

Cerebral Amyloid Angiopathy Associated with Cerebral Venous Thrombosis: A Rare Clinical Situation with Diagnostic and Therapeutic Challenges

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Abstract

Case Report

Cerebral amyloid angiopathy (CAA) is a small-vessel disease characterized by the deposition of β -amyloid in the walls of cortical and leptomeningeal vessels and is an important cause of lobar intracerebral hemorrhage in older adults. Cerebral venous thrombosis (CVT) is an uncommon cerebrovascular disorder for which anticoagulation represents the standard treatment. The coexistence of CAA and CVT is exceptionally rare and creates a major therapeutic dilemma because of the potentially high risk of intracranial hemorrhage. We report the case of an 84-year-old man with a history of two previous episodes of impaired consciousness associated with falls and confusion, requiring intensive care admission in 2021 and 2023, who presented with a two-week history of headache and cognitive impairment. Neurological examination revealed a Glasgow Coma Scale score of 13/15, brisk deep tendon reflexes in all four limbs, and severe cognitive impairment, with a Mini-Mental State Examination score of 11/30 and a Montreal Cognitive Assessment score of 10/30. Brain magnetic resonance imaging showed a right parieto-occipital intraparenchymal hematoma in the liquefaction stage, multiple predominantly lobar cerebral microbleeds, cortical superficial siderosis, a left capsulolenticular lacune, and vascular leukoencephalopathy. Magnetic resonance venography and cerebral angiography demonstrated thrombosis of the left transverse and sigmoid sinuses. The overall hemorrhagic pattern was highly suggestive of probable CAA according to the Boston criteria version 2.0. Given the high hemorrhagic risk associated with the previous lobar hemorrhage, multiple microbleeds, and cortical superficial siderosis, anticoagulation was not initiated. The patient received symptomatic management, including atorvastatin and donepezil, with clinical and radiological monitoring. The coexistence of CAA and CVT represents a rare and challenging clinical situation. Although anticoagulation remains the standard treatment for acute CVT, its use in patients with CAA and a high hemorrhagic burden requires an individualized assessment of the thrombotic and hemorrhagic risks and should preferably be discussed within a multidisciplinary team.

Keywords: cerebral amyloid angiopathy; cerebral venous thrombosis; intracerebral hemorrhage; cerebral microbleeds; cortical superficial siderosis; anticoagulation.

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INTRODUCTION

Cerebral amyloid angiopathy (CAA) is a small-vessel disease characterized by the deposition of β -amyloid peptide within the walls of small cortical and leptomeningeal arteries and arterioles [1,2]. It predominantly affects older adults and represents an important cause of spontaneous lobar intracerebral hemorrhage. Its radiological spectrum also includes strictly lobar cerebral microbleeds, cortical superficial siderosis, and characteristic white matter abnormalities [3,4].

The diagnosis of CAA in living patients is essentially clinicoradiological. The Boston criteria version 2.0 allow a diagnosis of probable CAA in patients aged 50 years or older who present with a compatible clinical syndrome and appropriate hemorrhagic lesions, with or without characteristic white matter features, in the absence of another cause that could explain the observed abnormalities [4].

Management of CAA primarily focuses on preventing recurrent hemorrhagic and vascular

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complications and controlling modifiable vascular risk factors. Decisions regarding antithrombotic therapy remain challenging and should be individualized, particularly in patients with multiple cerebral microbleeds, cortical superficial siderosis, or a previous lobar intracerebral hemorrhage [5,6].

Cerebral venous thrombosis (CVT) is an uncommon cerebrovascular disorder resulting from thrombosis of a cerebral venous sinus or cerebral vein. Its clinical presentation is highly variable and may include headache, seizures, focal neurological deficits, altered consciousness, or manifestations of intracranial hypertension. Computed tomography or magnetic resonance venography is generally required to confirm the diagnosis [8,9].

Anticoagulation is the standard treatment for acute CVT, including in patients with venous infarction or intracranial hemorrhagic lesions related to the venous thrombosis [8,9]. However, the coexistence of CAA and a substantial pre-existing hemorrhagic burden creates a particularly difficult therapeutic situation because anticoagulation may increase the risk of intracerebral hemorrhage [6,7].

The association between CAA and CVT appears to be exceptionally rare. Nevertheless, a small number of published observations have described this unusual coexistence and highlighted its diagnostic and therapeutic complexity [1,2].

We report the case of an elderly patient with probable CAA associated with CVT and discuss the diagnostic considerations and therapeutic dilemma raised by this uncommon association.

