

# Basilar Artery Stenosis Mimicking the Lacunar Syndrome of Pure Motor Hemiparesis

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**ABSTRACT: Background:** Stereotyped, repeated transient ischemic attacks manifesting as pure motor hemiparesis are most often attributed to ischemia of the internal capsule or ventral pons resulting from *in situ* disease of the small penetrating arteries. **Case description:** We report a 61-year-old man presenting with recurrent episodes of left-sided weakness consistent with the lacunar syndrome of pure motor hemiparesis. Subsequent neuroimaging revealed infarction of the right ventral pons and a critical basilar artery stenosis as the inciting lesion. Despite maximal antithrombotic therapy, he continued to have repeated symptoms. Angioplasty and stenting were attempted but both failed due to plaque recoil and technical difficulties. After the procedure, the patient had no further ischemic episodes and remained symptom-free at two months. **Conclusions:** This case illustrates the imprecise and discordant relationship between the mode of presentation of a stroke syndrome and its presumed pathophysiology. The lacunar syndrome of pure motor hemiparesis should be recognized by clinicians as a mode of stroke presentation due not only to small vessel disease, but also to large artery atherosclerotic disease such as basilar artery stenosis. Prompt institution of treatment can lead to a good clinical outcome.

**RÉSUMÉ: Sténose de l'artère basilaire simulant un syndrome lacunaire d'hémi-parésie motrice pure.**

**Contexte:** Des accès ischémiques transitoires cérébraux répétés et stéréotypés sous forme d'hémi-parésie motrice pure sont le plus souvent attribués à une ischémie de la capsule interne ou du pont antérieur résultant d'une maladie *in situ* des petites artères perforantes. **Description de cas:** Nous rapportons le cas d'un homme de 61 ans qui a consulté pour des épisodes récurrents de faiblesse du côté gauche compatible avec un syndrome lacunaire d'hémi-parésie motrice pure. La neuroimagerie a montré un infarctus de la portion ventrale droite du pont et une sténose critique de l'artère basilaire. Malgré une thérapie antithrombotique maximale, il a continué à présenter des symptômes. On a tenté une angioplastie et la mise en place d'une endoprothèse, mais sans succès à cause de la rétraction d'une plaque athéromateuse et de difficultés techniques. Après l'intervention, le patient n'a pas présenté d'autre épisode ischémique et il est toujours asymptomatique deux mois plus tard. **Conclusions:** Ce cas illustre une relation imprécise et discordante entre le mode de présentation d'un syndrome d'accident vasculaire cérébral et sa physiopathologie présumée. Le syndrome lacunaire d'hémi-parésie motrice pure devrait être reconnu par les cliniciens comme le mode de présentation d'un accident vasculaire cérébral dû non seulement à une maladie des petits vaisseaux, mais également à la maladie athéromateuse de grosses artères telle la sténose de l'artère basilaire. Un traitement rapide peut donner de bons résultats cliniques.

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The clinical diagnosis of stroke is usually made beginning with the history of a sudden onset of focal neurological deficit(s), followed by the neurological examination to localize the lesion. From the mode of presentation and localization, the affected artery and stroke mechanism are inferred. Neuroimaging then assists in confirming the clinical diagnosis and other ancillary tests help to delineate the precise etiology so that secondary prevention strategies can be optimally employed. From time to time, however, clinicians may be misled by a well-recognized stroke presentation linked to a specific pathology and pathophysiologic mechanism, when further investigations

demonstrate incorrect localization or an alternative stroke mechanism. This paper illustrates the imprecise and discordant relationship between the mode of presentation of a stroke syndrome and its presumed pathophysiology, namely, the

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lacunar syndrome of pure motor hemiparesis caused by a critical stenosis of the basilar artery. The recognition of such presentation as a result of basilar artery stenosis is important, as occlusive disease of the basilar artery requires a different approach to treatment. Left untreated, basilar artery stenosis can result in significant mortality and neurological morbidity.

#### CASE REPORT

A 61-year-old, right-handed man presented to a hospital emergency department with an episode of sudden onset left face, arm and leg weakness. There was some mild dysarthria attributed to his facial weakness. He described some transient blurring of vision the day before the episode. The weakness resolved completely within 30 minutes. He was started on aspirin, 325 mg daily, then discharged home.

