Appearance and Disappearance of Empty Delta Sign in Superior Sagittal Sinus Thrombosis

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SUMMARY The diagnostic value of the empty delta sign on post-infusion CT films was investigated in five patients with superior sagittal sinus thrombosis subsequently verified angiographically and/or pathologically. The empty delta sign, which has been considered to be unique and reliable in the diagnosis of cerebral venous sinus occlusion, was observed only on CT films taken one to four weeks after onset, and was not seen in the extremes of the acute or the chronic stage of the illness. These observations may explain why this sign has not been apparent in some reports concerning the CT findings of superior sagittal sinus thrombosis. Recanalization within the thrombus may be the reason why this sign was no longer apparent in the chronic stage of the patients with superior sagittal sinus thrombosis.

Although computed tomography (CT) has gained wide acceptance as an accurate and noninvasive technique for the evaluation of intracranial lesions, the value of CT in the diagnosis of superior sagittal sinus (SSS) thrombosis remains unsettled. Several reports have described CT findings in SSS thrombosis and emphasized the importance of the so-called empty delta sign, clot in the sinus, dilated cerebral veins, multiple focal bilateral parasagittal hemorrhages, and gyral and tentorial enhancement. Among these findings, the empty delta sign was considered to be unique and reliable in the diagnosis of SSS occlusion. However, this sign was sometimes not apparent even in patients in whom cerebral sinus and venous occlusion was subsequently verified pathologically.

The purpose of this paper is to describe this CT finding, especially in connection with the diagnosis of SSS thrombosis.

Report of Patients

Patient One
A 37-year-old male had suddenly developed high fever up to 38.4°C, diarrhea and headache. He was admitted to a hospital, but headache and diarrhea lasted for almost five days. Four days before transfer to our hospital, he showed right-sided hemisensory disturbance followed by hemiparesis on the right side and total aphasia. After that, repeated generalized convulsions developed and the level of consciousness declined gradually. On admission, the level of consciousness corresponded to stupor, and there was right hemiparesis including the face; there was total aphasia. CT scans on admission (four days after onset of hemiparesis) showed a large low density area and multiple hemorrhages over the left parieto-occipital region, but the so-called empty delta sign was not apparent on the enhanced CT films at regular window settings. A cerebral angiogram showed displacement of the anterior cerebral artery from left to right on the AP view of the arterial phase. The SSS together with the torcular herophili was not visualized and, instead, abnormal cork-screw-like veins which did not reach the sagittal sinuses were observed in the venous phase. Follow-up CT scans at regular window settings taken 10 days (fig 1, left) and two weeks after onset both showed a filling defect at the level of the torcular herophili corresponding to the so-called empty delta sign. The patient was submitted to a craniectomy to reduce the intracranial pressure, and at this time the obstructed SSS and engorged superficial cerebral veins were observed. The empty delta sign was still present on the enhanced CT films one month later, but it has disappeared three months later (fig 1, right). Since then, this sign has never been observed in spite of repeated CT examinations over a period of more than five years.

Patient Two
A 28-year-old female, eight weeks pregnant, suddenly developed severe nausea and headache together with lassitude, followed by the development of a slight right hemiparesis. On admission she had neck stiffness and a mild right hemiparesis. She had a known history of epilepsy and had had an episode of mild head trauma two months previously. CT scans taken two years before and two months before admission for evaluation of epilepsy and head trauma, respectively, showed no abnormal findings even on enhanced CT films. CT scans taken two and three weeks after the onset of nausea and headache both showed a low density area on the left parietal region, and the empty delta sign was visualized on the enhanced CT film, both at regular and at higher window settings (fig 2, left). A cerebral angiogram confirmed that this patient had an occlusion of the SSS, but the filling defect within the torcular herophili was no longer clearly apparent on the CT films taken one month later and had completely disappeared on those taken 3 months later even with increased window width (fig 2, right). CT examinations repeated at approximately three months and four years did not show the delta sign.

In addition, CT films of three other patients with SSS thrombosis verified angiographically were care-
fully examined retrospectively. The characteristic delta sign was not seen in the acute stage, within 5 days after the onset of symptoms, nor in the chronic stage, more than 2 months after the onset, despite repeated examinations. All five patients described here have survived to date.

Discussion

Barnes and Winestock\(^1\) were the first to describe the CT findings of SSS thrombosis, but they emphasized the diagnostic value of dynamic radionuclide scanning for sinus thrombosis rather than CT scanning. Thereafter, several authors\(^2\) to\(^{14}\) described CT findings in SSS thrombosis, but the reported findings ranged from normal\(^3\), \(^{10},^{12}\) to various specific\(^2,^{4,6-9,14}\) and nonspecific\(^3\) findings. Among the specific findings on CT, the empty delta sign of Buonanno et al\(^2\) is considered to be unique and reliable.\(^6,9,14\) However, even Buonanno et al\(^2\) found this empty delta sign in only 2 out of 11 patients. Many other workers\(^1,5,7,8,9,13\) failed to detect this sign despite careful examinations. This discrepancy could be explained by our results. Some of the above reports did not indicate precisely when the CT scans were taken in relation to the onset of the illness. When the CT examinations were repeated not only in the extremes of the acute or chronic stages, but also in the sub-acute stage, the frequency of appearance of the empty delta sign on CT in SSS thrombosis seems to be higher. When CT scans were taken between one and four weeks after the onset of symptoms this specific finding was seen more frequently. Furthermore, the empty delta sign was more apparent at high display levels and narrow display windows.\(^6,9,14\) of CT.

