


Two pediatric cases of epidural hematoma in the posterior fossa with extension along the sigmoid sinus groove: MR evaluation

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Abstract

To discuss the computed tomography (CT) and magnetic resonance (MR) findings of posterior fossa epidural hematoma (PFEDH) mimicking sinus thrombosis, we present two pediatric cases with the PFEDH extending along the sigmoid sinus groove evaluated by MR imaging (MRI) and MR venography (MRV). T2-weighted coronal MRI can diagnose both patency of the sigmoid sinus and epidural hematoma extending along the sinus groove. Phase-contrast MRV (PC-MRV) is also useful to evaluate the flow state in the dural sinuses but it should be diagnosed carefully whether low visualization of the dural sinus means only functional flow impairment or organized occlusion due to thrombus. To avoid an unnecessary anticoagulant therapy that may worsen epidural hematoma, it is important to recognize the pitfall that PFEDH extending along the sinus groove is easy to misdiagnose for a dural sinus thrombosis.

Keywords

Epidural hematoma, posterior fossa, sigmoid sinus groove, magnetic resonance imaging, magnetic resonance venography

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Introduction

Sinus thrombosis and sinus occlusion are well-known consequences of head trauma in children. A post-traumatic sinus occlusive lesion is thought to be associated with skull fracture crossing the venous sinus and epidural hematoma (1–6). Recently, Singh et al. reported that a posterior fossa epidural hematoma (PFEDH) compressing a dural venous sinus could mimic dural sinus thrombosis (3). We present here two pediatric cases with the PFEDH extending along the sigmoid sinus groove as diagnosed by magnetic resonance imaging (MRI) and phase-contrast magnetic resonance venography (PC-MRV), and discuss the usefulness of MRI including magnetic resonance venography (MRV) for diagnosis in the case of PFEDH with or without sinus thrombosis in children.

Case report

Case 1

A four-year-old girl was admitted to our hospital with headache, vomiting, and somnolence after falling from

a height of 4 m. No other neurological deficit was detected. Non-contrast computed tomography (CT) with three-dimensional volume-rendering clearly showed left occipital bone fracture crossing the transverse sinus groove (Fig. 1a). Non-contrast CT performed 3 h after trauma also showed a high-density area in the left transverse sigmoid junction, which suggested sinus thrombosis (Fig. 1b). However, MRI revealed high signal intensity indicating fresh epidural hematoma in the sigmoid sinus groove, which displaced the sigmoid sinus medially, on axial and coronal images

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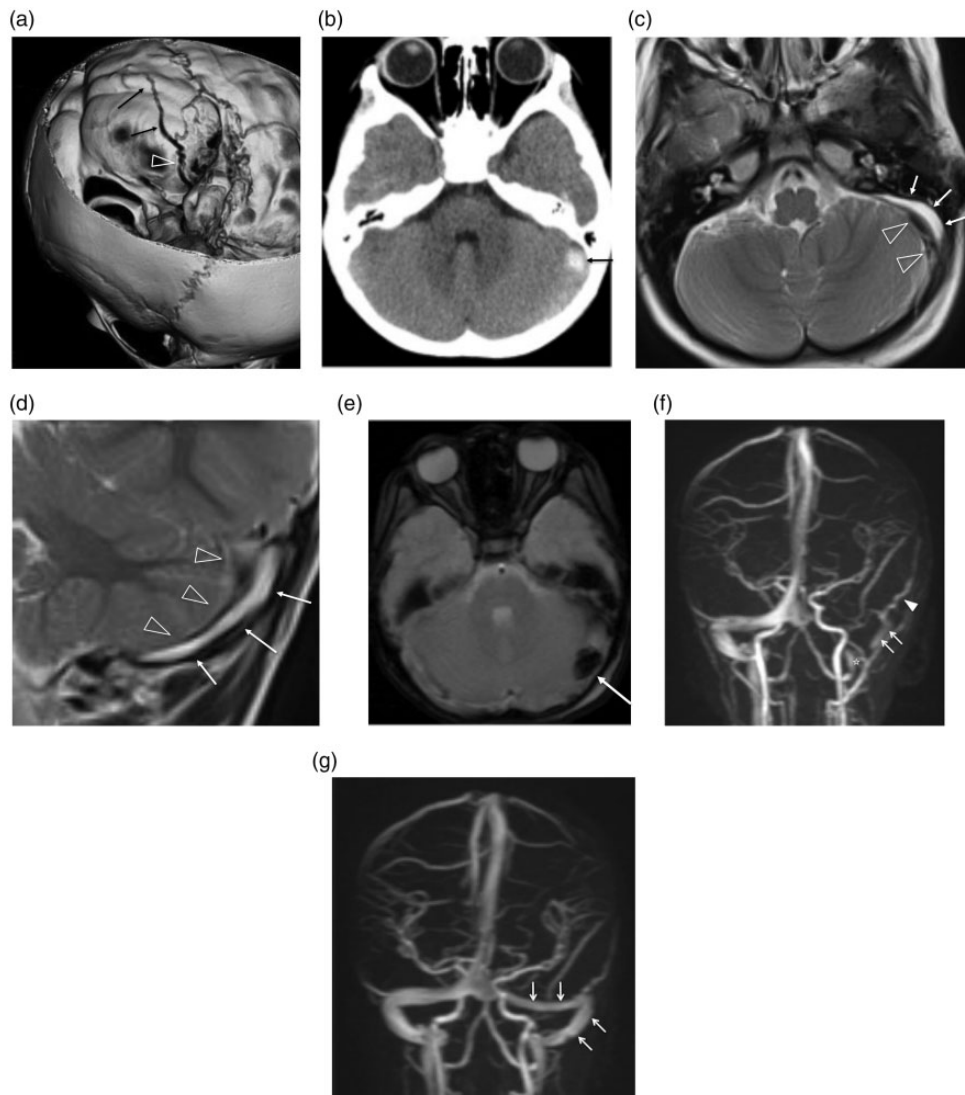


