CLINICAL AND RADIOLOGICAL APPROACH TO THE DIAGNOSIS OF BRANCH ATHEROMATOUS DISEASE: CASE REPORT AND REVIEW OF LITERATURE

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DOI: https://doi.org/10.32896/cvns.v7n1.11-18
Received: 10.12.2024
Revised: 26.03.2025
Accepted: 28.03.2024
Published: 31.03.2024

ABSTRACT

A form of ischemic stroke known as branch atheromatous disease (BAD) is brought on by plaque accumulation that obstructs or stenoses the entrance of penetrating branches. Despite its therapeutic importance, this concept is still poorly understood and underutilized in clinical practice and research. BAD is strongly linked to an increased risk of disability and early neurological degeneration (END). Based on clinical status, risk factors, laboratory results, and radiographic aspects of infarct and blood vessel morphologies, BAD has several characteristics. Determining treatment options and each patient's prognosis depends on early BAD identification.

Keywords: Branch atheromatous disease, stroke, imaging

INTRODUCTION:

A subtype of ischemic stroke known as branch atheromatous disease (BAD) is caused by proximal atherosclerosis of the arteries, which extends to the origin of perforating branches leading to occlusion [1]. Louis Caplan developed the idea of BAD in 1989 based on autopsy results described as an obstruction at the start of a deep penetrating artery in the brain related to a junctional plaque or micro atheroma and caused a small infarct in the internal capsule or pons [2]. Although it is considered a mild deep brain infarct, this condition can result from either atheromatous occlusion, as seen in Branch Arterial Disease, or lipohyalinotic degenerative changes, which are often associated with a true lacunar infarct [3]. Because the prognosis and treatment approaches for these two vascular disorders differ, it is essential to distinguish between them [4].

Ten to fifteen percent of cases of acute ischemic stroke (AIS) are BAD, a prevalent subtype of AIS [5]. BAD-induced stroke has been strongly associated with worse outcomes than lacunar strokes, including high rates of early neurological deterioration (END), recurrent transient ischemic events, and disability. Within 48 to 72 hours of the stroke's commencement, END happens in 17% to 75% of BAD-related stroke [1,6]. Based on their neurological symptoms or signs and the results of their admission magnetic resonance imaging (MRI), patients with BAD are frequently misdiagnosed as having lacunar infarcts brought on by lipohyalinotic degeneration. This leads to a miscalculation of the estimated prognosis and a delay in therapeutic therapy [7].

BAD is mainly determined by indirect imaging findings, such as particular morphological traits of the ischemic lesion thought to be produced by it because conventional imaging techniques cannot show tiny vessel alterations [2,8]. Infarctions in the subcortical regions in which blood perfusion dependent on deep and small perforating arteries were commonly referred to as BAD-related infarctions [2]. The lenticulostriate artery (LSA), anterior choroidal artery, thalamoperforating artery, paramedian pontine artery (PPA), and Heubner's artery are among the arteries that are classified as perforating arteries. The characteristics of ischemic lesions are used to indirectly study the involvement of LSA and PPA [6]. Recent studies have shown that high-resolution magnetic resonance imaging (HRMRI) with T2-weighted turbo spin echo (T2 TSE) sequence with black blood technique and 3D mapping can be used to study intracranial artery vessel morphology, including atherosclerotic plaque, irregular wall thickening, arterial remodelling, and focal geometrical features of the artery. These features are all expected to be crucial vascular risk factors in the development of atherosclerosis [4,9].

CASE REPORT:

A 58-year-old man with hypertension, diabetes, and dyslipidemia suddenly became weak on the left side of his body; however, he was still able to ambulate. The symptoms worsen in the evening, accompanied by dysarthria when the patient cannot ambulate. When he was brought to our hospital, a neurological test showed that he had left hemiparesis in his arm, leg, face, and dysarthria with GCS E4V5M6, blood pressure 142/78 mmHg, NIHSS score 8 – which increased to 10 after 3 hours of admission, and high C-Reactive Protein (CRP) level (164 mg/dl). The patient also routinely consumed direct oral anticoagulants (DOAC) because there was a history of deep vein thrombosis. MRI and MR angiography (MRA) revealed acute right basal ganglia, corona radiata infarction, and right terminal middle cerebral artery (MCA) segment M1 stenosis (Figure 1). Digital subtraction angiography (DSA) showed mild stenosis at the right MCA (Figure 2). Based on focal geometric characteristics of the middle cerebral artery, our patient is categorized as a straight type

of M1 on both sides (Figure 3). Despite starting a statin and antiplatelet medication, his left hemiparesis worsened, and the next day, he went flaccid. The patient was then discharged and continued with rehabilitation therapy.

