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Case Report

A case of cerebral amyloid angiopathy mimicking diffuse axonal injury

Abstract

A 61-year-old man became unconsciousness after falling down a flight of stairs due to the consumption of alcohol. The patient showed a deep comatose state, and therefore, he was transferred to this department. On examination, he showed a disoriented state and left hemiplegia. Computed tomographic scan of his head demonstrated 3 small intracerebral hemorrhages in the bilateral temporal and left frontal lobe as well as a subarachnoid hemorrhage near the left cerebellar tentorium, thus suggesting diffuse axonal injury. On the second hospital day, he regained full consciousness and his hemiplegia almost completely subsided. Magnetic resonance image of his head on the sixth hospital day indicated countless multiple hypointensity spots sparing basal ganglia and corpus callosum on T2*-weighted images, thus suggesting multiple microhemorrhages. He was diagnosed to have cerebral amyloid angiopathy because there was a dissociation between his clinical manifestations and the magnetic resonance imaging findings. This report discusses the differential diagnosis between diffuse axonal injury and cerebral amyloid angiopathy.

Cerebral amyloid angiopathy (CAA), hypertensive cerebral angiopathy, diffuse axonal injury (DAI), cerebral cavernous malformations, neurovasculitis, hemorrhagic metastasis, fat embolism, cerebral autosomal dominant arteriopathy with subcortical infarcts, and leukoencephalopathy can all cause multiple cerebral hemorrhages [1-4]. Diffuse axonal injury is most typical disease associated with multiple cerebral hemorrhages after blunt head injury [5]. Cerebral amyloid angiopathy is a condition characterized by amyloid depositions in the walls of leptomeningeal and cerebral cortical blood vessels. Clinically, CAA results in recurrent lobar hemorrhage, microhemorrhages, ischemic lesions, and dementia [6-8]. Multiple cerebral hemorrhages due to CAA can occur after a mild head injury [9]; however, there has been no report that describes how to make a differential diagnosis between CAA and DAI. This report presents a case of CAA mimicking DAI and details the clinical differences between these 2 diseases.

A 61-year-old man became unconsciousness after falling down a flight of stairs due to alcohol consumption. When health care providers checked the patient, he showed a deep comatose state, and therefore, he was transferred to this department. He had neither any particular family nor history of illness including hypertension. On examination, he had a sum score of 13 on the Glasgow Coma Scale. He had isochoric reactive pupils. His vital signs were as follows: blood pressure, 162/98 mm Hg; pulse, 66 beats/min; tympanic temperature, 36.5°C; and respiratory rate, 28 breaths/min. He showed left hemiplegia and a left temporal contusional wound. The remaining physiologic findings were unremarkable. The only abnormal results of the biochemical analyses of the blood were a high alcohol concentration (274.8 mg/dL) and creatine phosphokinase (310 IU/L). A computed tomographic (CT) scan of his head demonstrated 3 intracerebral hemorrhages of approximately 1 cm at the bilateral temporal and left frontal lobe as well as a subarachnoid hemorrhage near the left cerebellar tentorium, suggesting DAI (Fig. 1). On the second hospital day, he regained full consciousness and his hemiplegia almost completely subsided. He showed only a recent memory disturbance. The head CT findings demonstrated that each of the cerebral hemorrhages had only slightly increased in size. Surprisingly, on the sixth hospital day, magnetic resonance image (MRI) of his head indicated countless multiple hypointensity spots on T2*-weighted images (WIs), suggesting multiple microhemorrhages (Fig. 2). However, there were no hypointensity spots on the basal ganglia and corpus callosum. There was a dissociation between his clinical manifestations and the MRI findings [3] because cavernous hemangiomas or CAA had caused his multiple cerebral hemorrhages. Enhanced MRI did not depict any enhanced effect by contrast medium on the 10th hospital day, and thus, cavernous hemangiomas were ruled out and he was diagnosed to have multiple cerebral hemorrhages due to CAA induced by blunt head injury. His recent memory disturbance improved (Revised Hasegawa Dementia Scale

