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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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1. Background

Carotid artery dissection (CAD) is a serious fatal condition Q2 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 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

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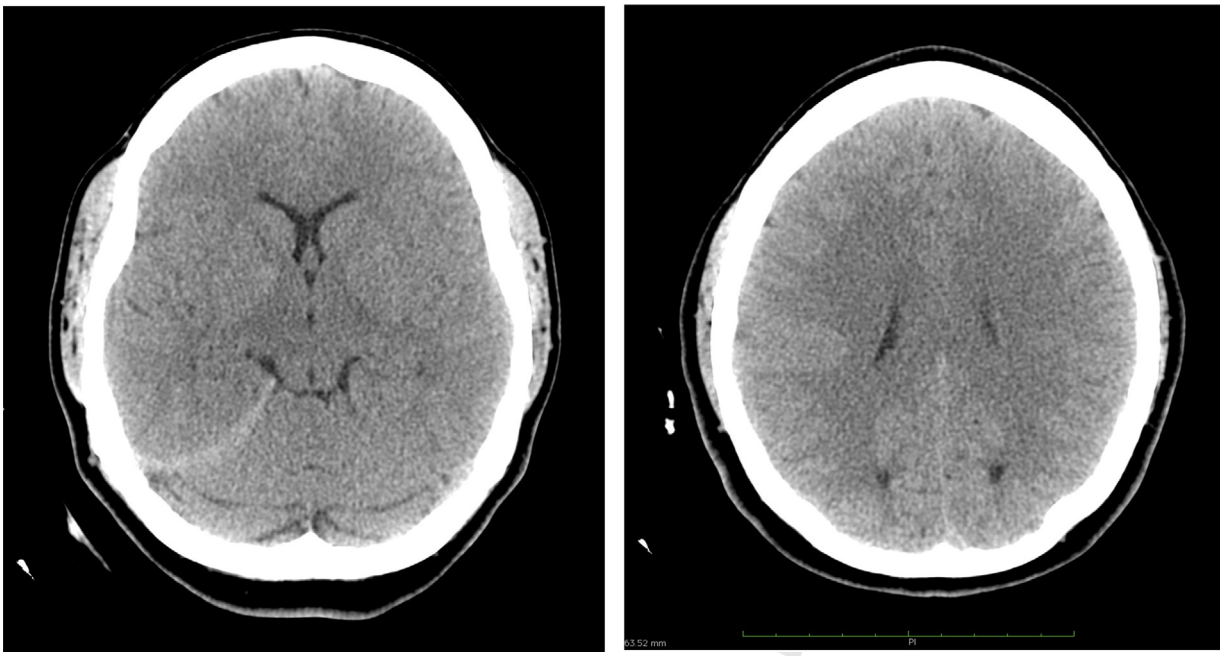

Fig. 1 – Axial non-contrast CT demonstrates brain without focal lesions and with no signs of intracranial hemorrhage. However, hematoma of soft tissues of left parietal area is present.

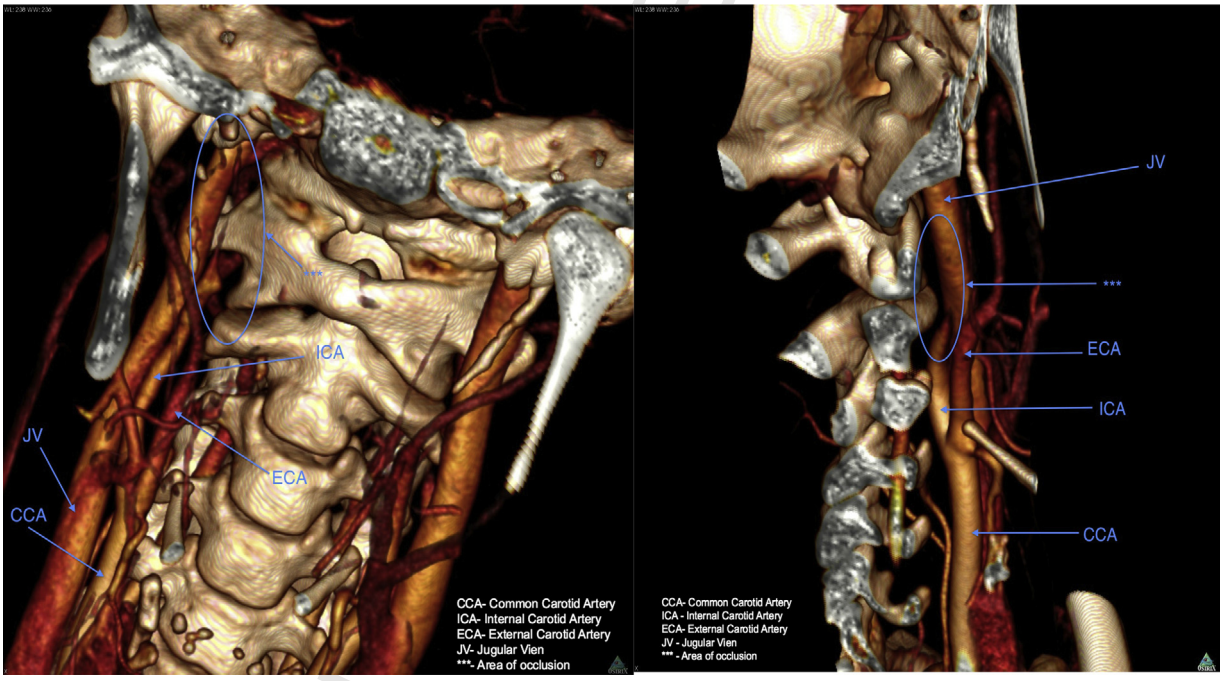

Fig. 2 – 3D-CTA showing bilateral occlusion of the internal carotid artery in anterior and lateral view. CCA: common carotid artery, ICA: internal carotid artery, ECA: external carotid artery, JV: jugular vein, *: area of occlusion.**

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

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