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Intracerebral Hemorrhage Secondary to Chronic Left Middle Cerebral Artery Occlusion: Diagnostic Challenges and Clinical Implications

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Abstract

Intracerebral Hemorrhage (ICH) is a severe form of stroke with high morbidity and mortality. While Middle Cerebral Artery (MCA) occlusion typically results in ischemic infarction, ICH secondary to chronic MCA occlusion without concomitant ischemia is an exceedingly rare phenomenon that challenges both diagnosis and management. We report a 45-year-old male with multiple cardiovascular risk factors presenting with acute focal neurological deficit, which emphasizes the importance of considering chronic MCA occlusion as a potential etiology of ICH, even in the absence of infarction. Radiological evaluation, especially with magnetic resonance imaging, is essential to guide prevention and management strategies.

Keywords

Intracerebral Hemorrhage, Middle Cerebral Artery Occlusion, Collateral Circulation, Cocaine, Neuroimaging

1. Introduction

Intracerebral Hemorrhage (ICH) represents a devastating form of stroke, characterized by the extravasation of blood into the brain parenchyma. This serious neurological condition is often precipitated by well-established risk factors, such as uncontrolled systemic arterial hypertension and diabetes mellitus, which contribute to cerebral vascular fragility. Although occlusion of the Middle Cerebral Ar-

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tery (MCA) is one of the most common causes of ischemic cerebral infarction, the occurrence of ICH in association with chronic MCA occlusion, without evidence of concomitant cerebral infarction, is a remarkably rare clinical and radiological phenomenon that poses significant challenges for both diagnosis and elucidation [1].

Understanding the pathophysiological mechanisms underlying this atypical presentation is of paramount importance for developing clinical management strategies that are appropriate and suitable for these patients [2]. The rarity of such cases in the medical literature highlights the need for in-depth investigation and documentation of clinical cases that can contribute to collective knowledge. The main objective of this article is to present a detailed case report of a patient who developed ICH secondary to chronic left MCA occlusion and, based on this case, discuss the radiological and etiological implications, with an emphasis on the importance of adaptive collateral circulation and atheromatosis as preponderant risk factors [1] [2].

2. Case Description

This report describes the case of a 45-year-old male patient with multiple cardiovascular risk factors, including chronic cocaine use, a history of Systemic Arterial Hypertension (SAH), type 2 Diabetes Mellitus (DM2), and alcoholism, admitted with sudden right hemiplegia and behavioral changes. The objective is to discuss the clinical and neuroimaging findings, in addition to exploring the diagnostic and etiological challenges related to chronic occlusion of the left Middle Cerebral Artery (MCA) and its implications for the development of a hemorrhagic stroke (HCV).

On admission, his blood pressure was 210/120mmHg, and laboratory tests revealed fasting glucose of 178 mg/dL, normal coagulation parameters, and mildly elevated total cholesterol. Initial computed tomography scan revealed the presence of a hematoma in the left temporoparietal region, with mass effect and a midline deviation of 0.6 cm. During hospitalization, additional tests were performed to elucidate the etiology of the event. Intracranial arterial and venous magnetic resonance angiography demonstrated a significant reduction in blood flow in the M1 segment of the left MCA, associated with a paucity of distal branches, suggesting chronic arterial occlusion. Small, slowed distal branches indicated the presence of collateral circulation. In addition, early bifurcation of the M1 segment of the right internal carotid artery was observed, with parietal irregularities and significant stenosis. Hypoplasia of the posterior communicating arteries was identified and considered an anatomical variation. The study of the vessel wall did not show signs suggestive of vasculitis, but signs of carotid and bilateral middle cerebral atheromatosis were found.

The patient was admitted to the intensive care unit for monitoring and supportive care. No neurosurgical intervention was performed. At discharge, he presented with mild cognitive impairment.

