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ORIGINAL ARTICLE

CT and MR imaging of primary cerebrovascular complications in pediatric head trauma

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Abstract The incidence of severe traumatic head injury in children has constantly increased over the last years. Diagnostic imaging has become an unrenounceable tool for the documentation and follow-up of intracranial lesions. The use of magnetic resonance imaging (MRI) in the early posttraumatic phase has led to a more thorough understanding of intracranial injuries. We retrospectively analyzed the cranial computed tomography (CCT) and magnetic resonance (MR)-studies of patients with traumatic head injuries for primary cerebrovascular complications. In 64 children (45 male, 19 female) with traumatic head injuries, CCT and MR examinations were available for analysis. The children's age ranged from 3 months to 15 years with a median age of 7 years. All patients had initial CCT on admission to the hospital with follow-up examinations depending on clinical state and initial imaging findings. All patients had at least one MR examination between 0 to 120 days after the trauma with a median time interval of 17 days. In five of 64 (7.8%) patients, cerebrovascular complications were found on imaging studies. Initial imaging within the first 24 h after the trauma detected a complete middle cerebral artery infarction in one patient and extensive sinus thrombosis after a complex skull fracture in another. In two patients, thrombosis of the transverse sinus appeared on MRI 4 to 6 days after the trauma. In another patient with open-skull injury, a posttraumatic aneurysm of the pericallosal artery was diagnosed on MRI 30 days after the trauma. Our study shows that, although primary cerebrovascular lesions after traumatic head injuries in children are rare, the radiologist should be aware of the characteristic injury patterns and the time appearance of imaging findings on CT and MRI.

Keywords Pediatric head trauma · Cerebrovascular lesion · MRI · CT

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Introduction

Advanced cross-sectional imaging techniques have led to a better understanding of injury patterns in traumatic brain injury with major influence on patient management and assessment of long-term prognosis [1]. Primary vascular complications of craniocerebral trauma are uncommon but serious complications often requiring further imaging studies and modification of therapeutic and diagnostic procedures.

Primary vascular complications can result from arterial or venous injury leading to bleeding, infarction, thrombosis, arteriovenous fistula, or posttraumatic aneurysm.

Although vascular complications can also be seen following mild head injury, they most commonly occur in severe traumatic head injury [2, 3].

Out of a retrospective analysis of the imaging studies in 64 children with traumatic brain injuries, we present three representative cases of posttraumatic primary vascular complications with emphasis on the imaging findings on CT and MRI.

Materials and methods

We retrospectively analyzed the imaging data of 64 children (45male, 19 female) with traumatic brain injuries, who were referred to our hospital in the time from 2000 to 2008.

On admission, the patients' age ranged from 3 months to 15 years with a median age of 7 years. Involvement in traffic accidents (53%) and falls (33%) were the most common types of injury.

According to the Glasgow Coma Score (GCS), 23% of the patients had a mild (GCS 13–15), 27% a moderate (GCS 8–12), and 50% a severe (GCS 3–7) traumatic brain injury.

All patients had at least one cerebral computed tomography examination on admission and one cerebral MR examination during their further clinical stay. The time interval between admission and MR-examination ranged from 0 to 120 days with a median time interval of 17 days.

All CT and MR images were evaluated for signs of posttraumatic vascular complications and their time of appearance.

Results

In 59 children (92.2%), cerebrovascular injuries were excluded by CT, MRI, and clinical follow-up.

Posttraumatic vascular complications were detected in five patients (7.8%). Three patients had dural sinus occlusion with involvement of the superior sagittal sinus in one and unilateral transverse sinus in two patients. All patients had skull fractures crossing the involved sinus. One patient developed a middle cerebral artery infarction several hours after a closed head injury. In all patients, coagulation disorders were excluded on blood examinations.

In one patient with recurrent intracranial bleeding after a severe open traumatic brain injury, we found a posttraumatic aneurysm of the pericallosal artery.

