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Current Overview of Clinical and Radiological Findings Associated with Cerebral Amyloid Angiopathy

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ABSTRACT:
Purpose: In this review, the pathophysiology, clinical manifestations, and radiological findings of sporadic CAA will be detailed.
Material and Methods: Cerebral amyloid angiopathy (CAA), the second most common cause of spontaneous acute intracerebral hemorrhage after hypertension in the elderly population is characterized by brain parenchymal damage secondary to hemorrhage and ischemia caused by the accumulation of Aβ protein in the walls of small arteries and arterioles. Advanced age is the most significant risk factor for CAA. While the definitive diagnosis requires histopathological examination through autopsy/biopsy, the probable or possible diagnosis of CAA is based on clinical features as well as characteristic neuroimaging findings.
Results: With the increasing elderly population and the growing prevalence of succesibility-weighted magnetic resonance imaging sequences in routine, it becomes imperative to have a thorough understanding of the imaging spectrum associated with CAA. Early diagnosis is extremely critical in patients with CAA who have not yet developed intracranial hemorrhage. Furthermore, patients with CAA may present clinically transient focal neurological episodes or cognitive impairment, which can be mistaken for transient ischemic attacks caused by convexity subarachnoid hemorrhage.
Conclusion: Additionally, before initiating newly introduced anti-amyloid monoclonal antibody drugs in Alzheimer's disease, it is necessary to exclude signs of CAA. Moreover, the anticipated side effects of these drugs often manifest imaging abnormalities resembling inflammation or bleeding associated with CAA, necessitating familiarity with the imaging findings of CAA.

Keywords: *Cerebral amyloid anjiopathy; magnetic resonance imaging; susceptibility weighted imaging; cerebral hemorrhage*

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INTRODUCTION

Cerebral amyloid angiopathy (CAA) is a vasculopathy first described by Oppenheim in 1909, characterized by the accumulation of beta-amyloid (Aβ) protein in the walls of cortical and leptomeningeal vessels, particularly associated with cortical and lobar hemorrhage. CAA is the second most common cause of spontaneous acute intracerebral hemorrhage (figure 1) after hypertension in geriatric population (figure 2) (Biffi and Greenberg, 2011). Alongside age-related sporadic types, there are also genetic forms characterized by the accumulation of different types of amyloid at earlier ages. The accumulation of Aβ protein in the walls of small arteries and arterioles leads to parenchymal damage by causing hemorrhage and ischemia. Histologically, deposition of amyloid within media and adventitia layers of vessel wall results in thickening, hyalinization, and smooth muscle loss (Vinters, 1987). Advanced age is the most important risk factor for SAA, with the frequency increasing from 7.3% in the first 10 years after the age of 65 to 17.6% and further to 34.6% after the age of 85 (Greenberg and Vonsattel, 1997). While CAA is a well-known significant cause of lobar

intracerebral hemorrhage, convexity subarachnoid hemorrhage (SAH) can present with less known clinical manifestations characterized by transient neurological symptoms in this patient group (Banerjee et al., 2017). While the definitive diagnosis of CAA requires histopathological examination through autopsy or biopsy, the probable or possible diagnosis of CAA is based on clinical features and characteristic neuroimaging findings (Charidimou et al., 2022).

The wide spectrum of additional clinical and radiological features of CAA makes its early recognition radiologically even more crucial. With the increasing elderly population and the widespread use of sensitivity-weighted magnetic resonance imaging (MRI) sequences in routine practice, familiarity with the imaging spectrum associated with CAA becomes imperative. It is known that antiplatelet and anticoagulant medications commonly used in elderly patients due to accompanying diseases increase the risk and recurrence of intracranial hemorrhage in CAA patients (Biffi et al., 2010). Therefore, early diagnosis is extremely critical, especially in CAA patients who have not yet developed intracranial hemorrhage. Furthermore, CAA patients may present clinically with transient focal neurological episodes or cognitive impairment, which can be mistaken for transient ischemic attacks caused by convexity subarachnoid hemorrhage. Additionally, the side effects of newly introduced anti-amyloid monoclonal antibody drugs in Alzheimer's disease often manifest imaging abnormalities resembling inflammation or bleeding associated with CAA. This necessitates familiarity with imaging findings to distinguish between these conditions (Withington and Turner, 2022).