CASE REPORT

An 84-year-old man was admitted to our department because of a two-week history of headache associated with progressive cognitive impairment. His previous medical history was notable for two episodes of impaired consciousness associated with falls and confusion, requiring intensive care admission in 2021 and 2023.

On admission, the patient was afebrile and had a relatively preserved general condition. Neurological examination revealed a Glasgow Coma Scale score of 13/15 and brisk deep tendon reflexes in all four limbs. No definite focal motor deficit was identified. Cognitive assessment demonstrated severe impairment, with a Mini-Mental State Examination (MMSE) score of 11/30 and a Montreal Cognitive Assessment (MoCA) score of 10/30.

Brain magnetic resonance imaging (MRI), including susceptibility-weighted sequences, demonstrated a right parieto-occipital intraparenchymal hematoma in the liquefaction stage (Figure 1).

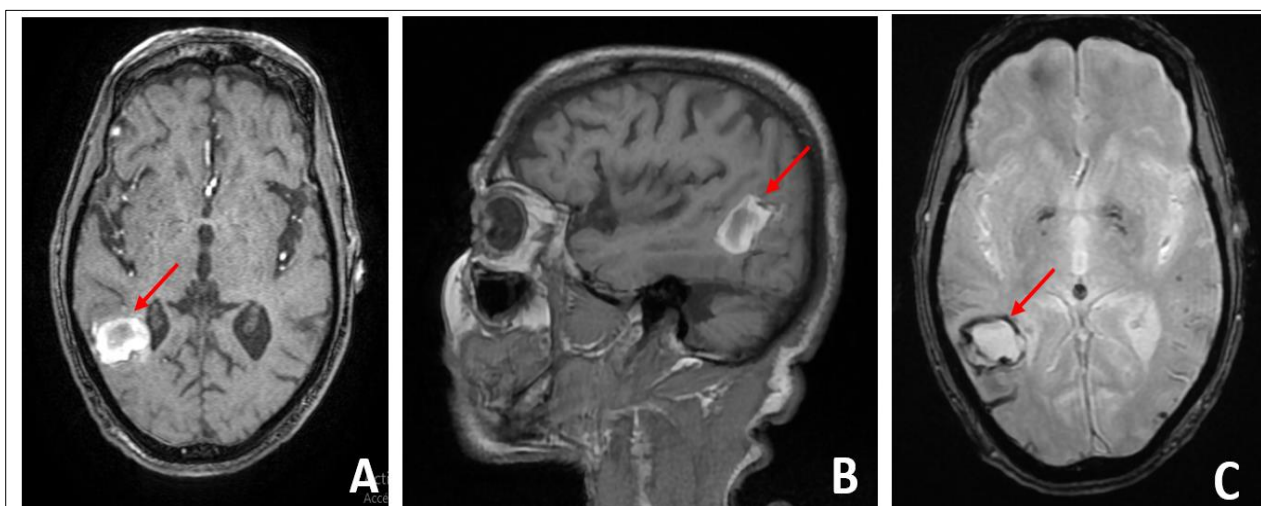


Figure 1: Brain MRI demonstrate a right parieto-occipital intraparenchymal hematoma in the liquefaction stage.

A: T1-weighted sequence, axial section, **B:** T2-weighted sequence, sagittal section, **C:** T2*-weighted sequence, axial section

Multiple cerebral microbleeds with a predominantly lobar distribution and cortical superficial siderosis were also identified (Figure 2).

