

## Short Communications

# Medial Medullary Infarction from Fibrocartilaginous Embolism to the Anterior Spinal Artery

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**SUMMARY** A previously healthy young woman presented with sudden onset of quadriplegia, anesthesia below the C3 dermatome, respiratory paralysis, vertical nystagmus, ocular bobbing and cortical blindness. After partial resolution of the latter deficits, she remained quadriplegic, with a C3 level of anesthesia, and in respiratory paralysis until death from complications of a fulminant pulmonary infection. Autopsy disclosed bilateral infarctions of the medial aspect of the medulla and the upper cervical cord, in the distribution of the anterior spinal artery. Multiple sites of arterial occlusion by fibrocartilaginous material were found in branches of the anterior spinal artery, in correspondence with the sites of infarction. A review of the literature disclosed only 2 examples of medial medullary syndrome from embolism to the anterior spinal artery.

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INFARCTION IN THE MEDULLA OBLONGATA is usually unilateral and limited to its dorso-lateral aspect. Medial medullary infarction occurs rarely, and it results from occlusion of the vertebral or anterior spinal artery (ASA).<sup>1,2</sup> Bilateral symmetric involvement of this area by vascular lesions has been described on three instances before,<sup>3-5</sup> in one of them as a result of embolic occlusion of ASA branches.<sup>4</sup>

This report describes the case of a young patient with bilateral embolic medial medullary infarctions. The topography of the medullary lesions and the associated vascular pathology were studied by serial sections, which disclosed multiple occlusions of the ASA branches by fibrocartilaginous emboli.

### Case Report

A 23 y/o female bank clerk complained of sudden occipital headache while at work, and vomited once. Taken immediately to the hospital, on her way she developed bilateral arm weakness, rapidly followed by weakness of the legs. By the time of her arrival in the emergency room a few minutes after onset, she had become unresponsive, had shallow respirations at a rate of 3 per minute and then suffered a respiratory arrest. Because of marked trismus, intravenous diazepam (Valium) was required to perform oral endotracheal intubation. After 10 minutes of mechanical ventilation she was able to open her eyes spontaneously. Physical examination showed regular pulse at 72 per minute, temperature 37 degrees C. She was able to open and close the eyes spontaneously and on verbal command, and the extraocular movements were conjugate and full in all directions. Testing of visual func-

tions showed an inability to fixate on a static or moving target, with an absence of blinking response to visual threat from either side, suggesting bilateral blindness. Pupils were 3 mm in diameter and reactive to light. She could answer to simple questions using a code of eye blinking. Ocular movements were full and conjugate in the horizontal and vertical planes, with irregular coarse upward vertical nystagmus in all directions of gaze. Irregular "bobbing" eye movements of conjugate down gaze occurred intermittently. She had mild bilateral facial weakness, more pronounced inferiorly, but the orbicularis oculi were also weak, and accounted for diminished corneal reflexes bilaterally. Cranial nerves IX, X and XII could not be examined because of oral endotracheal intubation. Her 4 limbs were paralyzed and flaccid, with the exception of traces of internal rotation of the hips and knee flexion to verbal command. Rotation and flexion-extension head movements were also absent. The deep tendon reflexes were absent, except for traces of right biceps and both ankle jerks. Plantar responses were minimal, with tendency to extension. Reactivity to pain was absent below the clavicular area bilaterally, and joint position sense was absent in the limbs. The neck was supple. There were no cervical bruits.

A CT scan on admission was normal, without signs of intracranial hemorrhage, infarction or mass effect. Cerebral angiography performed within hours showed patent extracranial and intracranial arterial systems; the vertebrals and basilar arteries and their branches showed no definite signs of occlusion, except for questionable slowing of flow in the right posterior cerebral artery.

