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Traumatic Basilar Artery Dissection and Hypertrophic Olivary Degeneration

CASE REPORT

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ABSTRACT

Head trauma is an important health issue during childhood and is the most frequent cause of mortality and morbidity during this period. Ischemic cerebral infarct following minor head traumas is seen at regions supplied by the middle cerebral artery, while the vertebra–basilar system is involved to a lesser degree. In this manuscript, we presented the case of a 7-month-old boy who presented with hemiparesis following head trauma, in which basilar artery dissection-related infarction and hypertrophic olivary degeneration were detected.

Keywords: Minor head traumas, basilar artery dissection, hypertrophic olivary degeneration

INTRODUCTION

Head trauma is an important health issue during childhood. It is the most frequent cause of mortality and morbidity during this period (1, 2). The annual rate of hospital presentation due to head trauma has been reported to be 12% (2). Minor head traumas (MHTs) comprise 70–80% of all head traumas (1, 2). As the head is proportionally bigger and the neck tissue supporting the head is weaker in children, the likelihood of incident cerebral infarct after MHTs is higher in the pediatric population than in adults. However, it is still a rare entity during childhood (2). Ischemic cerebral infarct following MHT is seen at regions supplied by the middle cerebral artery, while the vertebra–basilar system is involved to a lesser degree (2, 3).

Hypertrophic olivary degeneration (HOD) is a rarely encountered condition (4). It is often related to either dentate nucleus or contralateral cerebral pedicle and ipsilateral central tegmental tract lesions (5-9). This region is known as the triangle of Guillain and Mollaret (5). In literature, HOD has been reported to occur following infarction, hemorrhage, trauma, or tumor (5-9). It is thought that a pathological process is induced by the reduction of synaptic impulse to the olivary nucleus or transsynaptic degeneration. It has been reported that pathological olivary proliferation onset occurs 3 weeks later with a maximum duration of 8.5 months and that atrophy occurs several years later (9). In this manuscript, we report the case of a patient who with hemiparesis following head trauma, in which basilar artery dissection–related infarction and HOD were detected.

CASE REPORT

It was found out in the history that a 7-month-old boy had fallen from a height of 45 cm and that his mother brought him to a hospital 12 hours after the fall as she had recognized irritability in her son. Brain computed tomography (CT) was performed and reported as normal in the first center; the patient was brought to our hospital as his parents had recognized fixed leftwards gaze and weakness at the left upper and lower extremities. On physical examination, findings were as follows: body weight, 9000 g (75–90%); height, 70 cm (75–90%); head circumference, 44.5 cm (50%); normotensive anterior fontanella, 2x1 cm in size; heart rate, 136 bpm; systolic blood pressure, 90 mmHg; diastolic blood pressure, 60 mmHg; respiration rate, 36/min; fixed leftward gaze, isochoric pupils, bilateral positive light reflex, muscle strength of 2/5 left upper and lower extremities. Fundus examination was normal. No fracture was observed on the cervical vertebra radiograph. Cranial CT, which was performed within 12 h following trauma, was considered to be normal (Figure 1). On cranial magnetic resonance imaging (MRI) performed 24 h after the trauma (Figure 2), hyperintense signal changes consistent with infarction were observed at the cerebellum and brainstem (being more prominent at the right side; Figure 2a-c), and at the bilateral cerebellar hemispheres and pons on axial T2-weighted images. On diffusion-weighted images, limited diffusion (Figure 2d-f) was observed at the same areas; in addition, there was a focal lesion consistent with infarction, displaying limited diffusion adjacent to the corpus callosum at the right side, which was not observed on the T2-weighted images. Complete blood counts, lipid profile, and plasma and urinary homocysteine levels, which were performed to as-

