Atheromatous Emboli to the Lumbosacral Spinal Cord

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Abstract: Atheromatous Emboli to the Lumbosacral Spinal Cord

The lumbosacral spinal cords of 28 patients with atheromatous emboli to abdominal viscera and/or grafts to the abdominal aorta were examined by serial sections. In 12 patients, atheromatous emboli were found in spinal arteries, most commonly in the sacral cord, and most frequently in the anterior spinal artery. The general absence of spinal cord infarctions was attributed to the nature of the emboli, apparent good collateral circulation, and the absence of diffuse atherosclerosis. However, 38% of the patients had arteriosclerosis; this was generally focal and not associated with significant luminal narrowing. Only one patient had infarction, which was limited primarily to the gray matter. It would appear that hypoperfusion must exist in conjunction with atheromatous emboli in order for infarction to develop. Organized atheromatous emboli also caused focal ischemic atrophy of neurons. It is postulated that this change may be the morphological basis for some of the atypical lower motor neuron diseases found in the elderly.

Additional Key Words: spinal arteries, infarcts, gray matter

Introduction

Pathological alterations affecting the intrinsic spinal arteries have been reported rarely and, therefore, have been reputed to be extremely uncommon. For example, Blackwood,1 in reviewing 3,737 postmortem examinations, found no cases of atherosclerosis, hypertensive vascular disease, embolism, or any type of vasculitis involving the spinal arteries. The chance finding of multiple atheromatous emboli in the spinal arteries of the lumbosacral cord of a patient at autopsy directed our attention to these arteries. We suspected, despite the paucity of case reports (table I27), that atheromatous embolization to these arteries might be common, since atheromatous embolization of the lower extremities and to the kidneys and other abdominal viscera have been reported with ever-increasing frequency.5'8'17 This investigation, therefore, was undertaken to determine the frequency and consequences of atheromatous embolization to the lumbosacral cord. It also was anticipated that additional information might be garnered which could help fill the general hiatus in knowledge concerning pathological changes in the spinal arteries in this area.

Methods

Complicated atherosclerosis is most frequently observed in the distal portion of the aorta and may serve as a source of atheromatous embolization to various abdominal organs and the lower extremities. Since the arterial blood supply to the lumbosacral cord originates from the same region in the aorta, this part of the cord appeared to be a good candidate for investigating possible atheromatous embolization.

Two groups of high risk patients were chosen: (1) patients with histologically documented atheromatous emboli to abdominal viscera, and (2) patients with infrarenal aortic grafts replacing atherosclerotic aneurysms.

This retrospective study was made possible because preserved organs including entire spinal cords were generally available for the last 1,000 autopsies performed in this hospital.

Twenty-eight patients were found who fit into one or both of the above two groups.

Paraffin blocks of the entire lumbosacral cord were prepared from each patient, with an average of 17 sections being taken. The following staining procedures were used: hematoxylin and eosin, elastic van Gieson, Weil-Weigert, Luxol-fast blue, and Nissl.

The slides were examined for evidence of vascular or spinal cord pathology. In those patients showing alterations, an additional five serial sections were made on all blocks of the involved lumbosacral cord. The charts from each patient were reviewed for symptomatology indicative of distal spinal cord disease.

Results

CLINICAL

Twenty-three of the 28 patients in this study were found to have atheromatous emboli in various abdominal viscera and 11 had aortic grafts. In seven patients with grafts, atheromatous emboli also were evident. Twenty-five patients were white, three were black. There were 21 males and seven females. The average of our patient population was 69.9 years.

