Total basilar artery thrombosis presenting with recurrent transient ischemic attacks (TIAs): a case report

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ABSTRACT

Background: Managing acute ischemic stroke in patients with significant comorbidities, such as hypertension, heart failure, and substance abuse, presents unique challenges. Timely intervention is critical, but the risk of complications and poor prognosis is high.

Case Presentation: A 46-year-old male with a history of smoking, drug abuse, poorly controlled hypertension, and dilated cardiomyopathy with an ejection fraction of 25%-30% presented with sudden left-sided weakness, facial droop, and dysarthria. The initial National Institutes of Health Stroke Scal score was 6. Computed tomography (CT) brain showed no acute infarctions but revealed chronic lacunar infarctions and atrophy. CT angiography identified significant vascular abnormalities, including right internal carotid artery attenuation and non-visualization of both intracranial vertebral and basilar arteries. Intravenous thrombolysis (tPA) led to initial improvement, but recurrent strokes or transient ischemic attacks (TIAs) followed, leading to intubation and mechanical ventilation. Magnetic resonance imaging revealed evolving infarctions in the pons and cerebellum. A basilar artery thrombectomy was successfully performed, but postoperative complications included spontaneous pneumothorax and femoral artery pseudoaneurysm, requiring further interventions. Due to extensive posterior circulation infarction, the patient had a poor neurological prognosis and was placed on do not resuscitate status.

Conclusion: This case highlights the complexities of treating acute ischemic stroke in patients with substantial comorbidities. While interventions such as thrombolysis and thrombectomy are critical, they carry risks. A multidisciplinary approach is necessary to balance immediate treatment with considerations of long-term prognosis and quality of life.

Keywords: Acute ischemic stroke, dilated cardiomyopathy, thrombolysis, basilar artery thrombectomy, transient ischemic attacks, posterior circulation infarction, comorbidities, cerebrovascular disease, posterior circulation infarction, neurological complications, hypertension, drug abuse.

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Background

Basilar artery thrombosis (BAT) is a rare, life-threatening condition resulting from the occlusion of the basilar artery, which supplies blood to the brainstem, cerebellum, and occipital lobes. The basilar artery is a critical component of the posterior circulation of the brain, formed by the confluence of the two vertebral arteries. Thrombosis in this artery can lead to significant neurological deficits and high mortality if not promptly diagnosed and treated [1]. The pathophysiology of BAT involves the formation of a thrombus within the basilar artery, often due to underlying atherosclerosis, embolic events, or arterial dissection. Risk factors include hypertension, diabetes mellitus, hyperlipidemia, smoking, and conditions that predispose to hypercoagulability such as atrial fibrillation and certain genetic disorders [2]. Clinically, BAT can present with a wide range of symptoms due to its impact on the brainstem and cerebellum. Common presentations include vertigo, ataxia, dysarthria, dysphagia, and altered levels of consciousness. Severe cases can progress to "locked-in syndrome," characterized by quadriplegia and anarthria with preserved consciousness and vertical eye movements, or can lead to sudden death if the respiratory centers are affected [3]. Diagnosis is typically confirmed through imaging studies. Magnetic resonance imaging (MRI) and magnetic resonance angiography (MRA) are preferred for their detailed visualization of the posterior circulation. Computed tomography (CT) angiography is also useful, particularly in emergency settings. Early and accurate diagnosis is crucial as the prognosis significantly improves with timely intervention [4,5]. Treatment strategies for BAT include anticoagulation, thrombolysis, and endovascular procedures. Intravenous thrombolysis with tissue plasminogen activator (tPA) is an option

if the patient presents within the therapeutic window. Endovascular thrombectomy has become a mainstay of treatment, offering direct removal of the thrombus and restoration of blood flow [6,7]. Despite advancements in diagnosis and treatment, BAT remains associated with high morbidity and mortality. Early recognition and intervention are vital to improving outcomes, emphasizing the importance of understanding the risk factors, clinical presentation, and available therapeutic options [8,9].

This case highlights the variable clinical presentation of a patient with total basilar artery occlusion. We address how such patients can present and what the appropriate management is in such cases.