The appearance or disappearance of the empty delta sign has been described previously by some authors without comment. Crimmings et al\(^14\) described a patient with SSS thrombosis in which CT films did not show the empty delta sign at five days but shows it clearly after eight weeks at high display levels and/or narrow display windows. Brant-Zawadzki et al\(^8\) reported that the empty delta sign seen on a post-infusion CT scan was no longer apparent in a follow-up CT scan two months later, but did not discuss the reason for this.

Fresh blood clot is isodense to the brain\(^8\) or may have slightly increased attenuation on CT films.\(^14\) This might be one of the reasons why we could not observe the empty delta sign on post-contrast images in patients in the extremely acute stage although we could not demonstrate an increased density in the SSS on unenhanced CT films. The obstructed sinus in the chronic stage usually shows numerous channels of recanalization within the thrombus.\(^8,15\) This perhaps explains why the empty delta sign was no longer apparent in the chronic stage of SSS thrombosis.

The empty delta sign is very important for the CT diagnosis of SSS obstruction, but it may not be apparent in the extremes of the acute or chronic stages of SSS thrombosis.

References

SUMMARY In spontaneously hypertensive rats, we studied the participation of xanthine oxidase-linked free radical in ischemia and reperfusion-induced cerebral injury, using allopurinol, a xanthine oxidase inhibitor. The loss of righting reflex was noted in some animals after a 4 hour occlusion of bilateral common carotid arteries and 19 of 25 animals died within 72 hours after reperfusion. One hour after reperfusion, the cerebral water content increased significantly, with an increase in sodium content and a decrease in potassium content. In 7 animals treated with oral administrations of allopurinol (200 mg/kg) 24 hours and 1 hour before occlusion, no death was found either during occlusion or after reperfusion, and the loss of righting reflex was noted in only one animal 24—72 hours following reperfusion. The increase in cerebral water content and accompanied changes in electrolyte contents were clearly prevented by allopurinol. These results suggest the possibility that the production of xanthine oxidase-linked free radical participates in cerebral injury due to ischemia and reperfusion in spontaneously hypertensive rats.

Effect of Allopurinol on Ischemia and Reperfusion-Induced Cerebral Injury in Spontaneously Hypertensive Rats

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XANTHINE OXIDASE ACTIVITY is a major factor in the generation of superoxide radical, and the production of free radical from xanthine oxidase has been considered to play an important role in the genesis of tissue injury due to ischemia and reperfusion.1,2 This has been proved by the evidence that allopurinol, a tight-binding competitive inhibitor of xanthine oxidase,3 prevents the ischemia and reperfusion-induced tissue injuries in the stomach, intestine, kidney, and heart.2,4,6 Such a free radical also seems to be implicated in the genesis of ischemia-induced enzyme damage in the gerbil cerebral cortex.7

In the present study, we studied the effect of allopurinol, a xanthine oxidase inhibitor, on ischemia and reperfusion-induced cerebral injury after bilateral common carotid artery occlusion in spontaneous hyperpertensive rats in order to investigate the importance of xanthine oxidase-linked free radical in cerebral injury.

Materials and Methods

Fifty-nine male spontaneously hypertensive rats (Okamoto and Aoki,8 23—37 weeks old, weighing 350—450 g), which had systolic blood pressure of 160—200 mm Hg as measured with electrophygmonometer (PE-300, Narco Biochem Inc., Houston), were used.

The animals were anesthetized with ether and fixed in supine posture, the skin was incised in the ventrolateral neck region and the common carotid arteries were separated from the surrounding tissue in the bilateral manner. A cotton thread was put on both arteries and it was passed through a polyethylene tube (20 mm length, PE-160, Clay Adams) and then pulled to obstruct completely the blood flow. Then, the tube was exposed out of the ventrolateral neck region and the skin was sutured. After the operation under ether anesthesia, the animals were transferred to individual cages. Four hours following the occlusion, the thread passing through the tube was cut to reperfuse the blood flow and 1, 2, 3, 24 and 72 hours after reperfusion, neurological symptoms and mortality were observed.

For the study of water and electrolyte contents, animals were anesthetized with intraperitoneal administration of pentobarbital-Na (Somnopentyl, Pitman-Moore Inc.) 40 mg/kg at 1 hour after reperfusion. In this study, spontaneously hypertensive rats having the surgical preparation but without carotid artery occlusion were used as sham-operated animals. Brains were removed immediately after decapitation under anesthesia. The posterior two-thirds of the cerebral hemi-
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