

Post-traumatic syringomyelia

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Abstract

Post-traumatic syringomyelia was previously thought to be an infrequent but serious sequel to spinal cord injury. Clinical and CT studies have shown an incidence of between 1% and 5%, but more recently MRI has suggested an incidence of up to 22%. Twenty spinal cords have been examined after death from two days to 43 years after injury. Four had syrinxes, 20% of the series, approaching the incidence found by MRI. The acute and chronic pathological changes after trauma are described. Post-traumatic syringomyelia seems to develop from cores of necrotic tissue (myelomalacic cores) rather than lysis of haematoma. The mechanism of extension of syrinxes remains unexplained.

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Post-traumatic syringomyelia is a troublesome complication of severe spinal cord trauma. The exact incidence remains unknown.

Studies based on clinical examination and spinal CT have shown an incidence of between 1.1 and 4.5%,^{1,2} but since the introduction of MRI, incidences of 12-22% have been recorded.³⁻⁵ Few cases have been examined after death, and in the largest series published an incidence of 17% was recorded.⁶

We were able to find reports of only 35 detailed postmortem examinations in post-traumatic syringomyelia.^{1,6-9}

We present the pathological findings in 20 spinal cords examined between two days and 43 years after severe spinal injury. Four of these cases (20%) had cysts extending for at least two segments from the site of original trauma.

Cases and methods

Spinal cords were obtained from a single spinal injury unit over a period of six years. After fixation they were examined macroscopically and blocks were taken from the site of trauma and from multiple levels rostral and caudal to the lesion. Sections were stained with haematoxylin and eosin, luxol fast blue and cresyl violet, phosphotungstic acid haematoxylin, and, in a few selected cases, with the Marchi method.

Results

Seven cervical, nine thoracic, and three lumbar cord lesions were found and injury was at multiple levels in only one case. The table gives brief clinical details.

Four cases had cystic lesions extending for at least two segments from the site of trauma. These were seen in patients surviving from six weeks to 34 years after trauma and who were

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Nature of injury and survival time

Case No	Sex	Age at death	Survival since injury	Nature of injury	Main pathological findings in spinal cord
1	M	50	2 days	RTA. Fracture dislocation T2	Oedema. Neuronal shrinkage.
2	M	28	2 days	Rugby football injury. Fracture dislocation C4-5.	Oedema. Bilateral vertebral artery thrombosis.
3	F	30	5 days	RTA. Hyperextension injury. No direct trauma to spine or cord.	Bilateral dissecting aneurysms of vertebral arteries—see text.
4	F	44	15 days	Fall. Fracture T10.	Macrophage and capillary proliferation at site of injury. Haemorrhagic anterior spinal infarct two segments above injury.
5	M	18	6 weeks	RTA. Fracture dislocation T3-4. Incomplete paraplegia below T5.	Syrinx C5-T8—see text.
6	M	67	3 months	Fall. Fracture dislocation C5-6.	Syrinx C4-C7—see text.
7	M	55	5 months	RTA. Fracture dislocation C5-6. Incomplete paraplegia below C7.	Prominent macrophage infiltration. Anterior spinal infarction.
8	F	70	6 months	Fall. Fracture C2. Incomplete paraplegia below C4.	Cavitation in central grey tissue at site of injury.
9	M	29	6 months	RTA. Damaged cord L1-3. Incomplete paraplegia.	Evidence of haemorrhage at site of injury.
10	M	69	10 months	Fall. Fracture dislocation C5-6.	Gliosis.
11	M	61	2 years	Fall. Fracture dislocation C5-6.	Cysts containing macrophages at site of injury.
12	F	85	6 years	Fall. Fracture dislocation C4-5	Fibrillary gliosis.
13	M	58	13 years	Fall. Fracture L1 complete paraplegia below L4.	Fibrillary gliosis.
14	M	62	22 years	Fracture dislocation T12	Syrinx T10-T12—see text.
15	F	43	27 years	RTA. Fracture T2. Brain injury. Incomplete paraplegia below T5.	Fibrillary gliosis at multiple sites.
16	M	52	32 years	Tree fell on spine T6-7.	Fibrillary gliosis extending to two segments below injury site in dorsal columns.
17	M	59	34 years	Fall. Injury to lumbar spine.	Syrinx C8-L3—see text.
18	M	50	36 years	Fall. Complete paraplegia below T11.	Fibrillary gliosis.
19	M	60	40 years	Shotgun injury. Complete paraplegia below T10.	Fibrillary gliosis.
20	M	66	43 years	Shotgun injury. Fracture T11.	Fibrillary gliosis.