Within 12 hours following admission the ocular "bobbing" movements disappeared and she regained visual function bilaterally, but her motor function deteriorated further: all tendon reflexes were abolished, and no voluntary movements of the limbs were detected. Upbeat vertical nystagmus was less marked and only present on upward gaze. Her neurologic condition

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remained stationary for the rest of the hospital stay, with flaccid areflexic quadriplegia, anesthesia for pain below the C3 dermatome bilaterally, absent joint position sense, bilateral facial weakness, full visual fields and extraocular movements, discrete upbeating nystagmus in upward gaze, and respiratory paralysis.

Examinations of cerebrospinal fluid were performed on 3 occasions, in all being within normal limits. A cervical myelogram performed on day 2 was normal. Auditory and visual evoked responses performed on days 4 and 5 were normal.

On the day following admission, chest x-rays showed signs of bilateral basilar pneumonia, and treatment with nafcillin was started. However, a progressive decline in  $pO_2$  followed, in association with extensive signs of bilateral consolidation by x-rays. Despite the use of multiple antibiotic coverage and chest physiotherapy, she developed progressive hypoxia and hypercapnia which required increasing levels of oxygen in inspired air. On day 12, she became suddenly cyanotic and dyspneic, with sinus bradycardia followed by ventricular tachycardia and arrest.

#### Autopsy Findings

The cause of death was a left tension pneumothorax. In addition, there was marked diffuse fibrous proliferation in the interstitial tissues of both lungs, with almost complete obliteration of alveolar spaces. Examination of the heart was only remarkable for moderate dilatation and hypertrophy of the right ventricle. The heart valves and endocardium were normal.

The brain weighed 1200 gm, and appeared normal to external inspection. Sections of the formalin fixed cerebral hemispheres showed no abnormalities, and microscopic examination of multiple sections of cerebral cortex, basal ganglia, cerebellum, midbrain and pons was unremarkable. The brainstem cuts revealed discrete areas of symmetric softening at the medial portions of the lower one-half of the medulla. The cervical cord showed areas of softening of the anterior aspects of segments C1 through C5. The medulla and upper cervical cord were divided into 7 blocks which were serially sectioned at 7 micron intervals, and stained with HE, Heidenhain's method for myelin, and the Kluver-Barrera method. In addition, the distal 3 cm of both vertebral arteries, the origin of both ASA and their distal single (descending) trunk, and the proximal one-third of the basilar artery were embedded in one block, serially sectioned at 7 micron intervals, and stained with HE and Van Gieson's techniques. Microscopic examination of these vessels failed to reveal areas of arterial occlusion or inflammation of the vascular wall.

Microscopic examination of the medulla disclosed areas of infarction affecting the median region bilaterally, extending throughout the lower two-thirds of this portion of the brainstem. The uppermost level of medial infarction was located approximately 1.7 mm below the ponto-medullary junction, where the infarction was confined to the most dorsal aspect of the tegmentum, with slightly asymmetric involvement of the me-

dial longitudinal fasciculus and tectospinal tracts (fig. 1A). In addition, two smaller separate foci of infarction were present at the ventro-lateral portions of both inferior olivary nuclei (fig. 1A); these lesions disappeared several hundred microns below, whereas the medial infarcts enlarged progressively, attaining their maximal size at a level of section of the hypoglossal nuclei, where they involved both pyramids, medial lemnisci, tectospinal tracts, medial longitudinal fasciculi, the exiting hypoglossal fibers, and the ventro-medial portions of the inferior olivary nuclei (figs. 1B and 2). These large median infarcts remained unchanged for an extension of approximately 500 microns below the hypoglossal nuclei level. Caudal to this level the infarcts showed progressive reduction in size, especially in their ventral portion, eventually becoming confined to the lemnisci and tectospinal tracts at a level of section of the decussation of the medial lemnisci (fig. 1C). They became smaller and more ventrally located at the level of the cervico-medullary junction (fig. 1D). Sections of cervical cord revealed infarction of the central area at the C2-C3 level, with bilateral involvement of anterior horns and anterior and lateral funiculi. More caudally the cord infarction was limited to the anterior horns and medial portions of the anterior funiculi.