MATERIAL and METHODS

Purpose and Type of the Study

Systematic search was done via Pub med database on January 2023 by using the terms cerebral amyloid angiopathy (MesH) which returned 899 articles of related topic of which concerning radiological findings were further included. This review will detail the pathophysiology, clinical, and radiological findings of sporadic CAA.

Pathophysiology

In the pathogenesis of CAA, inadequate clearance of Aβ protein from interstitial fluid of central nervous system (CNS) is implicated. Perivascular spaces are areas of continuity between the tunica adventitia of arterioles, and subarachnoid space as well as cerebrospinal fluid. It is hypothesized that the removal of Aβ protein from the interstitial fluid in brain parenchyma primarily occurs through retrograde drainage of these perivascular spaces into the cervical lymphatic system (Morrone et al., 2015). Age-related conditions such as arteriosclerosis, previous traumas or infarctions contribute to decreased clearance of Aβ protein (Tarasoff-Conway, 2015). In CAA, there is an accumulation of soluble vascular Aβ derived abundantly from the amyloid precursor protein (APP), which is a transmembrane glycoprotein encoded on chromosome 21 and heavily produced in brain cells, consisting of 40 amino acids (Hawkes et al.,2014). This protein is different from the amyloid protein found in Alzheimer's disease, which accumulates in different locations, is 42 amino acids long, and has less solubility in water (Attems et al., 2004). Although they are distinct pathologies, CAA is detected in 90% of Alzheimer's disease cases, while only 25% of CAA patients have concomitant Alzheimer's disease (Ellis et al..1996). In CAA, vessels with a diameter smaller than 2 mm, primarily arterioles but less frequently capillaries, and rarely venules, are damaged by the accumulation of Aβ. The involvement is patchy and segmental, with both normal and abnormal vessels coexisting. Posterior brain regions, particularly occipital, posterior temporal, and parietal lobes, are prone to involvement. Cerebellar involvement typically emerges in later stages. Basal ganglia, thalamus, and white matter vasculature are preserved areas compared to the small vessel disease associated with hypertension (Charidimou et al.,2012a).

In the early stages, the accumulation of Aβ in the tunica adventitia leads to thickening of the vessel wall, while subsequent Aβ saturation of the adventitia results in decreased accumulation within the smooth muscles of the tunica media. Furthermore, the cytotoxic effect of Aβ leads to smooth muscle loss, resulting in vascular fragility and

thinning of the vessel Wall (Revesz et al., 2002).

For sporadic CAA, the presence of the apolipoprotein E (APOE) ε4 allele associated with vascular fragility, while APOE ε2 allele in relation with vascular accumulation of Aβ, have been identified as risk factors (Charidimou, 2015a). Light microscopy revealing presence of amyloid protein in the vessel wall with Congo red staining exhibiting green apple birefringence is the traditional diagnostic method. However, achieving a more sensitive and specific definitive diagnosis is feasible through immunohistochemical demonstration of anti-Aβ antibodies (Tarasoff-Conway et al., 2015).

In clinical practice, the updated Modified Boston Criteria are utilized to diagnose probable or possible CAA based on imaging and clinical findings without necessity for histopathology known as gold standard (Table 1) (Charidimou et al., 2022).

Table 1. Boston Criteria Version 2.0

Classification	Modified Boston Criteria (version V)
Final Diagnosis	Full postmortem examination • Spontaneous intracranial hemorrhage, cortical subarachnoid hemorrhage, transient focal neurological attacks, or cognitive impairment, or dementia development. • Severe CAA accompanied by vasculopathy. • Absence of other diagnostic lesions.
Pathology-supported probable CAA	Clinical findings and pathological tissue diagnosis (hematoma evacuation or cortical biopsy). • Spontaneous intracranial hemorrhage, cortical subarachnoid hemorrhage, transient focal neurological attacks, or cognitive impairment, or dementia development. • Presence of varying degrees of CAA in the specimen. • Absence of other diagnostic lesion.
Probable CAA	Age 50 or older, with clinical findings and MRI or CT • The clinical presentation in the form of spontaneous intracranial hemorrhage, cortical subarachnoid hemorrhage, transient focal neurological attacks, or cognitive impairment, or dementia; presence of at least 2 definite lobar hemorrhagic lesions on T2*-weighted MRI; concomitant presence of intracranial hemorrhage, cerebral microbleeds, or superficial siderosis of the convexity. OR • One lobar hemorrhagic lesion and one white matter feature (severely dilated perivascular spaces in the centrum semiovale or multifocal pattern of white matter hyperintensity). • The absence of other potential causes for hemorrhage or cortical superficial siderosis. • The absence of deep hemorrhagic lesions (intracranial hemorrhage or cerebral microbleeds) on T2*-weighted MRI. • Cerebellar lesions are not considered deep or lobar hemorrhages.
Probable CAA	Being over 50 years of age, with clinical findings and MRI or CT imaging: • With clinical presentation such as spontaneous intracranial hemorrhage, cortical SAH, transient focal neurological attacks, or cognitive impairment, or dementia, at least two of the following will be present; The presence of lobar hemorrhagic lesions on T2*-weighted MRI in association with any of the following: intracranial hemorrhage, cerebral microbleeds, or convexity subarachnoid hemorrhage (SAH) or cortical superficial siderosi, OR • "A white matter feature (severe visible periventricular white matter hyperintensity or a multifocal pattern of white matter hyperintensity) • The absence of other potential causes that could lead to hemorrhage or cortical superficial siderosis. • The absence of deep hemorrhagic lesions (intracranial hemorrhage or cerebral microbleeds) on T2*-weighted MRI. • Cerebellar lesions are not considered deep or lobar hemorrhages.

