Spinal cord embolism

LIONEL WOLMAN AND PETER BRADSHAW

From the Departments of Neuropathology and Neurology, United Sheffield Hospitals

Vascular accidents involving the spinal cord, in contrast to the brain, are extremely rare. Damage to the spinal cord from verified emboli seems to be an even greater rarity and no examples were found by Blackwood (1958) in a series of 3,737 post-mortem reports from the National Hospital, Queen Square, London. Embolic myelomalacia has been reported in bacterial endocarditis and other forms of heart disease such as mitral stenosis and coronary occlusion. When caisson disease and air embolism are excluded, the paucity of the literature on the subject indicates that spinal embolism is very uncommon, and in some reports confirmation of the emboli and the site of their impaction has been lacking. The purpose of this paper is to record two further examples of the syndrome and to present details of the nature and origin of the emboli and the lesions produced.

CASE REPORTS

CASE 1 T.D., a man aged 59, early in March, 1966, noticed progressive weakness first of the right and, a few days later, of the left lower limb. The disability was slight until six weeks later when he awoke with paralysis of both lower limbs and numbness up to the waist. The following morning he was unable to pass urine. He had suffered from a cough dating from bronchitis 20 years previously but otherwise the past and family histories contributed nothing. On May 2, he was admitted to hospital elsewhere and transferred to the neurological department on 26 May 1966.

On examination the spine, cranial nerves, and special senses were intact. Inconspicuous but coarse fasciculation was observed over the shoulder girdles and arms, but the upper limbs were otherwise normal. There was flaccid paralysis of both lower limbs. The deep tendon reflexes were normal in the upper and depressed in the lower limbs, the abdominal responses were absent and both plantars were extensor. Vibration sense was absent below and including the pelvis and joint position sense was absent in the toes and defective in the ankles. There was a cutaneous sensory impairment to pin prick, temperature and touch below the second dorsal segment and including the sacral area on both sides. There was retention of urine which necessitated the use of an indwelling catheter and numerous deep pressure sores over the sacrum, buttocks, and legs. General examination was

unremarkable and the blood pressure was 190/90 mm. Hg.

A plain radiograph of the chest revealed evidence of an old right basal pleurisy. Radiographs of cervical and dorsal spines were normal. A full blood count was normal but the erythrocyte sedimentation rate (E.S.R.) was 50 mm./hr. The lumbar cerebrospinal fluid pressure and manometric observations were normal and the fluid contained no white cells, 7 red cells/c.mm., protein 35 mg./100 ml.; Lange no change. The Wassermann reaction (W.R.) was negative in the blood and cerebrospinal fluid. A myelogram showed no abnormality between the lumbar sac and the foramen magnum.

In view of the negative results of investigation, it was thought that the patient was probably suffering from the effects of metastatic carcinoma, possibly arising from the bronchus. The patient's condition deteriorated and the course was complicated by both pulmonary and renal infections and by anaemia. He died on 10 August 1966 within five months of the onset.

Necropsy findings The body was that of a thin, wasted male whose appearance was older than the stated age. Extensive pressure sores were present over the sacrum, lateral aspects of both thighs and left heel. The left pleural cavity was obliterated by old dense adhesions. There was a severe terminal bilateral bronchopneumonia. The heart was of normal size and appearance and the coronary arteries showed minimal atheroma. The whole of the aorta was extremely atheromatous with many large erosions of the intimal surface on several of which fleshy fibrin thrombus had been deposited (Fig. 1). The lower abdominal aorta was most severely affected but the ascending aorta, the arch adjacent to the origin of the left common carotid artery, and the posterior aspect of the descending aorta round the openings of the lower intercostal and lumbar branches were similarly involved. Both common carotid arteries were very atheromatous and the origins of both internal carotid arteries were stenosed by fibrin thrombus deposited on severely atheromatous areas. The subclavian and vertebral arteries appeared healthy. The renal, coeliac, and mesenteric arteries were widely patent and only slightly atheromatous. The kidneys showed no gross abnormality and the urinary tract was free from infection. The other viscera appeared healthy.

