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Case Report

# Patient with basilar artery occlusion related to metastatic melanoma ☆

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ABSTRACT

Posterior circulation stroke accounts for approximately 20% of all ischemic strokes. The basilar artery, which is the main vessel of the posterior circulation, supplies most of the brainstem, occipital lobes and part of the cerebellum and thalami. We present a case of a 73-year-old man with known metastatic melanoma while undergoing immunotherapy presented to the emergency department with a history of progressive shortness of breath, generalize weakness, and dysphagia. The patient's imaging workup revealed brain metastasis. While hospitalized had a sudden onset of loss of consciousness which lasted a few minutes and was back to baseline. An hour later he had another episode of loss of consciousness with absence of brainstem signs. Urgent head computerized tomography showed basilar artery occlusion. Patient was transferred to the intensive care unit and started on intravenous heparin (DVT/PE dosing protocol) and supportive care. At present there is lack of high-quality evidence from randomized controlled trial to guide as how best manage patients with basilar artery occlusion.



## Introduction

Stroke is the leading cause of disability in developed countries [1]. Posterior circulation stroke accounts for approximately 20% of all ischemic stroke cases [2]. The basilar artery is formed by the confluence of the vertebral arteries at the ponto-medullary junction (Fig. 1), which is the main vessel of the posterior circulation, supplying most of the brainstem, occipital lobes, and part of the cerebellum and thalami. Basilar artery thrombosis and occlusion cause approximately 3% of ischemic strokes [3]. Despite recent advances in the treatment of acute anterior circulation stroke, the rate of death or disability associated with BAO is approximately 80% due to lack of evidence based on randomized controlled trials [3].

## Case presentation

A 73-year-old-man presents to the emergency department with shortness of breath, generalized weakness and dysphagia while undergoing treatment for metastatic melanoma with ipilimumab /nivolumab immunotherapy. Neurology was

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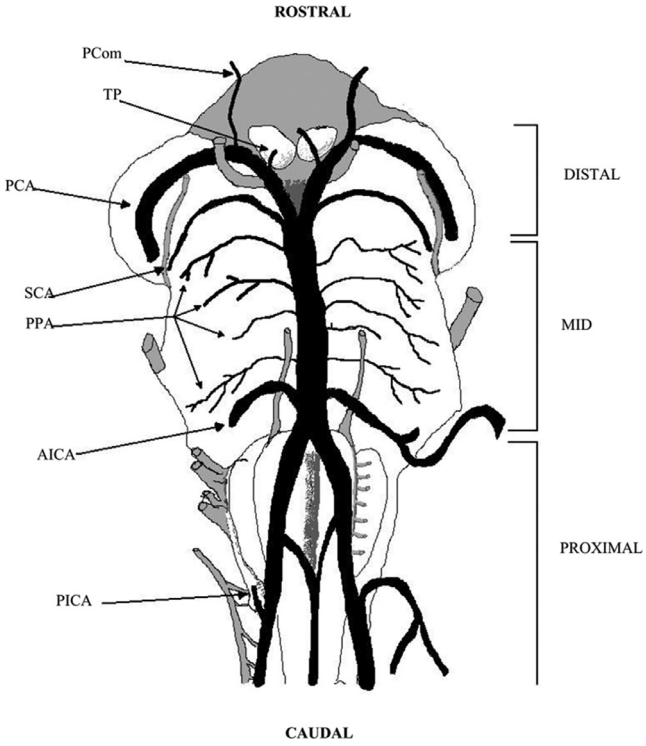


