Vertical Gaze Ophthalmoplegia from Infarction in the Area of the Posterior Thalamo-Subthalamic Paramedian Artery

MICHAEL WALL, M.D.,† THOMAS L. SLAMOVITS, M.D.,‡ LEON A. WEISBERG, M.D.,* AND SAMUEL A. TRUFANT, M.D.*

SUMMARY Three cases of a stroke syndrome of acute supranuclear vertical gaze ophthalmoplegia are presented. Voluntary vertical gaze and the vertical vestibulo-ocular reflex were absent or diminished initially in all three patients. The patients also had loss of convergence and alteration in their mental status. Computed tomography scans showed lesions in the region of the rostral midbrain and lower diencephalon in the area surrounding the third ventricle. The blood supply, ischemic stroke syndrome and function of this region with reference to the supranuclear control of vertical gaze are discussed.

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THE NEURAL SUBSTRATES for vertical gaze are located at the junction of the mesencephalon and diencephalon (fig. 1). The area currently thought to be of most importance in the function of vertical gaze saccades is the rostral interstitial nucleus of the medial longitudinal fasciculus (rostral iMLF).1 The rostral dorsolateral portion of this nucleus has been shown to be important in the production of downgaze.1,2 The caudal ventromedial aspect of this nucleus has been implicated in the premotor control of upward gaze3 as has the region of the posterior commissure and the pretectal area.3,4 This region of the rostral midbrain is supplied by the posterior thalamo-subthalamic branch of the basilar communicating artery, a branch off the basilar tip.5 In this report lesions are demonstrated in the region supplied by the posterior thalamo-subthalamic paramedian artery and the vertical gaze ophthalmoplegia and associated findings are discussed.

Case Reports

Case 1

This 56-year-old right handed man was in his usual state of good health until the evening prior to admission when he noted the sudden onset of double vision when looking straight ahead. He was aware of both vertical and horizontal components to the double vision which persisted in all fields of gaze. He then went to sleep, but when he failed to respond to his alarm clock his friend attempted unsuccessfully to awaken him and called an ambulance. While in the ambulance he awoke, confused and combative. At the time of admission to the hospital, he was drowsy but arousable when stimulated. He was disoriented with poor recall of the recent preceding events. There was slight flattening of the right nasolabial fold and mild weakness of the right upper extremity. His tendon reflexes were slightly brisker on the right than his left. He had flexor plantar responses bilaterally. There was slight dysmetria with movement of the left arm and left leg. Sensory examination was within normal limits.

Neuro-ophthalmologic examination showed normal visual acuity, visual fields and fundi. His pupils were 5 mm, slightly irregular and did not react to light or to a near target. Ocular motility examination (fig. 2) showed an alternating exotropia in the primary position of about 30 diopters. Lancaster red-green testing showed an eight diopter right hyperphoria which increased to 16 diopters on gaze up and to the right and a ten diopter left hyperphoria on gaze down and to the left. On command he could look fully horizontally in either direction with normal pursuit and saccades in either eye but there was no vertical pursuit or saccadic function. The doll's head maneuver failed to overcome the vertical gaze limitation. However, Bell's phenomenon was present bilaterally. Optokinetic nystagmus (OKN) was present horizontally but absent in both directions vertically. There was no ptosis. Collier's lid retraction sign was absent. Convergence was also absent.

The initial computerized tomographic (CT) head scan, done on the day of admission, with and without contrast, was normal. CT scan with contrast and 2 mm sections through the posterior fossa ten days later showed an isodense sharply marginated area of contrast enhancement in the rostral midbrain and contiguous diencephalon. The area of contrast enhancement surrounded the area inferior and lateral to the third ventricle in the subthalamic region. The lesion extended superiorly to involve the dorsal medial thalamus (fig. 3 and 4).

Two months later his ability to read was improving but he still had diplopia. He had a 15 diopter left exotropia, and had regained about 15 degrees of voluntary upgaze and had about 30 degrees of voluntary downgaze. There were full vertical eye movements when the oculocephalic maneuver was performed. His pupils were 4 mm, sluggishly reactive to light and briskly reactive to near. Two months later, he reported his double vision had improved. His extraocular movements were full at that time. There were some irregular
intermittent nystagmoid jerks on upgaze. He had slowed saccades bilaterally when he looked up or down and normal horizontal saccades.