The brain (weight 1,455 g.) showed bilateral frontal cortical atrophy with widened sulci. The vessels at its base showed only patchy atheroma at their junctions. There was slight *ex-vacuo* dilatation of the ventricles. The spinal cord showed slight swelling in the upper thoracic

FIG. 1. Severe diffuse atheroma of the aorta with erosions in the arch and abdominal part ($\times \frac{1}{4}$ th).

region and there was congestion of the pial vessels on the surface. The pia arachnoid was not thickened and there was no evidence of tumour infiltration or of any metastatic deposits in the extradural tissue or in the vertebral bodies.

Histology Although no lesions were recognized in the brain on naked-eye examination, several scattered minute areas of old softening were found in the routine sections. These were situated in the cortex of the frontal lobe and in the cortex on either side of the central sulcus, in the head of the caudate nucleus and in the putamen on the right side. They consisted of areas of dense gliosis, wedge shaped in the cortex, in which an occasional lymphocyte and histiocyte could be recognized. No thrombosed vessels were seen in relation to these infarcts.

Sections were examined from each segmental level of the cord and the upper thoracic segments were cut serially in view of the history of a sensory level at D.2. The sections were stained by haematoxylin and eosin, Nissl, Mallory's phosphotungstic acid haematoxylin, Masson's trichrome combined with Verhoef's elastic tissue, Holmes' silver impregnation, and Weigert's myelin method. In the cervical region and D.1 segment there was ascending degeneration in the posterior and antero-lateral columns. At D.2 there was a wedge-shaped area of necrosis involving the right anterolateral column of white matter extending from the surface into the superficial part of the anterior horn (Fig. 2). At D.3 the area was of similar shape but was separated from the anterolateral surface by a thin band of intact tissue. At D.4 the area was much larger, extending dorsally in the lateral column, and was accompanied by a transversely elongated area of necrosis in the centre of the posterior columns. This latter area became much more prominent at D.5, extending across the right posterior horn to link

FIG. 2. Transverse section of right half of spinal cord at D.2 showing necrotic area in anterolateral column. (myelin $\times 15$).

up with an elongated area in the right lateral column. At D.6 there were three discrete areas of necrosis involving a large wedge-shaped area in the right anterolateral column, a transversely elongated area involving the posterior columns and right posterior horn and a small wedge-shaped area in the dorsal part of the left lateral column. At D.7 the area in the right anterolateral column had become small and wedge-shaped beneath the surface, whilst extending across the posterior columns, left posterior horn, and left lateral column was a large crescentic area of necrosis separated from the surface by a thick band of intact white matter (Fig. 3). This crescentic-shaped lesion also involved D.8 becoming smaller and split into posterior and lateral components (Fig. 4). These extended into the D.9 segment only on the left side, whilst at D.10 the necrosis was confined solely to the left lateral column. Below this level the cord was intact apart from descending degeneration in the dorsal half of the lateral columns. The infarcted areas had a similar appearance consisting of pale-staining, vacuolated necrotic tissue in which were numerous histiocytes and scanty lymphocytes. In only one small intramedullary artery in the necrotic area on the right side at D.6 was fibrinous embolic material seen in the lumen of the vessel. The anterior and posterior spinal arteries and their branches seen in all these sections were patent, healthy, and free from atheroma.

The aorta was removed at necropsy together with the paravertebral tissues. Several of the pairs of intercostal and lumbar arteries were dissected out from their origins in the aorta and sectioned serially, some transversely and others longitudinally. Although the lumbar arteries originated in severely eroded areas of the aorta so that it was difficult to see their openings with the naked eye, the sections surprisingly showed the lumen to be widely patent and the atheromatous process either did not extend into the vessel or if it did it was minimal. In all the sections examined, however, embolic atheromatous material was seen lying free in the lumen or occasionally loosely attached to the wall (Fig. 5). Most of this material was eosinophilic and structureless, staining positive with P.A.S. and Luxol fast blue, and resembling in its appearance the soft grumous material lying on the surface of the eroded areas in the aorta. A few small cholesterol crystal clefts were seen in the embolic material (Fig. 6).

Although the kidneys appeared healthy, microscopically there were several minute wedge-shaped cortical infarcts and small arteries in relation to these contained cholesterol emboli (Fig. 7).

