

Case report

Multiple intracranial hemorrhages in a normotensive demented patient: A probable cerebral amyloid angiopathy

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Abstract

Cerebral amyloid angiopathy (CAA) is the most common cause of lobar intracerebral hemorrhage. Repeated bleeding may be presented with vascular dementia. We have reported a 68-year-old normotensive demented patient with probable CAA presented with hemiparesia, headache and vomiting. According to the experience of this case, it is recommended to consider CAA for normotensive elderly patients presented with multiple and superficial intracerebral hemorrhage.

KEYWORDS: Cerebral Amyloid Angiopathy, Intra Cerebral Hemorrhage, Dementia.

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Cerebral amyloid angiopathy (CAA) is the condition of β -amyloid deposition in the wall of cerebral cortex and leptomeninges vessels making them weak and more fragile.^{1, 2} The condition is more often asymptomatic.³ However, it can present as several clinicopathologic entities. The most common presentations are intracerebral hemorrhage (ICH) and dementia.⁴ Definite diagnose is depended on brain tissue sampling and this usually made after death or when a biopsy of the blood vessels of the brain during the surgery is done.⁵ Tissue sampling is not available for all suspicious patients; therefore, we should consider probable diagnosis based on Boston criteria.⁶ Here, we report a case of probable CAA that referred to Alzahra Hospital, Isfahan, Iran, in 2011.

Case Report

A normotensive 68-year-old male who suffered from dementia without parkinsonism sign and

symptoms, presented with hemiparesia without any decrease in level of consciousness. Symptoms started since two weeks before admission with headache, nausea, vomiting and hemiparesia. The severity of headache and permanent weakness forced the patient and his relatives seeking medical care. Past medical history revealed a progressive dementia since one year ago without history of hypertension or other related medical conditions. He did not have any history of trauma, but he had two attacks of transient hemiparesia and headache in previous year neglected as senile symptoms. Other than hemiparesia, neurological examination at the first day and at the time of discharge revealed that cognition impairment was compatible with subcortical dementia. Laboratory examinations, including: complete blood counts, electrolytes, liver and renal function tests, and coagulation tests were all within normal ranges. Computed tomography (CT) showed three well defined hyperdense cortical

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lesions over the both parietal lobes and left insular lobe without mass effect or surrounding edema (Figure 1). The further investigation using magnetic resonance imaging (MRI) with administration of contrast agent revealed neither further enhancements nor additional information compared to CT scan (Figure 2).

After 2 weeks, while the patient was hospitalized, we took another CT scan, and there was no evidence of previous lesions as shown in figure 3. Therefore, according to a documented multiple superficial intracerebral hemorrhage and other criteria, diagnose of probable CAA was introduced. Throughout the hospitalization, standard management of ICH was done ⁷ and finally, the patient was discharged with the order of cognitive enhancer drugs and rehabilitation.



Figure 1. Spiral brain CT scan on admission



Figure 2. Brain MRI with and without contrast agent on third day

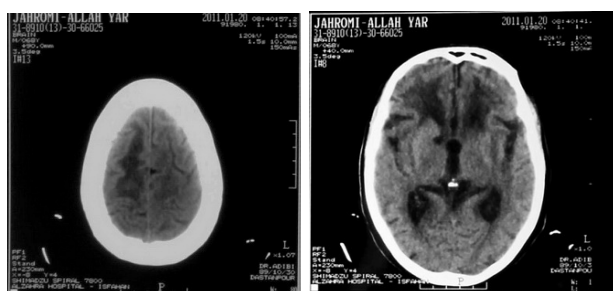


Figure 3. Spiral brain CT scan two weeks later

Discussion

Dementia and ICH are the main clinical presentations of CAA. ⁸ CAA is the most frequent cause of lobar ICH in the elderly. ⁹ The condition is typically observed in normotensive adults over 60 years old. ^{1, 3, 10} In contrast to deep distributed ICH resulting from hypertension, the location in CAA is generally superficial. ^{10, 11} CAA is more often asymptomatic ³ and in the brain autopsy of more than 40% of normotensive persons over the age of 70, we can find amyloid changes. ¹² Dementia may be seen earlier than symptomatic ICH in 25 to 40% of patients and can be slowly progressive, similar to that seen in Alzheimer disease. ³ The most common type of CAA is sporadic and associated with increasing age. Sporadic CAA can involve multiple lobes. ⁹ Angiography and MRI are conventionally used for assessment, but the angiography is not useful. ^{1, 3}

Multiple regions of the signal void in gradient echo MRI implies multiple distributed petechial hemorrhages and make the diagnosis of CAA. ³ For the standard diagnosis of CAA, Boston's criteria ^{3, 6} were made in 1990:

--Definite CAA: Full postmortem examination indicative of CAA + lack of other diagnostic lesions

--Probable CAA with supporting pathology: Clinical data and histopathological sample (evacuated hematoma or biopsy) indicative of some degree of CAA in the sample + lack of other diagnostic lesions.

