

Case Report

Precedence of Parenchymal Enhancement on CT Angiography to a Fatal Duret Hemorrhage

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We report a case of fatal duret hemorrhage (DH) in a patient with acute tentorial subdural hematoma and bilateral chronic subdural hematoma along the cerebral hemispheres. Preoperative CT angiography (CTA) revealed prominent parenchymal enhancement in the ventral pontomesencephalic area. After burr-hole drainage, a large hemorrhage developed in this area. The parenchymal enhancement in the CTA may reflect the pontomesencephalic perforating vessel injury, and may be a sign of impending DH of acute transtentorial downward herniation. Previous use of aspirin and warfarin might have potentiated the process of DH and increase the extent of the bleed.

Key Words : Duret hemorrhage · CT.

INTRODUCTION

Duret hemorrhage (DH) is a delayed hemorrhage in cases with transtentorial downward herniation of the brain stem. It usually occurs in the midline or anterior rostral pons and the lower midbrain¹. DH usually has a fatal clinical course leading to mortality or a vegetative state². Herein, we describe a case of fatal DH in a patient with acute tentorial subdural hematoma (SDH) and bilateral chronic SDH. Preoperative CT angiography (CTA) revealed a parenchymal enhancement in the ventral pontomesencephalic area, which preceded the fatal DH in this area. We discuss the significance of the parenchymal enhancement in patients with transtentorial downward herniation.

CASE REPORT

A 66-year-old man was admitted to the emergency department with a 3-day history of progressive headache, nausea and vomiting. He had been in a bed-ridden state with a bilateral posterior cerebral arterial infarction due to cardioembolism. He had taken aspirin (100 mg/day) and warfarin (4-5 mg/day) as well as hypertension medications. Physical examination revealed no definite neurologic deficits except for cognitive de-

fects and bilateral medial hemianopsia. Both of his legs were slightly weak due to disuse atrophy. His vital signs were stable. Laboratory investigations revealed a coagulation abnormality with a prolongation of prothrombin time (33.3 sec), prothrombin time international normalized ratio (3.14), and activated partial thromboplastin time (37 sec). Brain CT on admission revealed left-side tentorial acute SDH and bilateral hemispheric chronic SDH, as well as a transtentorial downward herniation of the uncus with signs of increased intracranial pressure, such as sulcal obliteration and sylvian fissure obliteration (Fig. 1A, B). The brain stem was unremarkable (Fig. 1C). CTA was acquired with precontrast and arterial phase 2 hours after the initial CT for the evaluation of stroke risk. CTA revealed a prominent, wedge-shaped, enhancing lesion in the ventral pontomesencephalic area arterial phase (Fig. 1D, E). During the first hospital day, his mental state rapidly decreased to stupor with pupil dilation and impaired light reflex. Subsequent brain CT revealed a small, acute hemorrhage in the tegmentum of the pontomesencephalic junction area and an increase in the amount of tentorial SDH (Fig. 1F). After the coagulation abnormality was corrected to normal with the administration of fresh frozen plasma, the chronic SDH was operated with trephination as usual. After the operation, the patient was still in a stupor with pupil dilation

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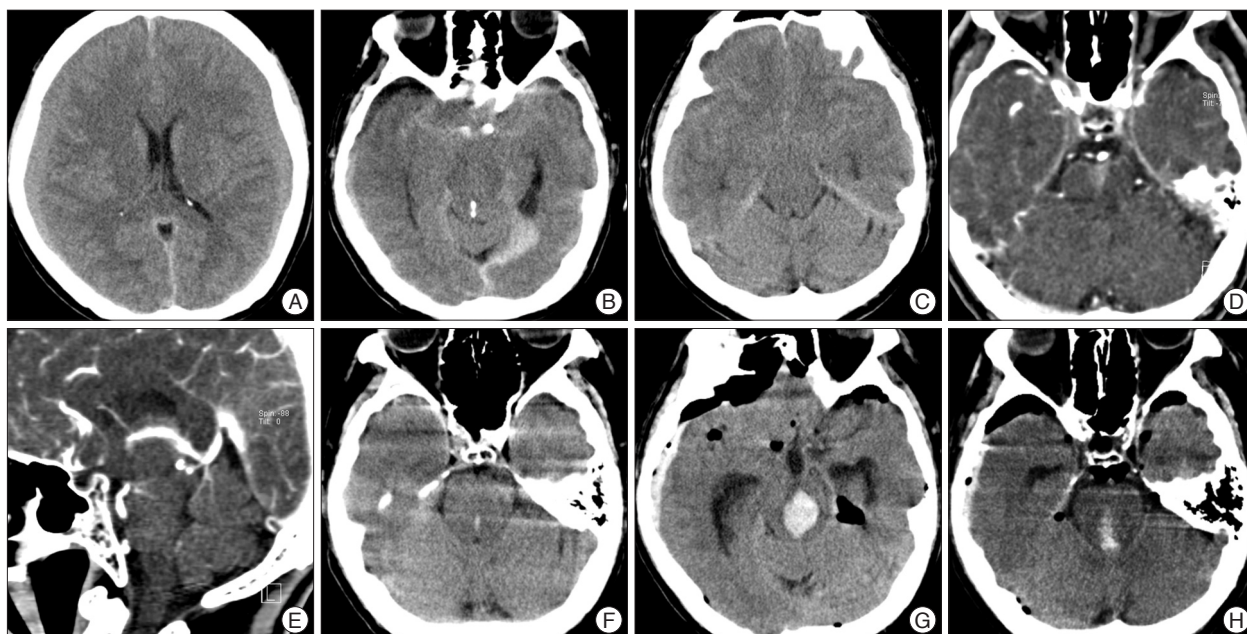