Fig. 1. (a) Three-dimensional reconstructed CT shows a left occipital bone fracture crossing the transverse sinus groove (arrows) and diastatic fracture of the occipitomastoid suture passing the sigmoid sinus groove (white arrowhead). (b) Plain axial CT performed 3 h after head trauma shows a high-density area in the left transverse sigmoid junction mimicking sinus thrombosis (arrow). (c, d) T2W axial (c) and coronal (d) images show the patency of the sigmoid sinus represented as flow void (white arrowhead), which is medially displaced by the epidural hematomas along the sigmoid sinus groove (white arrows). (e) Axial susceptibility-weighted imaging shows very low signal intensity, suggesting thrombus or clot in the distal end of the left transverse sinus (arrow). (f) PC-MRV shows normal signal indicating the patency of the left sigmoid sinus (arrow), which receives supratentorial venous drainage, such as from the vein of Labbe (arrow head), and loss of signal in the left transverse sinus suggesting occlusion; asterisk (☆) indicates jugular bulb. (g) Follow-up MRV three months after head trauma showed normal flow signal from the left transverse to the sigmoid sinus (arrows). CT, computed tomography; PC-MRV, phase-contrast magnetic resonance venography; T2W, T2-weighted.

(Fig. 1c and 1d). MRI also showed no intraparenchymal lesion. However, susceptibility-weighted imaging (SWI) showed very low signal-intensity foci, which suggested fresh thrombosis or clot in the distal end of the transverse sinus (Fig. 1e). Although PC-MRV showed flow signal of the left sigmoid sinus, which received supratentorial venous drainage in the temporal lobe, such as from the vein of Labbe, there

was no flow signal in the left transverse sinus (Fig. 1f). Therefore, we diagnosed that this case did not have left sigmoid sinus thrombosis, but rather transverse sinus occlusion due to thrombus or clot in the distal end of the transverse sinus. Her symptoms completely improved within several days without anticoagulant therapy. Follow-up MRI and MRV at three months after trauma showed resorption of the epidural

hematoma and normal dural sinus drainage (Fig. 1g). This patient showed no deterioration until the point of follow-up after 13 months.