Case Presentation

A 46-year-old Saudi male, smoker, and drug abuser. He is known to have poorly controlled hypertension, heart failure with the latest transthoracic echocardiography showing Ejection Fraction (EF) of 25%-30%, and dilated cardiomyopathy (DCM). Presented to the emergency room (ER) with 1 hour history of sudden onset of slurred speech, left-sided upper motor neuron type facial weakness, and left-side weakness affecting the upper and lower extremities.

Upon examination, he was having dysarthria and left-sided weakness with a power grading of 3/5 over upper and lower limbs with National Institutes of Health Stroke Scale (NIHSS) score of 6. Initial CT brain (Figure 1) showed no evidence of acute territorial infarctions with a background of age-related brain atrophic changes, and small cerebrospinal fluid-like hypodensity at the right caudate/anterior limb internal capsule nucleus and left frontal area likely represent old lacunar infarctions. CT angiography of the brain and neck (Figure 2) showed attenuation at the origin of the right internal carotid artery (ICA), petrous, and cavernous segment of the right ICA without definite thrombosis and no visualization of both intracranial vertebral artery and basilar arteries. The patient was within the window for intravenous tissue plasminogen activator (IV tPA). His blood pressure was on the higher side, more than 200 mmHg systolic, therefore it was lowered using IV labetalol to less than 180 mmHg and then the IV tPA was successfully given. After starting IV tPA, the patient showed gradual improvement in his neurological exam, with no post IV tPA complications. He was directly admitted to intensive care unit (ICU) for observation and blood pressure management. Upon reassessment, the patient was neurologically improving. He had residual mild dysarthria, mild pronator drift in the left side, power improved to 4/5 on the left side, and NIHSS dropped to 2 (mild left smile asymmetry and mild slurring of speech). Follow up CT scan, showed no evidence of acute territorial infarction or hemorrhage. Initial MRI/MRA (Figure 3) showed T2/FLAIR hyperintense foci at the right upper hemipons as well as the left cerebellar hemisphere exhibiting restricted diffusion on diffusion-weighted imaging/apparent diffusion coefficient images in keeping with acute infarcts, an attenuated intracranial segment of both vertebral arteries. No luminal illumination of the basilar artery (likely near total thrombosis). Marked attenuation of bilateral posterior cerebral arteries.

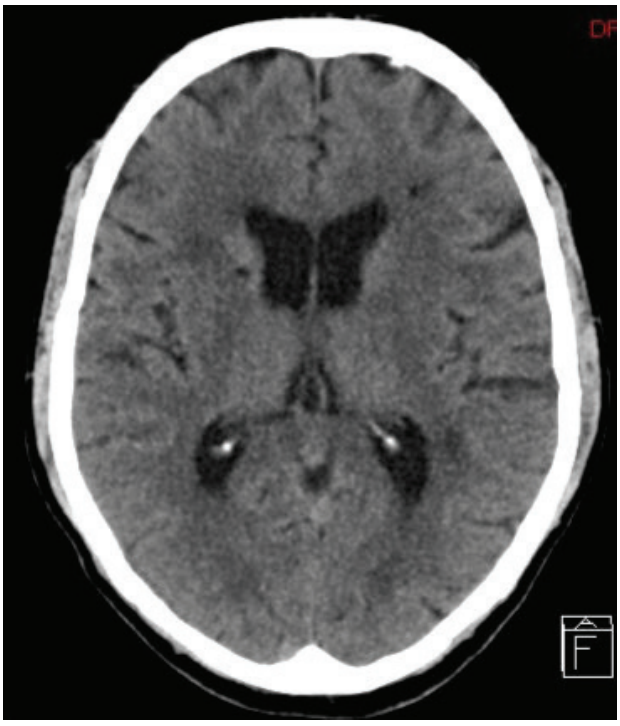

Figure 1. *No evidence of acute territorial infarctions with background of age-related brain atrophic changes, and small CSF-like hypodensity at right caudate/anterior limb internal capsule nucleus and left frontal area likely represent old lacunar infarctions.*

After clinical stabilization in ICU, the patient was shifted to the regular ward with the previously reported mild residual dysarthria and left-side body weakness. He had suddenly developed new onset of right-sided weakness and severe slurred speech, associated with dizziness. Accordingly, code stroke was activated.

