

Brain Abscess Associated with Cerebral Amyloid Angiopathy and Hemorrhage

Hsiao-Yue Wee¹, Che-Chuan Wang¹, Chien-Feng Li², Jinn-Rung Kuo^{1,3}

Abstract-

Purpose: We want to report an extremely rare condition, brain abscess associated with cerebral amyloid angiopathy and hemorrhage.

Case Report: We report on a 64-year-old woman who presented initially with moderate fever and headache. She was initially misdiagnosed with a bleeding tumor or arteriovenous malformation rupture and treated without antibiotic therapy. The mass was surgically drained due to neurological deterioration on day 14 after admission. *Streptococcus viridians* was isolated from the pus culture. The pathology evaluation showed cerebral amyloid angiopathy. She received intravenous antibiotic therapy for 4 weeks. She was eventually discharged home with left quadrantanopia.

Conclusion: This case reminds us early recognition of brain abscess formation at the site of intracerebral hemorrhage is very important for prompt and appropriate treatment to improve the overall prognosis.

Key Words: brain abscess, intracerebral hemorrhage, cerebral amyloid angiopathy

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INTRODUCTION

Cerebral amyloid angiopathy (CAA) is a disease of the elderly in which arteriolar degeneration occurs and amyloid protein is deposited in the media and adventitia layers of the cerebral arteries⁽¹⁾. CAA-related spontaneous intra-cerebral hemorrhage (ICH) represents only 2% of all ICH but is an important cause of hemorrhage in normotensive elderly patients without trauma⁽²⁾. Brain abscess associated with primary ICH developing at the site of preceding ICH is rare. Only 14 cases are reported in the literature^(3,4). The pathogenesis is explained by hematogenous spread of an infectious pathogen through

the fragile blood brain barrier after ICH. On the other hand, ICH due to brain abscess is very rare^(4,5). We report a case of brain abscess associated with cerebral amyloid angiopathy and hemorrhage. This presentation, to our knowledge, was rarely reported. The clinical picture and pathogenesis will be discussed.

CASE REPORT

64 y/o woman, denied of trauma history recently and any systemic disease before including hypertension, diabetes mellitus and others, but she received tooth replantation in the previous year. However, she was

From the Departments of ¹Neurosurgery, ²Pathology, Chi-Mei Medical Center, Tainan, Taiwan. ³Biotechnology Southern Taiwan University, Tainan, Taiwan.

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Corresponding author: Jinn-Rung Kuo, MD. Chi-Mei Medical Center, No. 901, Chung Hwa Road, Yung Kang City, Tainan, Taiwan 710.

E-mail: cmh7520@mail.chimei.org.tw

brought to the emergency room (ER) because of acute onset of severe headache and blurred vision. She had an intermittent fever (up to 38.9°C) of unknown origin for 5 days and received antipyretic and analgesic drugs at a local clinic. Upon arrival in the ER, her initial physical examination showed blood pressure of 119/73 mmHg, regular pulse of 94 beats/minute, and body temperature of 38.2°C. Neurologic examination showed clear consciousness, isocoric pupils with rapid light reflex and normal muscle power. A cranial nerve examination showed left homonymous hemianopsia and mild papilledema. Electrocardiography revealed normal sinus rhythm. Echocardiography showed no vegetation. Laboratory testing revealed white blood count of 8400/ μ L with polymorphonuclear leukocytes, 68%. C-reactive protein was 25.6 mg/l (hospital normal range, < 6 mg/l). Brain computed tomography (CT) revealed right parietal-occipital lobe hyper-dense lesion with perifocal edema (Fig. 1). The initial diagnosis was a suspected bleeding tumor or a ruptured arterio-venous malformation (AVM) related to the ICH. She was admitted to



Figure 1. Brain computed tomography showed a hyper-dense lesion with perifocal edema which was located in the right occipital lobe.

the neurological intensive care unit.

The patient was initially treated with betamethasone 4mg (initially Q8H and gradually decrease the dose), mannitol (20% 300 cc/btl) 75cc Q6H and gradually decrease dose, acetaminophen 500mg QID and other treatment. Tumor marker examinations (CA-125, CA-153, CA-199, carcinoembryonic antigen, and alpha-feto-protein) were negative. Abdominal sonography showed no significant findings. Brain magnetic resonance imaging (MRI) disclosed tubular-like enhanced components, perifocal edematous change, and a possible AVM causing an ICH (Fig. 2, A-B). The patient's general condition improved with conservative treatment.