CAA (cerebral amyloid angiopathy); MRI (magnetic resonance imaging); CT (Computed tomography); SAH(Subarachnoid hemorrhage); PVD (pervascular distance)

Ethical Approval

Because of nature of the study, ethical approval was vaived.

RESULTS and DISCUSSION

Hemorrhagic Radiological Findings of CAA

Lobar hemorrhage, microbleeds, convexity subarachnoid hemorrhage (cSAH) and cortical superficial siderosis (cSS) (figure 1) are radiologically identified lesions associated with cerebral hemorrhage detected by CT and/or MRI and integrated into the Modified Boston Criteria 2.0 version, serving as diagnostic criteria for "possible or probable SAA" without the need for biopsy (figure 1) (Charidimou et al., 2022). In computed tomography (CT), particularly cSAH and lobar hemorrhage, appear as high-density lesions in the acute phase and is initially preferred modality due to high sensitivity for diagnosis (figure 4,5) Additionally, in magnetic resonance imaging (MRI) MRI, T2*-weighted gradient recalled echo (GRE) sequences are preferred due to their sensitivity to magnetic susceptibility effects of blood degradation elements such as hemosiderin, showing lesions associated with acute hemorrhage like lobar hemorrhage and cSAH, as well as chronic blood elements such as cerebral microbleeds and cortical superficial siderosis (cSS). (figure 1)Susceptibility weighted imaging (SWI) is an advanced MRI technique that includes the T2*-weighted GRE sequence, providing three-dimensional, high spatial resolution, magnitude, and phase information. It has become widely used in recent years for visualizing hemorrhagic lesions (Schweser, 2010).

Lobar hemorrhages are located in the cortical, subcortical, or gray-white matter junction and typically develop secondary to involvement of cortical or leptomeningeal arterioles with a diameter of less than 2 mm. These lobar hemorrhages differ from those involving deep structures such as basal ganglia, thalamus or pons, which occur secondary to bleeding from perforating arterioles in hypertensive patients and have central locations. Lobar hemorrhages are more frequent and severe in the posterior location of the occipital lobes (figure 4,5). Lobar hemorrhages manifest with clinical symptoms of hemorrhagic stroke, such as headache, altered consciousness, focal neurological deficits, or seizures, which are often reported depending on the location and size of the hemorrhage. Recurrence of bleeding is possible and worsens the clinical presentation (figure 4,5) (Greenberg and Charidimou, 2018).