Only one patient had clinically recognizable symptoms which could be attributed clearly to lum-
Patient H. S., a 60-year-old white man, was in essentially good health until three years before death when he became hypertensive. This was treated satisfactorily with diuretics and other antihypertensive agents. A few days prior to his last admission, he suddenly had a sharp pain in the lower back that was followed by weakness in both lower extremities. Sensitivity to pain and temperature was lost in both lower extremities and buttocks. There was incomplete involvement of touch and position sense, and fecal and urinary incontinence were evident. The general clinical picture was interpreted as due to an acute vascular catastrophe involving the lower spinal cord, secondary to rupture or occlusion of an aneurysm in the lower aortic region. During the investigation of an abdominal mass, his neurological condition stabilized and there was a certain degree of improvement of motor function. The patient was operated on and a large infrarenal aortic aneurysm was resected and replaced with a graft. During surgery and the immediate postoperative period, a marked and prolonged period of systemic hypotension occurred, following which the neurological status deteriorated to total sensory and motor paralysis. The patient then had acute renal failure and was treated with dialysis. The patient's neurological status never improved and he died three months later of a cardiac arrhythmia.

MORPHOLOGICAL

Discussions concerned with morphology, composition, and source of atheromatous emboli have been described elsewhere. Atheromatous emboli, as observed in the spinal arteries of this series, showed three fairly distinct stages of evolution.

(1) Recent atheromatous emboli. Most frequently the emboli appeared as acicular spaces, representing alcohol-dissolved cholesterol crystals, outlined by erythrocytes. Occasionally the emboli consisted of amorphous thrombus material containing cholesterol clefts, lipid-laden histiocytes, tiny fragments of calcium and hematoxylin pigment. Neither type of emboli tended to occlude the arterial lumens.

(2) Organizing atheromatous emboli (figs. 1 to 3). These emboli were characterized by an eccentric proliferation of loose intimal fibrous tissue around cholesterol clefts. Frequently, cholesterol crystals were encompassed by giant cells and in all cases the internal elastica showed focal disruption. The periadventitial tissues frequently exhibited a lymphoid inflammatory response extending above and below the site of embolization as well as into the adventitial tissues of contiguous veins. Occasionally acicular spaces were detected in the adventitia of the affected arteries.

(3) Organized atheromatous emboli. These appeared as eccentric fibrous intimal plaques containing cholesterol clefts. The arterial lumens were severely narrowed by this process but recanalization of the plaques was very common. The internal elastica showed persistent disruption. At this stage the inflammatory response had generally disappeared, but

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**Table 1**

<table>
<thead>
<tr>
<th>Author(s)</th>
<th>Year</th>
<th>Cases</th>
<th>Spinal cord level</th>
<th>Spinal artery involved</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fisher et al., 1960</td>
<td>1</td>
<td>1st lumbar to 1st sacral</td>
<td>Complicated atherosclerosis</td>
<td>Infarction of posterior columns, posterior horn, and lateral columns</td>
</tr>
<tr>
<td>Soloway and Aronson, 1961</td>
<td>2</td>
<td>T12 to lumbosacral cord</td>
<td>Complicated atherosclerosis</td>
<td>Infarction of anterior column</td>
</tr>
<tr>
<td>Reich, 1964</td>
<td>2</td>
<td>T12 to S3</td>
<td>Complicated atherosclerosis</td>
<td>Infarction of anterior column</td>
</tr>
<tr>
<td>Mauris, Barker, and Trou hypocrit, 1968</td>
<td>1</td>
<td>T12 to S3</td>
<td>Complicated atherosclerosis</td>
<td>Infarction of anterior column</td>
</tr>
<tr>
<td>Harrington and Amplatz, 1972</td>
<td>1</td>
<td>Not stated</td>
<td>Complicated atherosclerosis</td>
<td>Infarction of anterior column</td>
</tr>
</tbody>
</table>
ACROMATOUS EMBOLI TO THE LUMBOSACRAL SPINAL CORD

Acicular spaces occasionally were recognized in the adventitia. Atheromatous emboli in the spinal arteries were seen in 12 of the 28 patients.

Several features merit special emphasis. The arteries of the sacral cord were more frequently involved by atheromatous emboli than those of the lumbar cord. Thus, in seven patients emboli were found solely in the sacral cord, four patients had emboli in both the sacral and lumbar segments, and in only one patient were the emboli limited to the arteries of the lumbar cord. Atheromatous emboli occurred most frequently in the anterior spinal artery (11 patients), but such emboli also were observed in the circumferential (nine patients), posterior spinal (eight patients), sulcal (five patients), and intraparenchymal neural arteries (three patients).

