

# Sigmoid Sinus Thrombosis After Mild Closed Head Injury in an Infant: Diagnosis by Magnetic Resonance Imaging in the Acute Phase

## —Case Report—

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### Abstract

Intracranial sinus thrombosis following a mild closed head injury without a skull fracture or intracranial hematoma is extremely rare. A 23-month-old girl presented with vomiting and gait ataxia 1 day after occipital trauma. Computed tomography revealed a slightly increased density area in the region of the left sigmoid sinus. T<sub>1</sub>-weighted magnetic resonance (MR) imaging demonstrated an isointense area in the left sigmoid sinus and T<sub>2</sub>-weighted imaging showed a hyperintense area reflecting the characteristics of oxyhemoglobin. MR angiography and cerebral angiography indicated occlusion of the left sigmoid sinus. After 4 days of conservative treatment, her symptoms subsided completely. Follow-up MR angiography and cerebral angiography showed recanalization of the sigmoid sinus. The MR images and MR angiograms were useful for both early diagnosis and follow-up. Treatment should reflect the severity of individual cases, and early diagnosis will help achieve a good outcome.

Key words: sinus thrombosis, sigmoid sinus, closed head injury, magnetic resonance imaging

### Introduction

Intracranial sinus thrombosis is a relatively rare event associated with a poor prognosis, largely because the diagnosis was difficult. Mild closed head injury without skull fractures or intracranial hematomas is an extremely rare cause, with only 11 reported cases.<sup>3,6,12,15,22,23,26</sup> We treated an infant with sigmoid sinus thrombosis complicated by mild closed head injury. The thrombosis was identified in the acute phase by magnetic resonance (MR) imaging.

### Case Report

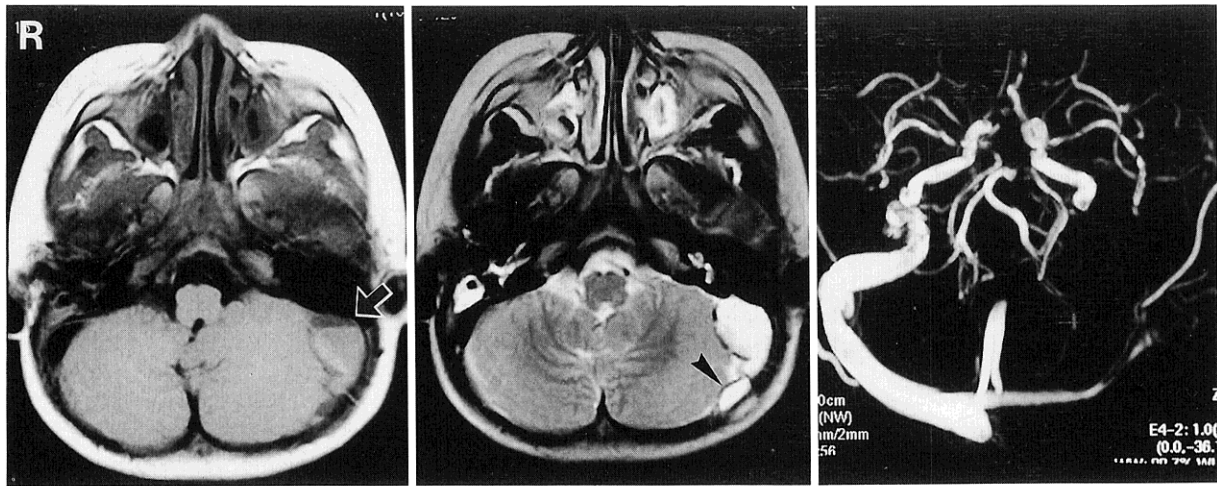
A 23-month-old girl fell from a height of approximately 1 m and struck her occiput on November 8, 1998. She suffered no loss of consciousness or

vomiting and was able to walk. However, vomiting and gait disturbance appeared by the next morning. The patient was first taken to the pediatric department of a local hospital and then referred to our institution for further evaluation. Her history included medical treatment for asthma for 3 months. Her family history was unremarkable.

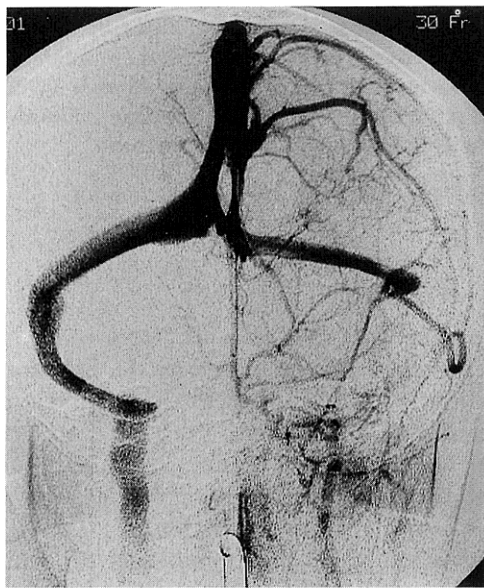
Neurological signs and symptoms on admission included moderate headache complaints, decreased muscle tone, and gait ataxia. There were no abnormalities of the cranial nerves or motor or sensory disturbances in the extremities. Fundoscopy showed no evidence of a choked disc. No signs of dehydration were present. Biochemical and hematologic screening tests disclosed no abnormal findings (hematocrit 34.6%, platelet count  $25.8 \times 10^4/\mu\text{l}$ , white blood cell count  $8.9 \times 10^3/\mu\text{l}$ , and C-reactive protein 0.1 mg/dl). All bleeding and coagulation test

