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## Reverse Locked-In Syndrome

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### Abstract

**Background**—Basilar artery occlusion can cause locked-in syndrome, which is characterized by quadriplegia, anarthria and limited communication via eye movements. Here, we describe an uncommon stroke syndrome associated with endovascular recanalization of the top-of-the-basilar artery: “reverse locked-in syndrome”.

**Methods**—We report the case of a patient with atypical neurological deficits caused by acute ischemic stroke of the brainstem tegmentum. We perform neuroanatomic localization of the patient's infarcts by mapping the MRI data onto a brainstem atlas.

**Results**—A 61-year-old man presented with acute coma and quadriplegia due to top-of-the-basilar artery occlusion. He underwent emergent endovascular thrombectomy, with successful recanalization of the basilar artery at 4 hours and 43 minutes post-ictus. The patient regained consciousness and purposeful movement in all 4 extremities, but the post-procedure neurological examination demonstrated bilateral ptosis with complete pupillary and oculomotor paralysis. Magnetic resonance imaging revealed infarction of the bilateral oculomotor nuclei in the midbrain

tegmentum. At nine-month follow-up, he had anisocoria and dysconjugate gaze but was living at home and required minimal assistance in performing all activities of daily living.

**Conclusions**—Since the patient’s deficits were the exact opposite of those described in locked-in syndrome, we propose the term “reverse locked-in syndrome” to describe this neurological entity characterized by bilateral ptosis, non-reactive pupils and ophthalmoplegia with preservation of consciousness and extremity motor function.

### Keywords

Top-of-the-basilar artery; locked-in syndrome; midbrain tegmentum; endovascular thrombectomy; ophthalmoparesis

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## Introduction

Basilar artery occlusion causes several of the most disabling and feared stroke syndromes. Neurological deficits vary depending upon the segment of the artery that is occluded. “Locked-in syndrome” is a particularly devastating consequence of basilar artery occlusion [1,2], characterized by quadriplegia and anarthria [3,4]. Locked-in syndrome is typically associated with mid-basilar occlusion, which results in infarction of the basis pontis but sparing of the pontine tegmentum. Less commonly, a distal, top-of-the-basilar occlusion may cause locked-in syndrome due to infarction of the bilateral cerebral peduncles [5,6]. Patients continue to process afferent stimuli in all sensory modalities and cognition is preserved, but efferent expression is limited solely to eye movements or blinking. This state of profoundly limited self-expression causes significant duress for patients and families [7,8].

Here, we present the case of a patient with acute coma and quadriplegia due to top-of-the-basilar artery occlusion who underwent emergent intra-arterial thrombectomy. The patient regained consciousness and purposeful movements in all four extremities after the procedure. However, the patient’s post-procedure neurological examination demonstrated an uncommon constellation of deficits characterized by complete bilateral ptosis as well as complete pupillary and oculomotor paralysis. Since the patient’s deficits were the exact opposite of those described in locked-in syndrome, we propose the term “reverse locked-in syndrome” to describe this neurological entity.

## Methods

### Clinical Data from Acute Hospitalization

A 61-year-old man with a history of left medial medullary infarction in 2006 and atrial fibrillation (on warfarin) collapsed while teaching in a classroom and became immediately unresponsive. Student witnesses described labored breathing while the patient lay motionless on the floor. Upon arrival of emergency medical services personnel his respiratory rate was 8/min and his systolic blood pressure was over 200 mmHg. Electrocardiogram revealed atrial fibrillation. On initial neurological examination, his left pupil was fixed at 6 mm, and his right pupil was 4 mm and sluggishly reactive. Endotracheal intubation was performed successfully in the field by emergency medical services personnel with etomidate, succinylcholine, lorazepam and fentanyl.

Upon arrival to a local hospital's Emergency Department, his blood pressure was 142/90 mmHg, heart rate 80 beats per minute, and glucose 138 mg/dl. The first neurological examination that was performed after expected clearance of the sedative, analgesic and paralytic medications revealed a Glasgow Coma Scale (GCS) score of 3T (Eyes = 1, Motor = 1, Verbal = 1T). His right pupil was fixed at 6 mm and left pupil was 4 mm with minimal response to light. Gaze was midline and the oculocephalic reflex was absent. There were no volitional vertical eye movements present. Reflexive vertical eye movement were not tested. His complete blood count and basic metabolic panel laboratory values were all within normal limits, and serum troponin was not elevated. Non-contrast head computed tomography (CT) scan showed a hyperdensity at the top of the basilar artery, concerning for thrombus (Figure 1A–B).

