

Acute Cerebral Infarction after Head Injury

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Cerebral infarction rarely occur following head injury. The authors present the case of a 39-year-old man with complete infarction in the middle cerebral artery(MCA) and anterior cerebral artery(ACA) territories occurred immediately after head injury. He had compound depressed fracture in right frontal bone with no neurological deficit. After the depressed bone elevation, postoperative computed tomography scan showed the right MCA and ACA territory infarction with midline shift. Cerebral angiography obtained on the day after emergent decompressive craniectomy showed the complete occlusion of the internal carotid artery(ICA) at the level of lacerum ICA segment. There was no evidence of neck vessel dissection and basal skull fracture. Cerebral infarction can occur in an ultraearly period after head injury without neck vessel dissection or basal skull fracture. We stress the need for attention to the cerebral infarction as the cause of a rare neurological deterioration of the head trauma.

KEY WORDS : Head injury · Infarction.

Introduction

Cerebral infarction caused by head injury has been rarely reported. In most cases, the major mechanism is the dissection of the carotid artery due to the accompanying injury in the neck area, vasospasm and the thrombosis developed in the vascular injury area are also known as a possible cause. In general, to identify the cerebral infarction in brain computed tomography(CT) after head injury, time interval to occurrence of cerebral infarction is necessary.

Cerebral infarction occurring immediately after head injury is extremely rare. Furthermore, cerebral infarction related to accidents may lead to legal problems because of an obscure causal relationship. Here we report a rare case of complete occlusion of internal carotid artery(ICA) in the patient who had no neck vessel dissection and basal skull fracture with a brief review of the literature.

Case Report

A 39-year-old man was immediately transported to emergency room after stroke by a car. The patient had compound depressed fractures in right frontal bone and co-

mplained of a headache. His vital signs were normal and no neurological deficits were found. He didn't have the history of diabetes, hypertension, etc. There was no specific family history. In routine chemical studies, lipid panels, blood coagulation tests, urine analysis, and fibrinogen study, any abnormalities were not detected, and the result of electrocardiogram(ECG) and chest X-ray film revealed normal findings.

Brain CT obtained one hour after trauma showed the right frontal depressed fracture without a brain swelling or hemorrhage(Fig. 1A). Depressed bone was reconstructed following massive saline irrigation under the general anesthesia. The dura underlying the fracture was intact. During the operation, his vital signs were stable and ECG monitoring showed no cardiac abnormalities.

Postoperatively, CT scan obtained 3hours after head injury revealed complete infarction in the right ACA and MCA territories associated with a severe brain swelling (Fig. 1B). The patient underwent an emergent decompressive craniectomy (Fig. 1C). After surgery, he was in a semicomatose and showed full dilation of the right pupil with no light response. On cerebral angiography obtained the day after the surgery, complete occlusion of the right ICA at the level of lacerum segment was detected (Fig. 2). He died 2weeks after the injury.