Repeated NIHSS was 7, systolic blood pressure (SBP) was 180 mmHg, power was 2/5 on the right side and 4/5 on the left, and CT brain (Figure 4) showed no signs of acute territorial infarction. After finishing the CT, the patient was back to his baseline with total improvement of the new neurological deficits. Presuming a diagnosis of transient ischemic attack (TIA) in the background of total basilar occlusion. The patient was started on heparin infusion as per protocol, and he was shifted to ICU for observation. New cardiac work ups were arranged, including holter, and transesophageal echo, which were unremarkable. In the ICU, the patient was sleepy and difficult to arouse, snoring, unarousable with painful stimuli for a short duration lasted for 3-5 minutes then he came back to his baseline. Presuming another new TIA, a few minutes later he developed another brief and reversable episode of

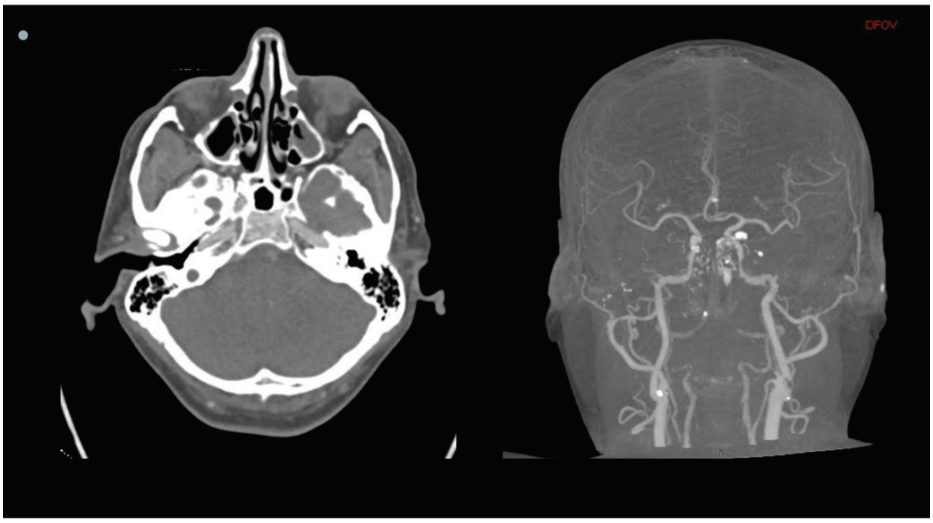

Figure 2. *Showing attenuation at the origin of the right internal carotid artery, petrous and cavernous segment of the right ICA without definite thrombosis and no visualization of both intracranial vertebral artery and basilar arteries.*

