Spontaneous Carotid Artery Dissection Presenting with Headache

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Introduction
Carotid artery dissection (CAD), an infrequent cause of transient ischemic attack and stroke among younger patients, is being increasingly diagnosed (1). The incidence of internal CAD ranges from 2.5 to 3 per 100,000 (2). The consequences of spontaneous dissection are critical as it may induce stroke in young patients. Thromboembolic events occur following carotid artery dissection due to the narrowing of the lumen, which is a potentially life-threatening condition that can cause long-term neurological disabilities. We report a case with internal carotid artery dissection and emphasize the importance of differential diagnoses for headache and neck pain in the young and middle-aged population.

Case Report
A 33-year-old female patient was admitted to the emergency department because of headache, neck pain, speech impairment, and weakness on her right side, which persisted for five hours. She had revealed signs of confusion for the last two hours. There was no history of trauma. We received the information from her husband. The neurological examination revealed a weakness on the right upper extremity and non-fluent aphasia. She had normal results from coagulation tests. Occlusion secondary to dissection with a cervical computed tomography angiography was indicated. Low molecular weight heparin and warfarin were administered.

Conclusion: It should be kept in mind that carotid artery dissection can be asymptomatic until ischemic symptoms occur. Emergency physicians should consider carotid artery dissection in young patients who present with neck pain and headache.

Keywords: Carotid artery, dissection, stroke

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ABSTRACT

Introduction: Carotid artery dissection, an infrequent cause of transient ischemic attack and stroke among younger patients, is being increasingly diagnosed. Thromboembolic events occur following carotid artery dissection due to the narrowing of the lumen, which is a potentially life-threatening condition that can cause long-term neurological disabilities. We report a case with internal carotid artery dissection and emphasize the importance of differential diagnoses for headache and neck pain in the young and middle-aged population.

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hypertension, diabetes, and stroke. We received the information from her husband. Cardiac and pulmonary auscultations were normal, and vital signs were otherwise within normal limits. There was no sign of arterial injury or any sign of cervical hematoma in her neck inspection. The neurological examination revealed weakness on the right upper extremity and non-fluent aphasia. Routine biochemical profile and urine analyses were within normal ranges. A non-contrast computed tomographic scan of her head did not reveal any pathological changes. There was a subtle restricted diffusion in the left centrum semiovale that is minimally hyperintense on the diffusion-weighted (DW) trace image and slightly restricting diffusion on the apparent diffusion coefficient (ADC) map (Figure 1a, b). Additionally, there was an absence of flow signals in the intracranial segment of the left internal carotid artery (ICA) and hyperintense signals and expansion in the distal cervical ICA (Figure 1c). A cardiac monitor had been placed, and intravenous access had been established for follow-up. Her vital signs were stable during the examination. There were no changes in the examination during the 6-h monitoring period, but the follow-up DW-MR scan highlighted ischemic changes in the territory of the left middle cerebral artery (MCA) (Figure 2a, b). Occlusion secondary to dissection with a cervical computed tomography angiography was indicated (Figure 2c). Raw-data axial images of the time of flight (TOF) angiography indicated the true lumen was compressed in the lateral part, and the three-dimensional (3D) maximum intensity projection (MIP) of the TOF indicated a true lumen narrowing secondary to long segment dissection in the cervical part of the left ICA (Figure 3a, b).

FIGURE 1. a-c. DW-MR (a), ADC (b) and T2-weighted axial image (c) show subtle restricted diffusion is present in the left centrum semiovale (long arrows), which is minimally hyperintense on the DW trace image and slightly restricting diffusion on the ADC map, absence of flow signal in the intracranial segment of left ICA, hyperintense signal and expansion in the distal cervical ICA (short arrow)

FIGURE 2. a-c. DW-MR (a), ADC (b) and MDCT angiography sagittal image (c) show hyperintense on the DW-MR in the left MCA territory, restricting diffusion on the ADC map (arrow), oblique sagittal MIP MDCT angiography image reveals luminal contrast filling end tapers from the second centimeter of the proximal ICA secondary to the dissection (red arrow)
The patient, who was not considered for interventional procedures, was admitted to the intensive care unit for 3 days. She was administered low molecular weight heparin and warfarin. After 3 days of anticoagulation therapy, all the symptoms and deficits were completely resolved. The patient had no chest pain or hemodynamic instability during the hospitalization period and was discharged on the 8th day. Hematologic, genetic, and immunologic parameters were within normal limits.
A follow-up neurological examination after 6 months revealed fully normal function, and MR arterial angiography revealed no more any compression or narrowing (Figure 3c, d). The patient filled the inform consent form for his data to be used in this case report.

Discussion
Cervicocerebral artery dissection is increasingly being seen, particularly in young patients presenting with ischemic stroke (3). CAD can either be traumatic or spontaneous. Spontaneous dissections can occur, usually due to a weakness in the vessel wall. Blunt traumatic vascular injuries occur most frequently following impact injuries and hyperflexion of the neck (4). Dissociation occurs from a rupture of the intima-media layer. Distal embolization of the thrombus may cause a transient ischemic attack or ischemic stroke (5). Headache and neck pain are common nonspecific symptoms (1). Presentation of CAD can vary across a wide spectrum, ranging from asymptomatic to massive cerebral infarction. Cerebral blood flow decreases because of compression of the hematoma and ischemic symptoms appear. Furthermore, sympathetic nerve fibers are compressed, resulting in Horner’s syndrome (2, 5). Genetic and environmental factors may play a role in the pathogenesis of ICAD (5). Even mild trauma, coughing, sneezing, exercise, and head turning may induce this disorder (6, 7). In our case, genetic and immunologic parameters were within normal limits. There was no history of trauma. Depending on the localization of dissection, a specific symptom may become prominent, such as neck pain. Horner’s syndrome may be associated with 25% of patients with CAD (8). A close follow-up is imperative for patients who present with atypical symptoms. Although four-vessel digital subtraction angiography (DSA) is the gold standard in diagnosis, multi-detector computed tomography angiography (MDCTA) and MR angiography are increasingly used in the ED. MDCTA has a good sensitivity for stenosis, intimal flap, and pseudo aneurysm (9). Other non-invasive tools, such as color Doppler USG, may be inadequate for precise diagnoses. Because clinical trials do not reveal any difference between antiplatelet and anticoagulant treatment groups, a treatment choice should be made regarding the clinical status of the patient (10).

Conclusion
It should be kept in mind that ICAD can be asymptomatic until ischemic symptoms are established. The emergency room physician should consider ICAD in young patients who present with neck pain and headache.