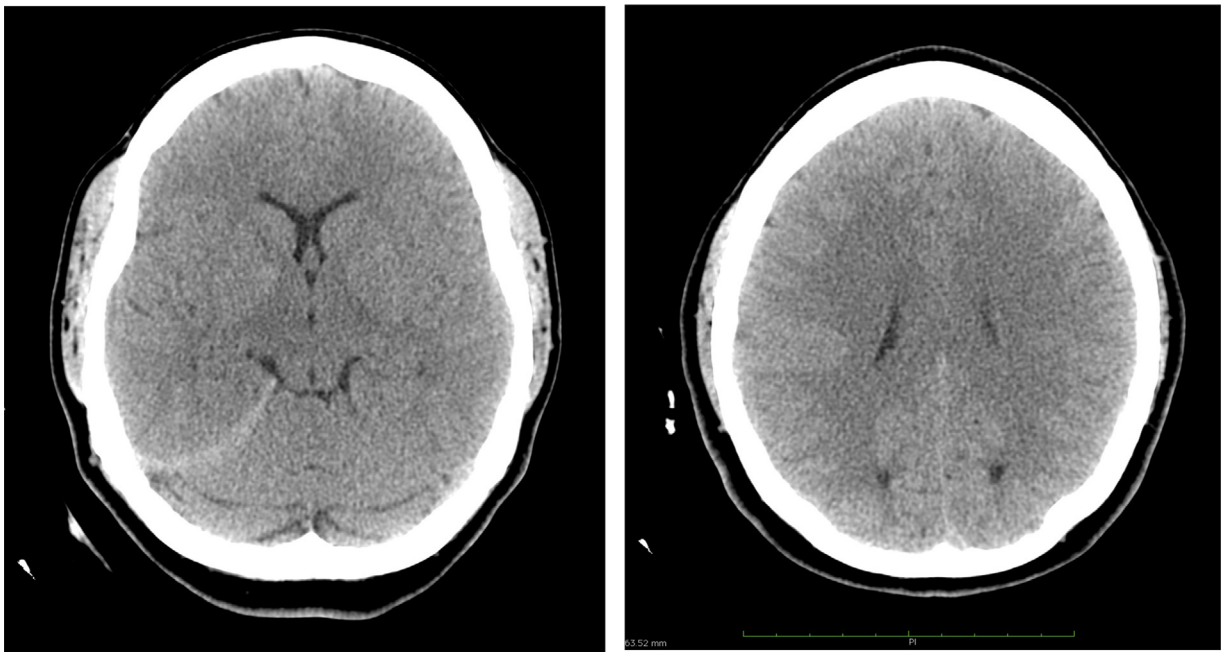

Fig. 1 – Axial non-contrast CT demonstrates brain without focal lesions and with no signs of intracranial hemorrhage. However, hematoma of soft tissues of left parietal area is present.