Within 1 hour and 15 minutes of symptom onset, the Emergency Department attending physician activated the Telestroke consultation service at our institution. Intravenous tissue plasminogen activator was not administered due an international normalized ratio of 1.9. The patient was transferred to our hospital emergently via helicopter. On arrival to the Emergency Department at our hospital at 2 hours and 8 minutes from his witnessed collapse, the patient remained comatose, with no motor responses to noxious stimuli (GCS score = 3T). Both pupils were fixed and dilated (right 6 mm, left 7 mm). There were no volitional or reflexive vertical or horizontal eye movements. Corneal reflexes were present. CT angiogram (CTA) of the head and neck showed an acute thrombus at the basilar terminus with filling defects extending into the posterior cerebral arteries bilaterally (Figure 1C–D).

The patient underwent emergent intra-arterial thrombectomy by two passes using a 4MaxAce (Penumbra, Inc, Alameda CA). Groin puncture to thrombectomy time was 64 minutes. Recanalization of the basilar artery was achieved at 4 hours and 43 minutes post-ictus (Figure 2). Two pieces of thrombus, each measuring approximately 5 mm, were returned from the aspiration catheter (Figure 2D). The final angiogram revealed thrombolysis in cerebral infarction (TICI) 3 perfusion, although a small luminal filling defect remained at the left side of top of the basilar artery (Figure 2B), suggesting residual adherent clot or underlying atheromatous disease. Post-procedure head CT showed a small hyperdensity in right thalamus, likely representing a mixture of hemorrhage and contrast extravasation, but no large territory infarct or hemorrhage. The patient was started on intravenous heparin infusion, without a bolus, for treatment of the potential residual, nonocclusive thrombus. The patient remained intubated.

When sedation was discontinued for a neurological examination 2 hours post-procedure, the patient was awake, alert, and able to follow simple commands such as showing two fingers and moving his toes. He was able to lift and sustain antigravity movement in his right upper and lower extremities and was moving his left upper and lower extremities purposefully in the plane of the bed. Cranial nerve exam revealed complete bilateral ptosis with fixed, dilated 6 mm pupils. When the examiner opened the patient's eyelids, his primary gaze was dysconjugate with vertical skew (hypertropia of left eye) without any spontaneous eye movements. There was complete ophthalmoplegia to voluntary gaze testing and to oculocephalic reflex testing in all directions. Visual fields were full to confrontation and the cough and gag reflexes were intact.

Magnetic resonance imaging (MRI) of the brain was performed on a 1.5 Tesla scanner 20 hours after clot retrieval. Diffusion-weighted imaging (DWI) and apparent diffusion coefficient (ADC) data were acquired with  $1.4 \times 1.4$  mm in-plane resolution, with 4 mm slice thickness and 0.4 mm inter-slice gaps (i.e. 4.4 mm between axial slices). Infarcts were identified as hyperintense foci on the DWI sequence and confirmed via the presence of hypointense foci on the corresponding ADC map. DWI and ADC analysis revealed multiple foci of infarction in the tegmentum of the rostral brainstem, bilateral medial thalami (right greater than left), right cerebral peduncle, left occipital lobe and bilateral cerebellar hemispheres (left greater than right) (Figure 3). The patient was extubated on post-stroke day 6, transferred from the neurocritical care unit to the neurology floor service on day 7, and discharged to an inpatient rehabilitation hospital on day 15.

At the time of transfer from the neurocritical care unit to the neurology floor service, the patient's neurological examination demonstrated normal arousal and ability to follow commands. Cranial nerve exam again showed bilateral ptosis requiring manual assistance to open the eyes. Both pupils were 5 mm with minimal reactivity. There was left-sided vertical skew on primary gaze. Rightward conjugate gaze, right supraduction and bilateral infraduction were impaired. There was right internuclear ophthalmoplegia. Visual fields were full to confrontation. There was left-sided facial palsy in an upper motor neuron pattern. The patient's speech was slightly dysarthric without aphasia. Cough and gag reflexes were preserved. Motor exam showed at least antigravity strength in all four extremities with mild weakness of left wrist flexion and extension. He had brisk reflexes throughout. Babinski sign was present on left. Sensory perception to light touch and painful stimuli was intact bilaterally.