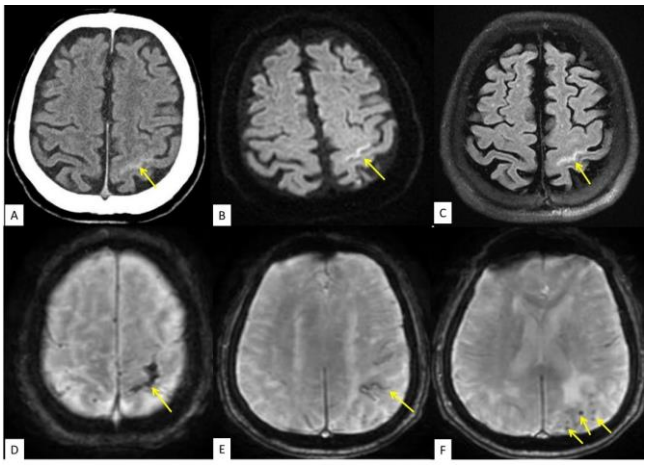

Figure 1. A 72-year-old male patient presented to the emergency department with sudden onset of speech disturbance, slurring of speech, and weakness in the right arm and leg, lasting approximately 8-10 minutes. On non-contrast cranial CT scan (A), a hyperdense lesion consistent with convexity subarachnoid hemorrhage (cSAH) is observed in the left precentral gyrus (arrow). On the same day, the hemorrhagic area identified on diffusion weighted imaging (DWI) (B) appears hyperintense (arrow), while it is indistinguishable on ADC (not shown). On the FLAIR sequence (C), the lesion is identified as hyperintense in the hemorrhagic area. On the same level on the successibility-weighted imaging (SWI) sequence, acute hemorrhagic area appears hypointense, while at lower levels, cortical superficial siderosis area (D) attributable to hemosiderin deposition on the surface of the gyrus, and multiple millimetric foci of susceptibility at cortical levels, which are typical findings accompanying cerebral amyloid angiopathy (E), are seen, not discernible on other sequences or CT. Additionally, there are patchy multifocal hyperintense lesions with partial coalescence at the level of deep white matter, indicative of white matter ischemic lesions.

Cerebral microbleeds are typically round or oval-shaped low signal foci, 2-10 mm in diameter, detected on SWI sequences located in or adjacent to the cortex. Patients are usually asymptomatic. Cerebral microbleeds, unlike the microbleeds associated with deep-seated arteriopathy in chronic hypertensive patients, are peripherally located similar to lobar hemorrhages. It is suggested that lobar hemorrhage arises from the unchecked progression of cerebral microbleeds. This also explains the bimodal occurrence of hemorrhagic lesions in CAA, presenting as either microbleeds or macrobleeds (lobar hemorrhage)(Greenberg et al., 2012).

Although cerebral microbleeds are known to be asymptomatic, they are suggested to be one of the factors responsible for cognitive impairment in CAA patients (figure 1,4,5) (Poels et al., 2012).

Besides hypertension (figure 2), many other etiologies can lead to cerebral microbleeds, each presenting with different demographic and clinical features. During the diagnostic process, a wide differential diagnosis list including diffuse axonal injury, cerebral vasculitis, acute hemorrhagic leukoencephalitis, hereditary arteriopathies, cerebral vasculopathy, cavernous malformations (figure 3), cerebral fat embolism, radiotherapy, encephalitis, hemorrhagic metastases, hypoxemia, Moya Moya disease, and posterior reversible encephalopathy syndrome (PRES) needs to be ruled out radiologically and clinically (Sharma et al, 2018). The cSAH refers to acute bleeding within one or more sulci at the level of the convexity. It appears as curved hyperdensity limited to thin sulci on CT scans. In MRI, fluid attenuated inversion recovery (FLAIR) sequences, acute hemorrhage has high signal intensity.

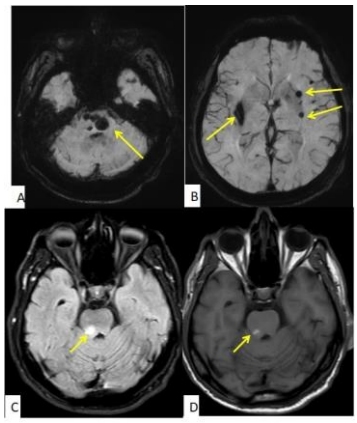

Figure 2. A 54-year-old male patient presented with complaints of headache and nausea. Successibility-weighted imaging (SWI) sections reveal bilateral hemorrhagic foci at the level of the brainstem in the pons (A) and at the slice passing through the basal ganglia (B). On the right side of the pons, hyperintense acute hemorrhagic focus is observed on FLAIR (C) and T1-weighted (D) sections. These findings were compatible with hypertensive hemorrhage.