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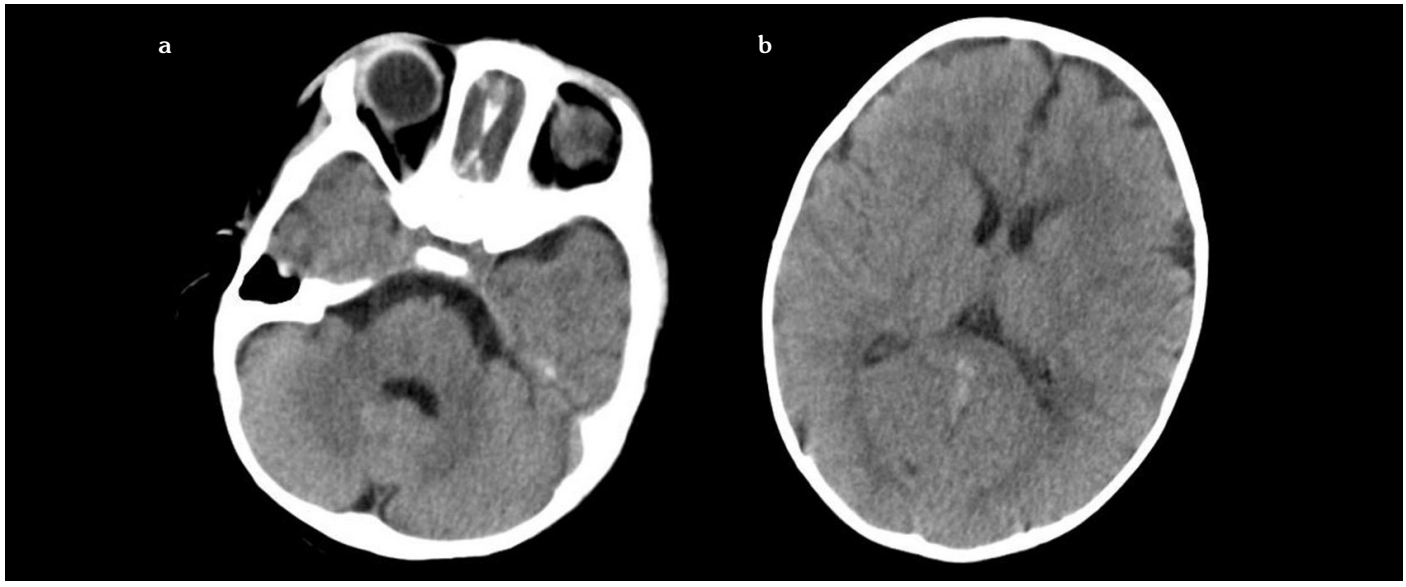

Figure 1. a, b. Axial non-enhanced CT images at the level of the pons (a) and lateral ventricles (b) does not show any abnormality
CT: computed tomography