On day 14 after admission, she had sudden onset of decreased muscle power, which reached zero, on her left side and blurred vision recurred again. Emergent brain CT revealed that the lesion became enlarged and brain edema became aggravated (Fig. 3). She underwent an emergent craniotomy. A large amount of pus (20 ml) was drained out from an abscess. Several abnormal vessels were located near the abscess wall. Bacteria cultures grew streptococcus viridians and the pathology results showed cerebral amyloid angiopathy (Fig. 4, A-B).

After surgery, the patient received intravenous antibiotics for 4 weeks (vancomycin 1000 mg Q12H + ceftriaxone 2 gm Q12H + metronidazole 500 mg Q8H for 2 weeks, and ceftriaxone 2 gm Q12H + ciprofloxacin 400 mg Q12H for another 2 weeks). Finally, she was discharged home with mild headache and left quadrantanopia.

DISCUSSION

Clinical course of brain abscess usually presents with initial non-specific cerebritis accompanied by signs of increased intracranial pressure. In most cases, neurological deterioration is late, fulminant, and very sudden. When the diagnosis is based on the initial clinical picture and imaging studies, delays often result from determining the definitive brain abscess diagnosis because it can be proven only during surgery.

We believe that our patient's brain abscess resulted from amyloid angiopathy. The probable explanation for

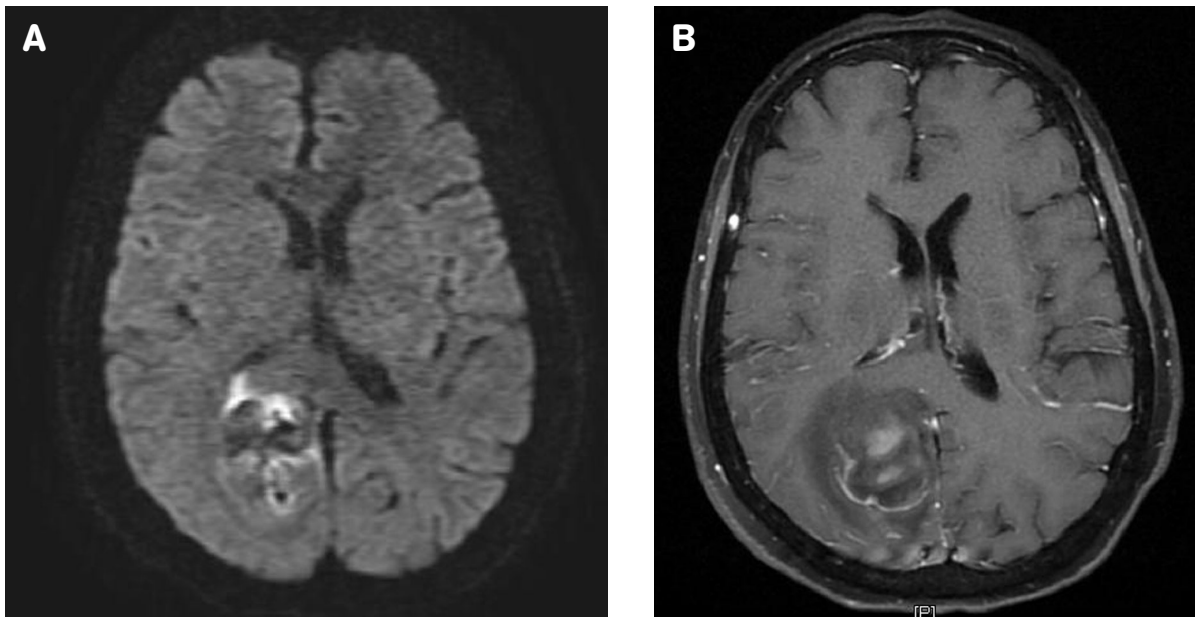


Figure 2. (A) Diffusion weighted magnetic resonance imaging (DWI) revealed heterogeneous density in the lesion. (B) MRI with T1 FLAIR and contrast showed that the lesion with suspicious tubular-like enhanced components, especially at the posterior part.