Below, we present the clinical and radiological features of three representative cases:

Case 1

A 5-year-old girl was run over by a car while crossing the street. Upon presentation, her GCS was 5. CT examination after arrival in the emergency department revealed fractures of the femur and pelvis. Initial CCT demonstrated beginning obscuration of the gray–white matter interface over the right hemisphere and of the right lentiform nucleus (Fig. 1a). A follow-up examination 12 h later showed increasing demarcation of a wedge-shaped low-density edema in the territory of the right middle cerebral artery suggesting acute infarction. Both CT examinations showed no dense media sign. An MR examination after another 30 h confirmed a complete infarction in the territory supplied by the right middle cerebral artery with restricted diffusion of gray and white matter structures including the lentiform and caudate nucleus (Fig. 1b). A three-dimensional time-of-flight MR-angiography showed narrowing of the supraclinoid internal carotid artery with two separate flow signals on transverse images (Fig. 1c). In addition, there was a signal void within the horizontal M1 segment of the right middle cerebral artery (Fig. 1d). After ruling out pathologic changes within the cervical portion of the carotid artery, the diagnosis of a posttraumatic right-sided media infarction due to dissection of the ipsilateral supraclinoid internal carotid artery was made. Four days after the trauma, a follow-up CT scan demonstrated progressive swelling and midline shift and the development of contralateral functional obstruction of the foramen of Monroi leading to decompression craniotomy.

Long-term follow-up examinations demonstrated extensive right hemispheric encephalomalacia and Wallerian degeneration of the ipsilateral cerebral peduncle.

Case 2

A 17-month-old boy fell from a height of 2 m onto a tiled floor. He was conscious immediately after the fall but developed progressive swelling over the skull. After admission to hospital, the patient immediately received a cerebral computed tomography scan. Still, during the CT examination, the child's consciousness deteriorated dramatically due to hypovolemic shock caused by bleeding.

CT scan revealed bilateral subdural hematomas over the parietal hemispheres and a large scalp hematoma. Additionally, there was a skull fracture which extended from the left temperoparietal region to the right parietal bone crossing the midline (Fig. 2a). After surgical evacuation of the hematoma, a follow-up CT was performed the next day. Non-enhanced scans now revealed a hyperdense thrombus in the superior sagittal sinus (Fig. 2b). Due to the associated risk of hemorrhage, anticoagulative therapy was not started. A follow-up MR examination 11 days after the injury confirmed an extended thrombosis of the superior sagittal sinus and of the parietal cortical veins with signs of collateralisation (Fig. 2c). A long-term follow-up MRI 8 months after the trauma showed increasing recanalization