DISCUSSION:

Branch atheromatous disease (BAD), which causes occlusion or stenosis at the penetrating branch's entrance due to localized atherosclerotic plaque-based thrombus, contributes 10.3%–10.8% of all cerebral infarctions [9]. BAD is distinguished from the other small and big arterial disorders by a single subcortical infarction greater than a lacunar stroke in the regions of deep perforators without accompanying severe arterial stenosis [5,10]. BAD-related stroke is categorized as an embolic stroke of unknown source (ESUS) under the Trial of ORG 10172 in Acute Stroke Treatment (TOAST) classification, which is commonly used to categorize cerebral infarction [10]. Since different vascular pathologies require distinct treatments, it should be possible to distinguish between lacunar infarct induced by lipohyalinotic degenerative changes (LD), and the primary differential diagnosis of BAD-related stroke [3]. Some epidemiologic studies indicate that BAD is more prevalent in male patients with age ≤ 60 years old who reside in Eastern Asia and Eastern Europe as opposed to Western nations. Patients with BAD are more likely than those with LD to have hypertension and hyperlipidemia [5,11].

Based on clinical symptoms, BAD is inferred when there is the progressive and gradual development of the ischemia, based on the signs and symptoms, suggesting intrinsic “thrombotic” disease rather than occult embolism [7]. Despite being understudied and lacking a recommended treatment, BAD-related stroke is linked to significant rates of early neurological deterioration (END) and disability. There are several criteria for END. First, the interval of symptoms is approximately seven days after the onset. The neurologic impairments worsen after the initial evaluation, which includes an increase of at least four points in the NIHSS score or a rise of at least one point in the NIHSS motor score for ischemic patients, or the attacks continue to occur at least three times after hospitalization or progress to persistence status for patients with internal capsule warning syndrome or pontine warning syndrome. Elevated inflammatory markers and CRP were also predictive of poor outcomes of the stroke [6,10].

The vascular territory and/or shape of the acute ischemic lesion are used to radiologically diagnose BAD because the branching arteries may not be readily visible by conventional imaging techniques [2]. LSA and PPA are the most frequently affected of the previously mentioned perforating arteries. The lenticulostriate arteries supply the lateral part of the globus pallidus and the head of the caudate nucleus, the anterior limb of the internal capsule, putamen, and the anterior part of the periventricular corona radiata [3]. In the LSA area, BAD is diagnosed using the radiological criteria listed below: a) A "comma-like" infarct lesion and has a "fan-shaped" extension that from bottom to top on the coronal slice with size more than 10 mm in diameter on axial slice, visible on 3 or more axial slices on diffusion-weighted imaging (DWI) of the LSA area; b) MRA or computed tomography angiography (CTA) or digital subtraction angiography (DSA) demonstrate that the parent artery of the diseased vessel (corresponding middle cerebral artery) does not have > 50% stenosis [9,10]. Additional radiological features can aid in diagnosing Branch Atheromatous Disease (BAD). Notably, leukoaraiosis and microbleeds, which are indicators of small vessel disease, are less frequently observed in patients with BAD [8]. BAD can develop from conditions such as arterial tortuosity, which can develop due to hereditary conditions, advanced age, and hypertension. It can also cause haemodynamic alterations and initiate the

formation of atherosclerosis in specific vessel [11]. It is anticipated that focal geometric features will also be a significant vascular risk factor in the onset of atherosclerosis. Because the shear stress is weak on the inside of the curve, plaque is likely to form at blood vessel curvatures.
The M1 segment morphology can be classified as either straight or curved based on pictures from three-dimensional time-of-flight magnetic resonance angiography (MRA) in coronal projection. From the anterior cerebral artery and M1 bifurcation site, a line was drawn to the M2 bifurcation point. The curve was categorized as upward type M1 if its vertex was above this line and downward type if it was below. If it had no vertices, it was classified as straight type. According to a study by Nagasawa, 2023, since the lenticulostriate artery typically branches out from the superior side of the MCA M1 segment, plaque is likely to form at the entry of the penetrating branch, making BAD more likely to occur in patients with downward type M1. Additionally, straight type M1 is more likely to be the site of plaque rather than upward type M1 [9].