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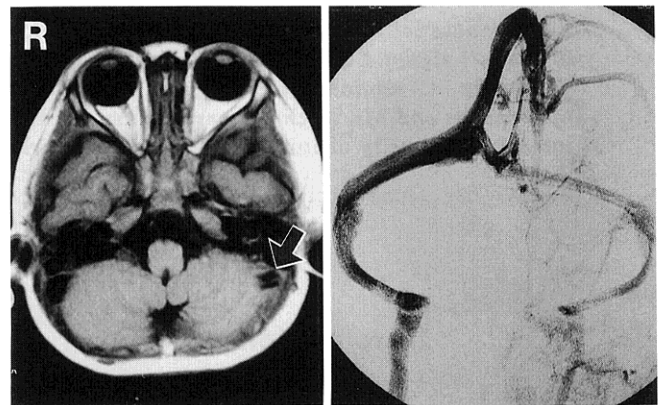
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**Fig. 1** T<sub>1</sub>-weighted magnetic resonance (MR) image on admission demonstrating an isointense area in the left sigmoid sinus (*left*), and T<sub>2</sub>-weighted image demonstrating a hyperintense area (*center*). Phase-contrast MR angiogram (velocity encoding gradient = 15 cm/sec) showing occlusion of the left sigmoid sinus (*right*). There is no midline shift of the fourth ventricle. *arrow*: lateral wall of the sigmoid sinus, *arrowhead*: small extradural hematoma.



**Fig. 2** Cerebral angiogram performed on the second hospital day also demonstrating occlusion of the left sigmoid sinus.



**Fig. 3** T<sub>1</sub>-weighted magnetic resonance image 15 days after admission (*left*) and cerebral angiogram on the following day (*right*) demonstrating that the left sigmoid sinus has recanalized. *arrow*: flow void phenomenon of the left sigmoid sinus.

results were within the normal ranges (bleeding time 2 min, prothrombin activity 82% of control, activated partial thromboplastin time 32.5 sec, fibrin degradation products 3.1  $\mu\text{g/ml}$ ). The control protein S activity was 102% and the control protein C activity was 123%, and the lupus anticoagulant was not detectable. The lumbar puncture opening pres-

sure was 240 mmH<sub>2</sub>O, and cell numbers and protein concentration in the cerebrospinal fluid were within the normal ranges. Computed tomography (CT) revealed a region of slightly increased density in the sigmoid sinus, which was enhanced by administration of contrast medium. T<sub>1</sub>-weighted MR imaging obtained on the same day showed an isointense area, and T<sub>2</sub>-weighted imaging demonstrated a hyperintense area in the left sigmoid sinus (Fig. 1 *left, center*). MR angiography (phase-contrast

method) demonstrated occlusion of the left sigmoid sinus (Fig. 1 right). Cerebral angiography confirmed occlusion of the left sigmoid sinus on November 10 (Fig. 2).

Intravenous drip infusion of glycerol and steroid was administered to treat the increased intracranial pressure. Headache and vomiting resolved 3 days after admission, and the patient was able to walk 4 days after admission. MR imaging and MR angiography 15 days after admission disclosed that the left sigmoid sinus had recanalized (Fig. 3 left). Conventional cerebral angiography on the following day showed the same finding (Fig. 3 right). The patient was discharged from the hospital 17 days after admission with no neurological deficits. MR imaging and MR angiography 3 months after hospital discharge confirmed patency of the sigmoid sinus.

### Discussion

Causes of sinus thrombosis include infection of the head and neck, pregnancy and delivery, oral contraceptives, and dehydration. More recently, sinus thrombosis has resulted from hypercoagulable states caused by protein S deficiency, protein C deficiency, or antiphospholipid antibodies.<sup>20</sup> Sinus thrombosis had been considered a very ominous event until recently, when mild cases have been resolved without residual neurological deficits because of technical advances.<sup>1</sup> Some cases of skull fracture or intracranial hematoma following head injury have caused thrombosis by direct compression of the sinus,<sup>2,9</sup> but thrombosis after mild closed head injury without such complications is exceptionally rare.