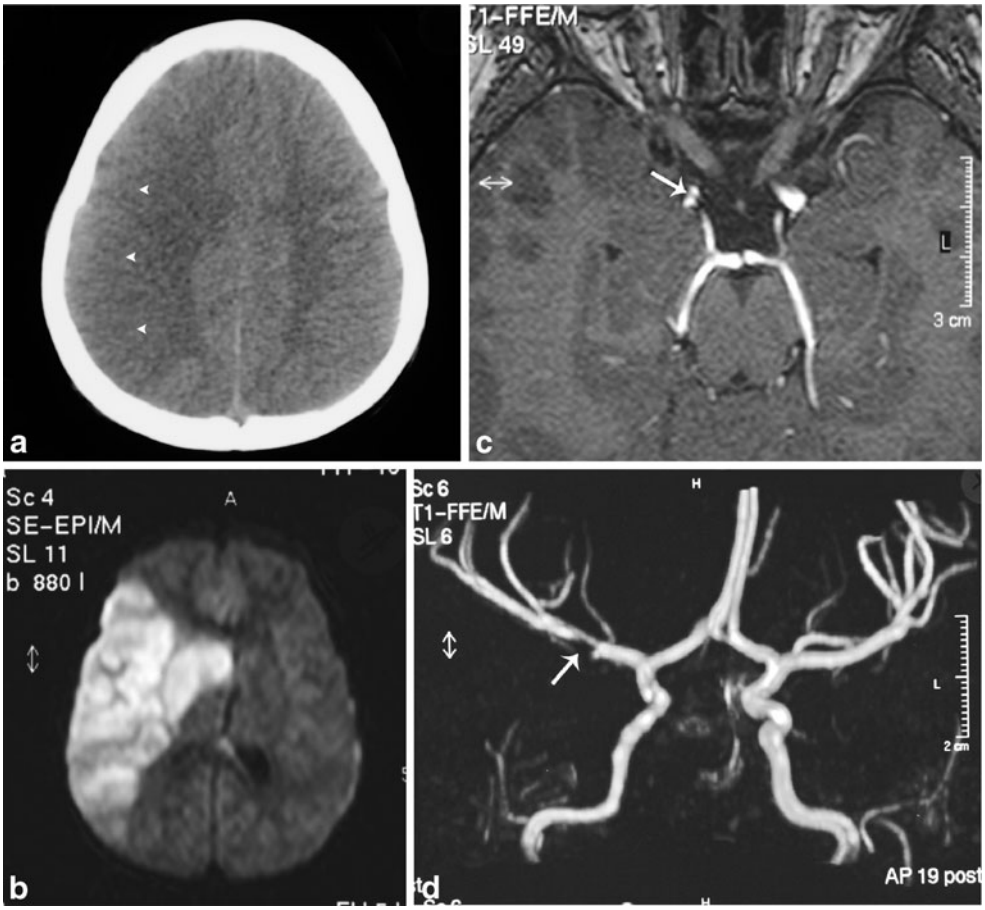

Fig. 1 Case 1: 5-year-old girl who was run over by a car. **a** CT scan 3 h after the injury reveals obscuration of the corticomedullary interface over the right hemisphere (*arrowheads*). **b** MR examination 42 h after the injury confirms complete right-sided middle cerebral artery infarction with high signal intensity on diffusion weighted image. **c** Axial source image from three-dimensional time-of-flight angiography demonstrates narrowing of the supraclinoid internal cerebral artery and two separate flow signals (*arrow*). **d** Maximum intensity projection image of three-dimensional-MRA shows irregular stenosis of the horizontal M1 segment of the right middle cerebral artery (*arrow*)

of the affected venous sinus (Fig. 2d). Posttraumatic parietoocipital parenchymal defects persisted bilaterally. Neurologic and ophthalmologic follow-up examinations revealed no distinct deficits.

Case 3

A 10-year-old unhelmeted boy suffered severe traumatic head injury and immediately lost consciousness when he lost control of his skis and hit an unsecured metal post. The initial CT scan after arrival in the emergency room showed a large depressed skull fracture on the left side associated with extensive parenchymal injury of the underlying brain tissue and cerebral herniation to the right side. He underwent left-sided craniotomy and lifting of the depressed skull fragment. On day 8, CT revealed increasing intraparenchymal hemorrhage in the left frontoparietal region causing increasing mass effect (Fig. 3a). Again, neurosurgical intervention with removal of hemorrhagic and necrotic brain tissue was performed. Sixteen days after the injury, intracerebral pressure values were rising again, and a non-enhanced follow-up CT demonstrated a progressive left-sided parenchymal bleeding in the frontal lobe (Fig. 3b). Contrast-enhanced images revealed a rounded contrast accumulation in the left frontal juxtafalcine region medial to the recurrent parenchymal bleeding site now leading to the suspicion of a posttraumatic vascular process (Fig. 3c). In the following days, the patient developed an increasing posthemorrhagic hydrocephalus which was treated by an external ventricular drainage. After stabilization of the patient's clinical state, an MR examination on day 30 confirmed an aneurysm of the left pericallosal artery (Fig. 3d). After preoperative digital subtraction angiography ruling out further vascular lesions, the aneurysm was clipped (Fig. 3e).

Discussion

Traumatic brain injury is a major cause of death and long-term neurologic sequelae in children [4]. Advanced imaging techniques have led to a more thorough understanding of injury mechanisms and have crucial impact on the clinical and therapeutic management of these patients.

The vast majority of traumatic head injuries in children are minor to mild and do not require further imaging studies [4]. In children, however, with severe traumatic brain injury or unexplained posttraumatic neurological deficit, cross-sectional imaging studies are necessary to confirm or rule out intracranial lesions requiring immediate surgical inter-

