Massive Infarction of Spinal Cord and Vertebral Bodies as a Complication of Dissecting Aneurysm of the Aorta

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Dissection of the aorta by hematoma may present protean clinical signs because of the fashion in which organ blood supply is modified.1 Intercostal and lumbar arteries supplying the spinal cord and vertebral bodies are commonly involved in the dissection, although no figures are available as to the specific incidence. Involvement of these vessels has been implicated as the cause of spinal cord necrosis seen in this entity.2 The incidence of involvement apparently exceeds the number of cases in which spinal cord necrosis has been observed. Only eight cases of spinal cord necrosis in association with dissecting aneurysm have been previously reported in the literature, and no instance of vertebral body necrosis could be found.2-9

The present case demonstrates involvement of intercostal and lumbar arteries by dissecting hematoma in which there was massive infarction of the spinal cord and vertebral bodies.

Case Report

An 80-year-old man entered the hospital 4 hours after fainting while walking down a flight of stairs. The period of unconsciousness is not known, but was considered to be no more than 1 or 2 minutes. He later recalled a mild, fleeting, "cramp-like" pain in the anterior chest immediately prior to collapse. There was no incontinence of urine or feces, nor were tonic or clonic movements observed. Upon regaining consciousness, he felt "cold" and had the urge to defecate. He was able to reach the toilet without assistance. He denied chest pain after the episode. On advice of his physician, the patient reluctantly came to the hospital some 4 hours later.

Past history revealed a moderate deafness for 20 years. He specifically denied cardiovascular complaints.

On physical examination the blood pressure was 100/84, pulse 108, rhythm regular, temperature 99.8° oral, respirations 22 per minute. There was slight periorbital edema. The anteroposterior diameter of the chest was slightly increased, and breath sounds were distant. No cardiac murmurs were heard. Peripheral pulses were present and equal. There was two plus enlargement of the prostate. The remainder of the examination was normal.

Laboratory data disclosed a white blood cell count of 11,000, with a moderate shift to the left; hemoglobin 12 Gm. per 100 ml. hematocrit value 39 volumes per cent. Initial urinalysis revealed three plus albumin, one plus sugar and 0 to 15 white cells per high-power field. Subsequent urinalysis was normal. Serologic test (VDRL) was nonreactive. The blood urea nitrogen was 38 mg. per 100 ml., while the fasting blood sugar was 100 mg. per 100 ml. Acid phosphatase was 1.3 King-Armstrong units. The electrocardiogram on admission showed sinus tachycardia and nonspecific changes associated with myocardial disease.

At the time of admission, it was proposed that the episode of syncope, which had occurred earlier in the day, was associated with a paroxysmal arrhythmia. The patient was placed on bed rest; no specific therapy was instituted. On the following day he developed fever. Medium rales were heard at the posterior left lung base, and the pulse was found to be irregular and rapid. Electrocardiogram now revealed atrial fibrillation with rapid ventricular response. This change in the cardiac mechanism gave credence to the impression of transitory arrhythmia. There was, however, no other clear evidence of cardiac abnormality at this time. Chest x-ray showed minimal infiltrate in both lung bases. A diagnosis of bronchopneumonia was made, and the patient was placed on penicillin and given digitoxin. Sputum culture revealed no specific organism. There was good response to therapy, with return of temperature to normal and slowing of the pulse rate. The cardiac rhythm reverted to a sinus mechanism.

On the seventh hospital day, the patient experienced sudden, severe, nonradiating anterior chest pain while at stool. An electrocardiogram at this time recorded no change from previous records. Approximately 10 minutes after this chest pain, there was paraplegia with loss of all neurologic modalities from the level of the sixth thoracic

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Sagittal section through the posterior wall of the thoracic aorta showing dissecting hematoma and thrombus in adventitial and para-adventitial portion of intercostal artery.

Ochre-yellow infarcts with peripheral hemorrhage in the vertebral bodies of the thoracic region.

vertebra caudally. The remainder of the physical examination was unchanged. Lumbar puncture and myelogram done at this time were normal.

A diagnosis of dissecting aneurysm was made clinically, but anteroposterior and lateral tomograms of the chest failed to show clear evidence of aortic dissection. The risk of contrast aortography was considered unwarranted.

After this episode of chest pain and paralysis there was gradual deterioration. There was no further pain. The patient developed pneumonia terminally and died 33 days after the onset of paralysis.