CONCLUSION:

Diagnosing and differentiating BAD with LD is essential to determine the patient's initial management. Since BAD is caused by arteriosclerosis-induced non-cardiogenic cerebral infarction, antiplatelet medicine is the primary treatment for this condition. Antithrombotic medication should be given if BAD is identified at admission to avoid END [10]. Classifying BAD-related stroke as a distinct stroke subtype and investigating whether these patients will experience different outcomes or treatment responses in comparison to other traditional stroke subtypes are suggested for further research. This underutilized idea can further be strengthened by developing and validating the diagnostic criteria for BAD using advanced imaging modality.

DATA AVAILABILITY:

Further information regarding the data used for this work can be obtained from the corresponding author upon reasonable request.

FUNDING:

This work received no external funding.

CONFLICT OF INTEREST:

The authors have no conflicts of interest to declare and is in agreement with the contents of the manuscript.

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FIGURE LEGENDS:

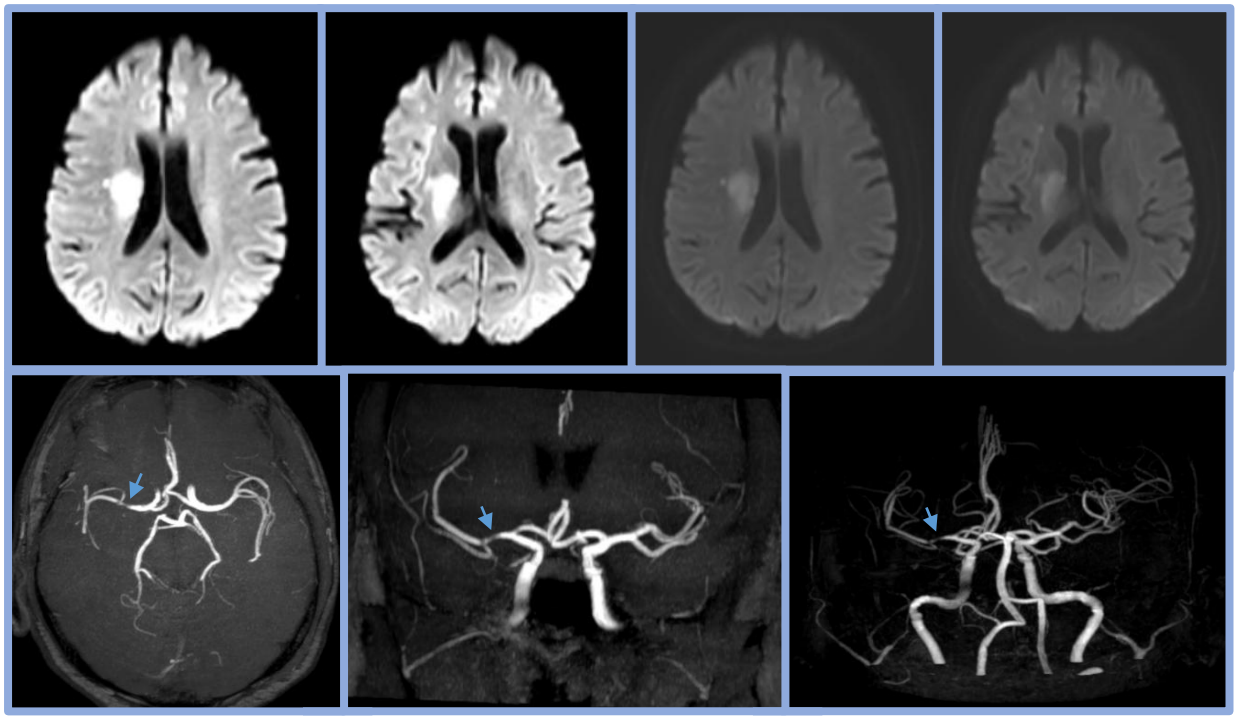

Figure 1: MRI and MR Angiography showed acute right corona radiata and basal ganglia infarction, and right terminal middle cerebral artery (MCA) segment M1 stenosis.