One year after the onset of his deficit he had returned to work as a clerk. There was no diplopia when he looked straight ahead but he complained of horizontal diplopia with a slight vertical component on lateral gaze. His examination revealed his eyes to be straight in the primary position. He had full extraocular movements with normal horizontal saccades, but slowed vertical saccades. He had a ten diopter left hyperphoria present on left gaze and an eight diopter left exophoria on left gaze. Pupils were 3 mm, slightly irregular and reacted sluggishly to light but briskly to a near target. Convergence was moderately decreased. Optokinetic nystagmus was normal horizontally; however, it was absent when the tape was pulled up and there were poorly developed saccades when the tape was pulled down.

Case 2

This 74-year-old, right-handed, retired coal miner, was in his usual state of health until one evening while having dinner, he became aware of a sensation of transient brightness bilaterally. He was weak and lethargic and needed assistance to get to his car. He spoke little. He went to sleep and the next morning he noticed he was unable to look up or look down. He also complained of some difficulty focusing, even when using his reading glasses. He denied any diplopia, weakness, numbness, or speech difficulty. According to the patient's wife, his mental state had returned to normal by the next morning.

Past medical history was significant for a left temporo-
peroparietal skull fracture with an associated right temporoparieto-occipital lobe hemorrhage one year prior. Anisocoria was noted then with the right pupil larger than the left.

On examination, he was oriented and able to make simple change. He could remember one of three words at three minutes but could remember three of three with some prompting. Similarities were performed well. Neurologic examination was remarkable for left sided ataxia and total paralysis of upgaze and downgaze to pursuits and saccades. This gaze limitation could not be overcome with a doll’s head maneuver or with forced eye closure (fig. 5a). Horizontal eye movements were full with saccadic pursuit and end gaze horizontal nystagmus. There was no skew deviation present. Retraction nystagmus was noted when the OKN tape was pulled down. This was also present on attempted upgaze. A poor response was present when convergence was tested. The pupils reacted minimally to direct light and somewhat better to a near target. There was minimal lid retraction in the right eye and the remainder of the neuro-ophthalmologic examination was within normal limits.

CT head scan (fig. 6) showed two small discrete contrast enhancing areas inferior and adjacent to the posterior portion of the third ventricle in the region of the rostral midbrain. There was a contiguous enhancing right posterior thalamic lesion.

Three months later there was a moderate deficit of upgaze with testing of pursuits and saccades (fig. 5b top). This gaze limitation could not be overcome by the doll’s head maneuver or by forced eye closure. There was improved but still deficient downgaze when pursuit and saccadic function was tested.

Nine months after onset of his deficit, voluntary gaze (pursuits and saccades) was again slightly improved (fig. 5b bottom). Ocular motility was otherwise unchanged except a seven diopter left hypertropia was noted. The pupils reacted poorly to light but well to a near target.

Case 3
This 55-year-old right-handed man presented with a history of rheumatic fever and aortic and mitral valve replacements, treated with Coumadin. He was in his
usual state of health until one morning when he could not be aroused from sleep. He awoke in the hospital and remembered having horizontal diplopia. He also stated that his speech was mildly slurred and he had some gait difficulty. He denied any weakness, numbness or tingling. When seen at an outlying hospital he was noted to be poorly responsive to verbal and painful stimuli which improved over two days. He was noted to have poorly reactive pupils and limited vertical gaze.

Five days later he was transferred to the Veterans Administration Medical Center in New Orleans at which time he was noted to be a somnolent but easily arousable man who was oriented in all spheres but mildly inattentive. Cardiac examination revealed that a regular rate and rhythm was present with a grade III/VI systolic ejection murmur which radiated to the apex and axilla and a IV/V harsh systolic murmur in the second intercostal space. No prosthetic clicks were noted.

Cranial nerve examination revealed right medial rectus weakness and limited upgaze and downgaze bilaterally. There was vertical diplopia and right exotropia. No abnormalities were noted on motor, sensory and reflex examinations. There was slight dysmetria to have poorly reactive pupils and limited vertical gaze.