### Follow-up Assessment

Six months later, repeat neurological examination demonstrated normal arousal and alertness, full orientation to self, place, and date, and normal comprehension with reliable command-following. There was mild anisocoria with the left pupil 1 mm smaller than the right, but both pupils were round and reactive to light. Primary gaze was dysconjugate with right exotropia and skew deviation. There was left-beating end-gaze nystagmus and mild left facial weakness. His speech was fluent but with non-volitional volume changes. His strength was full in all extremities but he required a walker due to left-sided appendicular ataxia. Sensory perception to light touch, vibration and joint position was intact. He was living at home and required minimal assistance in performing all activities of daily living by nine months.

### Neuroanatomic Analysis

We performed precise localization of the infarct, as detected by DWI, by manually tracing the infarct borders onto a template of brainstem nuclei in standard Montreal Neurological Institute (MNI152 1mm) space — the Harvard Ascending Arousal Network (AAN) Atlas. This anatomic template, which is available online ([www.martinos.org/resources/aan-atlas](http://www.martinos.org/resources/aan-atlas)), is based upon histologic and ultra-high resolution *ex vivo* MRI data from a human brainstem specimen [14], and has been used in recent studies of brainstem lesion mapping and brainstem connectivity [15,16]. Axial images from the AAN atlas were selected for infarct

tracing based upon visual confirmation of anatomic landmarks that were present on the axial DWI images. Infarcts that extended across multiple axial slices along the superior-inferior axis on DWI were traced on every 4th axial image in the atlas, since the spatial resolution of the atlas in this dimension is approximately 4 times greater than that of the DWI dataset (1 mm versus 4.4 mm).

## Results

The brainstem nuclei and fascicles affected by the infarction were: bilateral oculomotor nuclei and fascicles; bilateral medial longitudinal fasciculi (MLF); rostral interstitial nucleus of the MLF (riMLF); pretectal or midsection of posterior commissure; and bilateral Edinger-Westphal nuclei. The bilateral paramedian thalamic nuclei were also affected. Lesion mapping results on the AAN Atlas are shown in Figure 3.

## Discussion

We describe a rare constellation of neurological deficits associated with infarction of the midbrain tegmentum, for which we propose the terminology “reverse locked-in syndrome.” This syndrome is characterized by pupillary and oculomotor paralysis with preservation of consciousness and extremity motor function. It is important to distinguish this syndrome from other basilar stroke syndromes as it may be associated with a favorable outcome due to preserved consciousness and extremity motor function. In addition, this case adds to the growing body of evidence suggesting that rapid endovascular thrombectomy may be effective at improving outcomes for patients with basilar artery thrombosis [17].

The cerebrovascular territory implicated in reverse locked-in syndrome is that of the interpeduncular perforating arteries, which typically arise from the first segment of the posterior cerebral artery (P1) but can also arise from any artery in the interpeduncular fossa [18]. In our patient, these small interpeduncular penetrating arteries may have been occluded by the thrombus at the top of the basilar artery, which extended to the proximal P1 segments. Alternatively, these penetrating arteries may have been occluded by distal microemboli from the residual clot that partially occluded the top of the basilar artery after the thrombectomy. When the embolus extends into proximal posterior cerebral arteries (mesencephalic arteries), regions of the thalamus and cerebral peduncle may be affected, as in this patient. The temporal and occipital lobes are often involved if the thrombus extends beyond the posterior communicating arteries or if these are small in caliber [19].

There are several potential explanations for the multi-focal infarction of specific nuclei and fascicles within the midbrain tegmentum. First, the density of capillary networks may vary within the brainstem tegmentum, leading to selective vulnerability to ischemia for neurons and axons with less dense vascularization. Second, incomplete occlusion or rapid revascularization of the anteromedial and lateral group of mesencephalic arteries may have led to sparing of structures supplied by these arteries, with infarction of structures that were not as rapidly reperfused [20]. Further elucidation of the vascular anatomy underlying the reverse locked-in syndrome will likely require postmortem analysis of human brainstems

and in-depth characterization of anatomic variability in the brainstem tegmentum's vascular supply.