The subacute onset of paraparesis in a middle-aged man with a chronic cough, followed after six weeks by flaccid paralysis of the lower limbs and a sensory level over the trunk, suggested metastatic carcinoma. However, no primary carcinoma was detected, plain radiographs and myelography excluded spinal compression, and the normality of the cerebrospinal fluid pointed away from subacute necrotic myelitis.

At necropsy the detection of intense atheroma of the aorta suggested that it had blocked the

FIG. 3. Transverse section of D.7 showing crescentic area of infarction extending round spinal cord (myelin $\times 7$).

FIG. 4. Transverse section at D.8 showing necrotic areas in lateral and posterior columns (myelin $\times 7$).

FIG. 5. Longitudinal section of lower intercostal artery showing masses of atheromatous material lying free in the lumen (haematoxylin and eosin $\times 150$).

FIG. 6. Section of radicular branch of the upper lumbar artery showing a few small cholesterol crystal clefts in atheromatous embolic material in the lumen (haematoxylin and eosin $\times 150$).

openings of the lumbar or intercostal arteries or had extended into these branches. However, microscopy showed that this was not so and the walls of these small vessels, together with the anterior and posterior spinal arteries, were healthy. Instead, numerous small atheromatous emboli lay in the lumina of the lumbar and intercostal arteries. The spinal cord contained many small areas of infarction between D.2 and D.10. At the upper and lower limits these were wedge-shaped and confined to the anterolateral columns. Over the remainder of the affected cord, areas of infarction were crescentic and lay in the watershed territory between tissues supplied by the central branches of the anterior spinal artery and the penetrating branches of the peripheral circumferential pial vessels, extending between the posterior and anterior spinal arteries (Hassler, 1966). As both these spinal arteries originate from the radicular arteries, especially in the thoracolumbar region, atheromatous emboli would tend to be held up at the proximal origin and at the distal bifurcations of the radicular arteries, before migrating distally as has been demonstrated in the cerebral circulation. Only one small intramedullary vessel was found to contain embolic material in the present case but it is likely that showers of emboli had occurred, resulting in ischaemic myelopathy. Evidence of extraspinal embolism was detected in the brain and kidney.

CASE 2 A.B., a woman aged 65, early in March 1961 noticed progressive weakness of both lower limbs, unsteadiness of gait, and a sensation that she was walking on cotton wool. In July, she complained of slight clumsiness and weakness of the right hand without sensory disturbance. On 7 August, after a fall, she experienced urgency and hesitancy of micturition. There was no relevant past or family history and she was admitted to the neurological department on 12 August 1961.

On examination the spine, cranial nerves, special senses, and upper limbs were intact. There was relative weakness of both lower limbs without alteration of tone. The deep tendon reflexes were normal but the abdominal responses were absent and the plantars were equivocal. Vibration sense was absent in the toes, present at the ankles, and the threshold for joint position sense was elevated in the great toes. There was relative hypalgesia, thermalgesia, and hypaesthesia below the fourth dorsal segment and including the sacral area on both sides. General examination was unremarkable and the blood pressure was 150/80 mm. Hg.

Plain radiographs of the chest, cervical, dorsal, and lumbar spines were normal, together with a full blood count, E.S.R., urine analysis, and the serum electrolytes, urea, plasma acid, and alkaline phosphatases. There was a histamine-fast achlorhydria but the serum B₁₂ level was 466 $\mu\text{g.}/100$ ml. On 3 September painful swelling of the left calf was detected which was attributed to a

FIG. 7. Section of kidney showing cholesterol emboli in a small artery adjacent to a glomerulus (haematoxylin and eosin $\times 150$).

deep vein thrombosis. This was treated by rest and a pressure bandage but no anticoagulants were given. On 19 September, the pain and swelling had subsided. Re-examination then showed a severe spastic paraparesis with increased deep tendon reflexes and bilaterally extensor plantar responses. There was a dense cutaneous sensory loss, to all modalities, below the level of D.4. The lumbar cerebrospinal fluid pressure and manometric observations were normal, and the fluid contained no white cells, 7 red cells/c.mm., protein 25 mg./100ml.; Lange no change. The Wassermann reaction was negative in the blood and cerebrospinal fluid. A myelogram showed no abnormality between the lumbar sac and the foramen magnum. On 24 September, the patient had a major fit, collapsed, and never regained consciousness. She died on 26 September, six months after the onset of symptoms.