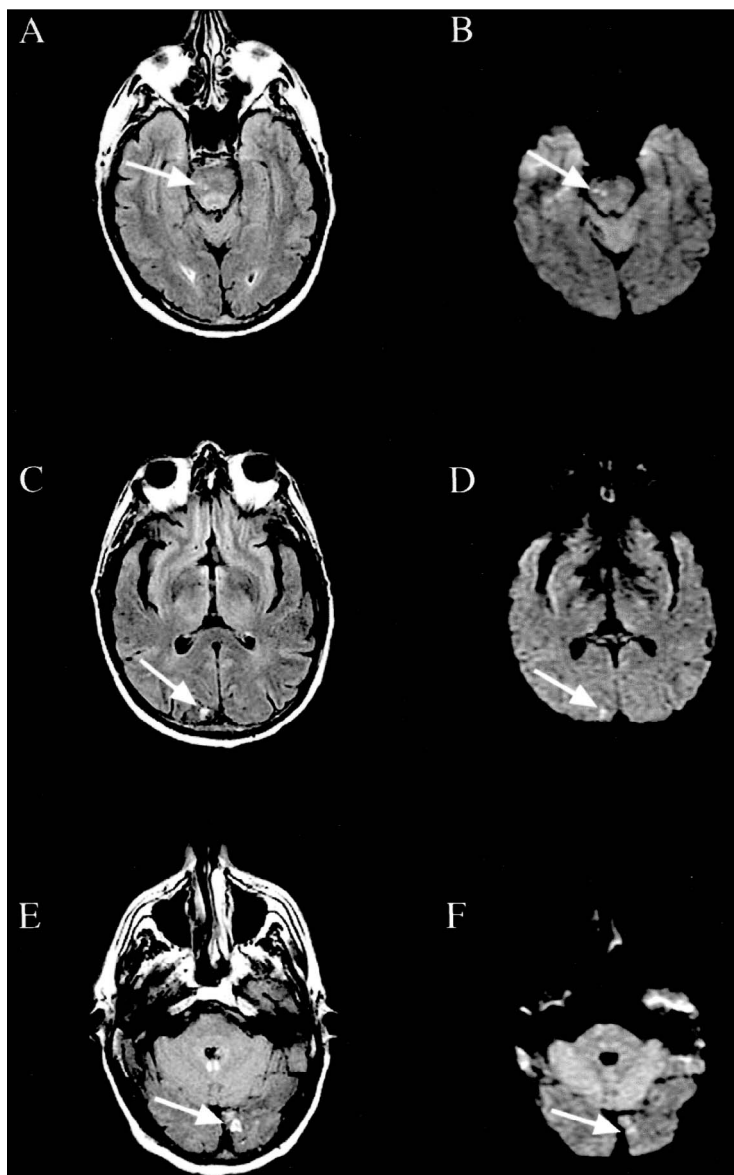
A day later, he had another episode of left hemiparesis. His symptoms resolved in 10 minutes. He was again assessed at the emergency department and discharged home on an increased dose of aspirin, 650 mg daily.

Two days later, he had a third episode. His symptoms transiently improved in the ambulance on the way to our hospital but then progressed to complete left hemiplegia. This again resolved completely in 20 minutes. Aspirin was discontinued, clopidogrel and intravenous heparin were started, and he was admitted to our hospital.

His past history was significant for hypertension, type 2 diabetes, and hypercholesterolemia. There was no history of previous transient ischemic attacks (TIAs). He was on no medications. On admission, his blood pressure was 155/82 mm Hg and his heart rate was 80 beats per minute and regular. His neurological examination was normal. His EKG showed normal sinus rhythm and evidence of an old inferior myocardial infarction.

The provisional diagnosis on admission was crescendo TIAs causing pure motor hemiparesis, suggesting ischemia involving the lenticulostriate or basilar perforators. One day after admission, he had another two-minute episode of left hemiparesis that resolved. Magnetic resonance imaging done the next day showed multiple areas of increased signal involving the right ventral pons and bilateral occipital lobes (Figure 1A, 1C, and 1E). The lesions were hyperintense on diffusion-weighted imaging, consistent with acute infarcts (Figure 1B, 1D, and 1F). Magnetic resonance angiography (MRA) revealed severe mid-basilar stenosis, which was not demonstrated on axial T2-weighted images at the level of the pontine lesion (Figure 2). Posterior communicating arteries were also absent bilaterally.

Despite maximal antithrombotic therapy, he had another episode of bilateral visual loss for three minutes, suggesting ongoing ischemia due to severe basilar artery stenosis. The decision was made to perform a cerebral angiogram with possible angioplasty. It confirmed mid-basilar artery stenosis of greater than 90%, with an ulcerated plaque (Figure 3). There were diffuse irregularities along the entire length of the basilar artery representing atherosclerotic disease. Stenosis was also noted at the origin of the right posterior cerebral artery with some distal flow. At this time, angioplasty was attempted. The balloon catheter was inflated successfully, but control angiography showed persistence of stenosis indicating plaque retraction into the lumen. Stenting was then attempted twice but was aborted due to the inability to negotiate the rigid self-