**Fig 1 – Basilar Artery classified according to the 3 anatomical segments of the artery as described by Archer and Horenstein [4]. 1. Proximal (caudal): from the confluence of the vertebral arteries to the origin of the AICA. 2. Mid basilar: from the origin of the AICA to the origin of the SCA. 3. Distal (rostral): from the origin of the SCA to the bifurcation into the PCAs. Anatomical representation of the segments of the basilar artery (PICA, posterior inferior cerebellar artery; AICA, anterior inferior cerebellar artery; SCA, superior cetebellar artery; PPA, pontine perforating arteries; PCA, posterior cerebral artery; TP, thalamoperforating arteries, PCom, posterior communicating artery.**

consulted to evaluate the patients for contrast-enhancing brain lesions on head MRI (Fig. 2). While hospitalized, he had a sudden episode of decreased level of consciousness (LOC) for a few minutes and then fully recovered. He was able to communicate and self-feed at breakfast. Two hours later, he experienced a similar episode of decreased LOC, with sinus tachycardia and normal blood pressure. However, this time his pupils were dilated and non-reactive to light in the absence of extra-ocular movements. He could move all 4 limbs in response to nociceptive stimuli. An urgent computerized tomography (CT) of the brain without contrast showed a hyperdense basilar and proximal right posterior cerebral artery, highly suggestive of acute thrombosis (Fig. 3). This was confirmed using by the CT angiography. Diffusion weighted magnetic resonance imaging (MRI) of the brain showed extensive diffusion restriction affecting the splenium of the corpus callosum, thalamus bilaterally, midbrain, dorsal pons, cerebellum, and occipital and temporal lobes (Figs. 4A and B). The patient was transferred to the ICU and received supportive care

**Fig. 2 – T1-weighted magnetic resonance imaging with contrast (Gadolinium) enhancing lesions in axial and sagittal views (white arrows).**

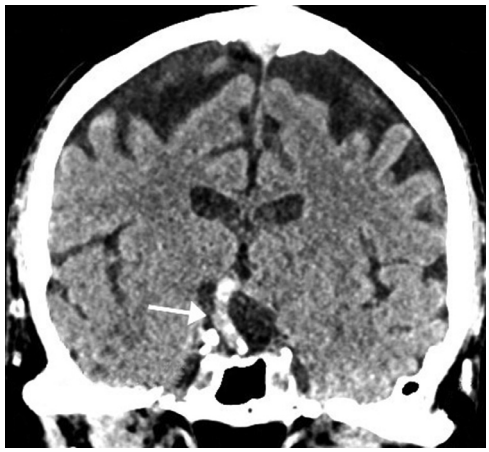

**Fig. 3 – Computerized tomography shows basilar artery occlusion in coronal and sagittal views (white arrow).**

and empirical treatment with intravenous heparin infusion (25,000 UTS/0.45% NS con:50 units/mL [DVT/PE dosing protocol] monitored with daily PTT level) plus his daily aspirin. The patient received hospice care per family's wishes and expired 1 week later.

## Discussion

Basilar artery occlusion (BAO) is classified according to three anatomical segments of the artery, as described by Archer and Horenstein (Fig. 1) [4]. The length of occlusion is classified as "short," if only 1 segment of the basilar artery is occluded, or "long," if 2 or more segments are occluded. The proximal basi-