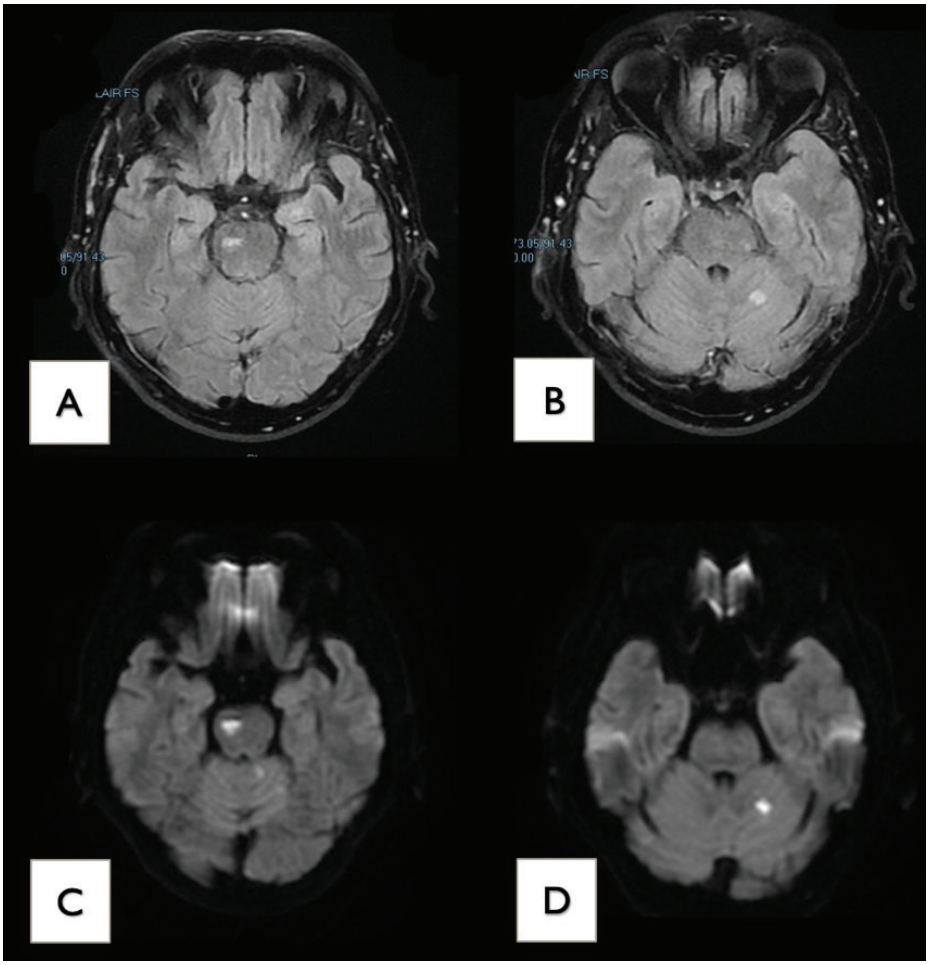

Figure 3. *MRI Brain T2/FLAIR hyperintense foci at right upper hemipons (A), as well as left cerebellar hemisphere (B), exhibiting restricted diffusion on DWI/ADC images seen in both right hemi-pontine area (C), and left cerebellar (D), in keeping with acute infarcts.*

sleep and unarousable. Code stroke was activated for the third time, it was planned to do urgent brain CT and MRI. However, before shifting the patient to imaging, he was drowsy, not opening eyes nor following command, snoring, glasgow coma scale (GCS) was 3/15, so was intubated and connected to mechanical ventilation (MV). After

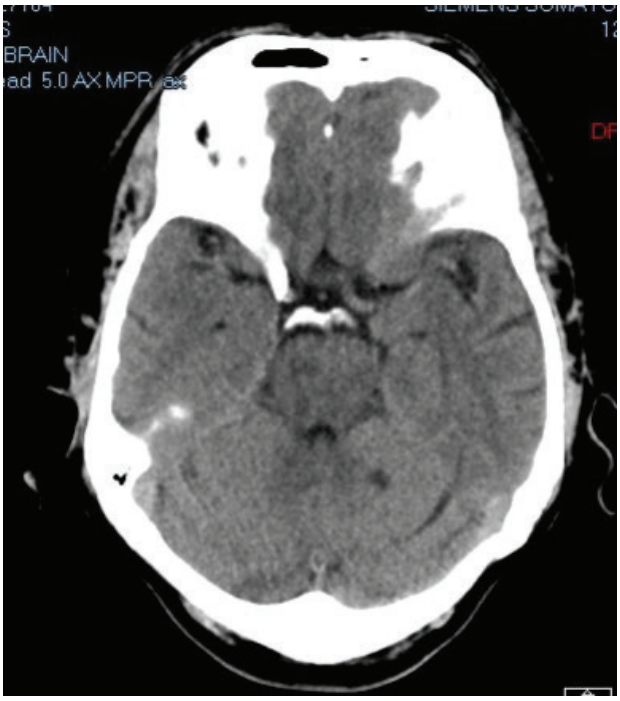

Figure 4. *Brain CT showing a newly seen hypodensity at the right hemi-pons and left cerebellar hemisphere (seen on the recent MRI) in keeping with acute infarcts.*