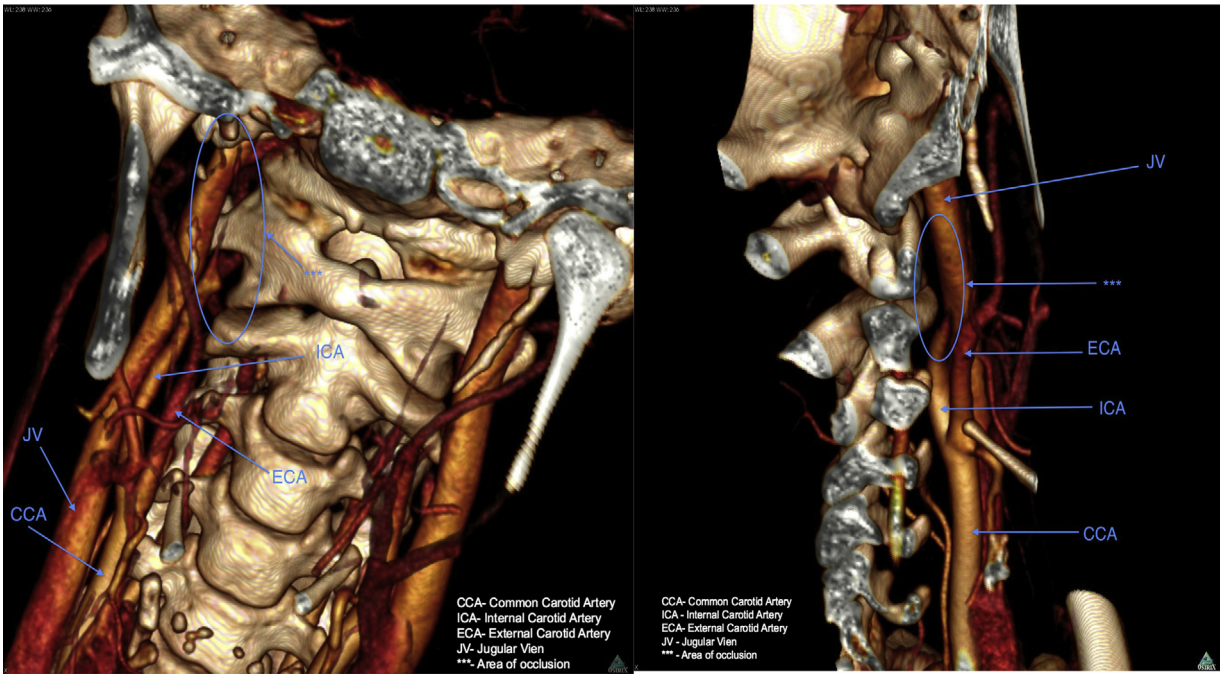

Fig. 2 – 3D-CTA showing bilateral occlusion of the internal carotid artery in anterior and lateral view. CCA: common carotid artery, ICA: internal carotid artery, ECA: external carotid artery, JV: jugular vein, *: area of occlusion.**

Potassium Chloride (KCl) infusion was added to the treatment regime to correct the hypokalemia in order to avoid any cardiac complications. To alleviate pain, an opioid (Dolargan) and acetaminophen (Pyralgin and Perfalgan) were given administered at the same time with a proton-pump inhibitor (PPI) as prophylaxis of acute peptic ulceration.

Moreover, a change was made from subcutaneous (SC) LMWH to oral warfarin after 7 days in order to maintain the long-term prevention of thromboembolism. The ischemic stroke affected the patient's Broca area and caused the motor dysphasia which was further evaluated by a speech-language pathologists (SLPs). Both, the motor dysphasia and the upper limb paresis had improved during the hospital stay of the patient. Two weeks following admission, the patient was discharged home with only minor dysphasia – 1 point in NHSS. Continuation of oral warfarin treatment for the next 6 months together with follow-up physiotherapy and speech therapy was recommended.

3. Discussion

3.1. *Pathogenesis*

CAD following a motor vehicle accident is thought to be associated with the mechanical tearing of the vessel due to the sudden hyperextension, hyperflexion or excessive lateral flexion of the neck [8]. The sudden extreme movements are responsible for stretching of the neck's vascular ties. The extracranial portion of the internal carotid artery (ICA) is the most vulnerable, because it is freely movable along the neck and its location at the anterior surface has no bony protection [2]. Once mechanical tearing of a vessel's intima takes place, blood may enter a secondary lumen forming an intramural hematoma and furthermore interfere with the blood flow in the primary lumen. Taken together, this may result in ischemic changes of the brain parenchyma [2]. Moreover, blood stasis in the secondary lumen will eventually lead to thrombotic formation and generate the risk for developing an embolic episode in smaller arteries of the brain [8]. Transient ischemic attack (TIA) and strokes are a common neurological finding in patients with Internal Carotid Artery Dissection (ICAD) with a rate of 49–84% of all patients [9]. They are mainly categorized into the embolic origin rather than those related to carotid artery stenosis by the false lumen and their prevalence is estimated with 92.2% and 7.7% respectively [2]. In 56% of patients, neurological symptoms related to ischemic lesions will appear immediately or within the first 24 h following trauma, while in the reminding cases symptoms develops in the longer run [5].

3.2. *Clinical presentation*

Diagnosis of CAD is frequently delayed due to the absence of initial signs and symptoms in majority of patients [6]. However, when symptoms precede the ischemic symptoms, headache and neck pain present as the most frequent clinical manifestation [10]. It is thought that pain occurs as the effect of hematoma causing arterial distension and further stimulation of the pain-sensitive receptors [2]. Therefore, it is believed that headache may appear as a localized warning sign, and if investigated on time, can offer a chance to treat the patient prior to development of TIA or cerebral ischemia, which is the most common neurological manifestation of ICAD [2,10].