--Probable CAA: Clinical data and MRI or CT indicative of multiple hemorrhages limited to lobar, cortical, or corticosubcortical areas (cerebellar hemorrhage allowed) in subjects aged > 55 years.

--Possible CAA: Clinical data and MRI or CT indicative of single lobar, cortical, or corticosubcortical area hemorrhage in subjects aged > 55.

At present, there is no confirmed treatment to stop the progression of CAA, ⁹ but prevention from further hemorrhage and symptomatic treatment of seizures and related symptoms are recommended. ¹ CAA usually has poor prognoses. Each experience of hemorrhage brings the increasing risk of disability and de-

pendence with permanent brain damage or even death.⁴ The management of ICH related to CAA is the same to the standard management of ICH with particular attention for usage of anticoagulant, management of intracranial pressure, and preventing complications. When the ICH causes a prominent mass effect and patient is impending to herniation, a prompt evacuation of hematoma can be life saving.⁴ Recurrence of ICH in CAA is common.¹³ A history of hemorrhagic stroke before the index lobar hemorrhage can predict early recurrence of ICH.⁴ Occasionally, some medications such as those used to treatment of Alzheimer's disease

may improve cognition and memory impairment. Seizures, occasionally termed "amyloid spells," can be treated with anticonvulsants such as phenytoin and carbamazepine.⁵

Conclusion

In summary, as the cerebral amyloid angiopathy is the most common cause of lobar parenchymal hemorrhage in elderly, we should be cautious and consider CAA in normotensive patients presented with multiple or superficial intracerebral hemorrhage, particularly when the patient is demented with a history of hemorrhagic stroke.

Conflict of Interests

Authors have no conflict of interests.

Authors' Contributions

ACh designed the study and also provided assistance in final editing of the article. RN gathered information about the patient and wrote the article. SMJM have reported the MRI and CT scan findings. MS reviewed related articles and gathered necessary information. SAF provided assistance in final editing of the article. All authors have read and approved the content of the manuscript.

References

1. Prasad BKD, Kejrival GS, Sahu SN. Cerebral amyloid angiopathy. *Indian J Radiol Imaging* 2006; 16(4): 745-7.
2. Tian J, Shi J, Mann D. Cerebral amyloid angiopathy and dementia. *Panminerva Med* 2004; 46(4): 253-64.
3. Chao CP, Kotsenas AL, Broderick DF. Cerebral Amyloid Angiopathy: CT and MR Imaging Findings. *Radiographics* 2006; 26(5): 1517-31.
4. Menon R. Cerebral amyloid angiopathy[Online] 2010. Available from: URL: <http://emedicine.medscape.com/article/1162720-print>.
5. MedlinePlus.Cerebral Amyloid Angiopathy[Online]2011.Available from: URL: <http://www.nlm.nih.gov/medlineplus/ency/article/000719.htm>
6. Knudsen KA, Rosond J, Karluk D, Greenberg SM. Clinical diagnosis of cerebral amyloid angiopathy: validation of the Boston criteria. *Neurology* 2001; 56(4): 537-9.
7. Blacker DJ, Musuka T. Management of intracerebral hemorrhage in 2020. *Future Neurology* 2011; 6(6): 745-56.
8. Yoshimura M, Yamanouchi H, Kuzuhara S, Mori H, Sugiura S, Mizutani T, et al. Dementia in cerebral amyloid angiopathy: a clinicopathological study. *J neurol* 1992; 239(8): 441-50.
9. Gürol ME. Cerebral Amyloid Angiopathy. *Turk Norol Derg* 2009; 15(1): 1-9.
10. Vinters HV. Cerebral amyloid angiopathy. A critical review. *Stroke* 1987; 18(2): 311-24.
11. Kim NR, Chung JG, Lee SK. Cerebral Amyloid Angiopathy. A case report. *Korean J Pathol* 2003; 37(2): 129-33.
12. Revesz T, Holton JL, Lashley T, Plant G, Rostagno A, Ghiso J, et al. Sporadic and familial cerebral amyloid angiopathies. *Brain Pathol* 2002; 12(3): 343-57.
13. Ishii N, Nishihara Y, Horie A. Amyloid angiopathy and lobar cerebral haemorrhage. *J Neurol Neurosurg Psychiatry* 1984; 47(11): 1203-10.