A distinguishing feature of the reverse locked-in syndrome described here is the complete recovery of consciousness and the near-complete recovery of extremity motor function. Basilar stroke syndromes with similar constellations of cranial nerve nucleus and/or fascicular involvement have been reported — including with bilateral ophthalmoplegia, bilateral ptosis, and fixed dilated pupils — but these previous reports also involve a disturbance of consciousness or a more severe disturbance of extremity motor function. For example, hypersomnolence with bilateral third nerve palsies was initially reported in 1958 due to infarction of the reticular formation in the midbrain tegmentum [21]. Bilateral ophthalmoplegia with bilateral ptosis has been reported in association with extremity motor dysfunction, involvement of additional cranial nerves or sensory disturbance leading to adverse outcomes including death [22,23]. The syndrome of wall-eyed bilateral internuclear ophthalmoplegia (WEBINO syndrome) has also been described with lesions in the midline of the midbrain tegmentum that affected the bilateral medial longitudinal fasciculi (MLF) and pretectum. In WEBINO syndrome, the primary gaze is dysconjugate due to exotropia of both eyes (wall-eyed) with bilateral internuclear ophthalmoplegia and impaired convergence [24,25]. Our patient did not have exotropia on primary gaze at the time of his acute examinations in the intensive care unit, probably due to extension of infarct to the midbrain-diencephalic junction, causing abnormal convergence tone from a “pseudosixth” phenomenon. This pseudosixth phenomenon or pseudoabducens palsy refers to failure of ocular abduction without dysfunction of sixth nerve when there is lesion near the midbrain-diencephalic junction [26,27].

The putative mechanism for oculomotor, pupillary and eyelid abnormality in reverse locked-in syndrome is infarction of the following brainstem nuclei and fascicles: bilateral oculomotor nuclei and fascicles, which causes paresis of adduction, supraduction, infraduction and ptosis; bilateral medial longitudinal fasciculi (MLF), which causes horizontal gaze palsy; bilateral rostral interstitial nuclei of MLF (riMLF), which causes downward gaze palsy; pretectal or midsection of posterior commissure, which causes upward gaze palsy; midbrain-diencephalic junction, which causes “pseudosixth” abduction paresis; bilateral Edinger-Westphal nuclei, which causes dilated or midposition fixed pupils [27,28]. All of these nuclei and fascicles were affected to varying extents by the infarction, as detected by post-thrombectomy DWI. However, it is important to consider that the full extent of the infarction could not be precisely defined by the DWI data due to their limited spatial resolution, particularly in the superior-inferior axis (4.4 mm between axial slices).

The mechanism underlying our patient's rapid recovery of consciousness was likely timely reperfusion of the brainstem tegmentum and paramedian thalami, which spared a sufficient number of brainstem and thalamic arousal nuclei to enable reemergence of wakefulness, and hence consciousness. Indeed, near-complete sparing of brainstem arousal nuclei was observed on our lesion mapping analysis of the MRI data using the AAN atlas (Figure 3). Except for a small region of the midbrain reticular formation that overlapped with the infarct, all other nearby brainstem arousal nuclei (e.g. pedunclopontine nucleus, ventral tegmental area, and dorsal raphe) were not affected by the infarct. Although the bilateral

paramedian thalami were more significantly affected than the brainstem arousal nuclei, the asymmetry of the thalamic lesions, with relative sparing of the left thalamus, may explain why our patient did not demonstrate the disorientation, confusion, hypersomnolence, coma or akinetic mutism that have been previously described in patients with bilateral paramedian infarcts [29]. Similarly, the relative sparing of the descending corticospinal tracts traveling through the cerebral peduncles, with only minimal involvement of the right peduncle, likely explains his rapid recovery of motor function.

Fundamentally important to our patient's recovery of consciousness and motor function was the rapid mechanical extraction of the basilar artery clot. Although he presented in a coma and quadriplegic with fixed, dilated pupils, within 2 hours of the procedure he had recovered consciousness, was following commands and was moving all 4 extremities purposefully. At 9 months, he had a favorable outcome characterized by near-complete independence for activities of daily living.