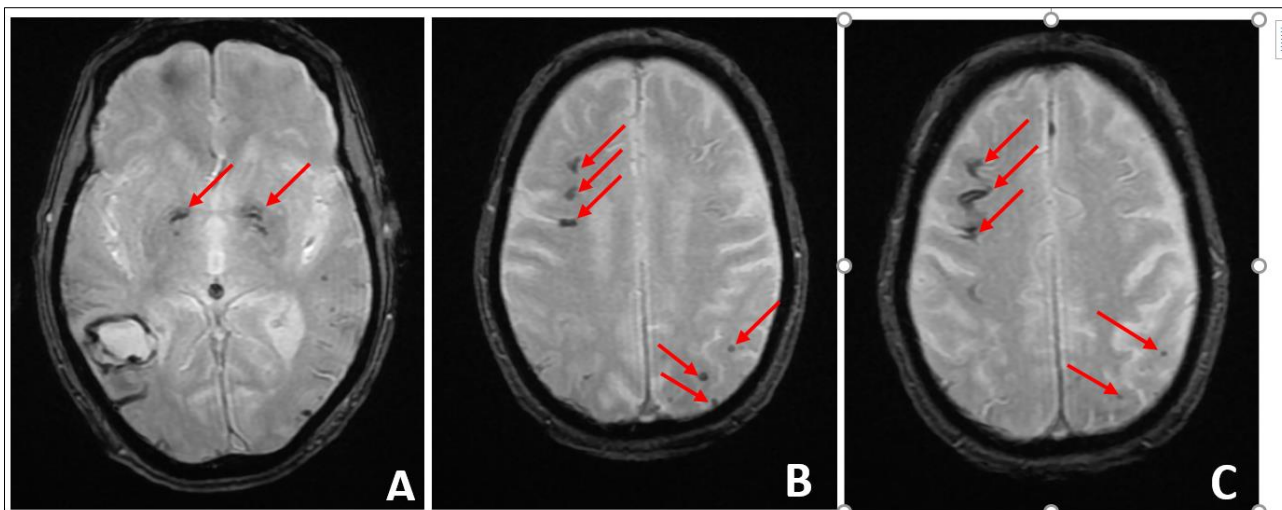


Figure 2: Brain MRI in T2*-weighted sequence, axial section demonstrates a multiple cerebral micro bleeds with a predominantly lobar distribution and cortical superficial siderosis.

A: cerebral micro bleeds with lobar distribution, **B and C:** cortical superficial siderosis.

Additional findings included a left capsulolenticular lacune and vascular leukoencephalopathy (**Figure 3**).

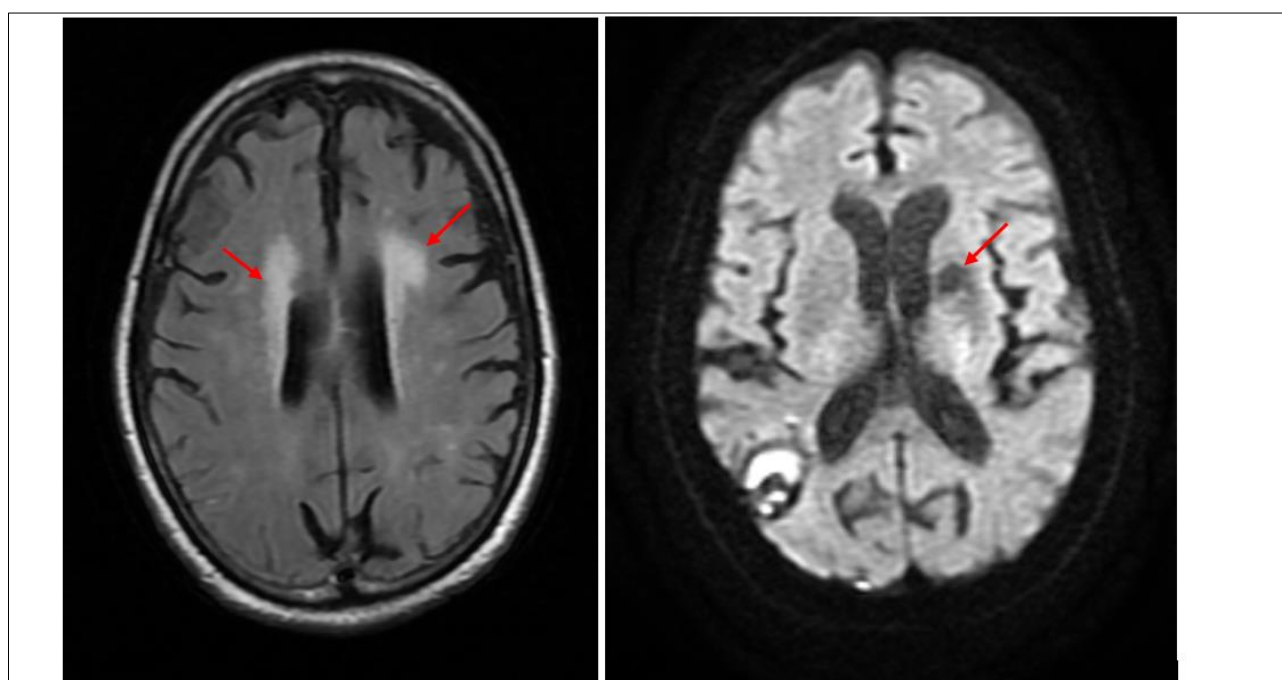


Figure 3: Brain MRI in axial section demonstrates a left capsulolenticular lacune and vascular leukoencephalopathy.