On examination, 15 days after onset of his symptoms, visual acuity was 20/15 OU. He had a 50 diopter alternating exotropia with no hyperdeviation. Horizontal eye movements were full to pursuit movements except for about 25% limitation of right medial rectus function. This could not be overcome with the doll’s head maneuver. Saccadic movements were hypometric in both directions horizontally. There were no vertical saccades present (fig. 7). There was about 10 degrees of upgaze to pursuit and about 20 degrees of upgaze to a doll’s head maneuver. Bell’s phenomenon was absent. There was no downgaze to pursuit or to the doll’s head maneuver. Optokinetin nystagmus testing showed a few beats horizontally in both directions and was absent vertically. Funduscopic examination was within normal limits. Visual fields were full to face and finger confrontation. Pupils were 4 mm and had a minimal reaction to both light and to a near target. Convergence was absent.

When his gait was tested, he was moderately unsteady. Romberg’s sign was absent. The remainder of the cranial nerve examination was normal except for diminished sensation to touch and pinprick on the left side of the face. Motor examination was normal. Sensory examination showed diminished sensation to touch and pinprick on the left side, however, proprioception and stereognosis were normal bilaterally. The tendon reflexes were hypoactive and symmetric. The plantar responses were flexor. Coordination examination revealed normal finger-nose-finger testing, rapid alternating movements and fine finger movements. However, there was mild difficulty with heel-knee-shin testing bilaterally.

CT scan (fig. 8) done 2 months after the initial onset of symptoms showed small lucent areas posterior to the third ventricle in the rostral mesencephalon and contiguous with the third ventricular region in the right thalamic region.

**Discussion**

The basilar communicating artery (mesencephalic artery, P1 segment of the posterior cerebral artery)5-9 is a single unpaired circumferential branch of the basilar artery arising from its bifurcation. It is that segment of the posterior cerebral artery proximal to the junction of the posterior communicating artery which is phylogenetically derived from the basilar system. The branches of this artery and branches of the P1 segment of the posterior cerebral artery supply the meso-diencephalic region. Many terms and classifications are used.5-10

There are three branches of the basilar communicating artery usually present. Two branches, the long and short circumflex arteries supply the cerebral peduncle, midbrain tegmentum at the level of the oculomotor nucleus, geniculate bodies and quadrigeminal plate.9 Using Percheron’s classification,4 these arteries are divided into rostral and caudal subdivisions and are termed the superior and inferior mesencephalic paramedian arteries (fig. 9a). Sieben and associates propose four subdivisions of the midbrain perforators.10

The third branch is the posterior thalamo-subtha-
FIGURE 5. Photographs of the eye movements in case 2, showing the evolution of partial recovery of vertical gaze. There is orthotropia in the primary position. Full horizontal gaze is present. Marked limitation of upgaze and absence of downgaze are shown as is impaired convergence (a). Partial recovery of vertical gaze at 3 months is shown in (b, top) and at 9 months in (b, bottom).

FIGURE 6. Post-contrast CT done one week after the onset of symptoms shows two nodular lesions located caudal to the posterior third ventricle. These are located in the pretectal dorsal midbrain region (a). In addition, there is an enhancing left (readers right) posterior thalamic lesion, present in the axial scan, but not shown. All three lesions are seen adjacent to the third ventricle on the coronal reconstruction (b).
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Figure 7. Photographs of the eye movements in case 3. A large right exotropia is present on gaze in the primary position. Lateral gaze is full except for moderate limitation of adduction of the right eye. The deficit of vertical gaze is seen in upgaze and downgaze. Absence of convergence is noted at the bottom of the sequence.

Lamic paramedian branch (thalamic paramedian artery) which penetrates the floor of the third ventricle and supplies the subthalamic region (including the rostral iMLF), medial rostral red nucleus, posteroinferior dorsomedial nucleus of the thalamus, nucleus parafascicularis and medial portion of the centromedian nucleus of the thalamus. Percheron has described three configurations (fig. 9b). Of eighteen dissections, there were nine cases of the type I configuration which has a single posterior thalamo-subthalamic paramedian artery coming off each basilar communicating artery. There were eight cases of type II in which both arteries come off one basilar communicating artery with or without a single stem.