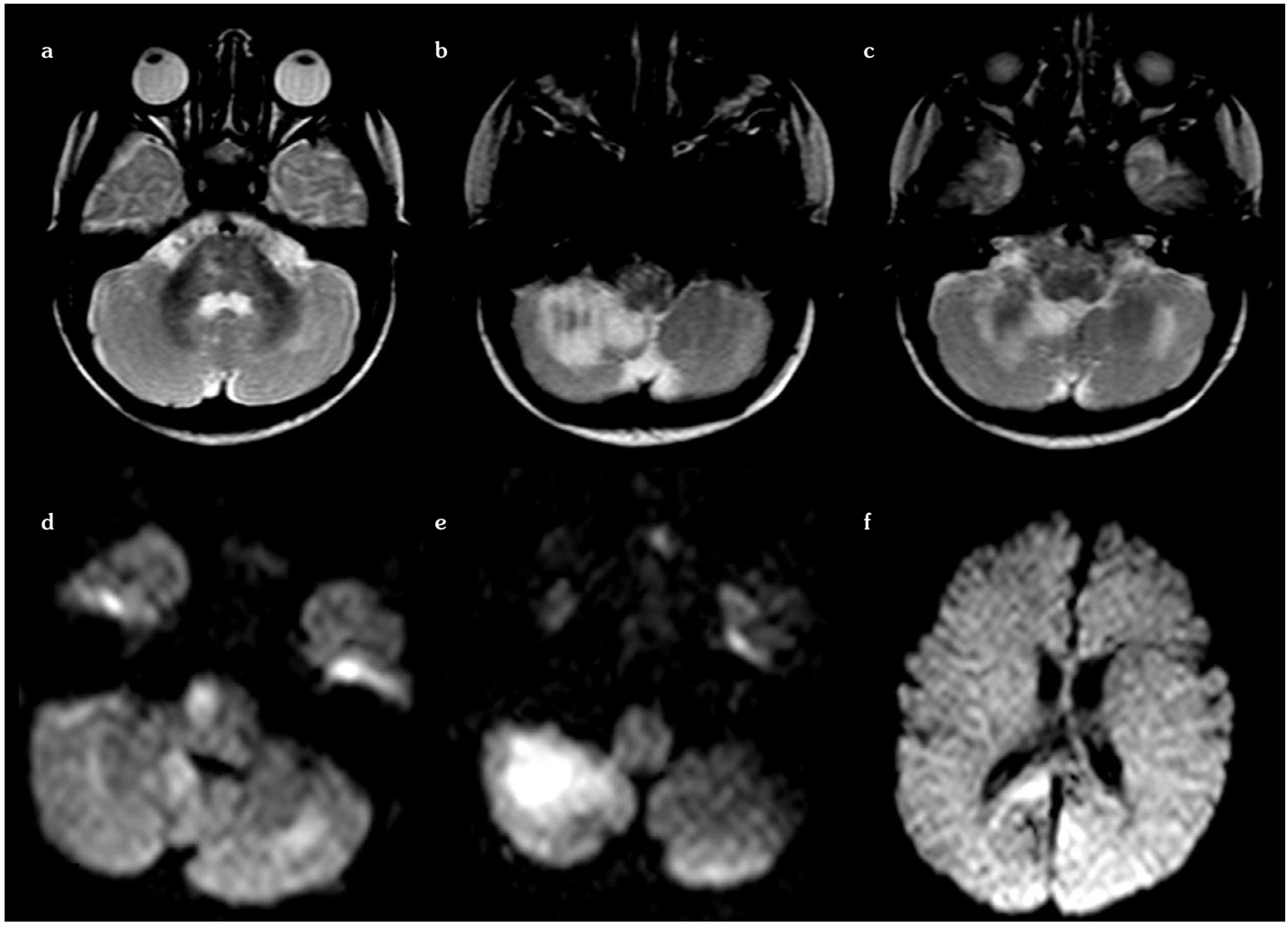

Figure 2. a, f. Axial T2-weighted images at the level of the cerebellum and brain stem (a-c) show hyperintense signal changes in the bilateral cerebellar hemispheres, which is predominant on the right side and in the pons, consistent with infarction. Diffusion-weighted images (b=1000) (d, e) reflect these changes as restricted diffusion. Besides, another focal area of infarction is demonstrated on the right side of the splenium of the corpus callosum (f) on diffusion-weighted image, which is not appreciated on T2-weighted images