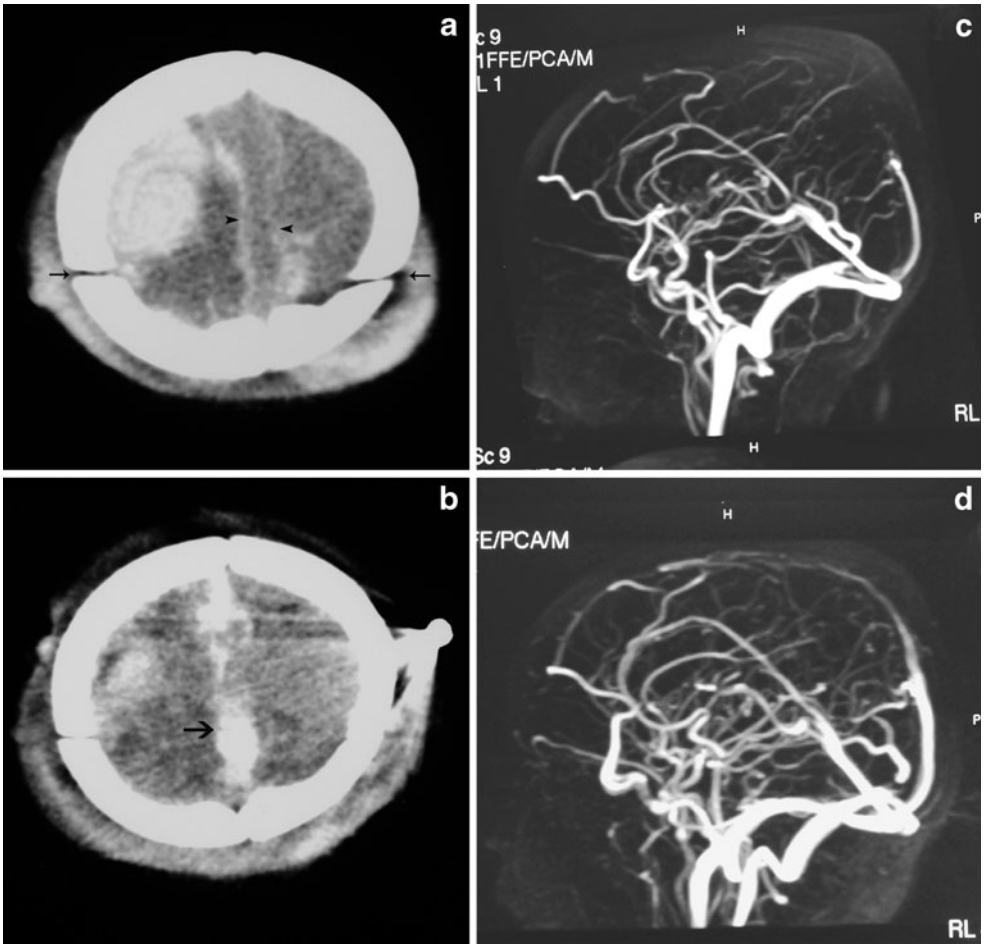

Fig. 2 Case 2: 17-month-old boy after a fall from a 2-m height with progressive swelling over the skull. **a** CT image after arrival in the emergency room shows bilateral parietal skull fractures crossing the midline (*arrows*). The superior sagittal sinus appears hypodense and broadened surrounded by subdural blood (*arrowheads*). **b** On follow-up CT 14 h later, a hyperdense superior sagittal sinus can be seen indicating dural sinus occlusion (*arrow*). **c** The two-dimensional phase contrast MR venography 11 days later confirms a subtotal occlusion of the superior sagittal sinus. **d** On follow-up, MR venography 8 months later, partial recanalization of the superior venous sinus has appeared

vention. Recent studies have shown that early posttraumatic MRI is especially valuable in demonstrating lesions like contusions or diffuse axonal injuries that are often not detectable by CT but are responsible for a wide range of posttraumatic neurological deficits [1].

The higher sensitivity and advent of new MR imaging techniques even demonstrating functional and microstructural changes after brain injury is especially helpful in judging the extent of the intracranial damage and for the assessment of prognosis. On the other hand, this requires a more detailed analysis and knowledge of trauma-induced changes by the radiologist [1].