Infarction in the region supplied by posterior thalamo-subthalamic paramedian artery has been shown to cause vertical gaze ophthalmoplegia presumably due to a lesion of the rostral interstitial nucleus of the medial longitudinal fasciculus (fig. 1, 10). Lesions causing isolated paresis of upgaze, isolated paresis of downgaze and combined upgaze and downgaze paresis have been reviewed by Büttner-Ennever and Pierrot-Deseilligny.1, 11 Upgaze paresis in monkeys can be produced by a lesion of the posterior commissure and its interstitial nuclei.1 Cases of isolated upgaze paresis in humans also involve these structures.1-4 Isolated downgaze paresis has been produced in monkeys2 and found in man11-19 with bilateral lesions in the region of the rostral iMLF. It has also been reported in a case with lesions of the periaqueductal gray and caudal portions of the nucleus of the posterior commissure, sparing rostral iMLF.19 Kömpf’s data from monkeys and Büttner-Ennever’s case support a rostral dorsolateral localization within the rostral iMLF for downgaze and a caudal ventromedial one for upgaze.1, 2 Pierrot-Deseilligny’s review suggests the converse.11 Combined vertical gaze ophthalmoplegia (upgaze and downgaze) has been found with lesions encompassing the rostral iMLF, interstitial nucleus of Cajal (INC) and the nucleus of Darkschewitsch.1

Six reported cases of ischemic combined vertical gaze ophthalmoplegia without associated third nerve nucleus or fasciculus involvement have been reported with post mortem examination (table 1, A). All of these cases had lesions of the rostral midbrain in the region just inferior to the third ventricle. This area encompassed the rostral iMLF. The vertical oculocephalic response was spared in one case. In this case, the INC was spared except for its extreme rostral tip.1 Two cases had absent vertical oculocephalic responses with lesions that may have involved or encompassed INC. Convergence was absent in four of the cases and pupillary reflexes were spared.

All three patients in the present study had vertical gaze ophthalmoplegia to pursuit and saccadic function which was complete in the first two cases and nearly complete in Case 3 (table 1, B). This gaze paresis could not be overcome by the doll’s head (oculocephalic) maneuver initially, but these oculovestibular eye movements returned after two months in Case 1. According to classical concepts, this loss of reflex eye movements occurs only with nuclear, internuclear and infranuclear disorders.

In a study of pretectal lesions in monkeys, Pasik noted similar discrepancies.3 Four monkeys had impaired upgaze with defective Bell’s phenomena and impaired upward oculocephalics and full voluntary downgaze. Three of these monkeys were shown to have no involvement of their oculomotor nucleus. Four of their monkeys had impaired spontaneous upward and downward movements with impaired vertical oculocephalics and defective Bell’s phenomena, however, three of these monkeys had lesions in the oculomotor nucleus. One of these monkeys had bilateral medial rectus paresis and bilateral MLF involvement. The other two had full horizontal eye movements.

Nashold and Gills23 described eight patients with...
Stereotactically produced midbrain lesions. These patients had loss of upgaze and all had absent Bell’s phenomena. One patient was autopsied and did not have involvement of the oculomotor nucleus. Nashold and Seaber in a later paper reported 16 patients with stereotactically produced rostral midbrain lesions as a procedure for pain. Thirteen patients had loss of voluntary upgaze, following a target and with the oculocephalic maneuver. Only three of these retained a Bell’s phenomenon. This loss of both voluntary and reflex eye movements with apparent supranuclear lesions has been noted in patients with presumed supranuclear monocular elevation paresis and loss of vertical gaze due to ischemic cerebrovascular disease. Jacobs’ patient with downgaze paresis had absent doll’s eyes and involvement of INC. The presence or absence of the vertical oculocephalic response in the autopsied cases of vertical gaze ophthalmoplegia is reviewed above (table 1A).