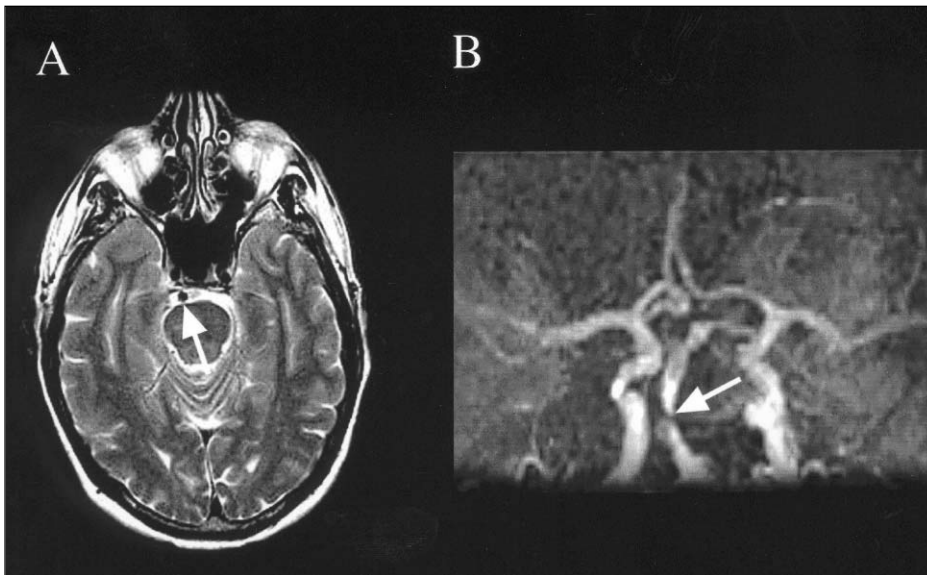
**Figure 1:** Fluid-attenuation inversion-recovery (A, C, E) and diffusion-weighted (B, D, F) images demonstrating acute infarcts: right ventral pontine infarcts (A, B), right occipital infarct (C, D), and left occipital infarct (E, F).

expanding stent through the tortuous extracranial vertebral artery. Following unsuccessful stenting, angioplasty was again attempted but resulted in minimal improvement in lumen calibre at the point of maximum stenosis.

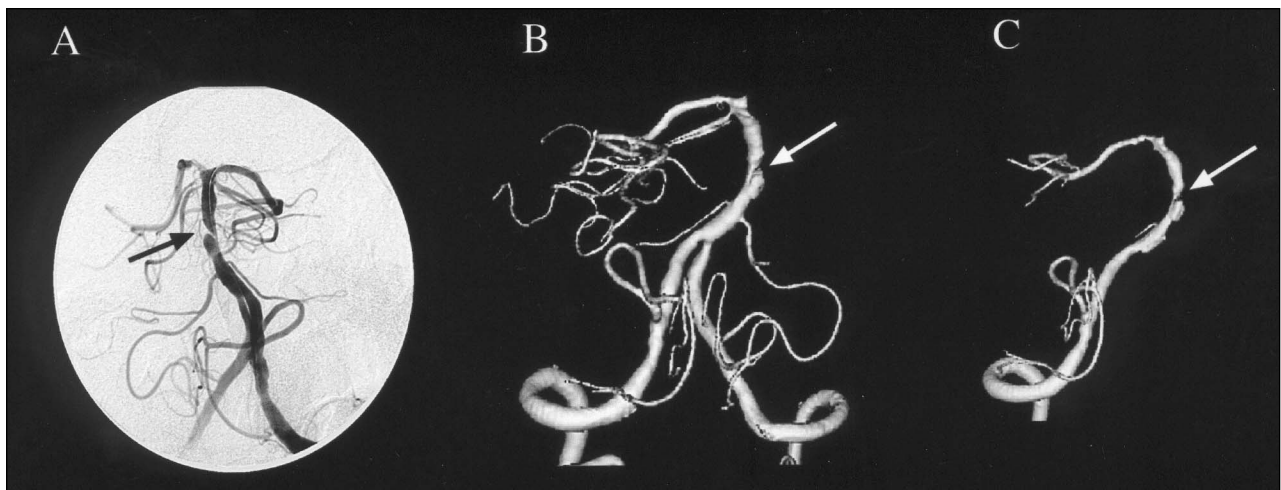
After the procedure, the patient had no further ischemic symptoms. He was discharged home on coumadin, clopidogrel and atorvastatin. Two months after discharge, he remained symptom-free with no further ischemic episodes.

#### DISCUSSION

Patients with symptomatic basilar artery stenosis often present with easily recognizable symptoms and signs.



**Figure 2:** T2-weighted axial image at the level of the pontine infarcts demonstrated normal calibre of the basilar artery (A), while magnetic resonance angiography showed critical mid-basilar stenosis at the same level (B).