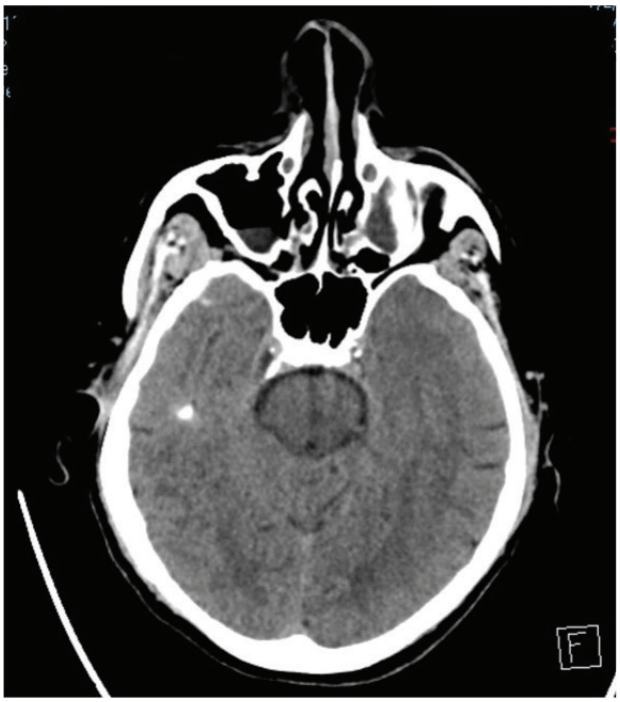

Figure 5. *Brain CT showing a new hypodensity at the mid-pons that represents acute infarct versus artifact.*

Intubation, he was initially hypotensive, SBP <100 mmHg (BP 77/43 mmHg), so boluses of phenylephrine (200 mcg were given) and levophed infusion was prepared to maintain SBP 160-180 mmHg. The patient was assessed post intubation, where dilated bilateral pupils were seen. CT Brain was repeated (Figure 5) and it showed new hypodensity at the mid-pons that could represents acute infarct versus artifact. A new brain MRI was obtained, and it showed an interval new appearance with an increase in the number of focal areas exhibiting restricted diffusion at pons as well as increased conspicuity of those seen at the left cerebellar hemisphere previously in keeping with acute infarcts (Figure 6). The patient was shifted for basilar artery thrombectomy the basilar artery was partially opened with no post operational complications. His examination post thrombectomy, GCS 5/15 with ongoing sedation infusion of fentanyl and propofol, with pinpoint pupils (unequally, smaller in the left), with absent oculocephalic and gag reflexes. In addition to bilaterally upgoing planters. During his stay in the ICU, the patient developed right-side spontaneous pneumothorax, hence intercostal chest tube (ICT) was inserted. Postoperative chest x-ray showed full expansion of the right lung. He also developed a right femoral artery pseudo aneurysm (at the catheter site) and aneurysm embolization was successfully performed. Eventually, the patient was tracheostomized and do not resuscitate (DNR) was activated given the patient poor functional status and decreases level of consciousness. Follow up Brain CT was done (Figure 7) and showed the development of bilateral symmetrical hypodensities involving both thalami, parasagittal parietooccipital, medial temporal lobes, splenium of corpus callosum, brainstem, and cerebellum causing mild mass effect upon the fourth and lateral ventricles (posterior and temporal horns) as well as effacement of adjacent sulci and basal cistern, these finding in keeping with acute to subacute infarction of posterior circulation.

Discussion

The case presents a complex clinical scenario involving a 46-year-old male with a significant history of smoking, drug abuse, hypertension, and poorly controlled heart failure, manifesting with a reduced ejection fraction indicative of DCM. The patient's acute presentation of left-sided weakness, facial droop, and dysarthria with an NIHSS score of 6 suggests an acute cerebrovascular event, prompting immediate intervention.

Initial imaging did not show acute territorial infarctions but revealed age-related brain atrophic changes and old lacunar infarctions. The CT angiography indicated significant vascular pathology, including attenuation of the right ICA and non-visualization of both intracranial vertebral arteries and basilar artery, highlighting the severity of the underlying vascular disease.

Timely administration of intravenous thrombolysis (tPA) led to an initial improvement in neurological symptoms, a favorable response that underscores the importance of prompt reperfusion therapy in acute ischemic stroke. However, the recurrence of symptoms and subsequent imaging suggested evolving infarctions, particularly