The number of atheromatous emboli ranged from few (less than four) to multiple (greater than four). Multiple emboli were observed in five patients, and in two of this group the embolization was massive with one or more emboli found in every section of the lumbosacral spinal cord. The arteries of the lumbosacral cord were most frequently involved in cases of multiple embolization, while in patients with few emboli, the process was limited primarily to the arteries of the sacral cord.

The incidence of atheromatous embolization to the lumbosacral cord as compared to other viscera is presented in table 2. Atheromatous embolization to the spinal cord was second in frequency to that of the kidneys. All patients with atheromatous emboli to the cord had atheromatous emboli in the kidneys, and 60% of the patients with atheromatous emboli to the kidneys had spinal cord emboli.

Spinal cord infarction was evident in one patient. The entire length of the sacral cord showed an organizing cystic infarct (fig. 4) limited almost entirely

![FIGURE 1](image1)

Anterior spinal artery. Organizing atheromatous emboli. The arterial lumen is almost occluded by a loose fibrous internal proliferation encompassing cholesterol clefts. Recanalization is evident. Periadventitial lymphocytes are present. Note the focal disruption of the internal elastic lamina, an important differentiating feature from ordinary arteriosclerosis. Elastic van Gieson, × 165.

![FIGURE 2](image2)


![FIGURE 3](image3)

Branch of sulcal artery in gray matter of lumbar cord. Acicular clefts appear in adventitial tissues. The arterial lumen is patent. Focal perivascular lymphoid infiltrates are present. The extrusion of the cholesterol crystals into the adventitia may preclude reactive intimal fibrous luminal narrowing. Hematoxylin and eosin, × 265.
to the gray matter except for a focus of unilateral infarction of the anterior and lateral columns in the most distal segment of the sacral cord. This infarct was accompanied by an extensive organizing atheromatous embolus in the anterior spinal artery as well as a focal recent embolus in a circumferential artery. Every segment of the lumbar cord (fig. 5), as well as T12, exhibited organizing infarcts of varying sizes and irregular distribution, limited exclusively to the gray matter. Atheromatous emboli in the lumbar cord were localized primarily to branches of the sulcal arteries within the gray matter. These organizing emboli, however, were related to many, but not all, of the infarcts.

Three patients showed ischemic changes involving the anterior horn, and areas of focal neuronal loss were evident in one of these patients.

Focal intimal sclerosis of the spinal arteries not associated with atheromatous emboli was found in ten patients. Six of these had hypertension.

Discussion
The results of this study indicate that atheromatous embolization to the arteries of the lumbar-sacral cord is common in patients with abdominal aortic grafts and in patients with complicated atherosclerosis of the abdominal aorta and concomitant atheromatous emboli to other viscera; the frequency is approximately equal to that of atheromatous emboli to the pancreas and spleen. The apparent general lack of recognition of this phenomenon presumably is related to the fact that these emboli are generally subclinical and involve most frequently the spinal arteries in the distal spinal cord, an area not usually examined in routine autopsy surveys of the spinal cord.

The predilection for atheromatous emboli to lodge in the sacral cord, particularly within the distal anterior spinal artery, may be attributable primarily to anatomical factors. Since the anatomy of the spinal arteries has been well described, only those features germane to this discussion will be included.

The anterior spinal artery at the level of the filum terminale bifurcates dorsally and each branch joins one of the two posterior spinal arteries. The direction of blood flow in the anterior spinal artery is caudalward, while that of the posterior spinal arteries at the level of the lumbar-sacral cord is cranial. These anatomical features tend to favor lodging of emboli in the distal anterior spinal artery.