Our Case 1 had alternating skew deviation with hypertropia in the abducting eye. Keane, in his study of 408 patients with skew deviation, had 47 cases with alternating skew deviation, 20 of which had a rostral dorsal midbrain localization. Eleven of these cases had a hypertropia of the abducting eye with 3 being due to stroke. He proposed that alternating skew deviation was due to involvement of the pathways from both utricles to the vertical-rotary ocular motor neurons. It is not likely that our patients’ loss of vertical gaze was due to bilateral involvement of the rostral portion of the oculomotor nuclear complex. According to Warwick’s map, the Edinger-Westphal nuclei and subnucleus for the inferior rectus occupy the rostral tip of the nucleus. The subnuclei for the superior recti are just caudal to inferior rectus subnuclei making vertical gaze ophthalmoplegia due to a lesion here a theoretic possibility. The loss of pupillary response to light with the initial loss to near in cases 1 and 3 could be due to oculomotor nuclear involvement. However it could also be explained by damage to the pretectal area and involvement of fibers for the near response as they descend through the midbrain. The medial rectus paresis in case 3, though, could be explained by involvement of the third nerve nucleus or fascicle. These cases did not, however, have a nuclear third nerve syndrome as described by Daroff. The overall clinical presentation is best explained by a rostral midbrain localization. This is supported by the locations of the lesions on the CT scans.

A more likely possibility, especially in cases 1 and 2 is that the supranuclear structures i.e. INC and rostral iMLF, may be more important in generation of the vestibulo-ocular reflex (VOR) than the elementary, direct 3 neuron arc (semicircular canals to vestibular nucleus — vestibular nucleus to nuclei of III, IV, and VI via the MLF — cranial nerves III, IV, and VI) classically proposed. Lesioning the vestibular nuclei...
in mammals produces degeneration not only in the nuclei of III, IV and VI and the MLF but also in the INC. A problem encountered in determining the function of INC with lesioning experiments and clinical pathologic correlation is whether to ascribe the dysfunction to the nucleus itself or to fibers passing through INC. Kainic acid lesions of the INC which destroy only the cell bodies of the neurons and leave the fibers in transit unaffected, in the cat, produce deficits of the vertical vestibulo-ocular reflex, especially in the upward directions. The oculomotor nucleus, MLF and posterior commissure fibers were spared in these animals. The INC is proposed to be a structure of the indirect VOR pathway which connects the vestibular nuclei and ocular motor nuclei. This pathway is thought to be multisynaptic, involving connections within the reticular formation of the brainstem and the cerebellum. The proposed connections of this pathway, important for vertical gaze, are diagrammed in Figure 10. Our patients may have had involvement of their INC as it lies adjacent to the rostral iMLF. The rostral iMLF might also play a role in this VOR integration as shown by experiments with radioactive tracer placed in the superior and lateral vestibular nuclei. The tracer labelled an ipsilateral portion of the rostral iMLF. Further study of the vertical VOR in animals and clinical pathologic correlation in humans will be necessary to clarify the pathways of this reflex.

All three of our patients had loss of convergence. The pathways mediating vergence are not well defined. Simulation studies suggest the pathway for convergence may begin at the temporo-parieto-occipital junction (Brodmann's areas 19 and 22) and descend to the third nerve nucleus anterior to the pretectum. Extracellular microelectrode recordings in monkeys have shown units near the oculomotor nuclear complex that have a firing rate related to the angle of convergence. This pathway may be involved in our patients. Loss of convergence has been noted with similar lesions in animals and humans.

Cases 1 and 3 had loss of pupilloconstriction to a near target. This pathway has been postulated to descend from cerebral cortex to the oculomotor nuclear complex along with the convergence fibers. This may account for the loss of the pupillary response to near in this series of patients.

All three of our patients had alteration in their mental status. The change from normal lasted only overnight in case two. This patient also had the smallest lesion noted on CT scan and medial thalamic involvement was not bilateral. The alteration of mental status in other similar cases has been attributed to bilateral lesions of the medial thalamus and adjacent periventricular gray. However, Castaigne has reported mood and behavioral changes with unilateral paramedian thalamic infarcts.

Our patients' lesions on CT scan conform to the area of distribution of the posterior thalamo-subthalamic paramedian artery. The sudden onset of the neurologic deficit, followed by gradual recovery and the development of the demonstrated low density and contrast enhancing lesions (with initially normal CT scans) are most compatible with a vascular ischemic etiology. Since both sides of the brainstem are involved in these cases, this may represent a Percheron type IIb occlusion. The mechanism of the patients' infarctions is unclear. Cases 1 and 2 could have been either embolic or thrombotic. The mechanism in Case 3 is presumed to be embolic as the patient had aortic and mitral valve replacements. Based on post-mortem findings, both thrombus formation and emboli have been implicated in occlusion of this vessel.