Multiple areas of arterial occlusion in ASA branches were detected throughout the medullary and cord infarctions. A pale eosinophilic granular material was frequently found inside the arteries (fig. 3), in some cases with total occlusion of the lumen and distension of the wall. In many instances, such occlusions were found at the proximal edge of an area of infarction. The occluded vessels were almost exclusively midline arteries, in either extraparenchymal or intraparenchymal location, corresponding to paramedian branches of the ASA. The presence of numerous foci of arterial occlusion in ASA branches, with local distension of an otherwise normal arterial wall, and in close correspondence with adjacent foci of infarction, all indicate multiple embolic arterial occlusions as the cause of the infarctions. The occluding material had the characteristics of fibrocartilaginous emboli, an aspect further suggested by its positive staining with the Alcian blue technique for acid mucopolysaccharides.

#### Discussion

The clinical presentation of this case was one of sudden onset of deficits initially involving areas of the brain stem and, probably, the occipital lobes. The combination of quadriplegia, trunkal and limb anesthesia, vertical nystagmus, ocular "bobbing" and probable cortical blindness suggest basal pontine and bi-occipital ischemia from basilar artery occlusion as the initial event. An embolic mechanism of the stroke is suggested by the transient character of some of the deficits and the finding of intra-arterial fibrocartilaginous material at autopsy. The failure of angiography in detecting the occlusions is thought to have resulted from migration of the embolic material into distal pa-

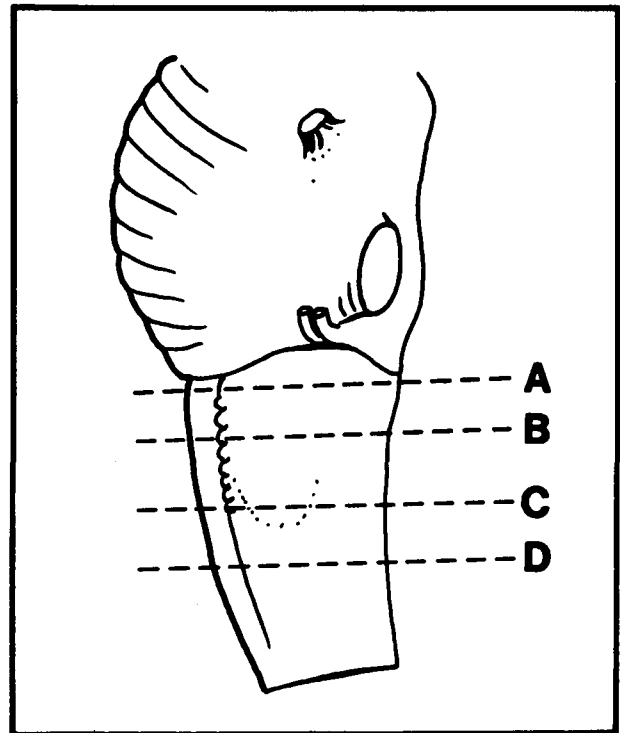
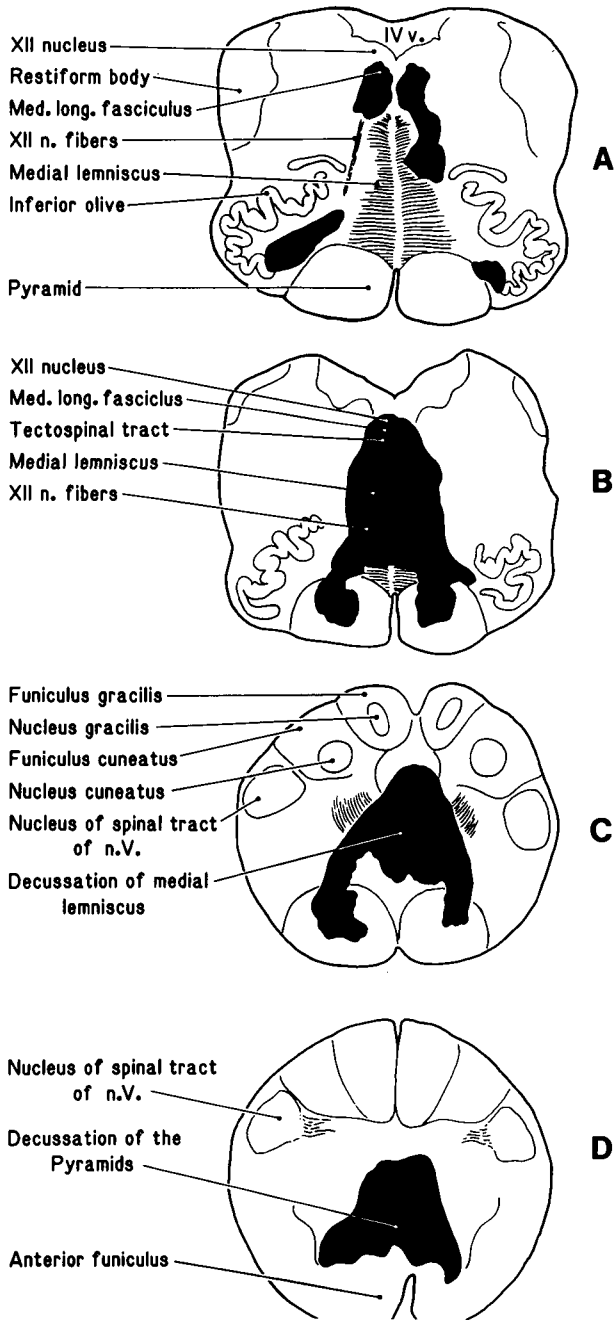