M = male; F = female; C = cervical cord; RTA = road traffic accident; T = thoracic cord; L = lumbar cord.

from 18 to 67 years old at the time of injury. Changes in the remaining 16 cases are described briefly under the headings of early or late pathological changes.

CASES WITHOUT SYRINXES

Early pathological changes

Cases surviving only a few days after injury showed oedema and disruption of the normal structure of the cord at the site of injury. Neuronal cell bodies showed shrinkage and loss of staining. By five days after injury axonal swelling and retraction balls were seen. Myelin sheaths were swollen and disrupted. Macrophage infiltration was seen first in a case surviving 15 days and macrophages were still present up to two and a half years after injury (case 11).

Reactive gliosis and capillary proliferation were first seen after 15 days and cyst formation six weeks or more after injury. Haemorrhage was seen in only two cases, in one (case 4) in an area of infarction in the territory of the anterior spinal artery two segments above vertebral dislocation. The other (case 9) showed haemorrhage at the site of cord injury.

In case 3 (who received a hyperextension injury in a rear shunt in a motor car accident) there was no evidence of direct trauma to the spinal cord and no bony injury or dislocation of the vertebral column but both vertebral arteries had extensive dissecting aneurysms. The spinal cord showed recent infarction in cervical segments 1–5 where it was severely swollen. A long tapering core of necrotic tissue, round on cross section, extended caudally to T8 at the base of the dorsal columns (fig 1).

Late pathological changes

Cases surviving for two years or more showed dense fibrillary gliosis at the site of injury. Where there had been severe cord trauma, the site of injury consisted of bundles of nerve fibres, dense fibrous tissue, and small islands of densely gliotic tissue as the only surviving remnants of spinal cord tissue. There was pronounced atrophy of ascending and descending

columns above or below the site of injury. In one case (case 16) ascending tracts were atrophic for two segments below the injury as well as above it. Corpora amylacea were prominent in the atrophic tracts. Evidence of old haemorrhage was seen in only one long surviving case

CASES WITH POST-TRAUMATIC SYRINGOMELIA

Case 5: C5-T8 syrinx (survival six weeks)

There was narrowing of the cord at T5. A central cystic cavity extended caudally to the level of T8. Histological examination showed total destruction of the cord at T5 with replacement by masses of foamy macrophages. Above the lesion foci of necrosis involving dorsal, anterior, and anteriolateral columns were seen up to the level of C8, where a single rounded area of infarction was present at the base of the dorsal columns, just dorsal to the central canal (fig 2). Below the lesion a rounded cavity packed with macrophages extended to the level of T8 at the base of the dorsal columns. There was no evidence of haemorrhage at any level.

Case 6: C4-C7 syrinx (survival 12 weeks)

The cord was narrowed, firm, and cystic at the level of C7. Just below this lesion at C7–8 two small central cysts were seen. Above the lesion a central cavity extended up to the level of C4. Histological examination showed that most of the central part of the cord was destroyed at C7, with cystic degeneration, gliosis, and collections of foamy macrophages, mostly around blood vessels. A few anterior horn cells survived and the peripheral zone of the cord remained normally myelinated. There was no evidence of haemorrhage. Above the lesion there was a rounded cyst at the base of the dorsal columns, filled with foamy macrophages. This extended up to C4. No connection with the central canal was seen. Above this there was atrophy of the dorsal columns. Below C8 atrophy of the descending tracts was the only change seen.