• Received : January 5, 2005 • Accepted : June 7, 2005

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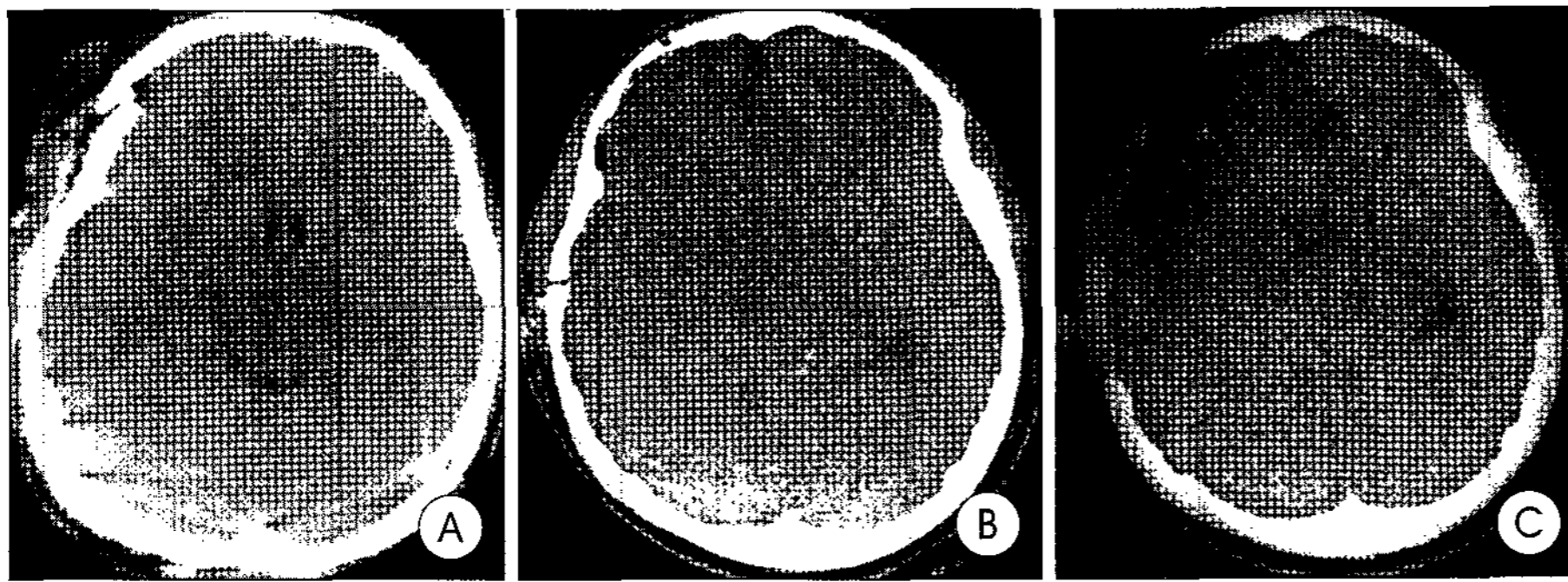


Fig. 1. A : Preoperative computed tomography(CT) showing the right frontal depressed fracture. B : CT obtained immediately after operation, showing the low density in the territory of anterior and middle cerebral arteries with the severe brain swelling. C : CT obtained after the emergent decompressive craniectomy.