A: FLAIR sequence demonstrates a vascular leukoencephalopathy, **B:** Diffusion-Weighted Imaging (DWI) demonstrates a left capsulolenticular lacune

Considering the patient's advanced age, previous lobar intracerebral hemorrhage, multiple predominantly lobar microbleeds, and cortical superficial siderosis, the overall imaging pattern was highly suggestive of probable cerebral amyloid angiopathy according to the Boston criteria version 2.0 [4].

Magnetic resonance venography demonstrated thrombosis involving the left transverse and sigmoid sinuses (**Figure 3**). Cerebral angiography confirmed poor opacification of the left transverse sinus, consistent with venous thrombosis.



Figure 4: Cranial MRA (Magnetic Resonance Angiography) demonstrated thrombosis involving the left transverse and sigmoid sinuses

It also demonstrated an aneurysmal dilatation of the terminal segment of the right internal carotid artery (**Figure 4**).



Figure 4: Cerebral angiography confirmed demonstrates an aneurysmal dilatation of the terminal segment of the right internal carotid artery

Laboratory investigations did not reveal any significant coagulation abnormality or inflammatory syndrome. The lipid profile showed an LDL cholesterol level of 1.36 g/L, total cholesterol of 1.90 g/L, HDL cholesterol of 0.64 g/L, and triglycerides of 1.28 g/L. Antinuclear antibodies and ANCA were negative. The remainder of the laboratory work-up was unremarkable.

Lumbar puncture was performed and revealed a normal opening pressure.

The coexistence of CVT and a substantial hemorrhagic burden suggestive of CAA created a major

therapeutic dilemma. Although anticoagulation is the standard treatment for acute CVT [8,9], the patient was considered to have a particularly high risk of intracranial hemorrhage because of his previous lobar hemorrhage, multiple cerebral microbleeds, and cortical superficial siderosis [5-7].

After an individualized assessment of the potential benefits and risks, anticoagulation was not initiated. Symptomatic treatment was prescribed, including atorvastatin 20 mg/day and donepezil 5

mg/day, together with clinical and radiological monitoring.

The patient was hospitalized for one week. The course of the illness was marked by an improvement in headaches, while cognitive impairment remained stable.

DISCUSSION

Cerebral Amyloid Angiopathy

CAA is a small-vessel disease characterized by the accumulation of β -amyloid peptide within the walls of cortical and leptomeningeal vessels. It predominantly affects older adults and represents a major cause of spontaneous lobar intracerebral hemorrhage [3,5,10].

Clinically, CAA may present with lobar intracerebral hemorrhage, transient focal neurological episodes, seizures, or cognitive impairment. It may also remain clinically silent and be detected incidentally on brain MRI performed for another indication [3,5].

The main radiological manifestations include lobar intracerebral hemorrhage, strictly lobar cerebral microbleeds, and cortical superficial siderosis. These imaging markers are particularly important because they identify patients at increased risk of recurrent intracerebral hemorrhage [4,5].

In the present case, the patient's advanced age, previous right parieto-occipital intracerebral hemorrhage, multiple predominantly lobar microbleeds, and cortical superficial siderosis strongly supported the diagnosis of probable CAA. In the absence of neuropathological confirmation, the term probable CAA is more appropriate than definite CAA, in accordance with the current diagnostic framework provided by the Boston criteria version 2.0 [4].

Cerebral Venous Thrombosis

CVT is an uncommon cerebrovascular disorder accounting for a small proportion of all strokes. It results from thrombosis of a dural venous sinus or cerebral vein and may present with a broad range of clinical manifestations [8,9].

Headache is the most common presenting symptom. Seizures, focal neurological deficits, altered consciousness, and manifestations of intracranial hypertension may also occur [8].

Neuroimaging is essential for diagnosis. CT venography and MR venography can demonstrate absent or reduced venous opacification and define the extent of the thrombosis [8,9].

In our patient, thrombosis involving the left transverse and sigmoid sinuses was demonstrated by venous imaging and confirmed by cerebral angiography, establishing the diagnosis of CVT.

Anticoagulation in Cerebral Venous Thrombosis

Anticoagulation is the standard treatment for acute CVT. Current international recommendations generally support the use of heparin during the acute phase, followed by an appropriate oral anticoagulant according to the underlying clinical and etiological context [8,9].