3. Discussion

Magnetic resonance imaging confirmed the presence of a subacute intraparenchymal hematoma in the left nucleocapsular and parietotemporal region, with surrounding vasogenic edema and compression of the left lateral ventricle (see **Figure 1** and **Figure 2**). Importantly, no areas of acute ischemia were detected. Considering the occlusion of the left MCA, an extensive infarction in this vascular territory would be expected if the occlusion were acute. However, the absence of ischemic changes supports the interpretation that the occlusion was chronic, with collateral circulation sustaining perfusion over time. These findings align with previous studies demonstrating that chronic MCA occlusion may predispose to ICH without infarction, due to the fragility of compensatory vessels [1] [2].

Recent evidence from advanced MRI protocols shows that peri-hematomal regions can exhibit dynamic injury and repair responses, despite the absence of frank ischemia (see **Figures 3-5**) [2] [3]. These imaging biomarkers highlight the importance of multimodal MRI not only in detecting ischemia but also in characterizing microvascular adaptations that may influence hemorrhagic risk [3]-[5]. In this case, the imaging findings of collateral vessels and microangiopathy reinforce the concept that vascular remodeling in chronic occlusion may prevent ischemia but predispose to rupture.

Proposed pathophysiological mechanisms for hemorrhage in chronic MCA occlusion include the formation of fragile microaneurysms within the collateral circulation and the impact of chronic hemodynamic stress on these delicate vessels. The long-standing hypoperfusion distal to the occlusion can lead to compensatory angiogenesis, forming new, often structurally weaker, collateral vessels. These newly formed vessels may be more susceptible to rupture under normal or

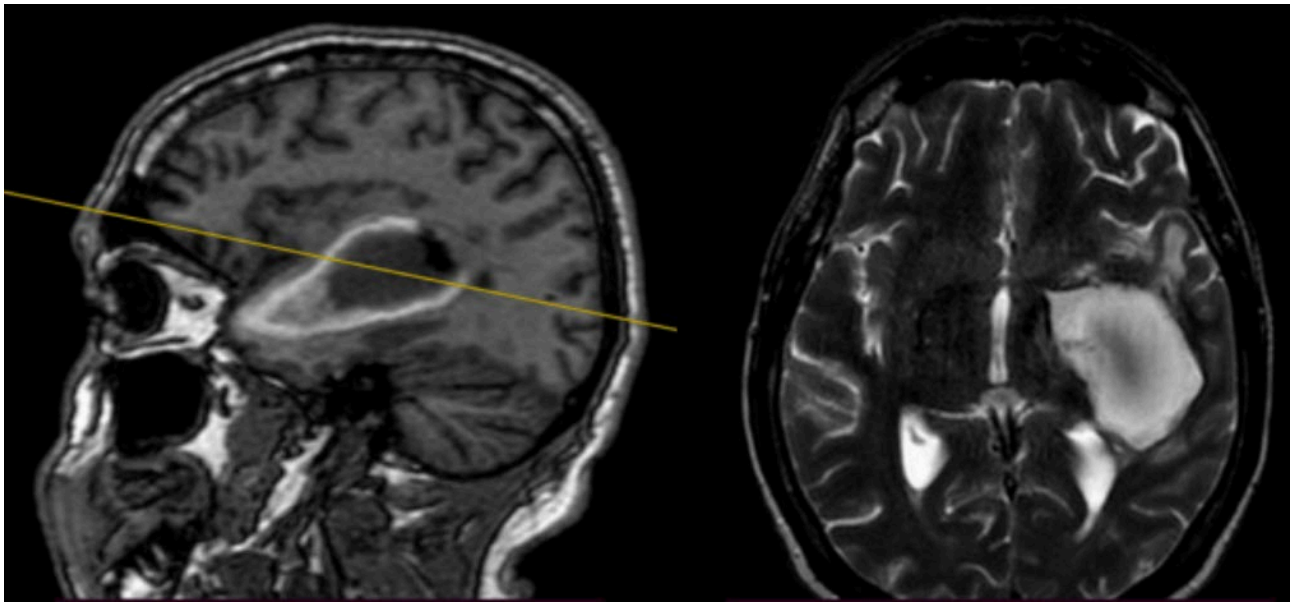

Figure 1. Subacute intraparenchymal hematoma in the image on the left, due to the evident hyperintensity in the sagittal T1-weighted sequence and the axial T2-weighted sequence.