Case 2

A five-year-old boy was admitted with repeated vomiting. He had suffered head trauma when he fell from a height of 2 m three days previously. He had no neurological deficit. Non-contrast CT (Fig. 2a and 2b) showed a high-density area mimicking sinus thrombosis in the right transverse-sigmoid junction and a high-density mass indicating epidural hematoma in the occipital convexity (Fig. 2a). Non-contrast CT with a bone window setting also showed diastatic fracture in the occipitomastoid suture (Fig. 2c). T2-weighted (T2W) coronal MRI revealed high signal intensity, which indicated fresh epidural hematoma along the sigmoid sinus groove, which medially displaced the right sigmoid sinus (Fig. 2d). PC-MRV clearly showed a normal flow signal in the right sigmoid sinus, which received supratentorial venous drainage in the temporal lobe. On the other hand, the right transverse sinus

was visible but very thin and narrow, which probably indicated decreased flow signal on MRV (Fig. 2e). Conservative therapy was applied and follow-up MRI and MRV one month after trauma showed resorption of the epidural hematoma and normal venous drainage in the dural sinus (Fig. 2f). This patient did not show any deterioration and this healthy condition persisted even after 12 months of follow-up.

The parents of each patient provided informed consent for all medical procedures and for inclusion in this report.

Discussion

Head trauma has been reported as a low-risk factor for venous sinus thrombosis in children (7). In their series of cerebral venous sinus thrombosis in children, of the 25 cases that were healthy before the onset, 23 cases were diagnosed as triggered by recent infection. Eight cases were triggered by dehydration, including six duplicates. Upon clinical presentation, symptoms included 40% seizures, 68% headaches, 28% vomiting,

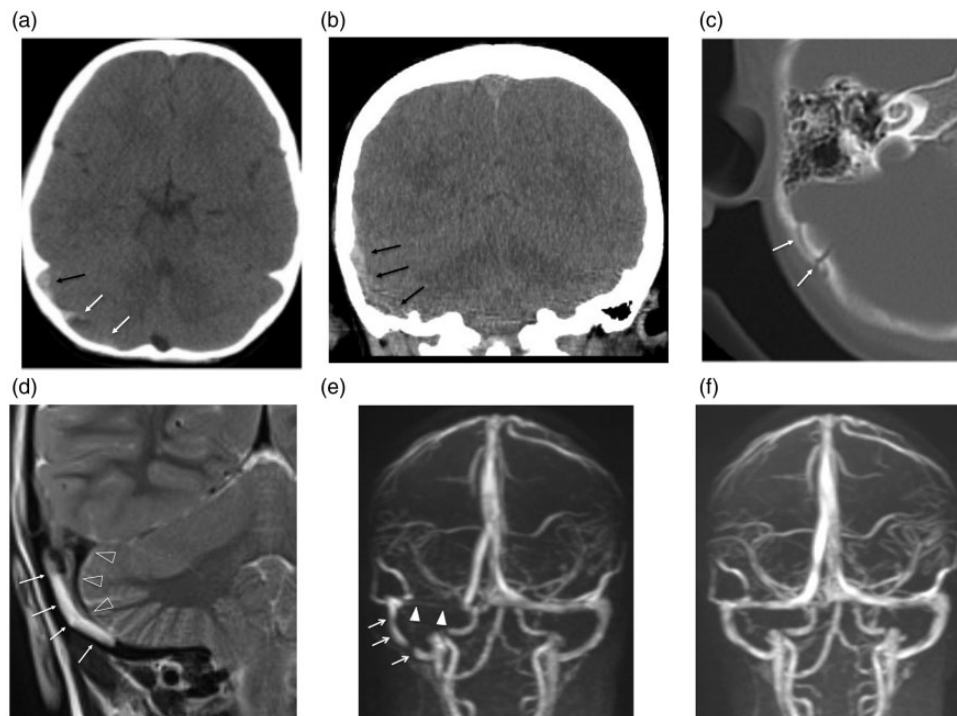


Fig. 2. (a, b) Plain axial (a) and reconstructed coronal CT (b) shows a high-density area mimicking sinus thrombosis at the transverse sigmoid junction (a; arrow) and along the sigmoid sinus groove (b; arrows). Axial CT (a) also shows epidural hematoma with high density in the calvarium of the posterior fossa (white arrows). (c) Bone window CT shows diastatic fracture of the right occipitomastoid suture and fracture in the right mastoid bone (arrows). (d) T2W coronal MRI shows patent right sigmoid sinus represented as flow void (white arrowhead), which is medially compressed by epidural hematomas along the sigmoid sinus groove (arrow). (e) PC-MRV shows decreased signal in the right transverse sinus (white arrowhead) and normal signal indicating a patent sigmoid sinus (arrows), which receives supratentorial venous drainage in the temporal lobe. (f) Follow-up PC-MRV one month after trauma shows normal venous drainage from the right transverse to the sigmoid sinus. CT, computed tomography; MRI, magnetic resonance imaging; PC-MRV, phase-contrast magnetic resonance venography; T2W, T2-weighted.