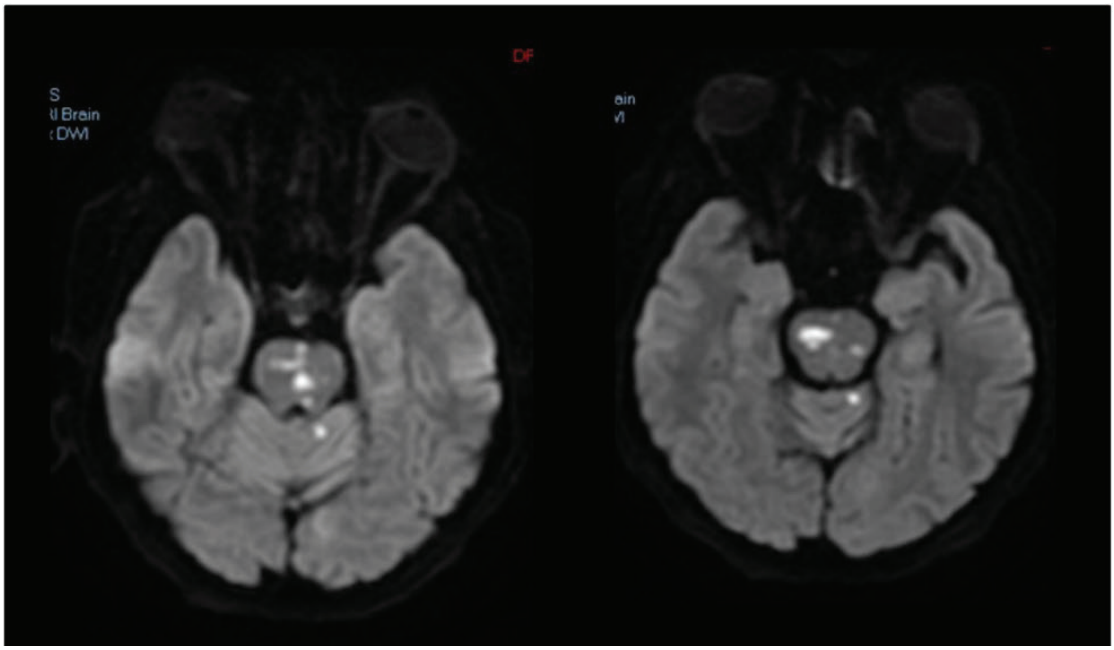

Figure 6. *MRI Brain DWI view, showing a new appearance with increase in the number of focal areas exhibiting restricted diffusion at pons as well as increase conspicuosity of those seen at left cerebellar hemisphere previously in keeping with acute infarcts for clinical correlation and management. Rest of the diffusion-weighted images are comparable to prior.*