Fig. 1. Initial precontrast brain CT reveals left-side subacute tentorial subdural hematoma and bilateral subacute subdural hematoma with gyral displacement (A and B). No significant abnormal density is seen in the ponto-mesencephalic junction (C) and a bilateral transtentorial downward uncal herniation is seen. Preoperative CTA was done and an enhanced axial CT and sagittal multiplanar reformatted image at the arterial phase reveals abnormal wedge-shaped enhancement in the midline pontomesencephalic junction (D and E). The 1-day follow-up CT just prior to the operation reveals newly developed a scanty amount of hemorrhage in the midline tegmentum of the rostral pons (F). Postoperative CT after burr-hole drainage reveals a large amount of ICH centered on the pontomesencephalic brain stem (G and H).

and impaired light reflex. A postoperative brain CT revealed a large pontine and midbrain hemorrhage with an intraventricular hemorrhage (Fig. 1G, H). He remained in a vegetative state for approximately 2 years and died due to respiratory distress.

DISCUSSION

This case can be regarded as a typical DH because the hemorrhage was developed in the typical location for a DH and under a typical clinical setting. Although there are several theories for DH, the widely accepted mechanism is an ischemic injury to the anterior perforating vessels and brain stem caused by acute downward herniation of the brain stem². Perforating vessels of the brain stem can be narrowed and stretched by the rapid downward displacement and anteroposterior elongation of the brain stem. Reduced flow and mechanical insult will result in ischemic injury to the brain stem and perforating vessels². If the blood flow is restored by physiologic compensation or even increased by the rapid removal of the supratentorial causative lesion, reperfusion to the injured arterial bed occurs and it eventually forms a DH^{2,6}. In our case, rapid alleviation of IICP by burr-hole drainage and/or hemodynamic fluctuation during recovery from anesthesia might have accentuated the DH and resulted in a larger DH compared to the preoperative CT. In addition, previous use of warfarin and aspirin might potentiate the process of DH and increase the extent of the bleed.

A postmortem angiographic study demonstrated contrast

agent extravasation from the terminal branch of the pontine perforator in patients with central herniation⁴. In addition, a human autopsy study revealed necrosis and ruptures of the pontine perforator after transtentorial downward herniation¹. We suggest that the parenchymal enhancement on the CTA in this case may be a sign of the extravasation of the contrast agent from the injured perforator and reflect the severity of the vascular injury in patients with transtentorial downward herniation. This parenchymal enhancement on the CTA may be comparable to the parenchymal enhancement on MRI after intravenous t-PA that reflects early reperfusion and damage to the blood-brain-barrier of microvessels; this is regarded as a highly specific sign of subsequent hemorrhage^{3,5}. In our opinion, the parenchymal enhancement may be used as a sentinel sign of the impending DH in the case of transtentorial downward herniation. Capillary telangiectasia may have a similar parenchymal enhancement as found in this case. However, its incidence is relatively rare, and in most cases, it does not cause fatal hemorrhage by itself. In addition, it has a very slow flow and cannot be strongly enhanced in the arterial phase CT.

CONCLUSION

The parenchymal enhancement in the rostral pontomesencephalic area on the CTA preceded the development of a fatal DH. The parenchymal enhancement may be the sign of contrast agent leakage from the injured microvessels, and reflect severe vascular injury in the setting of transtentorial downward

herniation. Further study may be needed to evaluate the usefulness of this finding as a sign of impending DH.

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