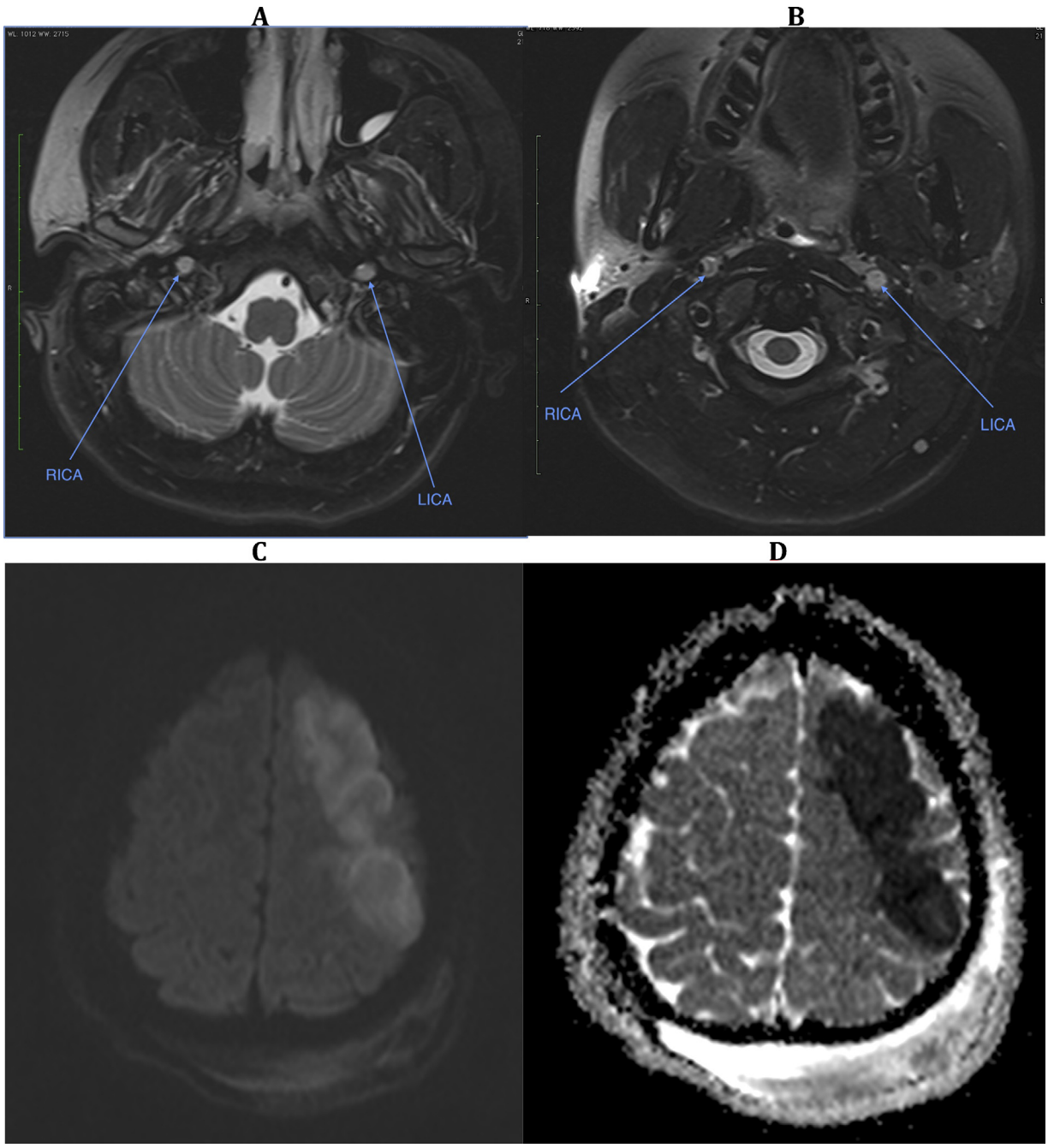

Fig. 3 – (A and B) MRI with technique of TSE FATSAT shows bilateral occlusion of carotid arteries. (C) T1 weighted axial MRI showing parenchymal changes corresponding with foci of infarct in subacute phase. (D) DWI ACE map is showing focal changes of the brain parenchyma in the left pole of the frontal lobe and in the posterior part of the left frontal lobe. RICA: right internal carotid artery, LICA: left internal carotid artery.

However, in many cases, an asymptomatic ICAD will result in delayed diagnosis, which will take place following the neurological symptoms related to the ischemic stroke [10].