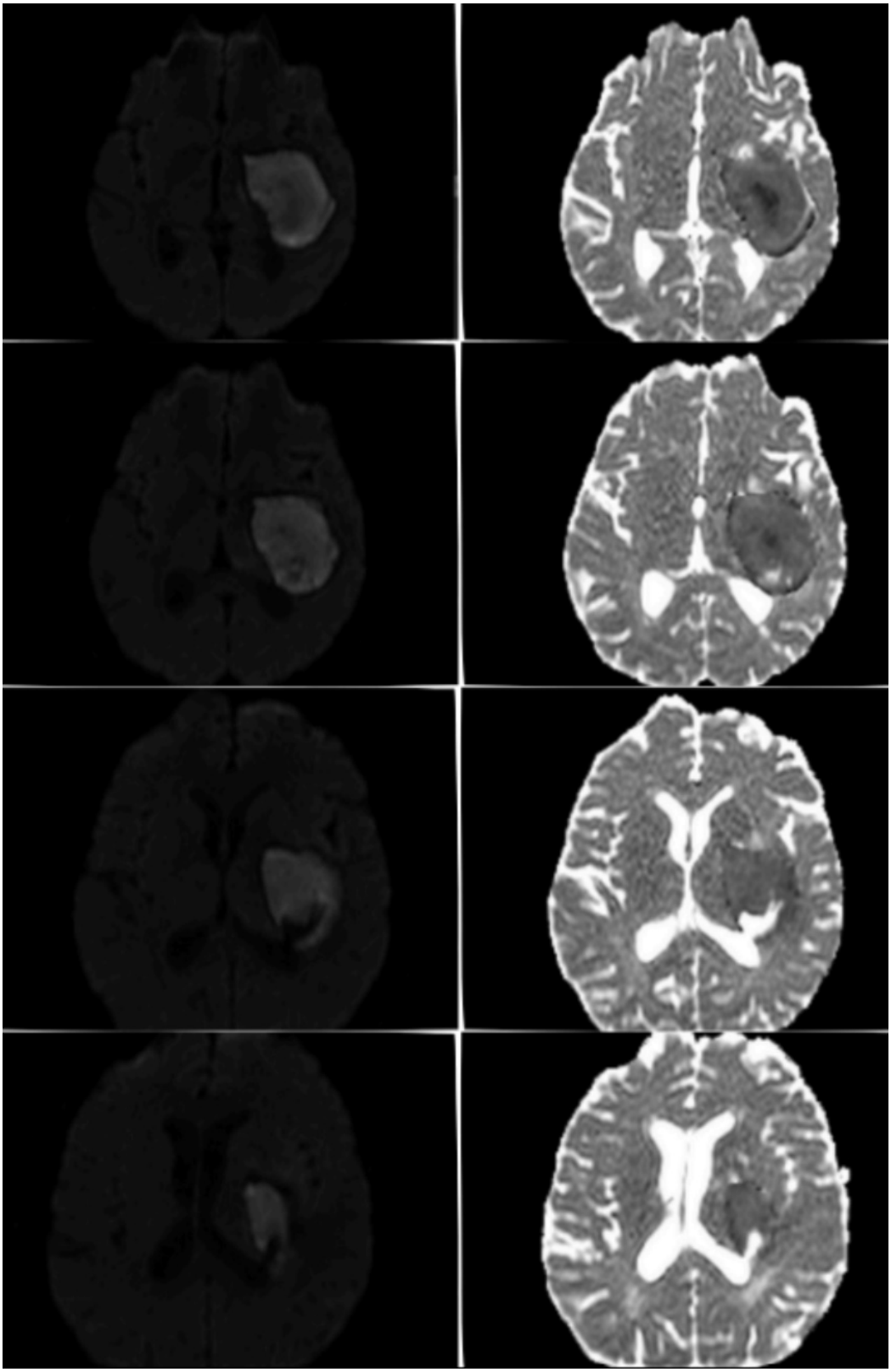

Figure 2. The diffusion/ADC sequence did not reveal any areas of diffusion restriction, ruling out the presence of an acute/subacute ischemic insult associated with the hematoma. This finding strengthens the hypothesis of chronic occlusion of the left MCA.