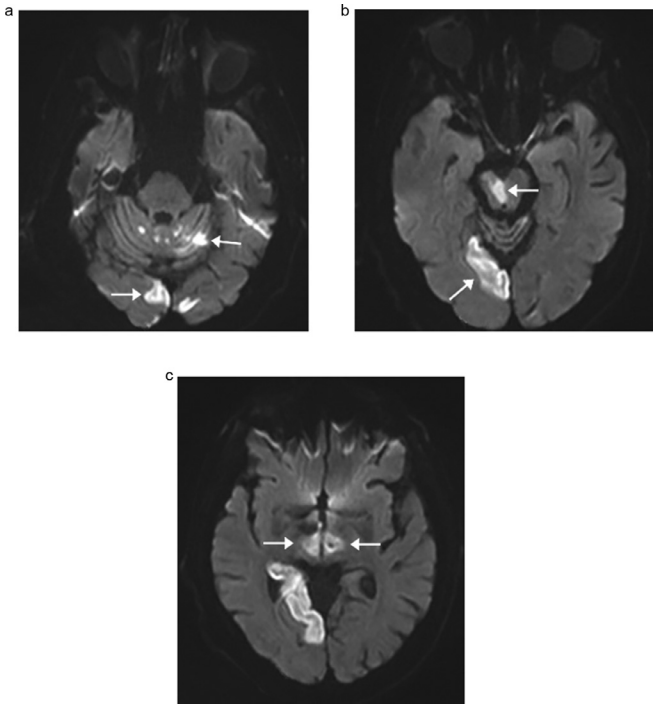


**Fig. 4 – Diffusion weighted images in axial view showing acute infarctions involving splenium of the corpus callosum, thalami bilaterally, midbrain, dorsal pons, cerebellum, and in the occipital and temporal lobes (white arrows).**

lar region is the most common location of atherothrombosis occlusion at the confluence of the vertebral arteries and mid-basilar region.

Stroke in patients with cancer is multifactorial and may result from underlying hypercoagulable state with paradoxical embolism, direct invasion of cancer into vasculature, infection, or endocarditis due to treatment-induced immunosuppression, or treatment-induced radiation vasculopathy. The pathogenesis of BAO is usually secondary to local atherothrombosis or cardioembolism. However, in this case we suspect it was due to the *hypercoagulable* state related to his metastatic melanoma. The *stuttering* symptom presentation of a rapid onset of reduced LOC, hemiparesis/plegia to quadriparesis/plegia, and progressive brainstem dysfunction is due to blockage of the penetrating brainstem arteries which are the end arteries, triggering a crescendo of multiple infarcts is unique to BAO, unlike typical anterior circulation strokes.

The use of noninvasive imaging techniques, such as computed tomography angiography (CTA) with a sensitivity and specificity of > 90% for demonstrating occlusion and magnetic resonance imaging (MRI/MRA), has improved the recognition of clinical syndromes associated with BAO. Intra-arterial digital subtraction angiography (DSA), traditionally used to confirm clinically suspected BAO, is still regarded as the gold standard. However, DSA is invasive, time-consuming, and has limited availability in specialist centers. General anesthesia may be required if the patient cannot cooperate with the procedure.

Care of patients with BAO should be undertaken in a stroke unit as vital brainstem structures are involved. BAO kills 80%-90% of patients in the absence of recanalization [5]. Symptomatic intracranial hemorrhage (sICH) which is more common in patients with BAO with an Alberta Stroke Program Early CT Score (which quantifies infarction volume) <8, most patients with sICH usually die. There is uncertainty as to whether no intervention, antiplatelet therapy [6], anticoagulation [6], intravenous or intra-arterial thrombolysis [7], transluminal angioplasty and stenting [8], or a combination of these [9] is the optimal treatment. Data from the International Stroke Trial support the use of antiplatelet therapy in all types of acute ischemic stroke, including posterior-circulation stroke [6]. Intravenous thrombolysis was not performed in this patient as he was on daily aspirin and had recent brain metastasis (Fig. 1) increasing the risk of sICH. There is a continued lack of proven treatment modalities based on randomized controlled trials for patients with acute BAO, and current clinical practice varies widely. Despite the lack of data demonstrating clinical benefits, many clinicians continue to advocate anticoagulants (ie, unfractioned heparin, low molecular-weight heparin) to further inhibit thrombogenesis and avoid clinical deterioration [10]. The recent Basilar Artery International Cooperation Study (BASICS) trial reported the outcomes of 27 BAO patients who did not receive any treatment. Of these 27 patients, 26 (96.3%) died within 1 month and 1 (3.7%) patient was institutionalized as bedridden and incontinent [9].

## Patient consent

Written informed consent was obtained from the patient for the publication of this case report.

## Author contributions

Dr. Meheroz H. Rabadi - Study concept and design, Data acquisition, Analysis and interpretation, Write-up of the manuscript for intellectual content, Study supervision

## Artificial intelligence

No AI and AI-assisted technologies were used in the preparation of this manuscript.

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