## Conclusion

We propose the term reverse locked-in syndrome to describe the constellation of neurological deficits experienced by our patient with bilateral ptosis, ophthalmoplegia, and pupillary non-reactivity to light but preserved consciousness and extremity motor function. This syndrome is associated with infarction of the rostral midbrain tegmentum affecting the bilateral oculomotor and Edinger Westphal nuclei/fascicles, medial longitudinal fasciculi, rostral interstitial nuclei of the medial longitudinal fasciculus, and pretectum/posterior commissure, all of which are structures supplied by interpeduncular penetrating arteries arising from top of the basilar artery and P1 segment of the posterior cerebral arteries. As endovascular thrombectomy of basilar clots is performed with increasing frequency, rapid recanalization is likely to limit the size of brainstem infarcts, which may increase the incidence of atypical stroke syndromes such as the reverse locked-in syndrome.

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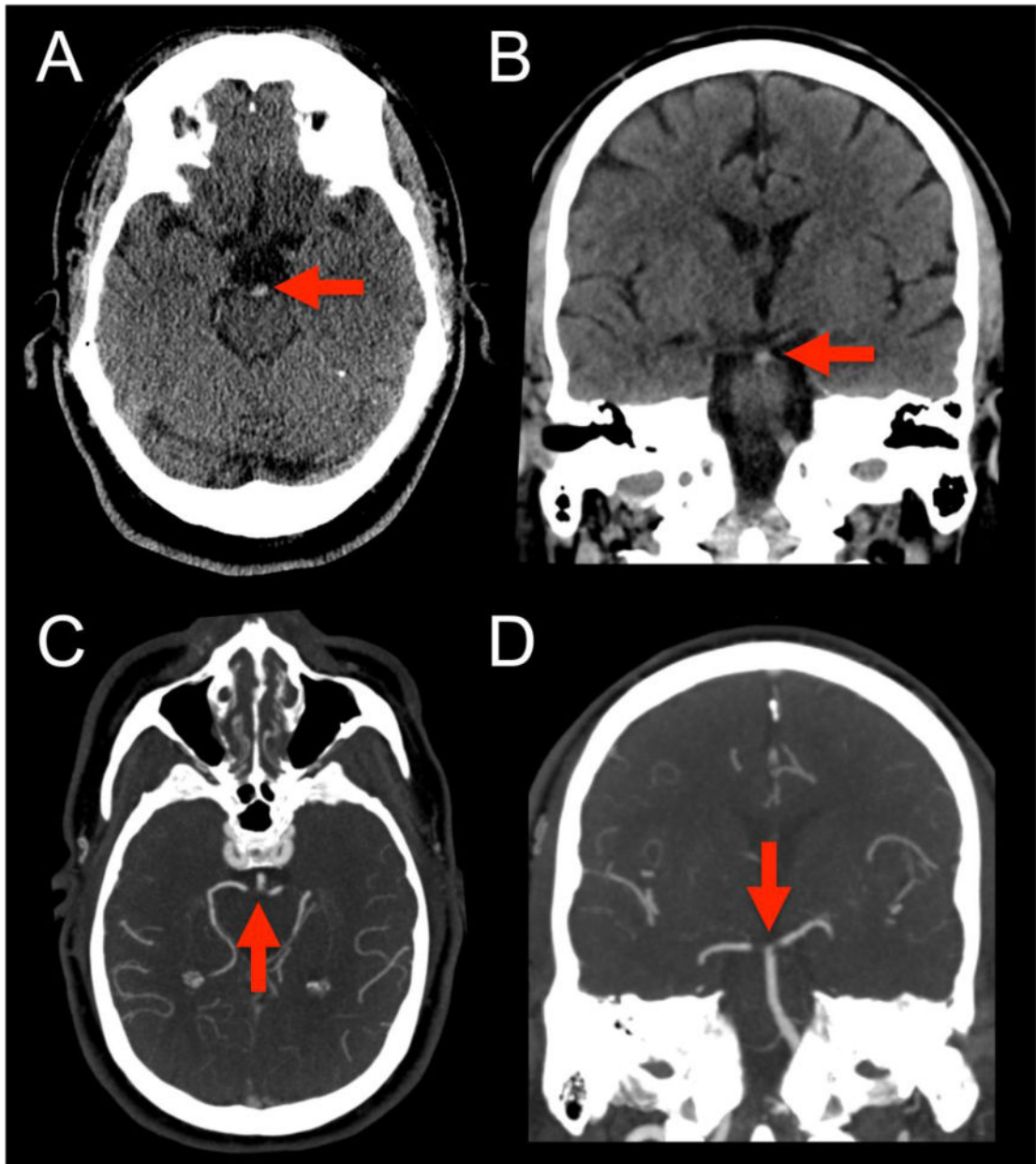
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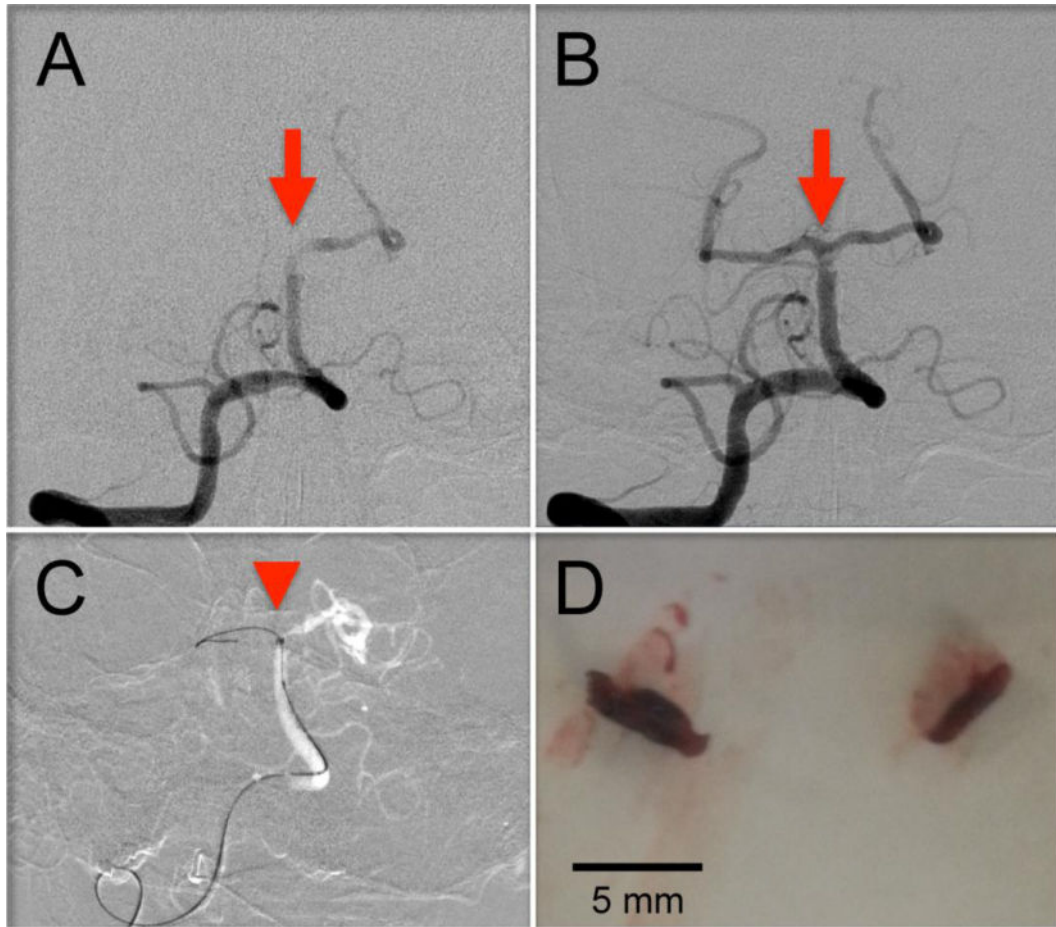
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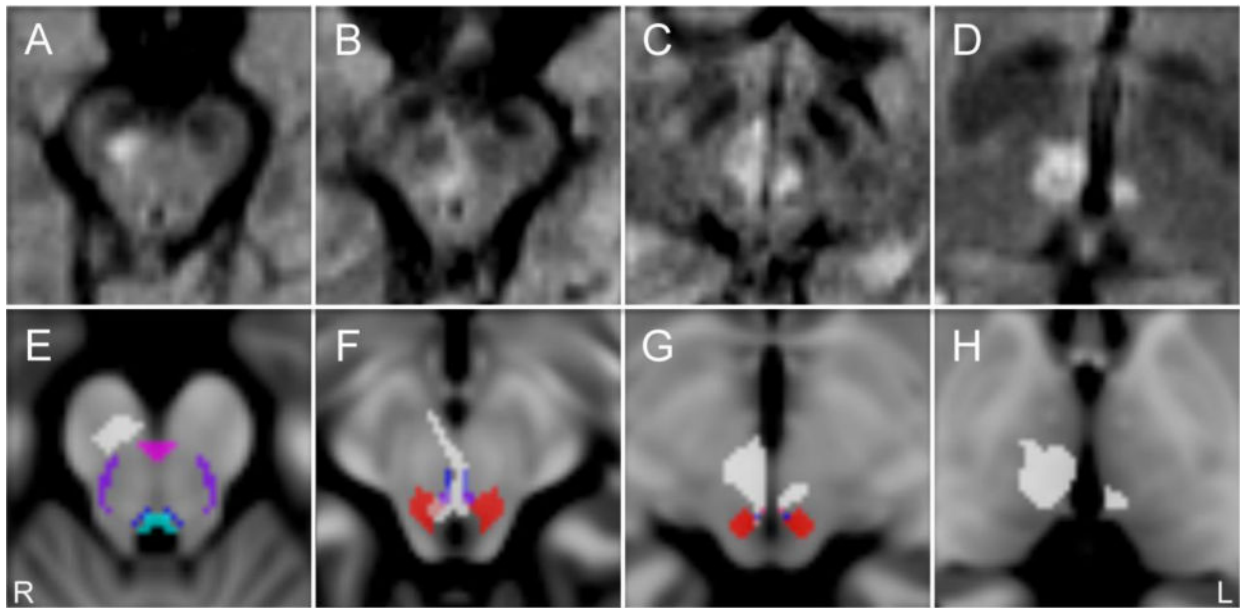