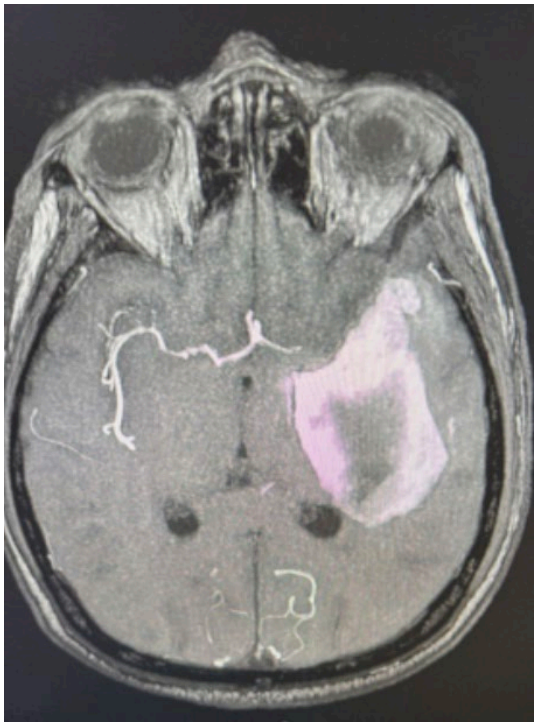

Figure 3. Significant reduction in the flow signal column of the M1 segment of the left middle cerebral artery, as well as paucity of its distal branches, denoting arterial occlusion. Signs of carotid atheromatosis of the intracavernous and supraclinoid segments are revealed.

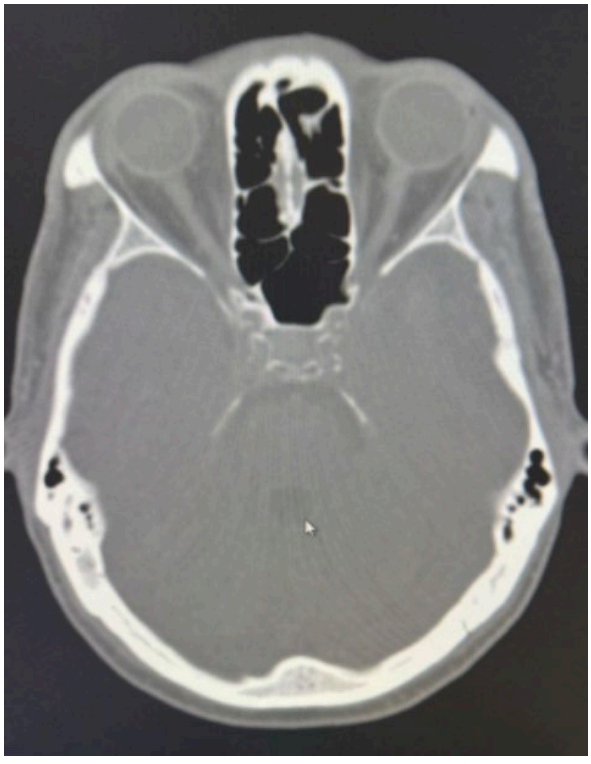

Figure 4. Signs of carotid atheromatosis in the intracavernous and supraclinoid segments are revealed.