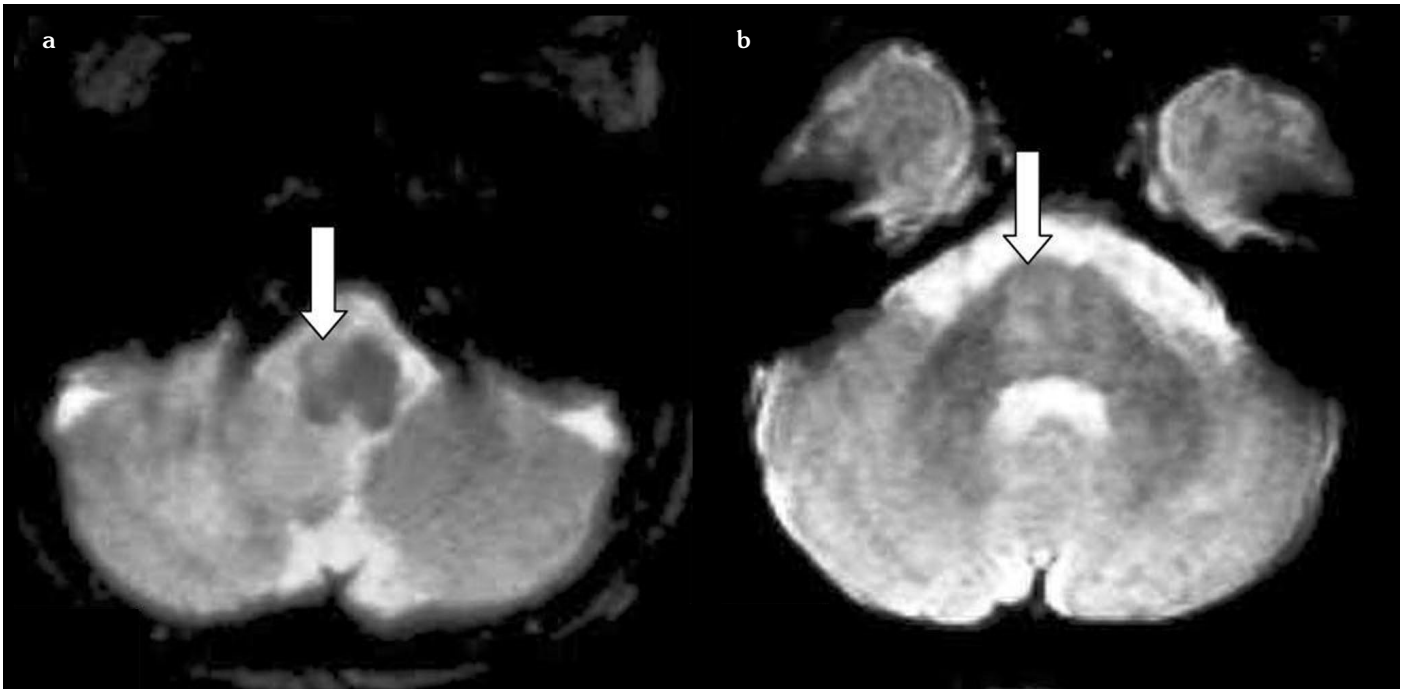

Figure 3. a, b. Axial T2-weighted images at the level of the medulla (a) and pons (b) show hyperintense foci of abnormality (arrows) in the right ventral medulla and in the right side of the pons

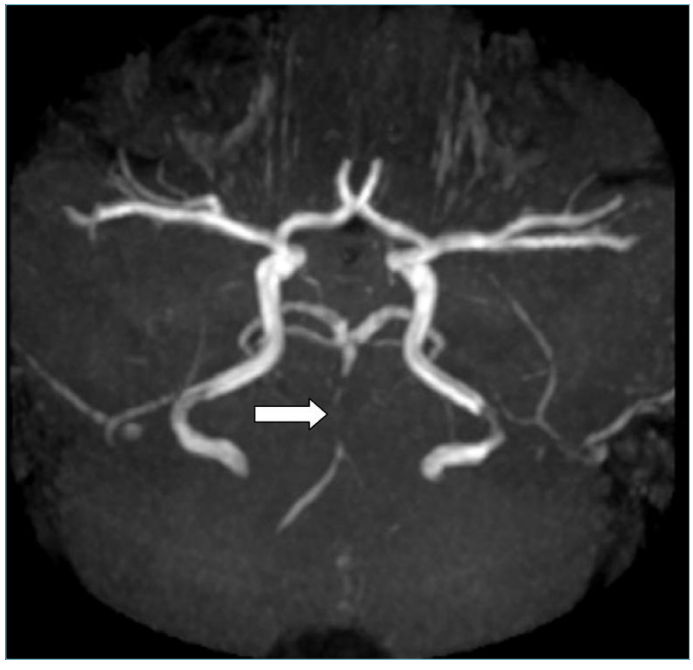

Figure 4. Oblique–axial MIP MR angiogram of the brain clearly demonstrates marked narrowing of the basilar artery, which is indicative of dissection (arrow)

sess genetic predisposition to thrombosis, were within the normal range. Moreover, tests (protein C and S deficiency, antithrombin III deficiency, active protein C resistance, factor V 1691 A mutation, prothrombin 20210A mutation, MTHFR [677 C-T) mutation, antiphosphplipid antibody) performed to assess causes for predisposition to hypercoagulability were also normal. Echocardiography was considered to be normal. On the magnetic resonance (MR) angiography performed 120 h after the trauma, a dissection was detected in the basilar artery (Figure 3). No congenital vascular aneurysm appearance was observed. A high-dose steroid was given to the patient as axonal injury could not be excluded. MR angiography was performed as MR images suggested involvement in posterior circulation, and basilar artery dissection was detected. Low-molecular-weight heparin (1 mg/kg; twice daily) was initiated. Steroid therapy was gradually discontinued. On the control MRI, hyperintense signals consistent with HOD were detected at the right side of the pons and the right ventral medulla on the axial T2-weighted images (Figure 4), and no expansion was detected on the infarction site. The patient was told to attend physical therapy session, and regular control visits were recommended.