FIGURE 1. Artist's drawing of the medullary infarction (outlined in black) at the various levels of section shown in the Insert. See text for explanation.

renchymal branches of a size below that of angiographic resolution.

Occlusion of the ASA with infarction restricted to the spinal cord has been documented in a variety of conditions: emboli of cholesterol<sup>6</sup> and fibrocartilaginous intervertebral disc material,<sup>7-14</sup> clamping of abdominal aorta during aneurysmectomy,<sup>15</sup> arterial compression from adjacent vertebral metastatic tumor,<sup>16</sup> as a complication of intrathecal phenol injection,<sup>17</sup> and in relation to schistosoma infection.<sup>18</sup> In these instances, the associated clinical findings have been a flaccid paraplegia with transverse level of sensory loss for pain and temperature, with preservation of posterior column function. However, cases of occlusion of the

ASA at the medullary level have been a rarity, presumably because the usually bilateral origin of the ASA from the vertebrals provides adequate collateral for a single vertebral artery occlusion.<sup>19</sup> Since the original clinical descriptions of Spiller<sup>20</sup> in 1908 and Dejerine<sup>21</sup> in 1914, 18 cases of the medial medullary syndrome have been documented pathologically, 5 of them featuring isolated infarction of the pyramid, the other 13 corresponding to infarction of the medial aspect of the medulla from occlusion of the vertebral artery or ASA (Table 1). The vascular pathology has been predominantly thrombosis of the vertebral artery rather than the ASA, accounting for an occasional combination with elements of lateral medullary infarction.<sup>1, 3, 23, 24</sup> Only 2 cases from embolism have been reported,<sup>3, 4</sup> one with unilateral infarction,<sup>3</sup> the other bilateral.<sup>4</sup> The case reported by Mizutani et al.<sup>4</sup> showed bilateral medial medullary infarcts similar to those of the present case, and the etiology was foreign-body embolization to intramedullary ASA branches. Our case disclosed more extensive medial medullary infarcts, as well as involvement of the cervical cord. This combination of lesions suggests that the embolic occlusion occurred in both the ASA trunk and its distal intramedullary branches, in addition to presumed transient occlusion of the proximal basilar artery. The neurological deficits that remained until the patient's death are accounted for by the bilateral infarcts of the medial portion of the medulla and cervical cord.