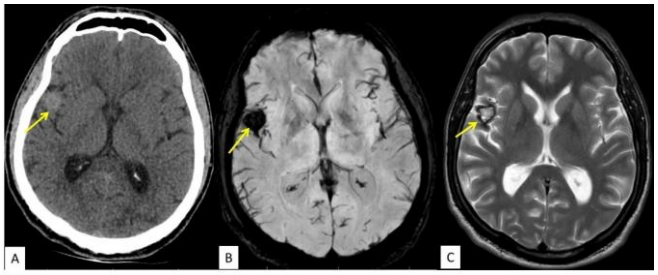

Figure 3. A 43-year-old male patient presented to the emergency department with facial and tongue twitching and difficulty speaking. On CT scan (A), a round lesion containing hyperdense bleeding is observed at the lobular level in the temporal lobe parenchyma adjacent to the right sylvian fissure. While the hemorrhage appears as a distinct signal-free area on SWI section, it is observed as hyperintense with a hypointense rim surrounding it on T2-weighted section (C). These findings were compatible with cavernoma

It develops as a result of extension from leptomeningeal vessels or more rarely, intralobar hemorrhage. Clinical manifestations are more specific and different from typical presentation of aneurysmal rupture or perimesencephalic subarachnoid hemorrhage in occurring in the basal cisterns or Sylvian fissure which patients present sudden severe headache typically described as, markedly headache. These patients typically describe transient focal neurological symptoms that spread within minutes, lasting less than 30 minutes. This is referred to as transient focal neurological episodes associated with CAA (figure 1). The pathophysiology involves cortical spreading depolarization phenomena. Headache may be mild or absent (Smith et al., 2021).

Distinguishing these transient symptoms from aura in migraine, focal Jacksonian seizures, and especially transient ischemic attacks (TIAs) can be challenging. Furthermore, these symptoms may herald future lobar hemorrhage, and failure to recognize them may increase the risk of lobar hemorrhage, particularly in patients using antiplatelet or antithrombotic drugs, which are more dangerous in these cases (Charidimou et al. 2012b).

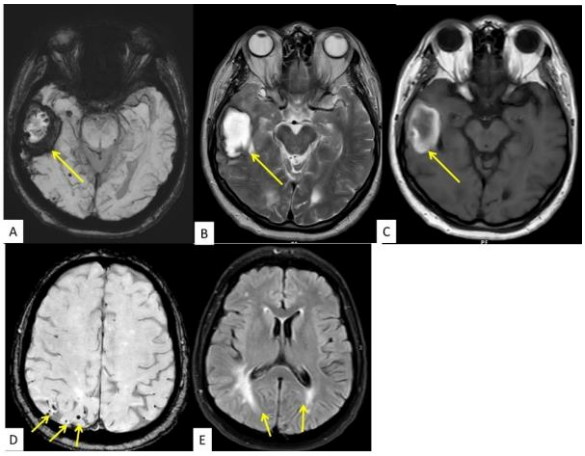

Figure 4. A 67-year-old male patient with a history of coronary artery disease and taking 100mg/day aspirin presents with headache and blurred vision. On SWI section (A), signal loss is observed in the right temporal lobe parenchyma consistent with a lobar hematoma; on T2 (B) and T1 (C) weighted sections, a large lesion containing signal indicative of acute parenchymal hematoma is seen. Multiple microbleed foci accompany on the SWI slices (D) passing from above. On FLAIR section (E) passing through the ventricular level, patchy hyperintense foci consistent with periventricular white matter ischemia are observed. These findings were compatible with propable cerebral amyloid anjiopathy related lobar haematoma.

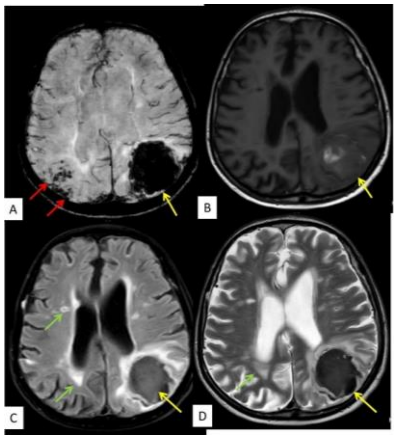

Figure 5. An 81-year-old male patient presented to the emergency department with confusion, agitation, and incontinence. A large lobar hemorrhage area is observed in the left occipital lobe, with distinct hypointensity on SWI (A), iso to hyperintensity on T1-weighted section (B), hypointensity on FLAIR (C) and T2-weighted (D) sections, surrounded by a hyperintense vasogenic edema area. Additionally, microbleed foci consistent with propable cerebral amyloid angiopathy (SAA) are visualized on SWI (A) in the right occipital region (red arrows). Hyperintense foci indicative of white matter ischemia are present on FLAIR and T2-weighted sections.