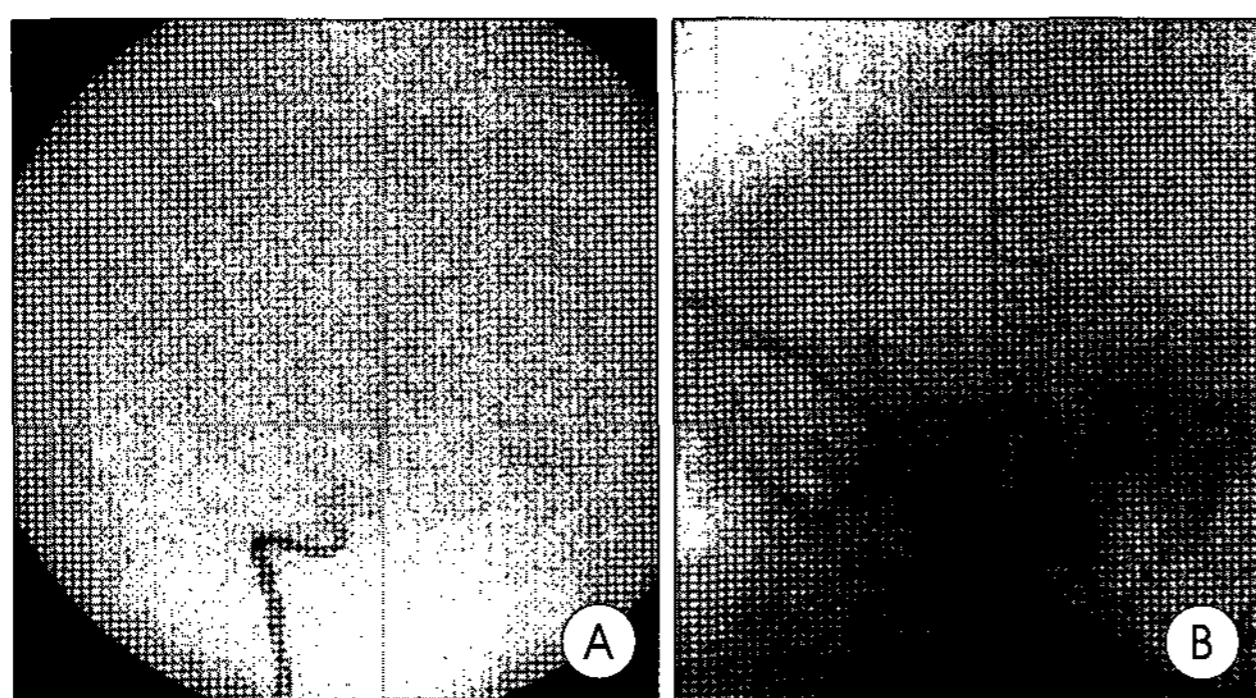


Fig. 2. Cerebral angiography on the day after decompressive craniectomy, showing the complete occlusion of the right internal carotid artery(ICA) at the level of lacerum ICA segment.

Discussion

Cerebral infarction can not be easily detected differently a cerebral hemorrhage or contusion in brain CT obtained immediately after accident. If cerebral infarction as the cause of an unexpected deterioration after accident was noted in a patient who had no neurological deficits at the time of admission, it may lead to legal problems because the casual relationship is not clear. Therefore, it is necessary to pay attention to the possibility of the development of cerebral infarction immediately after injury.

The incidence of cerebral infarction associated with trauma has been reported to be 1.9~2.9% in patients with the head and neck injury⁶⁾, and several possible mechanisms have been proposed. The relatively frequent cause is that the neck injury induces the hyperextension or vascular injury directly resulting in the dissection of the carotid artery or the development of pseudoaneurysm, which causes vasospasm or embolism that leads to cerebral infarction. It has been reported that this occurs more readily in the cases with a vascular lesion already⁷⁾. In Korea, the cases of the carotid artery infarction developed after the formation of saccular aneurysm following trauma, and the carotid occlusion caused by the vessel dissection have been

reported^{2,4)}.

In addition, after brain injury, even without the evidences of hemorrhage such as subarachnoid hemorrhage etc., vasospasm may occur, and the resulting cerebral ischemia is considered as the mechanism of cerebral infarction after accidents⁵⁾.

The cases of cerebral infarction due to the direct lesion of the intracranial vessel has been reported to be more scarce, and it has been

reported that the direct injury on the distal portion of ICA due to the basal skull fracture, or local thrombosis caused by the rupture of intima of the cerebral vessel due to the impact on the vessel wall during trauma induces cerebral infarction³⁾. Distinct from the cerebral infarction caused by the rupture of intima, the cerebral infarction caused the local injury on the vessel wall occurs within 24hours of injury in most cases, which is speculated due to the lag period from the vessel injury to the occurrence of secondary infarction due to the formation of thrombosis or pseudoaneurysm¹⁾. However, the development of minimal cerebral infarction 2hours after trauma has been reported, and in this case, the rupture of intima has been detected in autopsy³⁾. In addition, the MCA territory infarction by the fibrocartilaginous embolism from the nucleus pulposus has been reported⁸⁾.

In this report, hemispheric infarction was unexpectedly noted in the postoperative CT scan after the reduction of depressed skull bone. There were no warning signs and no time interval between the onset of trauma and the occurrence of infarction. Even though precise mechanism of cerebral infarction in this patient who had no neck injury or no basal skull fracture could not be evaluated, it is necessary to pay attention to the possibility of an unexpected cerebral infarction occurring immediately after head injury.

Conclusion

Cerebral infarction can occur in an ultraearly period after head injury without neck vessel dissection or basal skull fracture. We stress the need for attention to the cerebral infarction as the cause of a rare neurological deterioration after head trauma.

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