Necropsy findings The body was that of a thin, wasted female whose appearance was older than the stated age. Small superficial pressure ulcers were present over the sacrum and the left hip. A pulmonary embolus which had originated in the left calf was a terminal factor in the death. A mass of fibrin thrombus was adherent to the medial wall of the left auricle near its apex. The coronary and carotid arteries showed patchy atheroma but there was very severe atheroma of the aorta, especially in its lower abdominal part extending into the iliac vessels. Several small cysts were present in the cortex of the kidneys, the surface of which was granular. Some small uterine fibroids and a large yellow infarct of the lower half of the spleen were found.

FIG. 8. Section from left atrium showing fibrin deposit on myxoma (haematoxylin and eosin $\times 150$).

FIG. 9. Transverse section at C.4 showing necrotic area in right anterolateral column with ascending degeneration in posterior columns (myelin $\times 7$).

FIG. 10. Transverse section at D.1 showing necrosis in right lateral column extending into anterior horn (myelin $\times 7$).

FIG. 11. Transverse section at D.7 showing large infarct extending across the midline (myelin $\times 7$).

The brain (weight 1,300 g.) was swollen, with flattened convolutions and narrowed sulci. The vessels at its base showed severe patchy atheroma at their junctions. The upper part of the brainstem appeared swollen, softened, and haemorrhagic, and these haemorrhages had extended into the tegmentum of the pons on either side in its lateral part. The lateral and third ventricles were narrowed but showed no displacement. There were some small calcified plaques in the pia arachnoid over the posterior aspect of the lumbosacral cord but no other gross abnormalities were recognized.

Histology A section of the mass of fibrin thrombus attached to the wall of the left auricle showed that this had been deposited on a small myxoma growing on the medial wall of the auricle (Fig. 8). There was no evidence of infarction of the septal wall in this region. Several of the splenic vessels in the margin of the infarct were occluded with fibrin thrombus.

There was haemorrhagic necrosis of the tectum and tegmentum of the midbrain with numerous small haemorrhages scattered throughout the necrotic tissue in which were many foci of polymorphs, mainly around vessels. The haemorrhages had ruptured into the aqueduct. The basilar artery appeared healthy, patent and free from atheroma but several of the small perforating vessels had necrotic walls. The haemorrhages extended into the dorsal half of the upper pons and upwards to the subependymal tissue in the walls of the third ventricle. Small recent wedge-shaped infarcts were seen in the cortex of the cerebellum, right insula, left frontal and temporal lobes and right central region. No cellular reaction had occurred in these areas and no occluded vessels were seen.

The spinal cord showed several discrete areas of infarction of longer duration. At C.4 there was an area involving the right anterolateral column extending from the ventral nerve roots into the anterior horn of grey matter (Fig. 9). Many histiocytes were present between proliferated capillary vessels in the necrotic region. Ascending degeneration was prominent in the posterior columns. At C.6 a wedge-shaped area of necrosis was present in both lateral columns. A much larger wedge-shaped area of necrosis of more recent origin was present in the right lateral column at D.1 (Fig. 10) which extended into the anterior horn at D.2-3 and tapered out at D.4. At D.7 there was a large infarct involving the lateral and ventral column on the right side as well as the anterior horn and extending across the ventromedian fissure to include the medial part of the left ventral column and left anterior horn (Fig. 11). Similar but less extensive involvement was present at D.8. The remaining thoracic segments were intact, apart from D.12 (Fig. 12), where both lateral columns and the ventral part of the posterior columns contained infarcts which extended on the left side into L.1 (Fig. 13). In the lower lumbar segments the infarction was confined to the medial part of the ventral columns and anterior horns as well as the ventral part of the posterior columns in the territory supplied by the anterior spinal artery (Fig. 14).

The anterior and posterior spinal arteries were patent and free from atheroma. Several small intramedullary vessels in the infarcts had necrotic walls and numerous

FIG. 12. Transverse section at D.12 showing bilateral areas of infarction extending across the ventral part of the posterior columns (myelin $\times 7$).

FIG. 13. Transverse section at L.1 showing infarcted area extending across the left posterior horn (myelin $\times 7$).