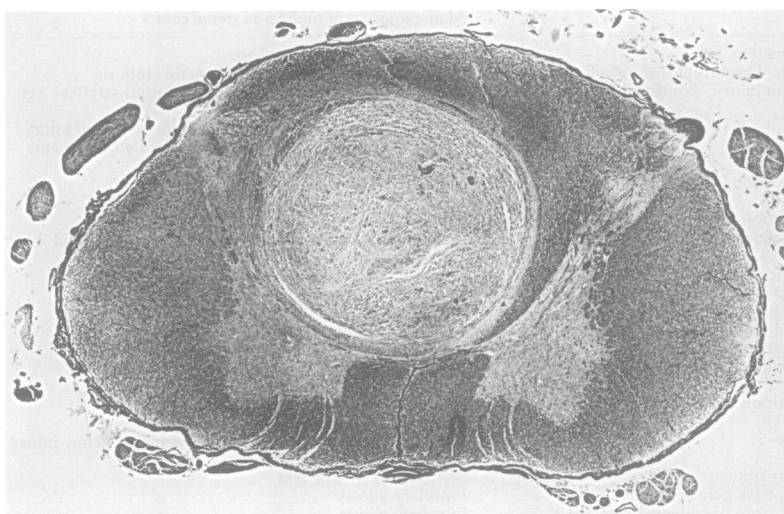


Figure 1 Case 3: a rounded core of necrotic tissue is seen at the base of the dorsal columns of the cervical cord (haematoxylin eosin, originally $\times 8$).

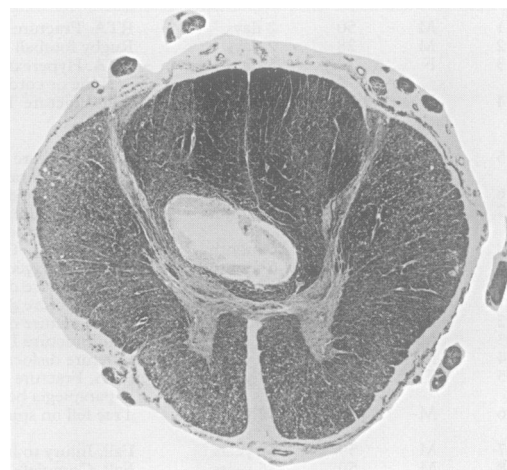


Figure 2 Case 5: a cystic space containing groups of macrophages at the base of the dorsal columns of the cervical cord (haematoxylin eosin, originally $\times 7.5$).

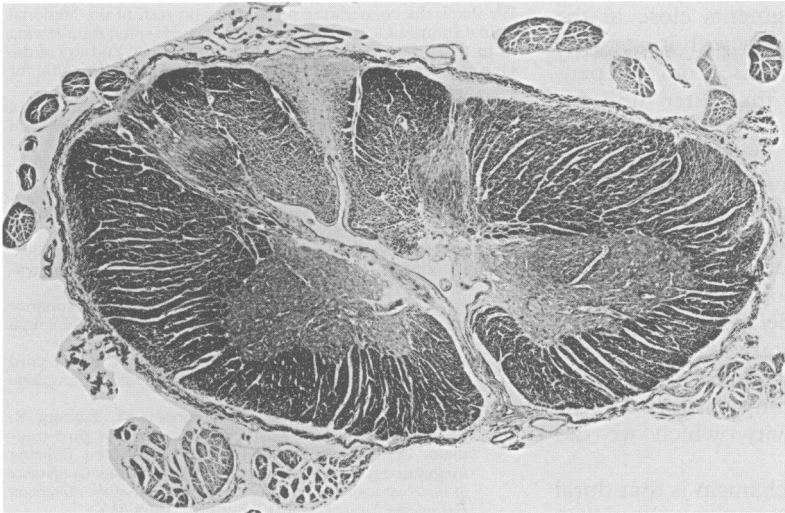


Figure 3 Case 17: A flattened cyst is seen in the central part of the thoracic cord. Ependymal cells form part of the lining. The cyst extends into connective tissue in the anterior median raphe, but is not continuous with the subarachnoid space (haematoxylin eosin, originally $\times 7.5$).

Case 14: T10-T12 syrinx (survival 22 years)

The surface of the cord was flattened and fibrosed at T12, where the dura was firmly adherent. Horizontal slicing showed a cavity extending from T12 to T10 with atrophy of dorsal columns above this level. The cord was macroscopically normal in lumbar and sacral segments. Histological examination showed complete disorganisation of the cord at T12 where only islands of gliotic tissue remained. Bundles of nerve fibres were seen among dense connective tissue and meningeal cells. The dura was thickened. There was no evidence of old haemorrhage. Above the lesion was a cystic cavity that extended to the level of T10 in the dorsal cord between dorsal columns and the posterior horn. The dorsal and contralateral lateral columns were pale and gliotic. Atrophy of the gracile tracts was noted throughout all segments above T10. Below the lesion there was atrophy of dorsal columns.

