Spinal Cord Infarction

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Vascular disease of the spinal cord occurs with less frequency than in the brain. Its manifestations are similar, however, and are often abrupt in onset, dramatic in scope, and frequently disabling.

For a better understanding of the clinical findings, it is worthwhile to review the vascular anatomy of the spinal cord. The major blood supply of the cervical cord is derived from the anterior spinal artery, which is formed at the level of the foramen magnum by two branches of the intracranial vertebral arteries. The anterior spinal artery supplies the ventral surface of the medulla and also the spinal cord. This artery is often considered a continuous structure, although at times this is not the case, particularly in the thoracic cord. Contributing to the anterior spinal system are a group of seven to ten radicular arteries. In the cervical area these are derived from the cervical branches of the vertebral arteries and the ascending cervical artery. In the rostral thoracic cord, contributions arise from the deep cervical and ascending cervical arteries. The radicular arteries supplying the middle and lower thoracic cord are less prominent. The lower thoracic and lumbar cord is supplied by the great anterior radicular artery of Adamkiewicz, which originates on the left and joins the anterior spinal system at the level of the ninth through the second lumbar cord segments. The lumbosacral cord is supplied by branches of the hypogastric and sacral arteries.

The irregular augmentation of the anterior spinal artery system results in watershed areas that may be vulnerable to hypoperfusion, most notably in the thoracic area. The anterior spinal artery gives rise to sulcal vessels, which alternately penetrate the cord to the right or left, and this system perfuses the anterior two thirds of the spinal cord.

The paired posterior spinal arteries supplying the posterior or dorsal part of the spinal cord originate from the vertebral or posterior inferior cerebellar arteries. The posterior spinal artery system is augmented by 10–20 posterior radicular vessels, which are more numerous than the seven to ten arteries of the anterior radicular system. The dorsal root ganglia and anterior roots are supplied by segmental arteries derived from the vertebral arteries, aorta, and iliac vessels.

The arterial system of the peripheral rim of the cord consists of dorsal and ventral penetrating vessels and a peripheral anastomotic ring. Collateral circulation here is adequate. Within the interior of the cord there are no anastomoses, and the penetrating vessels are essentially end arteries or arterioles (Figure 1).

The venous supply generally parallels that of the arterial vessels. In contrast to the paired posterior spinal artery arrangement, there is a single median posterior spinal vein. The anterior spinal vein receives contributions from the sulcal and circumferential veins. In general, the territories supplied by the anterior and posterior veins correspond to those of the anterior and posterior arteries, although the boundaries of the former appear to be somewhat broader.

Total cerebral blood flow of the human brain is 50 ml/min/100 g. Spinal cord total flow varies, depending on the particular species and the area of cord studied. In monkeys total flows in cervical, upper thoracic, and lumbar areas were 14.9 ± 1.4, 10.4 ± 0.8, and 19.7 ± 1.2 ml/min/100 g, respectively. As in brain, the gray matter of the cord is more highly perfused than the white, and the flow to the gray matter is fivefold that to the white. In all species lowest total flow is observed in the thoracic cord. In part, this difference has been attributed to the relative decrease in gray matter at this level.

Spinal cord blood flow is maintained by autoregulation. Hypoxia and hypercapnia increase blood flow while the reverse is the case during hypocapnia. Local increases in metabolic activity are paralleled by increases in cord blood flow.

Tempo of Cord Vascular Disease

The terms used to describe the tempo of brain vascular disease are also applicable to the spinal cord. There may be transient ischemic attacks with brief neurological deficit and recovery, probably due to emboli arising from the heart or the aorta and its branches.

Ischemic attacks may also occur in the setting of coarctation of the aorta and an associated steal syndrome. Intermittent neurological deficits have been reported with spinal cord arterial venous malformations. In addition to the shunting of blood to the lower-resistance venous part of the malformation, the enlarged vessels may cause fleeting deficits by compression. Dejerine described transient neurological deficits resulting from impaired flow as a consequence of arterial stenosis. Intermittent symptoms also occur with lumbosacral stenosis.

In some situations the neurological loss of function may be progressive and take place over hours. More
Anterior Spinal Artery Occlusion

Spiller in 1909 provided the classical description of this syndrome. In his report, the underlying cause was syphilis. Other causes of anterior spinal artery ischemia and occlusion are listed in Table 1.

Most episodes probably occur as the result of atherosclerosis of the aorta and its branches. The area of infarction may be remote from the site of the vascular occlusion in an area of the cord that was most vulnerable at the time the local circulation became insufficient.

The clinical picture of anterior spinal artery occlusion includes initial flaccid paralysis and depression or absence of the muscle stretch reflexes. With the passing of time the reflexes become hyperactive, and Babinski responses are elicited. Muscle atrophy of the upper extremities may occur when the infarction involves the cervical cord. A sensory level is found below which pain and temperature are not perceived; however, there is preservation of vibratory, motion, and position, and tactile stimuli. Bowel and bladder paralysis commonly occurs.

Posterior Spinal Artery Infarction

Ischemia and infarction in the territory of the posterior spinal arteries are uncommon. Hughes reported a patient who sustained posterior cerebral and posterior spinal artery thrombosis after an intraspinal injection of phenol; abrupt onset of weakness, bladder paralysis, and sensory loss were noted. The clinical findings consist of loss of sensation at the level of the injury and loss of associated segmental reflexes. Below the level of involvement, there is decreased appreciation of proprioceptive and vibratory stimuli.