3.3. *Radiological findings*

Secondary carotid artery dissection in the wake of severe trauma is rare and can be potentially life-threatening if not diagnosed and treated properly. Conventional angiography was once the golden standard in the diagnosis of pathological changes of vascular lumen. Nowadays, vascular imagining modalities including MRI and CT are preferred and hence becoming more popular in the early detection of CAD. In order to detect an ischemic brain lesion and its underlying cause, we should use a technique that allows for the visualization of the brain parenchyma with the supplying vessels. This can easily be obtained when performing a combination of CT and CTA or MRI and MRA [8,9,11].

3.3.1. *Angiography*

Conventional angiography, which is an invasive procedure, used to be the preferred method in diagnosing CAD. However, recently it has been replaced by noninvasive techniques for both primary assessment and follow-up [3,11]. The pathological changes observed on angiography including double lumen and intimal flap were only present in less than 10% of all ICAD cases [2,11]. Arterial stenosis, arterial occlusion and aneurysm formation are more common detectable angiographic changes, which constitute 41–75%, 18–49%, and 5–13%, respectively [2,11]. Arterial lumen and vessel wall irregularities are demonstrated in patients with ICAD; however, angiographic examination does not allow for direct vessel wall visualization in the presence of thrombus in a false lumen [11].

3.3.2. *Ultrasonography*

Color duplex ultrasound (CDU), which is a noninvasive, fast, easily implemented at the patient's bedside, free from radiation, inexpensive and reliable diagnostic tool is estimated to have an 86% detecting rate of ICAD [6,12]. It provides real time blood flow visualization with calculated blood velocity and resistance. Therefore, it can be used to estimate the risk of developing an ischemic brain lesion [2]. The pathological appearance of ICAD on ultrasound include intimal flap, floating thrombus, intramural hematoma and high resistance blood flow, which however, strongly suggest the presence of ICAD [3,2,11]. In addition to the aforementioned findings, ultrasound (US) examination can also visualize the false lumens even in the presence of thrombus [11]. In order to evaluate the recanalization or progression of the occluded vessels, serial monitoring with US is required [6]. Compared with other radiological methods, US examination does not require contrast medium, and is therefore the method of choice when contrast injection is contraindicated [11].

3.3.3. *Computed tomography*

Multisection computed tomography angiography (CTA) could provide both high-resolution and high-contrast images of the vascular system [11]. Compared to conventional angiography, CTA demonstrated 70% sensitivity, 67% specificity, and a positive and negative prediction value of 65% [6]. However, a recent paper showed that CTA achieved 100% specificity and sensitivity for stenosis and occlusion detection [11]. Typical pathological changes of ICAD demonstrated on CT scans include narrowed eccentric vessel lumen surrounded by mural thickening with an intimal flap or dissecting aneurysm [3,11]. Stenosis, occlusion, and thin annular contrast enhancement are additional findings of ICAD [11].

Three-dimensional CT angiography (3D-CTA) is the method of choice for evaluation of the arterial stenosis, which enables reconstruction of the carotid arteries. 3D-CTA in relation to cervical vascular pathology, provide a better presentation of the stenotic level and of the dissection site. Because 3D-CTA is both rapid and minimally invasive, it is suitable for regular follow-up examination [13].

A combination of non-contrast CT and CTA is the predominant diagnostic tool for the early detection of cerebrovascular changes, especially in relation to severely injured patients in the emergency room [8]. This is because CT can provide information at the acute phase of the dissection, which may be presented as hypointense on MRI, and therefore not accessible until a later stage. Good renal function is required for CTA, where contrast medium is administrated in order to visualize the vascular structures [9].

3.3.4. *Magnetic resonance imaging*

Nowadays, MRI and MRA are considered to be the gold standard technique for diagnosis of craniocervical arterial dissections with a sensitivity and specificity equal or better than CT and allows for a noninvasive visualization of the vessel walls [8,7]. Diffusion-weighted imaging (DWI) and perfusion-weighted imaging (PWI) are two different modes of MRI, which are helpful in detecting acute ischemic lesions and areas of hemodynamic compromise [11]. ICAD presents with typical findings on MRI/MRA, which include increased external diameter of the artery, luminal narrowing, aneurysms, and intramural hematoma [3]. On MRI, the intramural hematoma will appear as eccentric, crescent-shaped, hyperintensive area within the arterial lumen in addition to vascular expansion [2]. Furthermore, it is essential to mention that false negative diagnosis can be made following early MRI because the acute intramural hematomas appear as hypotensive on T1 and T2 weighted images and can therefore be easily missed [11].