At autopsy a single, large tear was located in the arch of the aorta just distal to and adjoining the orifices of the great vessels. From the tear a hematoma dissected proximally into the ascending portion of the arch, and distally into the thoracic and major portion of the abdominal aorta, chiefly through its posterior wall. The plane of dissection was through the external third of the medial layer and, although extensive, did not produce a thick hematoma. There was no external rupture or re-entry site. The large arteries were not damaged by dissection, but most of the intercostal and lumbar arteries had been torn from their intimal attachment, communicated with the dissecting hematoma, and contained early organizing thrombi in both the intimal and the adventitial portions (fig. 1).

There was massive anemic infarction of the bodies of the vertebrae from the fourth thoracic to the second lumbar. The ochre-yellow infarcts were sharply outlined by hemorrhagic borders (fig. 2). There was extensive infarction of the entire spinal cord distal to the midthoracic region and patchy involvement of the cervical area (fig. 3). The intervertebral disk showed no changes.

Discussion

Upon consideration of the blood supply to the spinal cord, one is surprised that there have been only eight reported cases of proved necrosis of the spinal cord as a complication of dissecting aneurysm of the aorta. Suh and Alexander pointed out that the anterior spinal artery in the midthoracic area is attenuated, and structures below this area depended, for the most part, on three or four anterior radicular branches, and five to eight posterior radicular branches of the intercostal and lumbar arteries for blood. These branches are not symmetrical, as are the intercostal and lumbar, nor do they arise from the same level in each individual. The posterior branches supply only the posterior portion of the posterior column and posterior horns. Therefore, the three or four anterior branches supply most of the cord substance below the midthoracic region. The largest of these is usually in the lower thoracic or upper lumbar region. Collateral circulation through the anterior spinal artery to the lower thoracic and lumbar area is precarious at best.

One would therefore expect involvement of the intercostal and lumbar arteries by a dissecting aneurysm to interrupt the blood supply to the lower thoracic and lumbar portions of the cord in almost every instance. Moersch and Sayre, however, reported seven cases in which considerable dissection of the intercostal vessels had occurred, but the spinal cord
revealed no lesions attributable to interference with its blood supply. They proposed that the disparity between the incidence of involvement of these vessels by dissection and resultant necrosis was due to the mechanical effects of the dissecting column.

Probably there is no single explanation for the variable response to this vascular insult. It seems reasonable, however, that involvement of these vessels will allow necrosis in structures supplied by them in terms of two variables: (1) whether or not an intercostal or lumbar artery bearing a significant anterior radicular branch is occluded and (2) the functional integrity of the collateral circulation in the particular individual.

This case also demonstrated necrosis of the vertebral bodies. In those cases in which spinal cord necrosis has been demonstrated histologically, other structures deriving their blood supply from the same source as the cord should similarly show necrosis. It should be noted, however, that in the reports of spinal cord necrosis, no mention is made of the vertebral bodies. It is possible that necrosis of these structures existed but was overlooked.

In all cases of dissecting aneurysm exhibiting neurologic signs and symptoms, the spinal cord and vertebral bodies should receive thorough study. Radiographic study during life of a segment of the vertebral column removed at postmortem did not disclose the infarcts in this case. These problems related to circulation assume added significance because surgical correction of dissecting aneurysm is a reality.\textsuperscript{13, 14}

Summary

A case of spinal cord and vertebral body necrosis as a complication of dissecting aneurysm of the aorta has been presented. Previous report of vertebral body necrosis complicating this entity could not be found. The incidence of intercostal and lumbar artery involvement in this condition is not known; a disparity appears between the incidence of involvement and necrosis of structures supplied by them. Factors believed to be important in assessing the significance of involvement of these vessels are mentioned. All cases of dissecting aneurysm in which there are neurologic signs should have thorough study of the spinal cord and vertebral bodies if they come to autopsy.

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References

7. Kalischer, O.: Aneurysma Dissecans der Aorta mit Paraplegie (Demonstration eines Pra-
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We have recalled . . . that science is the most powerful agency of change not only in the material world but also in the spiritual one; so powerful indeed, that it is revolutionary. Our Weltanschauung changes as our knowledge of the the world and of ourselves deepens. The horizon is vaster as we go higher. This is undoubtedly the most significant kind of change occurring in the experience of mankind; the history of civilization should be focussed upon it.—GEORGE SARTON. Horus: A Guide to the History of Science. Waltham, Massachusetts, 1952, p. 10.
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