As vascular complications after pediatric head trauma are rare, detection and therapy are often delayed. Besides bleeding caused by primary vessel injuries, the major vascular complications are arterial infarction, venous thrombosis, arteriovenous fistula, and arterial pseudoaneurysm. Among these injuries, posttraumatic cerebral infarction (PTCI) is commonly associated with a poor clinical outcome because ischemia is often irreversible involving complete territories of major vascular branches. The overall incidence of PTCI is ranging from 1.9% to 10.4% [5]. In a study by Server et al., the vast majority of PTCIs was caused by intracranial mass effects leading to mechanical shift or herniation with resulting vascular compression, most commonly affecting the territory of the posterior cerebral artery [5]. Another common mechanism leading to PTCI is intracranial arterial vasospasm associated with traumatic subarachnoid hemorrhage [6]. Over the last years, direct vascular injury has been increasingly found as a cause for PTCI especially in younger patients where the neurological deficit is often inappropriate with the initial imaging findings. PTCIs in posttraumatic vascular injuries are mostly due to dissections of arterial vessel walls leading to thrombosis or embolism in the corresponding vascular territory as shown in our case [7]. Traumatic arterial dissections are most commonly found in the cervical segment of the internal carotid artery, about 2 cm distally from the bifurcation and in the distal segment of the vertebral artery between C2 and the skull base. The most common site for intracranial dissection of the internal cerebral artery is the supraclinoid segment where the artery is relatively mobile and susceptible to acceleration/deceleration forces. Especially in children, traumatic vascular injuries leading to infarction are an extremely rare complication. Gümüs et al. reported a case of a 4-year-old child who developed a traumatic middle cerebral artery occlusion after a fall on the head [8]. They proposed