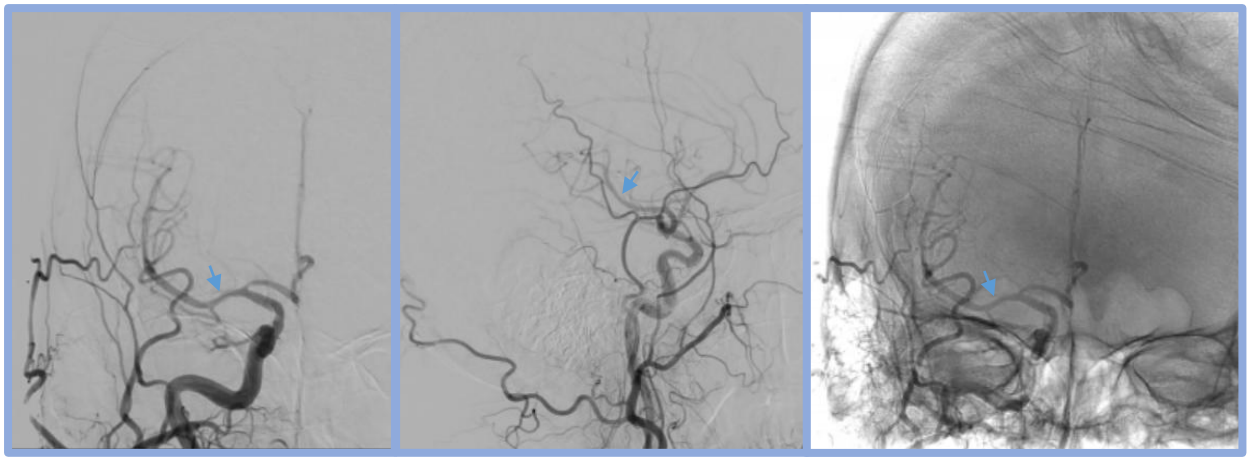

Figure 2: Digital subtraction angiography (DSA) showed mild stenosis at right MCA but was less than 50%.

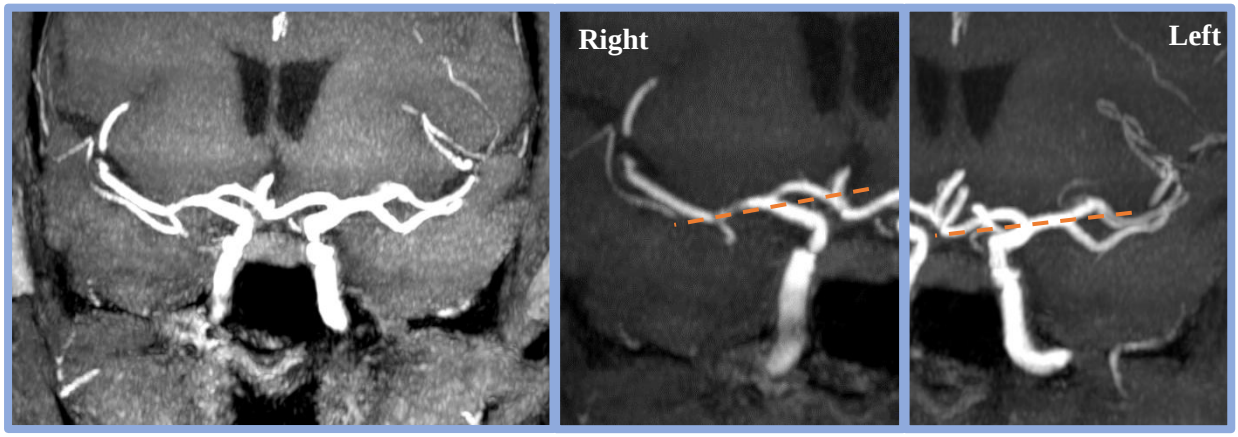

Figure 3: M1 segment shape classification on the coronal maximum intensity projection images of three-dimensional time-of-flight magnetic resonance angiography. A line was drawn from the M1 origin to the M2 bifurcation, and the curves' vertices were evaluated based on the position of the line. Our patient shows the straight type of both MCA (M1 segment).