FIG. 14. Transverse section at L.3 showing infarction mainly in the territory supplied by the anterior spinal artery (myelin $\times 7$).

FIG. 15. Section of spinal cord in area of infarction at L.1 showing organizing thrombus in the lumen of a small intramedullary artery with intense perivascular lymphocytic reaction and numerous histiocytes in surrounding tissue (haematoxylin and eosin $\times 285$).

lymphocytes and occasional polymorphs cuffed many of these vessels, some of which were filled with fibrin thrombus or with a cellular mass, interpreted as organization by endothelial reaction rather than by tumour cell emboli (Fig. 15).

This patient developed weakness and paraesthesiae of both lower limbs and, five months after the onset, exhibited a flaccid paraparesis with a sensory level at D.4. Investigations failed to establish a diagnosis. The condition deteriorated, spasticity developed in the lower limbs, and she died six months from the commencement of symptoms.

Although the atheroma of the aorta was severe in this case also, especially in the lower abdominal portion, and may have contributed to the spinal cord damage, the myxoma of the left auricle was probably the major source of emboli. Unfortunately the intercostal and lumbar arteries were not examined but the presence of multiple infarcts in the cerebral and cerebellar cortex, brainstem, cervical cord, and spleen, as well as in the thoracolumbar segments, supports the cardiac origin of the emboli. No myxomatous material was recognized in vessels

in relation to the infarcts and it may well be that only the fibrinous deposit on the tumour acted as embolic material. Many of the vessels in or near the infarcts had become necrotic and haemorrhage into the surrounding tissue in the case of the brainstem infarct had resulted in death.

The spinal cord infarcts were much more discrete, widely separated, and diffusely scattered than in the previous case. In addition they were more variable in their appearance, age, and situation. Thus in the cervical and upper thoracic cord they involved wedge-shaped areas in the lateral columns, whereas in the lower lumbar segments the infarct was confined to the anterior spinal artery territory. Only at the thoracolumbar junction had the infarction occurred in the arcuate watershed territory.

DISCUSSION AND CONCLUSIONS

The symptoms and signs relating to two patients with paraparesis due to spinal embolism have been presented. Although certain features were common to both, nothing came to light during life by which the correct diagnosis could have been achieved. Their ages were 59 and 65 respectively and neither displayed clinical evidence of significant hypertension, atheroma, or cardiac disease. Symptoms commenced insidiously and consisted of weakness of the lower limbs and impairment of urinary sphincter control. In case 2, there was progressive paraparesis; in case 1 there was sudden flaccid paraplegia within six weeks of the onset. Initial examination of both patients showed a flaccid paraparesis, without any increase in the deep tendon reflexes, and a sensory level extending to the upper dorsal region. The striking absence of spasticity, despite the presence of severe weakness, might at first have been attributable to spinal shock in case 1, but this feature remained throughout the course of the illness. It suggested the presence of a lesion extending over many segments of the spinal cord that had interrupted reflex arcs concerned with muscle stretch. Investigations served only to exclude more common pathologies; there was no evidence of primary carcinoma or of metastases; spinal compression was ruled out by the negative myelogram; and the normality of the spinal cerebrospinal fluid pointed away from a diagnosis of subacute necrotic myelitis. It is possible that an aortogram might have shown a reduction in the number of segmental vessels but, even with hindsight, it is difficult to determine how the nature of the vascular occlusions could have been revealed.

The concept that atheromatous material may become embolic has been attributed to Panum (1862), who quoted the gross necropsy findings in

the case of the Danish sculptor, Thorwaldsen, and ascribed the sudden death to rupture of an atheromatous plaque in a coronary artery with occlusion of that vessel. No microscopic confirmation was recorded. In recent years microscopic proof of the occurrence of atheromatous emboli in almost every organ of the body, especially the abdominal viscera, the lower limbs, and the brain, has been demonstrated. The interest and thoroughness of the investigator appear to be important factors in determining where such lesions are found. Mention of the spinal cord, apart from a few exceptions, is curiously lacking. Périer, Demanet, Henneaux, and Nunès Vincente (1960) described two examples of necrosis of the spinal cord in the territory supplied by the posterior spinal arteries. In the first the state of the aorta was not mentioned, but in the second, a man of 65, obstruction of the posterior spinal arteries by emboli of atheromatous material from a thoracic aortic aneurysm was demonstrated. Soloway and Aronson (1964) reported on 16 cases of atheromatous emboli to the central nervous system. In two of these, emboli were observed in vessels supplying the spinal cord though there was no associated myelomalacia. However, only three routine sections of the cord were examined.