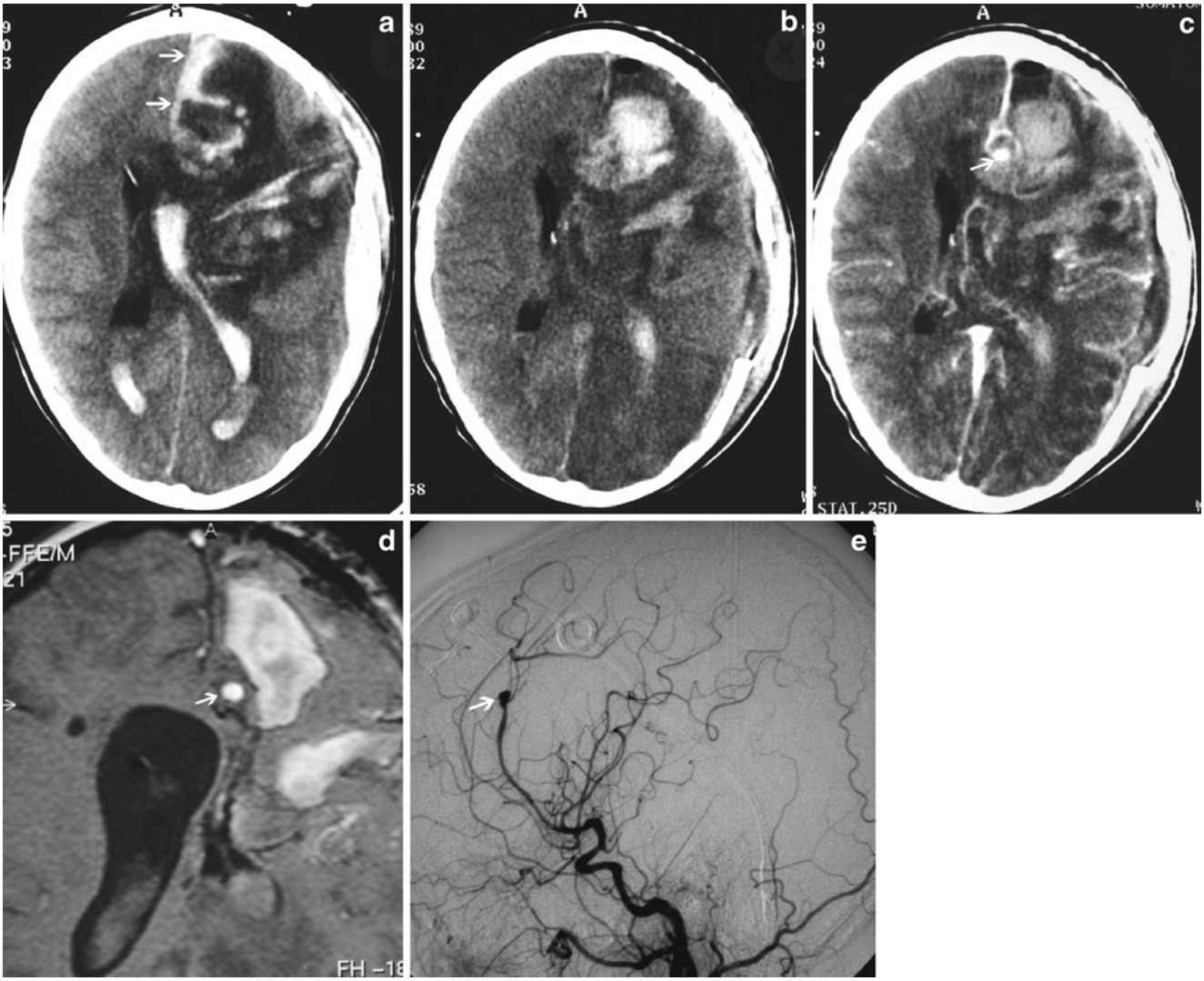

Fig. 3 Case 3: 10-year-old boy with open-head injury after skiing accident. **a** CT examination 8 days after the injury reveals increasing left hemispheric intraparenchymal hemorrhage with mass effect. Notice left frontal parafalcine bleeding and blood in frontal interhemispheric fissure (*arrows*). **b** Sixteen days after the injury, the patients clinical state deteriorated again. Control CT shows a progressive left frontal bleeding. **c** Contrast-enhanced images now reveal a rounded contrast accumulation in the anterior parafalcine region on the left side (*arrow*). **d**, **e** After external drainage of a posthemorrhagic hydrocephalus and stabilization of the patient's clinical state, axial source images from three-dimensional time-of-flight MR-angiography and preoperative conventional angiogram confirm the suspicion of posttraumatic left-sided pericallosal artery aneurysm (*arrows*)

posttraumatic mural thrombus formation in combination with slowing of the cerebral circulation as possible reasons for arterial occlusion. Another case of a 4-year-old child reported by Ahn et al. demonstrates that posttraumatic cerebral infarction in children can also be seen following minor head injury [2]. In this case, the child developed a basal ganglia infarction after a fall from a seesaw resulting in posttraumatic speech disturbance and progressive hemiparesis [2].

The difficulty in diagnosing a posttraumatic arterial injury early is that the neurological symptoms are often superimposed by other neurological impairments and that the initial imaging findings on posttraumatic CT are often normal or unspecific. Therefore, the correct diagnosis is often delayed. On the other hand, early diagnosis of vascular trauma is important because early treatment is the only way to prevent devastating outcome and long-term neurologic sequelae [9]. In children with unexplained neurologic deficits after head trauma, an MR examination should be performed as early as possible. Diffusion weighted imaging can demonstrate the site and extent of cerebral ischemia, and MR-angiography can demonstrate the major cerebral arteries. In addition, MR imaging is able to detect intramural hematoma in arterial dissection reducing the need for CTA or conventional angiography in most of the cases [9].

In general, the clinical outcome is poor, especially in patients who already have cerebral infarction on admission as demonstrated in our case [10]. Because of the potential risk of bleeding, anticoagulation is not recommended in these patients [11]. Only patients with mild to moderate symptoms might benefit from bypass surgery or endovascular treatment [10].

Delayed onset of neurological symptoms after head injury or worsening of preexisting symptoms several hours to days after minor head trauma can also be the first clinical sign of a posttraumatic cerebral venous thrombosis [3, 12]. The relationship between sinus thrombosis and head injury is well known, although there seem to be a number of predisposing factors that might be responsible for the development of trauma-induced thrombosis. It is generally accepted that skull fractures crossing venous sinuses are a risk factor for the development of thrombosis, although most of the linear fractures that cross sinuses do not lead to thrombosis. Therefore, in every case of posttraumatic sinus thrombosis, risk factors for thrombophilia should be investigated [13].

In contrast to septic thrombosis or venous sinus thrombosis appearing in patients with underlying chronic disease, the prognosis of posttraumatic thrombosis in otherwise healthy children is generally favorable. In a study by Huisman et al., all children with posttraumatic cerebral venous thrombosis showed spontaneous recanalization, and no patient developed an infarction or hemorrhage on follow-up examinations [14].

In our three cases, the posttraumatic cerebral venous thrombosis was associated with a skull fracture and developed within the first days after the trauma. The diagnosis of venous sinus thrombosis was in no case associated with clinical deterioration. On follow-up imaging performed between 2 to 7 months after the trauma, all patients showed complete recanalization of the thrombosed sinus without obvious defects related to the thrombosis. No patient received anticoagulative therapy.