Case 17: C8-L3 syrinx (survival 34 years)

The cord was flattened and distorted with dense adhesions to the dura in the lumbar and sacral regions. A large cavity with a thick lining extended upwards to the level of C8. Histological examination of the sacral cord showed bundles of nerve fibres between bands of dense fibrous tissue. No normal cord tissue was identified. At L3 there was dilatation of the central canal with atrophy and gliosis of gracile tracts but preservation of central grey tissue. Above this a large syrinx lined by flattened epithelium, resting on dense connective tissue, was seen adjacent to the central canal. In the thoracic level ependymal epithelium lined part of the syrinx which extended to the anterior median raphe at T6 (fig 3). The syrinx was in continuity with the central canal above this level. Above C8 the cord was intact but there was atrophy of the dorsal columns. A small amount of haemosiderin was seen in the cyst wall at C8. Similar pigment was seen in the leptomeninges above this level.

Discussion

Twenty spinal cords examined between two days and 43 years after trauma show a sequence of pathological changes similar to those previously described.^{10 11} Early reactive changes of oedema and tissue necrosis are followed by macrophage infiltration, capillary proliferation, and reactive gliosis which is the predominant finding one year after injury.

Four cases showed evidence of infarction; in one there was no evidence of direct trauma to the cord (case 3) and infarction was the result of dissecting aneurysms of both vertebral arteries. In two cases there was direct injury to the cord in segments adjacent to infarction in the territory of supply of the anterior spinal artery. In one other case (case 5) where there was syrinx formation, multiple small areas of infarction close to the main injury suggest small intraspinal vessel damage.

Four of the 20 cases examined (20%) had a cystic cavity extending for at least two segments from the site of original injury. This figure is slightly higher than the 17% described by Wozniewicz *et al* in the only large published postmortem series of 120 cases.⁶ Clinical and CT studies have shown a clinical incidence of post-traumatic syringomyelia of between 1% and 5%^{1 2} whereas MRI studies show a higher incidence of up to 22%,^{3-5 12} closer to the percentage found in postmortem studies.

Development of cysts after spinal cord trauma may occur as early as six weeks after injury. The cysts seem to result from cavitation of myelomalacic cores.^{2 7} These are cores of necrotic tissue that extend above and below the site of cord damage, first described by Holmes in 1915 in patients with gunshot wounds of the cord.¹³ Direct trauma is not, however, an essential prerequisite to formation of myelomalacic cores as one of our cases (case 3) had large cores after cervical cord infarction without evidence of direct injury. Myelomalacic cores are always found in the dorsal cord either at the base of the dorsal columns or between dorsal columns and dorsal horns. All four syrinxes in this series were in these sites.

The reason for location of myelomalacic cores at the base of the dorsal columns is not understood, but may be due to the presence of a watershed zone between the territories of the anterior and posterior spinal arteries.⁶ Mechanical restraints on lateral extension of necrotic cavities may be imposed by the arrangement of the fibre tracts of the cord.

Resolution of intramedullary haematoma has been suggested as a possible mechanism for cyst formation.^{14 15} In our cases evidence of haemorrhage was infrequent; in only three cases of trauma and in only one of four syrinxes. This suggests that it is not the usual precursor to cyst formation.

Our series provides no support for the role of central venous infarction in cyst formation as proposed by Davis and Symon.¹⁶

In some of the cases described at operation there have been multiple cavities and the origin of septation within cysts is disputed.^{2 15 16} The fact that at least one of our cases had

areas of infarction in segments close to the trauma, but not in continuity with it, suggests that separate cavities may arise from independent foci of infarction that later undergo extension.

Entry of CSF has been suggested as a cause of cyst extension^{2,9,15} but communications with CSF spaces have not so far been shown pathologically. In only one of our four cases was there communication with the central canal and no other communication with CSF pathways was identified. We have only examined representative blocks, however, and exclusion of communication would depend on serial sectioning throughout the entire extent of the cavity, which we did not do.

Another suggested mechanism is that dural adhesions at the site of old trauma cause alterations in intraspinal pressure differentials, which allow extension of the cavity² or allow fluid to be forced across the cyst wall through a relatively porous spinal cord.¹⁵ Dural fibrosis and adhesions at the site of original injury were seen in three of our four cases with syrinxes.

In conclusion, this study has illustrated the pathological reactions of the spinal cord between two days and 43 years after trauma. The finding of syringomyelia in 20% of cases selected for necropsy is close to the clinical incidence found by MRI, which is the method of choice for imaging the spinal cord. This method is thus more accurate than either CT or clinical examination in identifying the extent of syringomyelia, and suggests that post-traumatic syringomyelia is more common than previously thought.

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