and 43% drowsiness, and signs included 45% fever, 28% coma, 33% hemiparesis, and 33% cranial nerve abnormalities. Most were symptoms associated with elevated intracranial pressure. Factors related to the prognosis of cognitive function were transverse and/or sigmoid sinus as the affected venous sinus, absence of brain parenchymal injury, and anticoagulation therapy.

Several reports describe the relationship between fractures associated with dural sinus and venous thrombosis. A few published reports assessed possible differences between adult and pediatric populations with respect to this pathology. Revkin et al. presented 63 patients who had fractures overlying cerebral venous sinuses or the jugular bulb (1). Forty-nine adults and 14 children were included in the study. Sinus thrombosis was detected in 18 (36.7%) adult patients prevalently compared to 4 (28.6%) pediatric patients. In adults, 50% had complete obstruction of the dural sinus, whereas in children, no complete obstruction of the dural sinus was observed. They discussed that it was unclear why pediatric venous sinuses appear to fare better with traumatic events. The answer may be related to the sinus occlusive pathology due to PFEDH in children. In children, less damage can cause obstructive changes in the sinus due to PFEDH compared to adults. In the literature, PFEDH is caused by venous disruption in 85% of cases among all age groups (4,5,8). In children, since the meningeal artery is not in the calvarial groove, PFEDH is more likely to have a venous origin than in adult cases (4,9). Thus, PFEDH in children tends to arise from disruption of the wall of the dural sinuses, a diploic venous lake or meningeal veins. On the other hand, the dura mater is more tightly adhered to the inner table in children (10). However, since there is constant modeling and remodeling of the occipital bone along the sinus grooves, the outer wall of the dural sinus may be less adhered to the inner table of the occipital bone (3). Furthermore, compared to brain parenchyma, the dural sinuses can less

effectively opposed the outer membrane of the dura to the calvarium. Thus, these anatomic features may allow PFEDH to peel off the dura along the sinus groove in children (Fig. 3).

Based on these anatomic features, in 2016, Singh et al. (3) first reported that PFEDH in children tends to extend along the sinus groove and can mimic sinus thrombosis by compressing and displacing the sinuses. In our cases, non-contrast CT showed a high-density area mimicking sinus thrombosis in the transverse-sigmoid junction to the sigmoid sinus. However, T2W coronal MRI (Figs. 1d and 2d) revealed both the patency of the sigmoid sinus represented as flow signal void and fresh epidural hematoma, which has a high signal intensity, lateral to the sigmoid sinus.

In Case 1, SWI (Fig. 1e) showed that there was a thrombus or clot in the distal end of the left transverse sinus near the fracture line, which was detected by CT (Fig. 1a). In addition, PC-MRV revealed a loss of signal throughout the entire left transverse sinus, probably indicating transverse sinus occlusion at its distal end (Fig. 1f). These findings suggested that disruption of the outer membrane of the dural sinus in this area might be a source of bleeding for PFEDH, as well as a cause of transverse sinus occlusion. In addition, PC-MRV clearly showed the signal of the left sigmoid sinus, which indicated no sigmoid sinus occlusion (Fig. 1f), and clarified that the high density along the left sigmoid sinus on CT was not the thrombus in the sinus, but rather epidural hematoma. Thus, MRI and MRV are very useful diagnostic tools for distinguishing sinus thrombosis from epidural hematoma.