Occlusion of the posterior thalamo-subthalamic paramedian branch of the mesencephalic artery produces a distinct clinical stroke syndrome manifested by

### Table 1A

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### Table 1B

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+ = present; - = absent; ? = not reported; D = decreased to near target.
vertical gaze ophthalmoplegia and loss of convergence. This may occur with only a mild transient alteration in mental status and loss of pupillary reflexes. The characteristic lesion can be shown by computer- tomographic scanning of the area surrounding the mesencephalic-diencephalic junction.

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References

Major Cerebral Infarction from Tumor Embolus

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SUMMARY A major right hemispheric infarct developed in a 31-year-old man within forty-eight hours of lung resection for metastatic synovial-cell sarcoma. Post mortem exam revealed tumorous occlusion of the right internal carotid artery. Major stroke from cerebral tumor embolus should be seriously considered in patients with primary or metastatic lung cancer who have had a very recent pneumonectomy, especially when there are symptoms and signs of multi-organ or extremity ischemia.

 strokes with malignant solid tumors is usually secondary to a hypercoagulable state or emboli from infectious or nonbacterial thrombotic endocarditis.1-4 Tumor emboli that occlude major cerebral vessels are rare and, except for atrial myxoma, are often not emphasized in the differential diagnosis of nonatherosclerotic stroke.5-7 or of CNS vascular disease in cancer patients.2-4 Occasionally, tumor mucin can embolize to cerebral vessels8-9 and multiple microscopic tumor emboli to smaller vessels may present as an encephalopathy.3-10 Here, we present a case of synovial-cell sarcoma with a tumor embolus resulting in total internal carotid occlusion shortly after a partial pneumonectomy.

Report of a Case

This 31 year old black man developed swelling under his right toe in March of the year of admission. In mid-June, biopsy of the lesion revealed "clear-cell sarcoma of the tendon". At that time there was no evidence of metastatic disease on bone and gallium scans, or on chest x-ray.

Three weeks later, chest x-ray revealed two large nodular densities in the left lower lobe. He underwent a right below the knee amputation, and 12 days later, a left lower lobectomy with five wedge resections of the left upper lobe. Pathologic examination of the lung tissue confirmed metastatic synovial sarcoma with extensive vascular invasion. On the same day, he had a bone marrow storage procedure in preparation for intensive chemotherapy.

Two days after the pneumonectomy, he developed a dense left hemiplegia, hemianesthesia, and hemianopia with left sided hyperreflexia and an extensor plantar response, but remained alert. There was no clinical evidence of emboli to viscera, skin or limbs. On the day of this event, head CT with and without contrast was normal; however, repeat scan, four days later, revealed a non-enhancing low-density lesion in the distribution of the right middle cerebral artery. The patient did not improve neurologically.

Subsequently, he developed melena and his hematocrit fell from 33 to 27. Radiological studies revealed partial small bowel obstruction by metastatic nodules just distal to the duodeno-jejunal junction. He became febrile and died one month after his pneumonectomy.

Postmortem Examination

The patient had extensive spread of synovial-cell sarcoma. A metastatic deposit in the jejunum had perforated, with resulting peritonitis and abscess formation. Additional metastases were present in other bowel areas, lungs, spleen, both kidneys and adrenal glands. No major visceral or limb arteries were occluded by tumor emboli.

Neuropathologic examination showed the intradural portion of the right internal carotid artery to be completely occluded by firm, yellowish-white tissue (fig. 1). The occlusion extended into the first three centimeters of the right middle cerebral artery. Microscopic examination of the occluding material showed that it consisted of synovial-cell sarcoma (fig. 2). The anterior portion of the right temporal lobe, basal ganglia, and internal capsule showed infarction with early liquefaction necrosis. In addition, two hemorrhagic metastatic lesions, 3 cm in size, were present in both frontal lobes; there was one smaller non-hemorrhagic metastasis in the right occipital lobe.

Comment

Fifteen prior cases of major cerebral infarction from malignant tumor emboli were reviewed (table 1).3,11-23
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