Anatomical factors also may explain why certain patients with atheromatous emboli in their kidneys...
and other abdominal viscera may escape atheromatous embolization to the spinal cord. The anterior spinal artery is usually not continuous, and the blood supply to each segment is derived from a variable number of unpaired medullary arteries. The most constant and largest of these arteries is the great artery of Adamkiewicz. This artery conducts blood to the anterior spinal artery serving the lumbosacral cord. The origin of the artery of Adamkiewicz is variable, arising from either an intercostal or a lumbar artery between segments T9 and L4. Since complicated atherosclerosis is generally more severe in the abdominal portion of the aorta, it would seem reasonable to suppose that atheromatous emboli to the lumbosacral cord would be more prone to occur in patients having a lumbar origin to this artery. Essentially the same applies to the great posterior medullary artery, an unpaired smaller artery originating from either an intercostal or a lumbar artery and delivering blood to the posterior spinal arteries of the lumbosacral cord.

Although atheromatous emboli to the brain may cause cerebral infarcts, spinal cord infarction occurs only rarely following atheromatous emboli to the spinal arteries. There are three possible reasons to explain this. The first is related to the structure of the emboli. Most of the atheromatous embolic material observed in the spinal arteries consisted chiefly of cholesterol crystals. This type of embolic atheromatous material is usually found in small arteries having diameters of 150 to 200 μ, as is the case in arteries of the spinal cord. The irregular crystalline material usually is not occlusive, and as a rule, such emboli are not associated with secondary thrombosis.

Second, the general absence of infarction, even in the face of massive embolization, strongly suggests the presence of a good collateral circulation in the lumbosacral cord. Compared to the thoracic cord, the sulcal arteries in the lumbosacral region are more numerous, larger and extensively arborized in the gray matter. This anatomical feature may be of significance in the establishment of an efficient collateral circulation in the presence of embolization. The development of this collateral circulation, furthermore, is promoted by the nature of the atheromatous embolic process which is usually partial and slowly occlusive.

The third factor is the absence of severe, diffuse atherosclerosis of the spinal arteries, a process which occurs with some frequency in the cerebral arteries. Although intimal sclerosis of the spinal arteries was not uncommon, occurring in 38% of our patients, the arterial alteration was generally focal and usually without severe luminal narrowing.

Infarcts in the lumbosacral cord, in association with atheromatous emboli, did occur in one of our patients and has been documented in three other patients (table 1). The extremely rare occurrence of spinal cord infarcts with atheromatous emboli suggested that additional factors must be present which act in concert with the emboli. In our patient, this appeared to be a prolonged bout of hypotension. Additional factors inferred from case reports are myocardial infarction and reclamping of the aorta.

Hypoperfusion of the spinal arteries probably represents the main aggravating factor in the production of infarctions. It is of some interest that spinal cord infarction has been reported as a complication of a Stokes-Adams attack and a myocardial infarct. Hypotension within the spinal arteries may be caused by systemic factors such as heart failure and shock, but local events such as prolonged clamping or pressure on the aorta also are important. Atheromatous narrowing of lumbar, intercostal, or medullary arteries, which is expressed clinically by the syndrome of intermittent claudication of the cord, also may be an important factor in local hypoperfusion.

It has been asserted that occlusion of the anterior spinal artery causes infarction of the anterior two-thirds of the spinal cord. In our case, occlusion of the anterior spinal artery by atheromatous emboli in the sacral cord produced mainly gray matter softening. The reasons for the almost exclusive gray matter infarction in this patient may depend on systemic hemodynamics, distribution of medullary arteries, collateral circulation, and the relative susceptibility to ischemia by the gray and white matter (the latter being much more resistant).

Noninfarct-producing atheromatous emboli to the lumbosacral arteries are not always inconsequential. Initially they may be associated with sudden onset of neurological signs and symptoms which may improve or resolve, as demonstrated in Patient H. S. More insidiously, severe arterial luminal narrowing by organization may induce ischemic atrophy of neurons in the gray matter, particularly involving the anterior horns. Such changes could cause subtle neurological symptoms and could lead to a false diagnosis of frontal lobe disease, peripheral neuropathies or distal aortic occlusive disease. It is valid to speculate that organization of atheromatous emboli in the arteries of the lumbosacral cord with accompanying ischemic neuronal atrophy may be the morphological basis for some of the slowly progressive spinal cord syndromes found in the elderly. Painless progressive flaccid areflexic paraplegia and other atypical motor neuron diseases could fall in this category.

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References


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