Figure 3. The right parieto-occipital hemorrhage with extensive white matter edema was complicated by the progressive mass effect, as compared with Figure 1.

the neurological deterioration of our patient is as follows. Initially, amyloid protein deposition gradually replaced the smooth muscle in the blood-vessel wall resulting in angiopathy. Cracks then developed in the wall, and plasma enzymes leaked into the cracks, digesting the wall and producing fibrinoid necrosis⁽³⁾. Based on this, we assumed that the involved area became a 'leaky' blood-brain barrier (BBB) with micro hemorrhages or small abscesses that transmitted *streptococcus viridians* via the blood stream concurrently with both processes. When the small abscesses or hemorrhages formed a cyst wall or hemosiderin wall, more severe intraparenchymal hemorrhage occurred secondary to the neovascular breakdown in the abscess cyst wall or hemosiderin wall. Finally, the bacteria in the hematoma directly infected the perifocal brain parenchyma and the abscess formed quickly leading to sudden neurological deterioration in our patient. Based on the initial imaging studies (Figure 1) in this case, the high density area could be clotted hematoma or hidden evidence of the underlying abscess, producing the high signal intensity on DWI and the partial contrast-enhanced MR images of the lesion, which mimicked tubular vessels. Thus, the undetected and

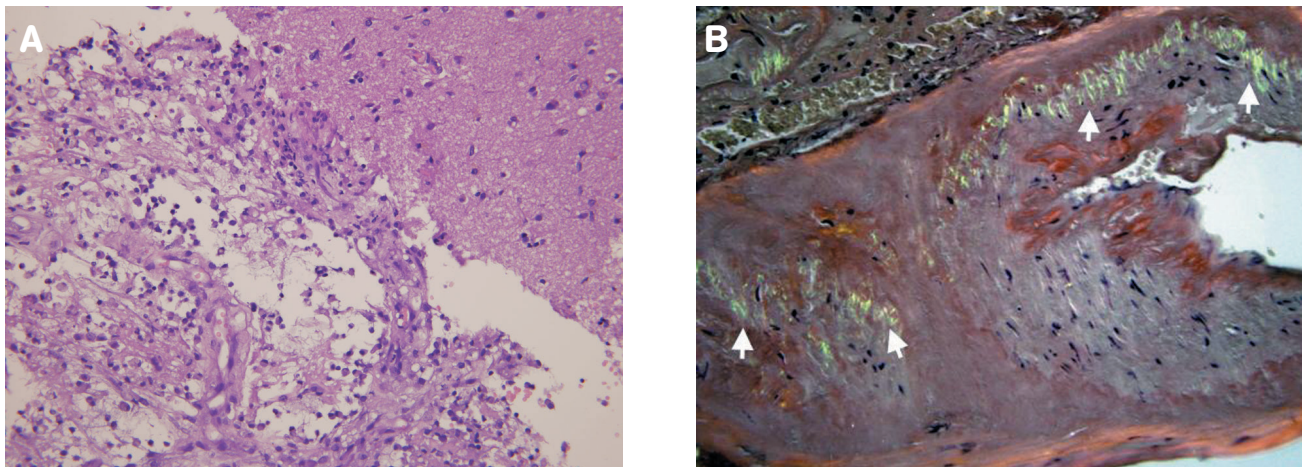


Figure 4. (A) Pathological examination showed mainly necrotic debris admixed with brain tissue (origin 10 x 20 magnification) (B) Congo red staining shows the vascular walls with apple-green birefringence under polarized lights and the abnormal deposition of amyloid protein (small white arrow indicate).

untreated abscess became larger and symptomatic 2 weeks after the ICH. Although the patient did not exhibit any dental symptoms, as streptococcus viridans is one of the normal flora bacteria species inside the oral cavity, we suspected that tooth replantation in the previous year might be the potential infection source.

In conclusion, brain abscess associated with a primary ICH, secondary to cerebral amyloid angiopathy is a rare condition. We want to emphasize that in a normotensive elderly patient without trauma, early recognition of brain abscess at the site of ICH is very important for prompt and appropriate treatment to improve the overall outcome.

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