Some clinical features of this and other documented cases of the medial medullary syndrome are of interest:



FIGURE 2. Full bilateral medial medullary infarcts at a level of section of the hypoglossal nucleus. HE, original magnification  $\times 5$ .

(1) Presence of upbeat vertical nystagmus on primary position, accentuated by upward gaze. This sign was present throughout the entire duration of our patient's illness, a feature also noted in prior cases.<sup>4, 5, 24, 31</sup> This form of nystagmus is considered to be a manifestation of lesions involving either the anterior cerebellar vermis or the medulla.<sup>32</sup> The medullary lesions associated with vertical upbeat nystagmus have been of tumoral<sup>33, 34</sup> or vascular<sup>4, 5, 24, 31</sup> character, and the constant unilateral or bilateral involvement of the medial longitudinal fasciculus (MLF) has been implicated in its causation.<sup>23</sup> The specificity of the medially-located medullary lesions for the production of pure vertical upbeat nystagmus is further supported by its absence in lateral medullary infarctions,<sup>35</sup> which spare the MLF. This structure was bilaterally involved in our patient. (2) Facial paresis without pathological evidence of a pontine lesion. In a number of instances, a mild contralateral predominantly inferior facial paresis has been

described in association with complete<sup>5, 24, 26, 30</sup> or partial (pyramidal)<sup>26-29</sup> medial medullary infarcts. A bilateral facial palsy was present in our case, and the lack of evidence for infarction above the ponto-medullary junction suggests that some corticobulbar fibers directed to the contralateral facial nucleus may exit from the medullary pyramid at levels corresponding to the upper portion of the olive.<sup>36</sup> This low medullary course of some corticobulbar fibers probably represents a small proportion of this tract's contingent since the degrees of facial paresis have been uniformly described as slight or mild, never to a level of frank paralysis.

The topography of medullary infarction in our case conformed to the vascular distribution of the anteromedial and anterolateral medullary arteries.<sup>37, 38</sup> These arise from the ASA at the middle and inferior portions of the medulla,<sup>28, 37, 38</sup> whereas in the upper medulla and ponto-medullary junction they originate from the distal vertebrals and proximal basilar, forming an arte-

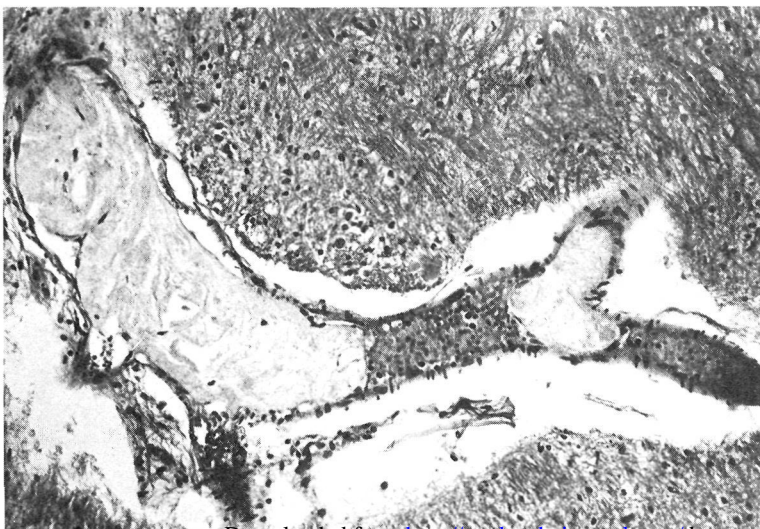


FIGURE 3. Fibrocartilaginous embolic material in an intramedullary branch of the ASA. Endothelial lining of the embolus is present. HE,  $\times 40$ .