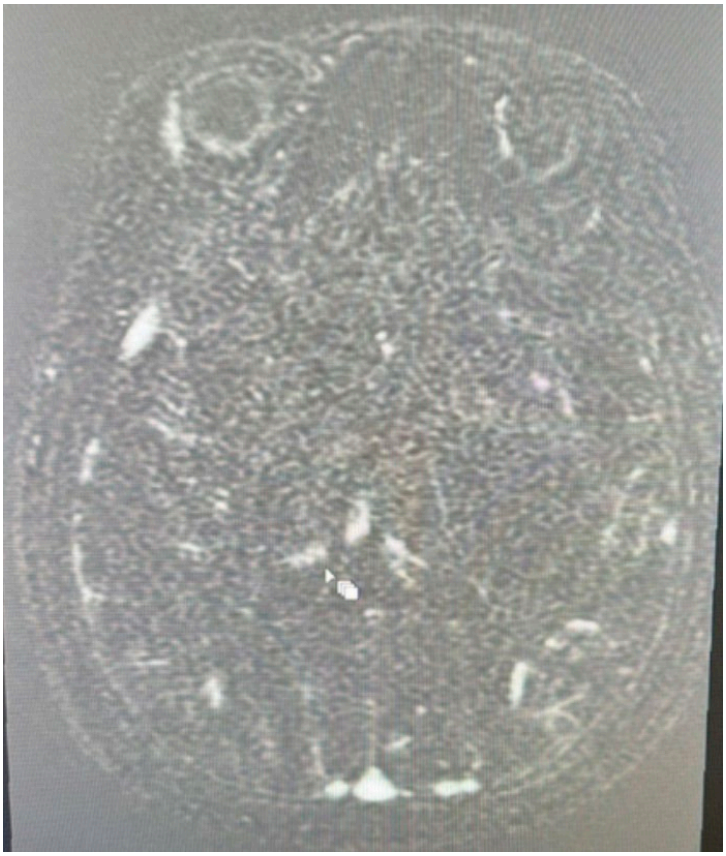

Figure 5. In the contrast phase (subtraction), small distal branches are seen, with slowed flow, which may correspond to collateral circulation.

elevated blood pressure, especially in the presence of underlying vascular risk factors like hypertension and diabetes. Furthermore, chronic hemodynamic stress, characterized by altered shear stress and increased transmural pressure within these collateral pathways, can contribute to endothelial dysfunction and vessel wall remodeling, ultimately increasing the risk of hemorrhage. This interplay between microaneurysm formation and chronic hemodynamic stress provides a plausible explanation for the occurrence of ICH in the absence of acute ischemia in such cases [5].

The patient also reported a history of cocaine use, which is a significant risk factor for hemorrhagic stroke. A recent systematic review and meta-analysis confirmed that cocaine is strongly associated with both ischemic and hemorrhagic stroke, due to its potent vasoconstrictive and hypertensive effects [4]. Case series have further demonstrated that cocaine-related ICH tends to occur in subcortical locations, often with intraventricular extension, and presents with worse clinical outcomes compared to spontaneous hemorrhages [5] [6]. Therefore, cocaine use remains a critical diagnostic consideration in younger patients presenting with unexplained hemorrhage.

In addition, studies evaluating outcomes of medium-vessel occlusion strokes treated with endovascular therapy have demonstrated that hemorrhagic transfor-

mation can occur even in the absence of ischemia, particularly when collateral circulation is insufficient [5] [6]. Vascular instability rather than ischemic burden may be the main determinant of hemorrhagic events in certain contexts of MCA pathology.

4. Conclusion

This case highlights the importance of a detailed investigation into the etiology of hemorrhagic cerebrovascular events, especially in the presence of arterial occlusions without evidence of acute ischemia. The absence of extensive infarction in the left MCA region indicates that the occlusion was chronic and that there was hemodynamic adaptation over time. Furthermore, multimodal MRI is crucial for differentiating ischemia from chronic occlusion, and recognition of associated risk factors—such as atherosclerosis and cocaine use—is essential to guide prevention and management strategies.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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