**Figure 3:** Conventional angiography demonstrating the mid-basilar stenosis (A), with three-dimensional reconstruction of the same lesion in anteroposterior (B) and lateral (C) views.

Atherosclerosis is the most important pathology involving the basilar artery.<sup>1</sup> In certain cases the initial presentation can be unilateral hemiparesis, often accompanied by subtle bilateral signs, before evolving into the full-blown syndrome. Fisher<sup>2</sup> coined the term “herald hemiparesis” for this. Patients often have prior TIAs more typical of posterior circulation ischemia. In contrast, our patient had repeated stereotyped episodes of transient left-sided weakness, resembling the lacunar syndrome of pure motor hemiparesis. This mode of stroke presentation has also been termed the capsular warning syndrome.<sup>3</sup> However, this is a misnomer since the responsible lesion is not invariably located in the internal capsule. The crescendo nature of multiple stereotyped episodes is thought to be the characteristic presentation of lacunar infarction, reflecting a pathophysio-

logically distinct mechanism related to *in situ* small penetrating vessel disease that involves either the lenticulostriate perforators<sup>3</sup> or the paramedian pontine perforators.<sup>4</sup> The lacunar hypothesis states that lacunar infarcts present as specific lacunar syndromes, with the underlying pathology being penetrating artery disease.<sup>5</sup> The validity of this clinicopathophysiological correlation has been evaluated by various studies. Lacunar syndromes are highly predictive of lacunar infarcts, with the positive predictive value ranging from 84% to 90%.<sup>6-8</sup> On the other hand, the lacunar syndrome of pure motor hemiparesis diagnosed within 12 hours of symptom-onset has been found to be less predictive of lacunar infarcts, with a positive predictive value of only 58%.<sup>9</sup> It has been suggested that lacunar infarcts could be the result of other pathophysiological mechanisms, such

as microembolism<sup>10</sup> or occlusion of penetrating arteries by atherosclerosis in the parent artery.<sup>11</sup> Lacunar infarcts have been shown to predict small penetrating artery disease as the mechanism of infarction only 75% of the time, with a significant alternative mechanism being large artery atherosclerosis.<sup>8</sup> Our case again illustrates that the relationship between stroke symptomatology and pathophysiologic mechanism is less than precise, as critical basilar stenosis due to large artery atherosclerotic disease can mimic the presentation of the lacunar syndrome of pure motor hemiparesis.

The mechanism of cerebral infarction in our patient could be multiple in nature. The occipital infarcts were most likely artery-to-artery embolism from the basilar artery atherosclerotic plaque. The ventral pontine infarcts could either be embolic, related to the atherosclerotic plaque encroaching on the origin of the perforating vessel, or hemodynamic, related to the critical stenosis of the more proximal basilar artery. The latter mechanism is somewhat analogous to that inferred by Fisher<sup>12</sup> in incipient internal carotid occlusion producing repetitive neurological events due to hemodynamic insufficiency.

Magnetic resonance imaging and MRA have led to enormous advances in both the diagnosis of stroke and the understanding of the underlying vascular pathology. An interesting observation in our patient is that the basilar artery stenosis was readily recognized on MRA, while axial magnetic resonance images corresponding to the same level did not identify any changes in the diameter of the artery to indicate stenosis. Axial images are insufficient to exclude a focal arterial stenosis, as these sections are 5 mm in thickness with 2 mm spacing in between. This is in contrast to the MRA images, which are produced by reconstructing thin contiguous axial images that are 0.9 mm in thickness. This observation illustrates the importance of employing MRA in assessing vascular pathology in the context of stroke, without relying solely on the axial images.

Percutaneous transluminal angioplasty is an innovative technique in the treatment of atherosclerotic disease when medical therapy fails. Good clinical outcomes with low risk of procedure and complication rates have been reported, especially in selected patients.<sup>13</sup> Endovascular stenting has been used to improve luminal diameter in atherosclerotic basilar stenosis after angioplasty has failed due to elastic recoil.<sup>14</sup> Unfortunately, this was attempted but was unsuccessful in our patient for technical reasons. New flexible types of stents designed for intracranial use are now available and could be employed in future cases.

Patients with symptomatic basilar artery stenosis have a high annual risk of stroke, despite the use of antithrombotic therapy.<sup>15</sup> Our case illustrates that basilar artery stenosis can present as the lacunar syndrome of pure motor hemiparesis, thus demonstrating that this mode of presentation is not specific to stroke due to small vessel pathology in the lenticulostriate or paramedian pontine perforators. Recognition of this is important, as prompt medical therapy is essential and new interventional strategies are available to help reduce the associated mortality and morbidity of basilar artery occlusive disease.

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