Venous Infarctions

Venous infarctions also occur infrequently and are rarely diagnosed with certainty in life. Back pain and lower extremity weakness are the most common symptoms. Paralysis may be progressive over hours or days. Bowel and bladder paralysis invariably occurs. Kim et al in a recent review could not differentiate with certainty between hemorrhagic, nonhemorrhagic, and embolic venous infarction on clinical grounds. Hemorrhagic venous infarctions were associated with marked pain at the time of onset and progressed rapidly; survival time was relatively short. Nonhemorrhagic infarctions were less often painful and evolved slowly. Survival time was longer than in hemorrhagic infarction. Embolic venous infarction was associated with pain of abrupt onset. The neurological deficits were less symmetrical in their extent than in the other two varieties.

The potential association of malignancy and venous spinal cord occlusion is well recognized, particularly in the case of pancreatic neoplasm.

Table 1. Causes of Ischemia and Infarction in Territory of Anterior Spinal Artery

<table>
<thead>
<tr>
<th>Condition</th>
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<tbody>
<tr>
<td>Syphilis</td>
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<tr>
<td>Giant cell arteritis</td>
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<tr>
<td>Polyarteritis</td>
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<tr>
<td>Sickle cell anemia</td>
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<tr>
<td>Intervertebral disc herniation</td>
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<tr>
<td>Temporary cervical subluxation</td>
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<tr>
<td>Mitral valve disease and emboli</td>
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<tr>
<td>Atherosclerosis of aortic vessels and branches</td>
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<tr>
<td>Hypotension</td>
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<tr>
<td>Cardiac arrest</td>
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<tr>
<td>Traumatic rupture of aorta</td>
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<tr>
<td>Dissection of ascending aorta</td>
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<tr>
<td>Angiography</td>
</tr>
<tr>
<td>Therapeutic renal artery embolization</td>
</tr>
<tr>
<td>Surgery for aortoiliac occlusive disease</td>
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Laboratory Investigations

Abrupt neurological deficit of spinal cord origin requires prompt investigative measures, including basic laboratory tests to evaluate the presence of underlying infection and alterations in blood coagulation. It is essential to determine as quickly as possible whether a focal process is causing spinal cord compression. A chest x-ray and complete spinal films followed by myelography are indicated. Tomography of the spinal canal and magnetic resonance imaging may be helpful. In some clinical situations these newly developed diagnostic tests may not be available, and traditional myelography is essential to determine whether spinal cord compression is present.

Myelography will help demonstrate the presence of malformations of the cord. Angiography to demonstrate stenosis of specific radicular arteries requires selective arteriography. The procedure is time consuming and may be potentially dangerous because of the amount of contrast medium needed to demonstrate the arterial lesion. At present there is no surgical treatment for the stenosed arteries that might be demonstrated by angiography.

The spinal fluid should not be ignored. Analysis of the spinal fluid may help establish central nervous system infection as well as recent or past spinal subarachnoid hemorrhage. In addition to the serology of the spinal fluid, cytological studies should be done to determine whether tumor cells are present. If it is concluded that the event is of vascular origin, then studies should be performed to establish a potential source of emboli, particularly within the heart.

Treatment

There are no good clinical studies on specific therapeutic regimens in patients who have sustained spinal cord ischemia or infarction. If the source is judged to be embolic, the use of anticoagulant or antiplatelet drugs should be considered.32 As with other forms of vascular disease, the usual risk factors should be sought for and treated: hypertension, heart disease, and diabetes mellitus.

In addition to rehabilitative measures, meticulous care must be given to bowel and bladder function, as well as to the skin.

In contrast to the clinical situation, many laboratory investigations have focused on models of hypoperfusion and trauma to help our understanding of pathology and possible therapy. Although several interesting observations have been made, their therapeutic applicability in man has yet to be established. These observations include the protective effect of hypothermia on spinal cord function and the use of methylprednisolone after ischemic injury.31 Both hypothermia and steroids are effective in animals, but treatment must be undertaken within a very short time after injury to achieve any protective effect.

Many studies have focused on treatment prior to injury. Corticosteroids do not increase spinal cord blood flow, and their positive effect is attributed to the protective action of steroids on cell function or on cell membranes. Corticosteroids also help reduce the presence of free radicals, which occur after ischemic injury.33

In animals the opiate antagonist naloxone31,32 has been used to prevent and reduce neurological deficits. Naloxone increases blood flow to the spinal cord and may also prevent entry of calcium into nerve cells after injury. Vacanti and Ames32 showed that pretreatment with hypothermia and magnesium had a protective effect, reducing neurological deficit after inducing cord ischemia. The precise role of magnesium is unclear. It has been postulated that pretreatment with magnesium may prevent the release of certain neurotransmitters and may also alter some of the deleterious effects of calcium that occur in the setting of ischemic injury.

Prognosis

Recovery after spinal cord infarction is extremely variable and for the most part tends to be dependent on the severity of the initial deficit. Many patients have the potential to recover and function independently.34

Summary

Spinal cord infarction occurs infrequently and may have diverse causes. It is necessary to establish whether an intrinsic or extrinsic lesion is responsible for the impaired cord function. Although therapy is limited at this time, the long-term prognosis is not necessarily unfavorable.

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