Chronic myelopathy is seldom recognized as a consequence of aortic atheroma (Wells, 1966) but the condition may be commoner than these two reports indicate. This is suggested by the examples collected by Corbin (1961) and by Gruner and Lapresle (1962). Garland, Greenberg, and Harri-man (1966) also raised the possibility of an embolic causation of the necrosis of the spinal cord in their cases where it was associated with aortic disease but they were unable to prove this. They stressed the importance of examining thoroughly the full extent of the blood vessels supplying the spinal cord from the aorta in addition to the spinal arteries and their branches in the leptomeninges.

Severe atheroma of the aorta, especially in its abdominal part above the bifurcation, is a common condition whereas atheromatous embolism is apparently relatively rare. Several explanations could account for this discrepancy. Smaller emboli may be without clinical significance or they may migrate through the vasculature in a solitary fashion and larger leptomeningeal arteries may dilate in response to emboli contained therein, as has been observed by Luessenhop, Gibbs, and Velasquez (1962) in the brain. Further, the long and tedious examination of the spinal cord and the tracing of its blood supply from the aorta which is required to prove the embolic process has deterred many from comprehensive necropsy study. The other factors concern difficulties in recognizing the embolic material which

is eosinophilic, grumous, and structureless, resembling fibrin. It may not contain definite cholesterol crystal clefts or they may be most conspicuous. The foreign body giant cell reaction to these crystals usually disappears after a few days and the reaction which persists is an intense inflammatory reaction in the vessel wall and extending into the surrounding tissue, in which eosinophils predominate. In the spinal cord this panarteritis could be easily misinterpreted as a form of myelitis.

Thus if atheromatous embolization of the spinal cord can occur in cases of severe aortic atheroma, it ought to be more commonly found and may well account for the slowly progressive spinal cord syndromes found in older patients. If the watershed territory is mainly affected then unilateral or, more likely, bilateral, motor or sensory symptoms may ensue.

Involvement of the nervous system by emboli from cardiac tumours is a much less common event owing to the rarity of these growths, which are predominantly myxoma arising in the left atrium. Thus in a recent review, Joynt, Zimmerman, and Khalifeh (1965) were able to collect from the literature 11 cases of cardiac tumours with cerebral emboli of tumour fragments and to add one of their own. Six additional cases with cerebral infarcts but no definitely identified tumour emboli were also found. In our second case multiple embolic softening were found in the brain and spinal cord as well as in the spleen. No tumour fragments were found in relation to these but several vessels were occluded with fibrin thrombus. This was not surprising in view of the large amount of clot deposited on the surface of the myxoma. Progressive thrombosis of blood with organization on the surface of these tumours is a common occurrence so that many have been regarded as organized mural thrombi. The necrotic damage to vessel walls in most of the infarcts was in striking contrast to the first case. The associated perivascular cellular infiltration resembled that seen in myelitis, but it appears to be a non-specific response as vasculitis has been reported with other types of embolic material such as cholesterol and fibrocartilage (Feigin, Popoff, and Adachi 1965).

SUMMARY

A clinical-pathological study of two patients with spinal cord embolism is described.

In the first, the embolic material originated in severe eroding atheroma of the aorta and consisted mainly of grumous material containing some cholesterol crystals. It was found in the intercostal and lumbar arteries which were themselves free from

atheroma. It is suggested that owing to the frequency of aortic atheroma compared with the infrequent finding of atheroma of spinal arteries this mechanism may be more prevalent in producing chronic ischaemic myelopathy.

In the second case the emboli consisted of fibrin originating from a mass deposited on a myxoma of the left atrium.

Several similar patterns of infarction in the spinal cord were seen in both cases. They consisted of a wedge-shaped area extending in from the lateral surface, or a central area incorporating both ventral columns, but a crescentic area in the watershed territory between anterior and posterior spinal arteries was the most typical. It would account for both the motor and sensory aspects of the syndrome.

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