An important feature of CVT is that intracranial hemorrhagic lesions resulting from venous congestion or venous infarction do not, by themselves, constitute an absolute contraindication to anticoagulation. The rationale for anticoagulation is to prevent thrombus propagation, facilitate recanalization, and reduce the risk of further thrombotic complications [8,9].

However, this general principle becomes more difficult to apply in patients with a pre-existing hemorrhagic small-vessel disease such as CAA, particularly in the presence of a previous lobar intracerebral hemorrhage and a high burden of cerebral microbleeds [6,7].

Cerebral Amyloid Angiopathy and the Risk of Hemorrhage During Anticoagulation

CAA is an important risk factor for lobar intracerebral hemorrhage. This risk is particularly concerning in patients with a previous intracerebral hemorrhage, multiple strictly lobar microbleeds, or cortical superficial siderosis [5,6].

The use of anticoagulants in patients with CAA therefore remains a complex clinical issue. Available evidence does not support considering CAA as an absolute and universal contraindication to anticoagulation. However, the presence and extent of hemorrhagic imaging markers should be carefully considered when assessing the individual risk-benefit balance [6,7].

In our patient, several high-risk features were present, including advanced age, previous lobar intracerebral hemorrhage, multiple cerebral microbleeds, and cortical superficial siderosis. The combination of these findings suggested a particularly high risk of intracranial hemorrhage if anticoagulation were initiated.

Association Between Cerebral Venous Thrombosis and Cerebral Amyloid Angiopathy

The coexistence of CVT and CAA is very rare. The available literature mainly consists of isolated clinical observations.

Mendel *et al.*, reported a case of intracerebral hemorrhage occurring in the context of CAA and cerebral venous thrombosis, illustrating the diagnostic and therapeutic difficulties associated with this coexistence [1].

Bernejo-Guerrero *et al.*, subsequently reported cerebral venous thrombosis in a patient with Iowa-type CAA, further supporting the possibility of an association between amyloid angiopathy and cerebral venous thrombosis [2].

Although these reports suggest that the coexistence of the two conditions is possible, the association remains exceptional. More importantly, their coexistence creates a therapeutic conflict: CVT generally requires anticoagulation, whereas CAA may confer a substantial risk of intracerebral hemorrhage, particularly in patients with a high hemorrhagic burden [1,2,6-9].

Therapeutic Dilemma and Individualized Management

The management of this association cannot be determined by a single therapeutic rule. Several factors should be considered, including the extent and clinical activity of the CVT, evidence of thrombus progression, the presence of intracranial hypertension, previous intracerebral hemorrhage, and the burden of CAA-related hemorrhagic markers on MRI [5-9].

In patients with acute CVT without major pre-existing hemorrhagic risk, anticoagulation remains the standard treatment [8,9]. Conversely, in patients with CAA, particularly those with a previous lobar intracerebral hemorrhage, multiple lobar microbleeds, and cortical superficial siderosis, the hemorrhagic risk associated with anticoagulation may become particularly significant [5-7].

In our patient, the decision not to initiate anticoagulation was based on an individualized assessment of the thrombotic and hemorrhagic risks. This decision should not be interpreted as evidence that anticoagulation is generally contraindicated in CAA, but rather as a patient-specific therapeutic decision in the presence of multiples markers of high hemorrhagic risk.

A multidisciplinary approach involving neurologists, neuroradiologists, and, when appropriate, hematologists or vascular specialists may be particularly useful in such complex cases.

CONCLUSION

The coexistence of cerebral amyloid angiopathy and cerebral venous thrombosis represents an exceptionally rare clinical situation that may pose major diagnostic and therapeutic challenges.

Recognition of CAA-related imaging markers, particularly lobar cerebral microbleeds, cortical superficial siderosis, and a history of lobar intracerebral hemorrhage, is essential for assessing the risk of recurrent intracerebral hemorrhage [4-6].

Although anticoagulation remains the standard treatment for acute CVT [8,9], its use in patients with

CAA and a high hemorrhagic burden requires an individualized assessment. CAA should not be considered an absolute contraindication to anticoagulation, but selected clinical and radiological profiles may justify a more conservative strategy after multidisciplinary discussion [6,7].

This case highlights the importance of a personalized approach to this rare association, balancing the risk of thrombotic progression related to CVT against the potentially severe hemorrhagic risk associated with CAA.

DECLARATIONS

Informed Consent: Informed consent for publication of this case report and the associated clinical and radiological data should be obtained from the patient or his legal representative.

Conflict of Interest: The authors declare that they have no conflict of interest related to this manuscript.

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