Symptoms of sinus thrombosis can be classified into two groups: manifestations of increased intracranial pressure such as headache, choked disc, and impaired consciousness<sup>18</sup>; and focal signs including convulsive seizures and hemiplegia caused by venous infarction, which is often hemorrhagic.<sup>1</sup> Cerebellar ataxia is not common, but gait ataxia occurred in all five cases with sigmoid sinus thrombosis (including three with skull fractures),<sup>26</sup> suggesting that gait ataxia is characteristic of sigmoid sinus involvement. The time interval between injury and symptom onset varies from moments after injury to 8 months,<sup>22</sup> which is an obstacle to accurate diagnosis of traumatic sinus thrombosis.

The severity of the manifestations of sinus thrombosis depends on the extent of the thrombus, the anatomy of collateral veins, and the rate of progression of thrombosis.<sup>28</sup> Sinus thrombosis following mild closed head injury in the 12 reported cases, including the present patient, originated in the superior sagittal sinus in five cases, the transverse si-

nus in five, the sigmoid sinus in four, and the straight sinus in one (multiple sinuses were involved in 2 cases). Condition on discharge was described in 11 patients. No deaths occurred, and neurological sequelae were noted in four patients, three of whom had superior sagittal sinus thrombosis. All three patients with sigmoid sinus thrombosis showed favorable outcomes on discharge.

Proposed mechanisms of sinus thrombosis after mild closed head injury have involved endothelial damage within the venous sinus that causes thrombosis by activating the coagulation system.<sup>5</sup> The brain also contains an abundance of thromboplastin that is released after injury. Thromboplastin induces an abnormal hypercoagulable state like the destruction of platelets and erythrocytes.<sup>11,24,25</sup> However, fibrinolytic activity is higher in normal venous walls than in artery or capillary walls, and thrombi in the sinuses frequently recanalize with time due to fibrinolysis.<sup>24</sup> This mechanism may account for the mild cases of sinus thrombosis.

The characteristic CT findings of sinus thrombosis include the cord sign, dense vein sign, and empty-delta sign following the administration of contrast agents.<sup>4,10,29</sup> Indirect evidence such as hemorrhagic infarcts, venous infarcts, extensive cerebral edema, and narrowing of the cerebral ventricles suggest sinus thrombosis in many cases.<sup>1</sup> Cerebral angiography, particularly digital subtraction angiography, is the method of choice for diagnosing sinus thrombosis. However, complete absence of the transverse and sigmoid sinus may be a normal anatomic variation, so diagnosis of thrombosis in these areas should proceed cautiously.<sup>1</sup> MR imaging findings in sinus thrombosis have also been reported.<sup>17,19</sup> Four phases were identified based on sequential changes.<sup>13</sup> In the first few hours, the thrombi appear as isointense to slightly hypointense on the T<sub>1</sub>-weighted images, and hyperintense on the T<sub>2</sub>-weighted images, reflecting the presence of oxyhemoglobin.<sup>27</sup> Several hours after thrombus formation, the thrombus appears as hypointense on the T<sub>2</sub>-weighted images as deoxyhemoglobin becomes dominant. In the present case, the MR imaging findings were extremely valuable to establish the diagnosis during the acute phase, within several hours of thrombus formation, as confirmed by the signal intensities characteristic of oxyhemoglobin. Moreover, the phase-contrast MR angiography was used to depict veins selectively by appropriate flow-velocity settings.<sup>21,22</sup> MR imaging was superior to CT or cerebral angiography in four ways: thrombus in the sinus was visualized with no contrast medium; time elapsed since thrombosis could be estimated; the procedure was noninvasive and safe

for performance in a child; and sequential follow-up imaging was facilitated.

No consensus has been reached for the treatment of sinus thrombosis, because the severity varies greatly from case to case. Hypertonic diuretics and steroids are useful to treat increased intracranial pressure, but severe cases may require barbiturate therapy and hypothermia. Anticoagulant therapy with heparin or warfarin is frequently administered to limit extension of the thrombus. Secondary extension of the thrombus to cortical and deep veins has been found in autopsy cases, stressing the need to prevent extension.<sup>14)</sup> Anticoagulant therapy for sinus thrombosis improves outcome, even in patients with hemorrhagic infarct, and does not increase the risk of exacerbation hemorrhagic components of infarcts.<sup>8)</sup> However, since traumatic sinus thrombosis can be accompanied by direct hemorrhagic injury, anticoagulant therapy should proceed carefully with adequate monitoring for complications such as new bleeding. Local thrombolytic injections of urokinase or tissue plasminogen activator to facilitate recanalization may be given directly into the occluded sinus by intravascular techniques,<sup>7,16,28)</sup> but evaluation of the therapeutic effects and complications including pulmonary embolism is required.

The present case of sigmoid sinus thrombosis following mild closed head injury in an infant was treated successfully as MR imaging allowed early diagnosis and observation of sequential changes. Treatment must be both prompt and appropriate for the individual patient to achieve a good outcome.

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