The hematoma will appear on the following days with signal intensity on T1 weighted images for approximately 2 months before it becomes unrecognizable after 6 months [11]. Apart from the great advantages of MRI, there are a few limitations, which include time and money consumption, around the clock availability, accessibility in medical centers, and the presence of false negative results in the early course of the CAD [8].

3.4. *Prognosis and treatment*

ICDA is directly related to CNS complications due to disrupted blood perfusion causing either hemodynamic changes or formation of thrombus and emboli [7]. Therefore, the management of such patients relies on the prevention or progression cessation of the accompanied neurological deficits [11]. Brain edema is one of the early sings of brain injury and may lead to serious complications such as brain herniation causing worse prognosis.

Based on literature reviews, emboli are the major underlying cause of strokes in patients with ICAD, thus anticoagulant and antiplatelet therapy are considered the cornerstone of management, unless contraindicated [3]. The use of anticoagulants therapy at the acute stage of intracranial dissection is controversial since it might promote subarachnoid bleeding. However, a single center retrospective observational study involving 81 patients suggested that intracranial dissection in the absence of aneurysm or subarachnoid bleeding (SAH) (based on clinical and brain CT/MRI findings) can be safely treated with anticoagulants [14]. The risk of SAH is higher in intracranial compared with extracranial artery dissection, and several studies have reported patients with intracranial artery dissection with initial ischemic manifestations who subsequently or concurrently developed SAH. The choice of antithrombotic therapy in patients with intracranial artery dissection without SAH has been evaluated neither in

randomized controlled trials nor in meta-analyses of observational studies. However, our patient presented bilateral total occlusion of ICA mainly in extracranial segments, so we decided to initiate anticoagulation in this case.

The regimen usually starts with SC LMWH for 7 days followed by 3–6 moths of oral warfarin or aspirin [11]. In case of a relative contraindication of heparin as mentioned above, administration, aspirin or clopidogrel are possible substitutions [7]. In a paper published by Biffl et al., 71% of patients who were treated with a systemic heparin showed better outcomes [9]. On the other hand, long-term administration of anticoagulants and antiplatelets may increase the risk of internal bleeding [2]. Thus, it is recommended to keep the therapeutic levels of heparin within aPTT of 50–70 s and warfarin with a target international normalized ratio (INR) of 2.0–3.0 for 3–6 months [8,10]. There are only a few researches, which indicates that NOAC can be safe and reasonable alternative in the treatment of patients with ischemic stroke due to ICAD compared with the treatment using vitamin K antagonists (VKAs) [15,16]. Thus, using novel anticoagulants in cervicocerebral dissection patients seems reasonable, however, further studies are required. In the treatment of this patient, we decided to use classic vitamin-K antagonist, as it is the most popular therapy of artery dissection in Poland.

Currently, there is no control study demonstrating a superiority of anticoagulants use over antiplatelets and vice versa [2]. Anticoagulants are more expensive, need continuous monitoring in order to keep coagulation parameters within the therapeutic range and may affect the patient's lifestyle. Therefore, antiplatelets, seems to be the better option in the course of a long-term treatment, unless another study shows an advantages of anticoagulants over them [17].

However, pharmacological management is not always sufficient to manage CAD and surgical interventions are then indicated. Surgery is also preserved for complicated presentations of CAD such as: expanding pseudoaneurysm or sever stenosis in addition to those with contraindications to the pharmacological treatment [18]. Different surgical approaches could be introduced to regain the ICA potency including ICA ligation, clipping, venous graft bypass, and endovascular stenting [2]. The latter option has the advantage over prior ones when it comes to auditory and facial disorders [5]. Immediate recanalization and obligations of the secondary lumen are another advantages of endovascular stenting. However, it carries a risk of embolization of thrombotic fragments during stent placement and reperfusion injuries in about 5.2% of implantations [3]. Those surgical procedures are difficult with an associated high risk in acute injuries. They are therefore not considered to be the first line treatment unless pharmacological management fails [8].

4. Conclusion

In summary, approximately 4% of all CADs result from trauma; however, it is an important cause of ischemic stroke, particularly in younger population. Extremely rarely, traumatic CAD will develop bilaterally as in the aforementioned case. The diagnosis of CAD becomes faster and easier due to the recent advances in noninvasive vascular imaging techniques. However, the lack of specific presentation delays the diagnosis until the occurrence of neurological symptoms caused by the ischemic stroke. Considering the early detection and treatment as well as proper management of the patient there was observed the improvement of the patient's symptoms.

Conflict of interest

None declared.

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