Delayed Post-Traumatic Anisocoria

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Post-traumatic carotid artery dissection is one of the major causes of ischemic stroke in young patients; its diagnosis remains a challenge for clinicians because of its variable clinical presentation. An otherwise healthy 37-year-old man was referred to the intensive care unit of our faculty for the management of multiple trauma because of a car accident. At 11 days from admission, his doctor noticed the advent of anisocoria. A prompt treatment was instituted with anti-platelet and-coagulant agents. The patient had a complete resolution of symptoms. The prognosis was good, and the patient achieved a complete clinical recovery. He was discharged without any sequelae.

Keywords: Anisocoria, internal carotid artery, dissection

Introduction

Although the symptoms of anisocoria in 10–20% of cases may be physiological, it is known that the symptoms may also show intracranial injury that requires immediate diagnosis and treatment and that such symptoms are alarming for clinicians. Because of cerebral vascular damage, many circumstances, such as aneurysms, mass lesions, head or direct eye traumas, mydriatic drug use and Adie's pupil, may cause anisocoria.

Traumatic cerebral artery dissection is a rare situation that can be overlooked during the acute period. Symptoms may occur after 2–3 weeks (1). The patient we present was admitted to our intensive care unit because of a motor vehicle accident, and on the 11th day of his stay, without any neurological deficits, he developed acute anisocoria and was diagnosed with carotid artery dissection.

Case Presentation

Because of an extravehicular accident, a 37-year-old male patient was brought to the emergency room. During his first examination, he was conscious, cooperative and oriented, and his artery pressure was 110/70 mm Hg, heart rate was 117 beats min⁻¹, respiratory rate was 16 min⁻¹ and temperature was 36°C.

Rale, rhonchus and additional noises were not observed in the lungs, and his abdominal examination was normal. According to the laboratory analysis, the haemoglobin level was 13.7 g dL⁻¹, platelet count was 412 000 mm³, INR was 1.11, serum Na level was 140 mEq dL⁻¹, K level was 4.73 mEq l⁻¹, creatinine level was 0.9 mg dL⁻¹ and Ca level was 8.3 mEq dL⁻¹. The patient had displaced fractures in the left femur and left fibula, and a multisegmental fracture on his left tibia, and the fractures were detected on the C6 spinous process and T2–T4 corpus. His computed brain tomography (CT) did not show any pathology in the parenchyma and other bone tissues.

Because of multiple traumas, he was monitored from the very first day of his hospitalization. He was operated under general anaesthesia for his fractures in the leg, and 6 h after the surgery, he was extubated in the intensive care unit. General anaesthesia was preferred instead of regional anaesthesia because the surgical position was planned to be the lateral decubitus position and the duration of the surgery could have been long. On seeing atelectasis in his chest X-ray in parallel with hypoxia development in postoperative blood gas follow-ups, the patient was put under non-invasive mechanical ventilation.

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Figure 1. Developing anisocoria on the 11th day of hospitalization

On the 11th day of the patient's hospitalization, his physical examination revealed anisocoria, and his field of vision and facial nerves were observed to be normal in the detailed examination. Light reaction was identified as bilaterally weak, and while the indirect light reaction on the miotic side was weak, it was negative on the mydriatic side (Figure 1).

Following anisocoria, because somnolence increased, the patient was administered 6 lt h⁻¹ O₂ via a mask. The patient's neurological examination was normal, and CT detected a lesion compatible with a minimal infarct that fell in the irrigation area of the anterior branch of the right middle cerebral artery (Figure 2). Similar to the CT findings, bedside ultrasound imaging detected an image on the same side of the internal carotid artery wall's bulbous lumen that may be consistent with dissection or thrombus (Figure 3). In total, 300 mg day⁻¹ acetylsalicylic acid was added to his 2 x 0.4 U of enoxaparin treatment.

The patient with stable haemodynamic, respiratory and neurological assessments was transferred to the neurology service for further evaluation and treatment. Fifteen days after the trauma, he was discharged healthy.

Discussion

Blunt trauma of the carotid artery is a rare condition. It is reported that carotid trauma is observed in 0.08–1.03% of head trauma cases (2, 3). Ranging from 53% to 82%, the primary reason of these traumas that lead carotid artery dissections is reported to be motor vehicle accidents (2). In only 10% of trauma-induced cerebral artery dissection, symptoms are quickly noticed. Situations characterized by anisocoria, such as neck swelling, local tenderness or Horner's syndrome, can result in a prompt diagnosis. However, except the neck, just like in our case, it can be entirely normal. Ischaemic attacks are often seen during early stages, but they may develop during the later period as well (4). While prognosis in younger patients and extracranial carotid artery dissections is better, in intracranial dissections, the mortality rate is as high as 75% (5).

In majority of cases that do not show neurologically pathological findings, a diagnostic image on CT may not be seen (6). Our patient was also evaluated to be normal when his CT images were first examined, and after a detailed examination, uncommon infarct symptoms were detected in the feeding areas of the internal carotid artery. Apart from acute onset anisocoria, another reason causing difficulty in the diagnosis is the absence of a pathological neurological examination

Figure 2. Infarct-compliant CT image in the right middle cerebral artery (MCA) irrigation area

Figure 3. Common carotid artery filling the defect in the lumen (dissection or thrombus)

finding. Carotid artery imaging with bedside ultrasound examination appears to be one of the intensive care practices as a diagnostic method as it supports and accelerates the process.

If there is no contraindication in terms of thrombosis risk in acute treatment, anticoagulation is recommended. Studies have shown that following heparin treatment during the acute phase, oral warfarin use reduces the risks of stroke and complications (7). Despite anticoagulant therapy, for persistent ischaemic patients or patients in whom anticoagulant agent use is contraindicated, surgical treatment or stenting may be indicated (8). It is suggested that low molecular weight heparin therapy that we had started during the early postoperative period prevented probable major ischaemic lesions. We thought that 300 mg/day aspirin added to the treatment after the definitive diagnosis would provide adequate anticoagulation treatment.

It is generally not easy to detect a carotid artery dissection that may develop during trauma. Although typical carotid artery dissection symptoms may appear during the early period, as it was experienced in our case, they can appear during the late period as well.

We believe that despite supporting the patient by means of radiological exposure of small infarction areas, the reasons for the absence of severe neurological deficits are the patient being young, early diagnosis of anisocoria and rapid initiation of anticoagulation during the early postoperative period.

Studies show that during the follow-up period of blunt trauma-induced internal carotid arteries dissections, in case of alarming situations such as Horner's syndrome and presence of asymmetric ptosis and new manifested-focal deficits, an immediate CT imaging is the right choice to make a diagnosis (9, 10). In our case, on the other hand, angiography was not performed because of the absence of ischaemic symptoms, early detection of thrombus or dissection and absence of growth in infarct areas in the control CT.

Conclusion

Bedside ultrasound applications are being increasingly used in intensive care units, and they assist in diagnosing possible pathologies in a fast and practical way. However, it should be noted that even if there is no neurological deficit development, in spine and/or head trauma patients, in patients with vascular dissection or thrombus formation-induced high-risk infarction development, diagnosis should be reinforced with advanced imaging techniques such as CT.

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