**Fig. 1.** A hyperdense top of the basilar artery is seen on axial and coronal images from the noncontrast head computed tomography (CT) scan in panels A and B, respectively (red arrows). A contrast filling defect is seen at the top of the basilar artery and bilateral posterior cerebral arteries on axial and coronal sections of the CT angiogram in panels C and D, respectively (red arrows).



**Fig. 2.**

Cerebral angiography with anteroposterior view of a right vertebral artery injection demonstrates a contrast filling defect at the top of the basilar artery (red arrow), right posterior cerebral artery and very mild opacification of the left posterior cerebral artery (panel A). Post-thrombectomy anteroposterior view of a right vertebral artery injection reveals antegrade flow in the basilar and bilateral posterior cerebral arteries, thalamoperforator branches and bilateral superior cerebellar arteries (panel B). There is a small, residual non-occlusive filling defect at the distal, left aspect of the basilar artery (red arrow), without evidence of impairing flow to the left posterior cerebral artery. Panel C shows the penumbra catheter being passed from the right vertebral artery to the right posterior cerebral artery past the basilar artery thrombus (red arrow-head). Two pieces of thrombus, each approximately 5 mm in maximal diameter, were aspirated (Panel D).



**Fig. 3.** Infarct Lesion Mapping. The brainstem and thalamic components of the infarct are shown on the diffusion-weighted images (top row, A–D) and on an atlas of brainstem nuclei, onto which the infarct was manually traced (bottom row, E–H). The infarct appears hyperintense on the diffusion-weighted images and is shown in white on the Harvard Ascending Arousal Network Atlas ([www.martinos.org/resources/aan-atlas](http://www.martinos.org/resources/aan-atlas)). The following cranial nerve nuclei and fasciculi are shown on the atlas: cranial nerve III (purple, F), medial longitudinal fasciculus (blue, F–G), and rostral interstitial nucleus of the medial longitudinal fasciculus (pink, G). The infarct overlaps with each of these nuclei and fascicles bilaterally. Also shown on the atlas are arousal nuclei of the ascending arousal network: pedunculo-pontine nucleus (purple, E), ventral tegmental area (pink, E), dorsal raphe (turquoise, E), and midbrain reticular formation (red, F–G). Except for a small region of the midbrain reticular formation that overlaps with the infarct (F–G), the infarct spares the arousal nuclei, which may explain the patient’s rapid recovery of consciousness after initially presenting in a coma.