In Case 2, PFEDH was placed beneath the occipital calvarium and in the sigmoid sinus groove (Fig. 2a and 2b). CT with a bone window setting (Fig. 2c) showed a fracture line of the diploic space in the mastoid bone and a diastatic fracture at the occipitomastoid suture which runs across the sigmoid sinus groove. Therefore, we suggest that disruption of the

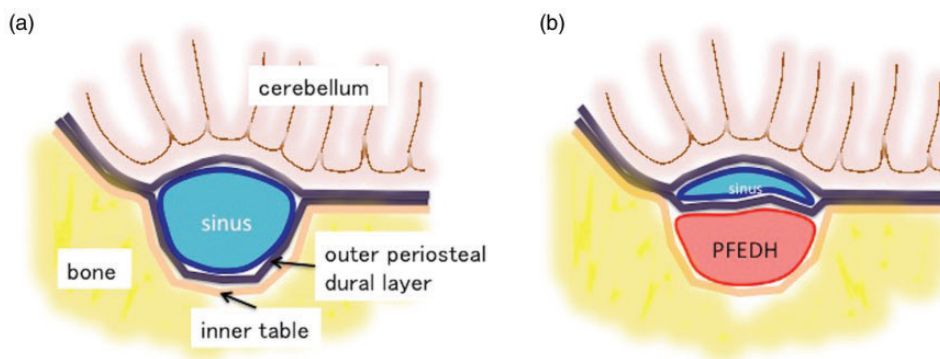


Fig. 3. Local anatomy around the sinus groove: (a) normal anatomy; (b) PFEDH to peel off the dura along the sinus groove compresses the dural sinus via the outer periosteal dural layer. PFEDH, posterior fossa epidural hematoma.

diploic or emissary vein near these fractures may cause PFEDH extending along the sigmoid sinus groove. In contrast to Case 1, although MRI did not reveal any occlusive change in the dural sinuses, PC-MRV showed a thinned right transverse sinus mimicking sinus thrombosis (Fig. 2e). However, since PC-MRV only reflects the state of venous flow, the sinus, which looks narrow, may not represent occlusive change and instead may indicate decreased flow in the right transverse sinus. The decreased flow in the right transverse sinus may result from a change in flow direction in the sinuses. Thus, the slight impairment of venous flow at the transverse-sigmoid junction due to compression of the sinus wall by epidural hematoma may make almost all of the venous flow from the superior sagittal sinus drain into the contralateral transverse sinus via a confluence.

Singh et al. (3) also stated that CT venography and MRV are each useful for differentiating between PFEDH and dural sinus thrombosis. In our experience, although CT venography was not performed, conventional MRI clearly revealed PFEDH extending along the sigmoid sinus, which displaced the sigmoid sinus medially (Figs. 1c, 1d, and 2d). In particular, we emphasize that a T2W coronal image is the most important view for determining the patency of the sigmoid sinus and its compression medially by epidural hematoma along the sinus groove. PC-MRV is also useful for evaluating the blood flow state in the dural sinuses. In theory, although PC-MRV can be used to visualize blood flow and provide a true velocity map in which voxel signal intensity is proportional to the true flow velocity in a specific direction along the x, y, or z axis (11), it may be difficult to visualize stationary or slow-flowing blood. Thus, since PC-MRV cannot display the venous sinuses themselves, the caution must be taken to assess whether low visualization of the dural sinus on PC-MRV indicates functional flow impairment or organized occlusion due to thrombus. In particular, the transverse sinus commonly shows asymmetry due to unilateral hypoplasia or aplasia as a normal variant (12).

Byrne et al. (13) reported it is safe to administer low molecular weight heparin to patients with severe head trauma within 72 h after injury, as a prophylactic measure for venous thromboembolism. However, if the cause of the venous sinus occlusive change is not primary thromboembolism but secondary compression by the epidural hematoma, anticoagulant therapy may be contraindication because of worsening the epidural hematoma. This dilemma shows that detailed and careful image assessment is important to decide on treatment policy.

Finally, to avoid unnecessary anticoagulant therapy which may worsen epidural hematoma, it is very

important to recognize that PFEDH in children tends to extend along the sinus groove and is easy to misdiagnose as dural sinus thrombosis on CT.

In conclusion, PFEDH in the sinus groove in children tends to extend along the transverse and sigmoid sinus grooves and can mimic sinus thrombosis on non-contrast CT. It is very important to recognize this pitfall to avoid unnecessary anticoagulant therapy which may worsen epidural hematoma. T2W coronal MRI can be used for a definitive diagnosis of both the patency of the sigmoid sinus and epidural hematoma along the sigmoid sinus groove.

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