TABLE 1 Pathologically-documented Cases of Medial Medullary Infarction

Author, year	Infarct location	Vascular pathology
Davison, <sup>1</sup> 1937 (2 cases)	Full R medial territory	Thrombotic occl. R VA and ASA, extending into proximal BA
	Full L medial territory, R pyramid	Occlusion L ASA
Davison, <sup>22</sup> 1944 (3 cases)	Full R medial territory	Thrombosis R VA and ASA
	Bilat. pyramid, partial ML	Occl. ASA trunk, after junction of branches from VAs
	Bilat. pyramid and ML	Compression origin both ASA by meningioma
O'Brien and Bender, <sup>23</sup> 1945	L pyramid and ML, R pyramid	Thrombosis medullary ASA
Brown and Fang, <sup>24</sup> 1961	R pyramid, medulla, pons	?Basilar branch occlusion
Meyer and Herndon, <sup>5</sup> 1962	Bilat. full medial territory	Arteritis (syphilitic) of branches of ASA
Fisher and Curry, <sup>25</sup> 1965 (Case 5)	R pyramid and ML	Thrombosis R VA
Trelles et al., <sup>26</sup> 1973 (2 cases)	Full R medial territory	Thrombosis R VA
	Full R medial territory, L pyramid and ML	Thrombosis R VA and ASA
Chokroverty et al., <sup>27</sup> 1975	R pyramid	Undefined
Leestma and Noronha, <sup>28</sup> 1976	L pyramid	Undefined
Hauw et al., <sup>3</sup> 1976 (2 cases)	R pyramid, ventral paraolive	Embolus distal R VA and proximal BA
	Bilat. full medial territory, R olive	Thrombosis both distal VAs and proximal BA
Ropper et al., <sup>29</sup> 1979	R pyramid	?Occl. medullary branch of R VA
Mizutani et al., <sup>4</sup> 1980	Bilat. full medial territory	Emboli (foreign body), intraparenchymal branches ASA
Ho and Meyer, <sup>30</sup> 1981	Full L medial territory, partial olive	Undefined
Present case	Bilat. full medial territory, partial olives	Emboli (fibrocartilaginous), intraparenchymal branches ASA

Abbreviations: VA = vertebral artery; BA = basilar artery; R = right; L = left; ML = medial lemniscus; ASA = anterior spinal artery.

rial plexus at the level of the foramen caecum.<sup>3, 38</sup> The latter vascular territory was spared in our case, as the whole extent of the infarct was restricted to the distribution of medullary and spinal branches of the ASA.

The source of the fibrocartilaginous embolic material remains unclear. All authors<sup>7-14</sup> agree that the material originates in the nucleus pulposus of the intervertebral disc, but the mechanism of its embolization into the ASA system is controversial. Naiman et al.<sup>7</sup> suggested two possible mechanisms: 1) a traumatic rupture of the annulus fibrosus, followed by tearing of the adjacent radicular artery with extrusion of nucleus pulposus material; 2) "injection" of disc material into arteries of the disc space, with further retrograde migration into a radicular artery. Subsequent reports<sup>8-11</sup> have stressed the importance of extrusion of disc material into the vertebral body (Schmorl's nodes), with access to sinusoids and veins of the bone marrow as the primary phenomenon,<sup>13</sup> leading ultimately to both ve-

nous and arterial embolization, as a result of arteriovenous communications normally present at the level of the spinal cord.<sup>39</sup> Trauma has occasionally preceded the onset of the myelopathy,<sup>7</sup> but most cases have affected individuals of sedentary habits, as was the case with our patient. Furthermore, retrospective review of cervical spine x-rays and myelogram failed to document degenerative disc pathology or evidence of trauma to this region. This form of vascular occlusion affects females predominantly, and the cervical cord is the most common site of infarction.<sup>12, 13</sup> This condition should be considered in the differential diagnosis of infarction in the ASA distribution.

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