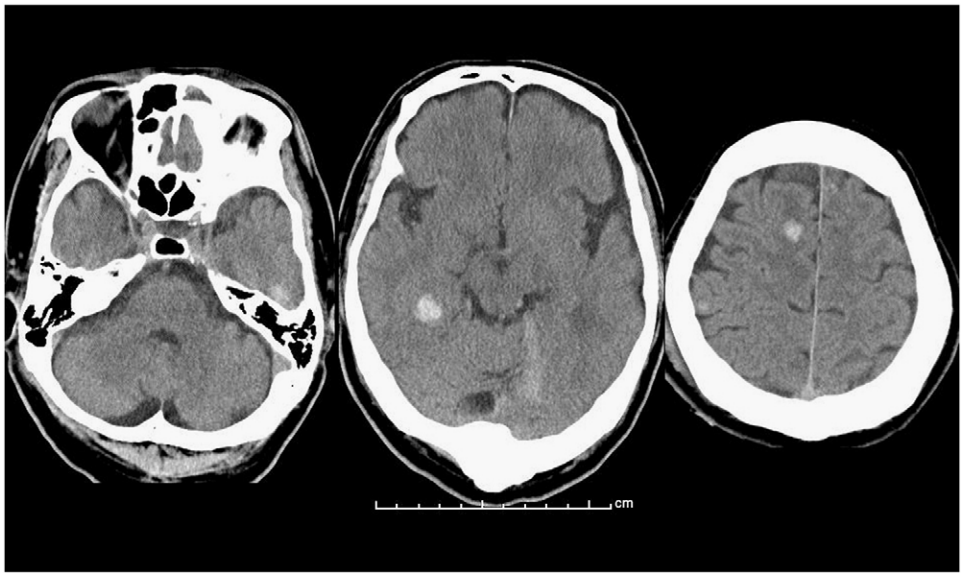

Fig. 1 Computed tomographic scan of the patient's head on arrival. The CT demonstrates 3 intracerebral hemorrhages measuring approximately 1 cm in size at the bilateral temporal and left frontal lobe as well as a subarachnoid hemorrhage near the left cerebellar tentorium, suggesting DAI.

[10]: 29 /30 points), and he was thereafter discharged under his own power on foot with only subtle motor weakness in his left leg on the 11th hospital day.

Cerebral amyloid angiopathy was diagnosed based on 3 findings. First, a quantitative analysis of the head injury using T2*WI revealed that the number of lesions detected by T2*WI correlated positively with both the duration of unconsciousness and Glasgow Outcome Scale in DAI [3,11]. He had more than 100 lesions on the T2*WI, and he should have shown a long-term comatose state and severe disability if the lesions had been DAI. Second, DAI tends to involve lesions at the corpus callosum; however, there were no such lesions observed in his brain [12]. In contrast, hemorrhages of CAA are located in subcortical lesions, and therefore, they rarely occur at the corpus callosum [13]. Third, DAI tends to occur in younger patients; however, the present patient was an older person [14]. In addition, CAA occurs more commonly in older patients than in younger ones [15]. The criterion standard of diagnosis of CAA is an autopsy or biopsy of the brain; however, a biopsy of CAA may result in a lethal hemorrhage due to the fragility of vascular tissue [16]. The clinical diagnostic criteria for CAA have been proposed by the Boston group [17]. According to this, CAA was diagnosed in this case on the basis of multiple hemorrhages in a patient older than 55 years for which no other cause can be found. Because the current patient had no history of hypertension, migraine, and malignancy and no enhanced lesions by contrast medium in his brain, and DAI was not causative of his multiple cerebra hemorrhages, the patient therefore fulfilled the Boston Criteria for probable CAA.

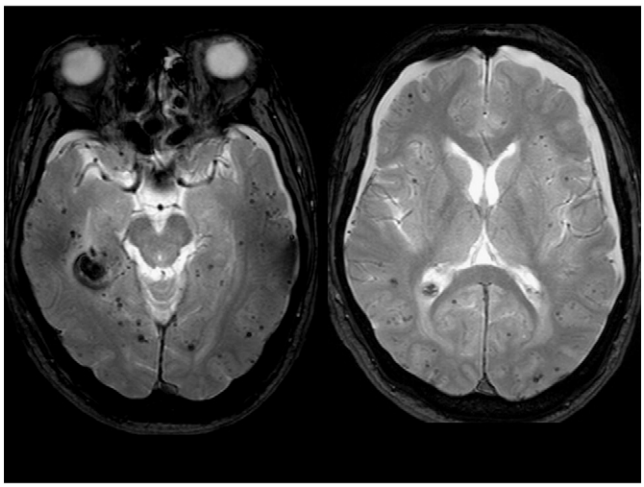

Fig. 2 Magnetic resonance image of the head on the sixth hospital day. The MRI indicates countless multiple hypointensity spots in a T2*WI, suggesting multiple microhemorrhages. No hypointensity spots were observed in either the basal ganglias or corpus callosum.