In a study of Taha et al., five patients with sigmoid sinus thrombosis developed neurological symptoms or worsening of existing symptoms 1 to 4 days after the trauma. Three patients had occipital fractures, and two patients with mild head injuries had no fracture. Although no patient received anticoagulative therapy, the thrombus resolved in four cases on follow-up examinations 4 to 6 weeks after the trauma [12].

As the proposed mechanism leading to cerebral venous thrombosis in moderate to severe head trauma is traumatic disruption of sinuses or compression by adjacent hematoma, there are unusual cases of cerebral venous thrombosis even after minor head injury. In these patients, conditions that predispose to thrombus formation especially acquired and inherited causes of thrombophilia have to be ruled out [3, 13].

Whereas the diagnosis of a posttraumatic venous thrombosis or a posttraumatic arterial infarction does not usually cause greater diagnostic problems if clinical observation and imaging is performed adequately, the diagnosis of a posttraumatic aneurysm is more challenging, and often, there is a considerable delay between clinical symptoms and correct diagnosis. In general, intracranial aneurysms in children are very rare, accounting for about 5% of all cases [15]. Concerning their etiology, size, localization, and clinical presentation, pediatric intracranial aneurysms are different from adult aneurysms. In children, the incidence of traumatic, mycotic, and giant aneurysms is higher, and aneurysms are more often located in the region of the internal carotid artery bifurcation and in the posterior circulation [16, 17].

Although aneurysms in children are rare compared with adults, the frequency of posttraumatic aneurysms in the pediatric age group is quite high. According to the literature, traumatic aneurysms account for 14–39% of all pediatric aneurysms [18, 19]. Most traumatic aneurysms are pseudoaneurysms that develop after a traumatic disruption of the vessel wall. They are commonly found at the skull base or along the distal anterior cerebral artery. The proposed mechanism for the development of distal anterior cerebral artery or pericallosal artery aneurysms is shearing forces between the vessel and the inferior margin of the falx cerebri. They usually cause clinical symptoms 2–4 weeks after the injury, and the diagnosis should be suspected in recurrent juxtafalcine bleeding [20]. Some authors suppose that the lack of visualization on initial imaging studies is due to compression and thrombosis of the aneurysm right after the injury and that recurrent bleeding due to the aneurysm is often obscured by accompanying hemorrhagic brain contusion [20, 21]. On the other hand, Nakstad et al. demonstrated that posttraumatic aneurysms demonstrate an increase in size over the first weeks, explaining the higher detection rate on follow-up studies [20]. In general, the suspicion for a bleeding posttraumatic aneurysm should lead to dedicated follow-up imaging studies with contrast-enhanced computed tomography or, in clinically stable patients, MR imaging including MR-angiography.

In contrast to nontraumatic aneurysms of the pericallosal artery, posttraumatic aneurysms are usually not found at vessel bifurcations. In our case, the aneurysm of the pericallosal artery was found distal to the bifurcation into the pericallosal and callosomarginal artery whereas Nakstad reported on two cases proximal to the bifurcation and one being located in the proximal callosomarginal artery [20].

Due to the high percentage of potentially fatal rebleeding in posttraumatic aneurysms and their tendency to grow over time, operation should be performed as soon as possible depending on the patient's clinical state [22].

In summary, our cases show that some specific primary cerebrovascular lesions rarely appear in pediatric craniocerebral trauma. As these lesions are potentially associated with the development of severe complications and long-term neurologic sequelae, early and correct diagnosis is important. Therefore, dedicated cross-sectional imaging especially early posttraumatic MRI is necessary in patients with unexplained neurological symptoms or if the neurological symptoms are inappropriate to the initial imaging findings.

Cerebral venous thrombosis should be suspected in patients with skull fractures crossing venous sinuses or in patients with delayed neurological symptoms or worsening of existing symptoms. In patients with hemiparesis or focal neurologic deficits, posttraumatic cerebral infarction should be suspected. Similar to venous thrombosis, PTCI is not inevitably associated with severe head trauma but might also appear in cases with minor or mild injury. In contrast, the development of a posttraumatic arterial aneurysm is most commonly associated with severe penetrating or non-penetrating trauma. The presence of a posttraumatic aneurysm should be suspected if recurrent posttraumatic bleeding especially in the parafalcine region occurs.

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