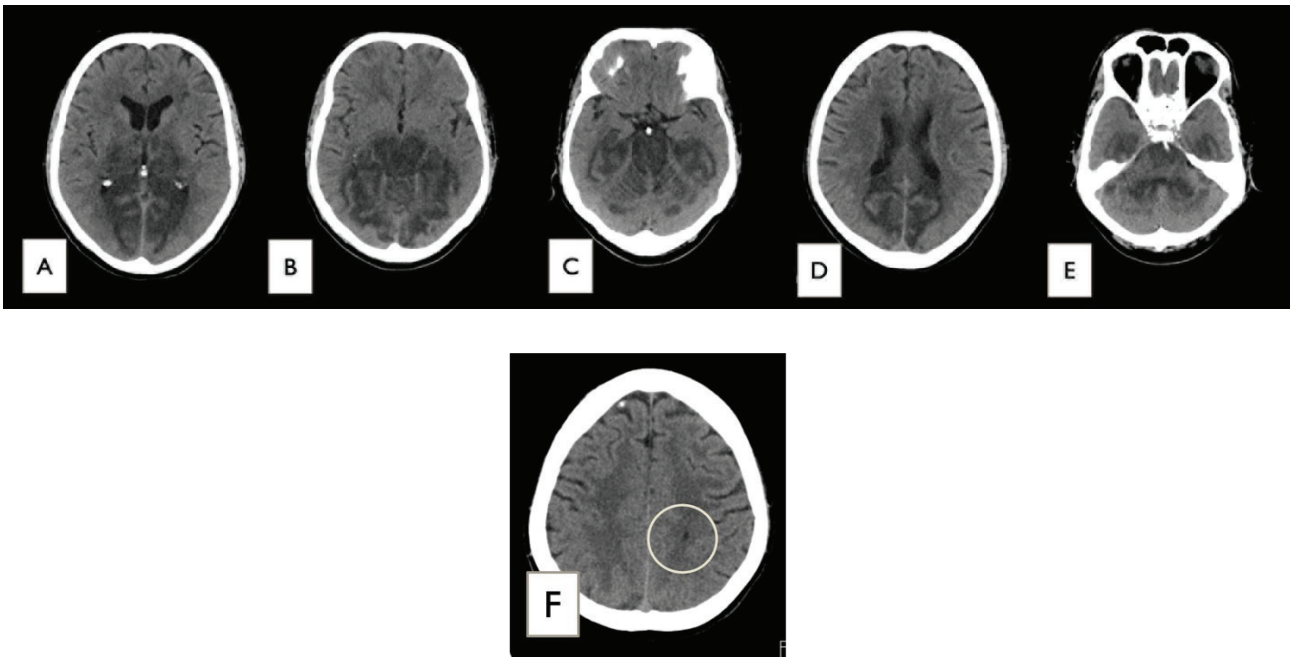

Figure 7. *Status post basilar artery thrombectomy and stenting. D evelopment of bilateral symmetrical hypodensities involving both thalami (A), parasagittal parietooccipital (B), medial temporal lobes (C), splenium of corpus callosum (D), brainstem and cerebellum causing mild mass effect upon the fourth and lateral ventricles (E) (posterior and temporal horns) as well as effacement of adjacent sulci and basal cistern, these finding in keeping with acute to subacute infarction of posterior circulation. Newly seen old insult seen at the left partial region (F). No intracranial hemorrhage could be seen. No midline shift. No hydrocephalus. Rest of the exam remains unchanged from previous.*

affecting the posterior circulation, as evidenced by MRI findings of hyperintense foci in the pons and cerebellum.

The patient's clinical course was complicated by fluctuating neurological status, leading to repeated strokes or TIAs. The deterioration necessitated intubation and MV due to compromised consciousness and respiratory effort, compounded by hypotensive episodes requiring vasopressor support.

Interventional radiology played a crucial role in basilar artery thrombectomy, though post-procedure complications, including spontaneous pneumothorax and femoral artery pseudoaneurysm, added to the patient's morbidity.

These complications required surgical interventions, including aneurysm embolization and ICT.

The development of bilateral symmetrical hypodensities involving critical brain regions, including the thalami, parietooccipital lobes, brainstem, and cerebellum, indicates extensive posterior circulation infarction. This extensive infarction correlates with the patient's poor neurological outcome, leading to the decision for DNR status due to poor functional prognosis and reduced level of consciousness.

Physicians should be aware of the variable clinical presentation of a patient with total basilar artery occlusion. Early recognition and early treatment and have better outcomes.

Conclusion

This case highlights the complexity of managing acute ischemic strokes in patients with significant comorbidities and extensive cerebrovascular disease. The initial successful thrombolysis followed by thrombectomy underscores the critical role of timely interventions in improving outcomes. However, the patient's extensive posterior circulation infarction and subsequent complications, including spontaneous pneumothorax and pseudoaneurysm, illustrate the challenges and potential complications in the management of such high-risk patients.

Despite aggressive management, the patient's poor neurological prognosis led to the activation of DNR status, reflecting the importance of considering overall prognosis and quality of life in treatment decisions. This case underscores the necessity for a multidisciplinary approach in managing severe cerebrovascular events and highlights the importance of prompt, effective intervention balanced with considerations of long-term outcomes and patient quality of life.

What is new?

A drug abuser presented with total basilar artery occlusion and recurrent TIAs in a short period of time. Recurring IV tpa and mechanical thrombectomy.

List of Abbreviations

CT	Computed tomography
DCM	Dilated cardiomyopathy
DNR	Do not resuscitate
GCS	Glasgow coma scale
ICA	Internal carotid artery
ICT	Intercostal chest tube
ICU	Intensive care unit
IV tPA	Intravenous tissue plasminogen activator
MRA	Magnetic resonance angiography
MRI	Magnetic resonance imaging
MV	Mechanical ventilation
NIHSS	National Institutes of Health Stroke Scale
SBP	Systolic blood pressure
TIA	Transient ischemic attack

Conflicts of interest

The authors declare that they have no conflict of interest regarding the publication of this case report.

Funding

None.

Consent for publication

Written consent was obtained from the patient.

Ethical approval

Ethical approval is not required at our institution to publish an anonymous case report.

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Summary of case

1	Patient (gender, age)	Male, 46
2	Final diagnosis	Total basilar artery occulsion
3	Symptoms	Focal weakenss, coma
4	Medications	Iv TPA
5	Clinical procedure	Mechanical thrombectomy
6	Specialty	Neurology