When head CT demonstrates multiple cerebral hemorrhages mimicking DAI after a head trauma, age, a dissociation between the number of microhemorrhages on MRI and clinical findings, and hemorrhages on MRI sparing the corpus callosum, then such findings are considered to be a key to making a differential diagnosis between DAI and CAA.

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References

[1] Blitstein MK, Tung GA. MRI of cerebral microhemorrhages. AJR Am J Roentgenol 2007;189:720-5.

[2] Yanagawa Y, Kaneko N, Sakamoto T, et al. Fat embolism syndrome with multiple hypointensity signals detected by head magnetic resonance imaging demonstrating a favorable outcome: a case report. Am J Emerg Med 2007;25:217-8.

[3] Yanagawa Y, Tsushima Y, Tokumaru A, et al. A quantitative analysis of head injury using T2*-weighted gradient-echo imaging. J Trauma 2000;49:272-7.

[4] Yanagawa Y, Sakamoto T, Okada Y, et al. Relationship between stroke and asymptomatic minute hemorrhages in hypertensive patients. Neurol Med Chir (Tokyo) 2001;41:13-7.

[5] Parizel PM, Ozsarlak, Van Goethem JW, et al. Imaging findings in diffuse axonal injury after closed head trauma. Eur Radiol 1998;8: 960-5.

[6] Felwick R, Amar K. A case of cerebral amyloid angiopathy. Med Sci Monit 2007;13:CS24-6.

[7] Smith EE, Greenberg SM. Beta-amyloid, blood vessels, and brain function. Stroke 2009;40:2601-6.

[8] Pezzini A, Del Zotto E, Volonghi I, et al. Cerebral amyloid angiopathy: a common cause of cerebral hemorrhage. Curr Med Chem 2009;16: 2498-513.

[9] Wakui K, Seguchi K, Kuroyanagi T, et al. Multiple intracerebral hemorrhages due to cerebral amyloid angiopathy after head trauma. No Shinkei Geka 1988;1287-91:16 (in Japanese).

[10] Tsukamoto R, Akisaki T, Kuranaga M, et al. Hasegawa Dementia Scale—Revised, for screening of early Alzheimer's disease in the elderly with type 2 diabetes. Geriatr Gerontol Int 2009;9:213-5.

[11] Yanagawa Y, Sakamoto T, Takasu A, et al. Relationship between maximum intracranial pressure and traumatic lesions detected by T2*-weighted imaging in diffuse axonal injury. J Trauma 2009;66:162-5.

[12] Rutgers DR, Fillard P, Paradot G, et al. Diffusion tensor imaging characteristics of the corpus callosum in mild, moderate, and severe traumatic brain injury. AJNR Am J Neuroradiol 2008;29:1730-5.

[13] Wagle WA, Smith TW, Weiner M. Intracerebral hemorrhage caused by cerebral amyloid angiopathy: radiographic-pathologic correlation. AJNR Am J Neuroradiol 1984;5:171-6.

[14] Adams JH, Doyle D, Ford I, et al. Brain damage in fatal non-missile head injury in relation to age and type of injury. Scott Med J 1989;34:399-401.

[15] Vernooij MW, van der Lugt A, Ikram MA, et al. Prevalence and risk factors of cerebral microbleeds: the Rotterdam Scan Study. Neurology 2008;70:1208-14.

[16] Jellinger K. Cerebrovascular amyloidosis with cerebral hemorrhage. J Neurol 1977;214:195-206.

[17] Knudsen KA, Rosand J, Karluk D, et al. Clinical diagnosis of cerebral amyloid angiopathy: validation of the Boston criteria. Neurology 2001;56:537-9.