In the subacute and chronic stages, blood products appear as low signal hemosiderin on SWI sequences on the cortical surface, termed cSS. C SS may not always manifest clinically. Many diseases presenting cSAH and cSS should be considered in the differential diagnosis. Several entities affecting supratentorial vascular structures, such as reversible cerebral vasoconstriction syndrome, vasculitis, venous thrombosis, infective endocarditis, Moya Moya disease, and posterior reversible encephalopathy syndrome (PRES), are among the leading causes (Sharma et al., 2018).

Distinguishing cSS, which appears as hypointense in SWI, from flow-related signal loss, calcifications, hemorrhagic infarcts, aggregated microbleeds, and thrombosed cortical vessels radiologically is also necessary (Charidimou et al., 2015b).

Cortical superficial siderosis of the central nervous system (CNS) is a different entity is characterized as superficial siderosis that occurs at infratentorial level and is characterized by hearing loss, ataxia, and pyramidal symptoms.

Non-Hemorrhagic Radiological Findings in CAA

In CAA, cerebral deep white matter areas, such as the periventricular white matter, becomes vulnerable to ischemia and infarction due to involvement of penetrating arterioles (Atttems et al., 2011).

Cerebral white matter is predominantly supplied by penetrating arterioles that cross the cortex via leptomeningeal arteries without any anastomoses. In cases of CAA, particularly in moderate to severe cases, stenosis or occlusion of these arteries can leads to ischemia in the white matter. Furthermore, loss of smooth muscle in the vessel wall leads to impaired cerebral autoregulation and the expected vascular wall response to focal ischemia does not occur (Atttems et al., 2011).

This results in chronic ischemia in the white matter and leads to acute infarct areas in the form of lobar lacunes or microinfarcts (Lee and Markus ,2006, Kimberly et al.,2009).

In cases where smooth muscle loss is severe, gray matter is also involved in the process, particularly resulting in atrophy in the posterolateral cortex. Chronic ischemic areas in the white matter appear as hypodensities on CT and hyperintensities on MRI. Multiple focal hyperintense ischemic lesions in the white matter are one of the non-hemorrhagic lesions integrated into the Boston criteria (V2.0) in the possible or probable diagnosis of CAA (Charidimou, A et al.,2022).

When lesions merge, leukoaraiosis occurs; subcortical U fibers are preserved due to dual vascular supply. (figure 1,4,5). Lobar lacunes, which are larger than microinfarcts, typically range from 3 to 15 mm in diameter. They show diffusion restriction in acute phase, appearing as hyperintense on diffusion-weighted images and hypointense on apparent diffusion coefficient (ADC) maps, representing areas of acute cytotoxic edema (Kimberly, W. T., et al.,2009).

Table 2. The hemorrhagic and non-hemorrhagic lesions detected on brain imaging in cerebral amyloid angiopathy

Hemorrhagic lesions	Non-Hemorrhagic lesions
Lobar hemorrhage	Cerebral white matter hyperintensities related to ischemia
Cerebral microbleeds	Lobar lacunar infarct/Microinfarct
Convexity subarachnoid hemorrhage	Dilated perivascular spaces in the centrum semiovale
Cortical superficial siderosis	Cortical atrophy

Dilated periventricular spaces (PVS) in the centrum semiovale

The removal of amyloid proteins from the interstitial fluid via perivascular spaces has been defined as one of the most important clearance pathways (Morrone, C. D., et al.,2015). As a result of damage to perivascular drainage, the saturation of vessel walls with Aβ protein leads to expansion of perivascular spaces through retrograde flow, further impairing perivascular drainage and perpetuating a vicious cycle (Charidimou, A., et al.,2014).

Dilatation PVSs are another non-hemorrhagic lesion

integrated into the Modified Boston criteria for the possible or probable diagnosis of CAA. While perivascular spaces are indistinguishable radiologically on CT, on MRI, they appear as linear or oval hyperintense vascular structures parallel to the course of the vessels on T2-weighted images. It has been demonstrated that dilated PVSs at the level of the centrum semiovale are associated with more severe CAA histopathologically. Additionally, an increase in the frequency of cortical microbleeds and cSS has been reported to correlate with dilated PVSs (Koo, 2016).

Dilated PVSs observed at the level of the basal ganglia have been reported to be associated with hypertensive arteriopathy (Martinez et al.,2013).