DISCUSSION

In studies on pediatric population, the reason of infarction was detected as trauma in 22% of the patients (10). There may be hemiparesis, speech disorders, cerebellar symptoms and temporary loss of consciousness in these patients (2, 10). In our patient, there was hemiparesis at the left upper and lower extremities following the minor head trauma. A detailed anamnesis and physical examination are essential in children with head trauma. Imaging modalities should then be performed. A cranial CT scan should be the first choice (2). MRI should be preferred in symptomatic patients despite a lack of abnormal findings on the cranial CT scan (1, 2). In our patient, the cranial CT scan was normal, but further imaging studies were performed as the patient was symptomatic. There were findings consistent to infarction on MRI, and MR angiography revealed basilar artery dissection.

Basilar artery dissection is rare during childhood, although it is commonly seen in adult patients (3, 11-14). Vascular injury is rare following head trauma, and traumatic vascular injury can occur through 3 mechanisms: traumatic vascular aneurysm, trau-

matic arterial dissection, and diffuse vasospasm (1, 11-16). Head trauma rarely causes vascular injury as in our patient. In patients who present with hemiplegia or hemiparesis, cerebral angiography or MR angiography is performed following cranial CT scan and/or MRI, and it is of importance in determining anatomic localization (1-3, 11-15). In our patient, infarction was detected after MRI performed at 24 h. Subsequent MR angiography revealed basilar artery dissection. In our patient, the mechanism predisposing to thrombosis was unclear. No risk factor was detected in evaluations directed to etiology. In literature, it has been reported that oral anticoagulant, low-molecular-weight heparin, and recombinant tissue plasminogen activator within the first 3 h should be used for the provision of cerebral perfusion in case of cerebral infarction following acute cerebral thrombosis (3, 10, 16). However, data are limited regarding the use of recombinant tissue plasminogen activator in the pediatric population (16). The role of prophylactic anticonvulsant use and steroid therapy is unclear in patients with cerebral infarction (16). Steroid therapy was given as axonal injury could not be excluded in our patient who presented with hemiparesis following trauma. Significant clinical improvement was noted in our patient. It was considered that there is a combination of axonal injury and dissection.

On the control MRI, a hyperintense signal consistent with HOD was detected at the right side of the pons the and right ventral medulla on the T2-weighted images. HOD is a rare entity during childhood (4). Palatal myoclonus is an important clinical finding in patients with HOD (4-9). Although ataxia and cranial nerve injury, especially the sixth cranial nerve involvement, are common in such patients, clinical findings may vary according to comorbid conditions and etiological factors (4-9). In our patient, palatal myoclonus was not detected.

In HOD, diagnosis is usually made by MRI, and there is unilateral involvement in most patients; however, it can be bilateral in some patients (4-9). In particular, ischemia, demyelization, and neoplasm should be considered in differential diagnosis (4-9). In HOD, increased signal intensity with or without olivary proliferation is observed at the ventral medulla on T2-weighted images (4-9). On T1-weighted images, a slightly increased signal intensity can be seen. Increased signal intensity may appear 3 weeks after the event and may persist for years. Olivary hypertrophy appears 4 months after event, but it may not be present in all patients (7, 9). In our patient, increased signal intensity was detected on T2-weighted images on the control MRI following trauma.

In literature, HOD is generally reported as case reports in adults (4-8). The first pediatric case was reported by Phatouros et al (9). Our patient is a very rare case reported from Turkey to have an association of cerebellar infarction with basilar artery dissection and HOD.

CONCLUSION

Patients with hemiparesis following trauma, despite a normal CT scan, advanced imaging modalities such as MRI, diffusion-weighted images and MR angiography or angiography could be needed to determine the dissection and infarction. Cerebral infarction following minor head trauma is a rare entity during childhood. We intended to emphasize the association of HOD with cerebellar infarction following basilar artery dissection as it is rarely seen.

Informed Consent: Written informed consent was obtained from patients parents who participated in this study.

Peer-review: Externally peer-reviewed.

Authors' contributions: CM, PH, KS, YA, KŞ and GH planned the study and prepared the first draft. CM, YA, KŞ and ÇA collected the patient data. CM, EA, PH, GH and ÇA prepared the manuscript for publication and reviewed the literature. All the authors were involved in and contributed to patient monitoring and treatment.

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