Cognitive Impairment/Cognitive Decline

In patients with CAA, cognitive impairment or functional losses are attributed to lesions such as microbleeds, chronic white matter ischemia, and lobar lacunes. Due to the strategic locations of these lesions, they are believed to alter cognitive functions or progressively decrease them due to disruption in neural network flow. In lobar hemorrhage (Maia et al., 2007) or CAA related inflamation (Auriel et al., 2016), cognitive decline is sudden and progressive. It is also known that the presence of CAA contributes to cognitive decline in patients with Alzheimer's disease (Viswanathan and Greenberg, 2011).

CAA related inflammation

Inflammation associated with CAA is the perivascular inflammation resulting from an autoimmune reaction to vascular Aβ protein. The presence of the APOE e4 allele is particularly implicated in this autoimmune process (Eng et al., 2004).

The clinical presentation includes subacute or rapidly progressive cognitive impairment, headache, focal neurological signs, seizures, and alterations in consciousness. MRI typically reveals asymmetric, subcortical hyperintense lesions on T2 and FLAIR sequences. On CT, the corresponding findings for lesions are hypodense subcortical areas. Although it may be challenging to differentiate from entities like vasculitis or PRES both clinically and radiologically, the presence of other imaging findings associated with CAA supports the diagnosis (Auriel et al., 2016).

Amyloid-Related Imaging Abnormalities (ARIA) associated with Alzheimer's drugs

Even though both CAA and Alzheimer's disease stem from abnormalities in amyloid clearance mechanisms, the mechanisms of brain damage and resulting clinical manifestations differ (Greenberg et al.,2020).

Recently approved by the FDA for disease-modifying treatment, anti-amyloid monoclonal antibody drugs like aducanumab and lecanemab are being used in patients with mild cognitive impairment or mild Alzheimer's dementia who test positive for amyloid. Before treatment, MRI is commonly used non-invasively to visualize structural changes. In MRI, it is of critical importance not only in identifying non-neurodegenerative etiologies but also in evaluating the pattern and extent of neurodegeneration, and determining whether the patient is suitable for anti-amyloid therapy. In brain MRI imaging, patients should not exhibit the defined findings associated with CAA, such as five or more microhemorrhages, any superficial siderosis, lobar macrohemorrhage, large territorial infarct, small infarcts, or widespread white matter disease, in order to be considered suitable for treatment. One of the most concerning side effects associated with anti-amyloid monoclonal antibodies approved by the FDA is the occurrence of amyloid-related imaging abnormalities (ARIA) presenting in two distinct patterns. The risk of developing ARIA increases with higher doses of anti-amyloid therapy, the presence of the APOE ε4 allele, and the presence of more than four microhemorrhages or severe white matter disease on imaging prior to treatment. It typically occurs within the first 6 doses. While often asymptomatic, mild cases appear to be less common compared to moderate and severe cases (Brashear et al.,2018).

ARIA-edema (ARA-E) manifests as vasogenic edema in the posterior cortical-subcortical white matter and resembles the imaging findings of inflammation associated with CAA. Most patients are asymptomatic, and it typically resolves within 4-16 weeks(%74). ARIA hemarorrhage (ARIA-H) ,on the other hand, is characterized by the development of microscopic or macroscopic areas of hemorrhage or siderosis in the brain parenchyma While ARIA-E is almost always transient, ARIA-H tends to be stable

over time (Charidimou et al.,2012b).

CONCLUSION

CAA is one of the common causes of hemorrhagic stroke in the elderly population. Particularly in recent years, with the routine use of hemorrhage-sensitive SWI imaging, a diagnosis of possible or probable CAA can be made based on clinical and radiological findings without histopathological confirmation. With increasing familiarity among radiologists and neurologists regarding the different imaging and clinical findings associated with CAA, clinicans can weigh the risks and benefits of anticoagulant medication use based on individual patient profiles before the onset of lobar hemorrhage. Consequently, this approach can prevent recurrent bleeding and prove beneficial for patients. Other conditions apart from CAA, which cause microbleeds and cortical superficial siderosis in the brain, should also be considered in the differential diagnosis. The imaging findings associated with Alzheimer's disease drugs in clinical use can manifest as hemorrhagic or inflammatory features similar to those seen in CAA.

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The authors do not have any comments regarding this article.

Conflict of Interest

Authors declared no conflict of interest

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