Pathology of spinal cord injuries due to fracture and fracture-dislocations of the cervical spine

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Autopsy was performed on 31 patients with spinal cord injuries at Chugoku Rosai Hospital between 1957 and 1987. The pathology of intervertebral disc injuries and hyperextension injuries of the cervical spine have been reported previously. This paper describes and discusses the pathological features of spinal cord injuries of 11 patients with fracture and fracture-dislocations of the cervical spine. Sex, age, cause of injuries, bone injuries seen on roentgenograms, level and grade of neurological deficits, survival time, and findings of postmortem studies are presented in the table. Roentgenograms and pathological features of five cases are illustrated in color.

Keywords: pathology of spinal cord injuries; cervical spine; fracture and fracture-dislocations.

Methods

The methods employed in the present study are the same as those used in my previous report on the pathology of cervical intervertebral disc injuries.¹

Case reports

Case 1

A 47 year old man fell from a height of 3.5 m and struck his head. He immediately developed complete paralysis below C4 and was admitted to our hospital 3.5 h after the injury.

Roentgenograms revealed dislocation of C5 on C6 with bilateral facets locking and fracture of the bilateral inferior articular processes of C5. Anterospondylolisthesis of C5 body was 40% of the width of the vertebral body. The dislocation was immediately reduced by application of crutch-field calipers. However, respiratory failure developed 5 days later and he died 11 days after the injury.

Autopsy showed extensive haemorrhage in and around the C5–6 vertebrae with rupture of all ligaments and facet joint capsular ligaments between C5 and C6, except the anterior longitudinal ligament. The cervical spine removed at

autopsy was stable in retroflexion (Fig 1a), but when anteflexion with rotation was applied, C5 vertebra could be readily dislocated at C6, reproducing the same X-ray findings as those observed at the time of initial examination (Fig 1b). The spinal cord was swollen from C2 to C7 and the surface of the cord between C4 and C6 was softened and discolored bluish-red. Sagittal sections of the cord revealed complete transverse haemorrhagic necrosis between C4 and C6 segments and haemorrhage localised in the grey matter at C7 T1 (Fig 2).

Case 2

A 23 year old man fell on to his head while practising judo. He was unconscious at the time of admission and as breathing had almost ceased, tracheotomy had to be immediately performed. Roentgenograms revealed dislocation of C4 on C5 with bilateral facets locking and anterior marginal fracture of C5 (Fig 3). His critical condition did not permit any attempt to reduce the fracture-dislocation. After resuscitation from apparent death he regained consciousness and survived for 26 days with respiratory assistance and thereafter for 3 months with spontaneous breathing. He could not raise his head nor maintain a sitting position. He died 4 months after the injury.

Autopsy revealed rupture of all ligaments except the anterior longitudinal ligament, but the site of bone injury was united with fibrous

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Figure 1 Lateral radiographs of the cervical spine removed at autopsy. (a) By retroflexion the spinal column shows normal alignment and stable spine. (b) By anteflexion with rotation, dislocation of C5 on C6 is reproduced.

tissue and the cervical spine was stable. The vertebral arteries and veins on each side were compressed by narrowing of the deformed vertebral foramina with occlusion by thrombus (Fig 4).

Macroscopically, the cut ends of the spinal cord at the level of the atlanto-occipital joint revealed a small cavity with discolouration at the central portion of the cord (Fig 5). Microscopically, cross sections of the cord at the level of C2 and C3 showed small cavities in the central portion of the cord and demyelination in the white matter (Figs 6a, b). Complete destruction of the spinal cord with loss of cord parenchyma was observed at the level of C4 (Fig 6c).

Case 3

A 68 year old man fell from a height of 3 m and sustained a laceration of his frontal region. When he arrived at our hospital 4 h after the injury, his respirations were weak and he became unconscious whilst being taken to the ward after the X-ray examination. Artificial respiration was immediately commenced and a tracheotomy was performed. He regained con-

Figure 2 Sagittal section of the spinal cord, showing complete transverse haemorrhagic necrosis at C4-6 segment and haemorrhage localised in the grey matter at C7 and T1.

sciousness with the support of a respirator. Roentgenograms revealed severe dislocation of C5 on C6. Displacement was the full width of the vertebral body with bilateral locked facets of C5-6, which was reduced by skull traction within 12 h (Fig 7). However, he was unable to breathe spontaneously and died 6 weeks thereafter.

Autopsy revealed extensive bleeding around the C5-6 vertebrae. Hemisected vertebral spine showed rupture of all ligaments and intervertebral disc between C5 and C6 vertebrae and the cervical spine was unstable. The entire length of the spinal cord was extremely swollen even 6 weeks after the injury. The leptomeninges was wrinkled due to shrinkage of the cord at and below C3 (Fig 8a). Spinal cord sections revealed severe transverse haemorrhagic necrosis below C3 to C7, showing *café au lait* discolouration (Fig 8b).

Case 7

A 54 year old man wearing a helmet fell from a height of 3.5 m and struck his head. He

Figure 3 Lateral radiograph of the cervical spine, showing dislocation of C4 on C5 with bilateral facet locking and marginal fracture of C5.

Figure 4 Lateral view of the cervical spine, showing occlusion of the vertebral artery due to narrowing of the dislocated vertebral foramina.

suffered complete tetraplegia below C4 and was admitted to our hospital 4 h after the injury. Roentgenograms revealed subluxation of C4 on C5 with slight displacement, which was reduced by skull traction (Fig 9). However, his neurological deficit deteriorated and respiration ceased 40 h later. After being resuscitated by tracheotomy and respiratory control, the patient survived but without spontaneous respiration. He could not move his head and neck and the level of anaesthesia ascended to C1. He died 8 weeks after the injury. He was conscious until 1 day before death.

Autopsy revealed haemorrhage surrounding C4-5 vertebrae. The posterior ligament complex was ruptured and excessive ante flexion of C4-5 could be demonstrated. The entire length of the spinal cord was reduced in size due to colliquative necrosis (Fig 10). The cord parenchyma became amorphous with soft mushy hardness. The anterior spinal artery and vein and minute vessels in the spinal cord at

Figure 5 Cross section of the cervical spine at the level of atlanto-occipital joint, showing a small cavity in the central portion of the cord.

each segment were occluded with coagulated blood cells (Fig 11).

Case 9

A 34 year old man struck his head when he dived into a shallow pool and sustained a

Figure 6 Cross section of the spinal cord at the level of C2 and C3, showing small cavities in the region of the central canal and demyelination in the white matter. C4, showing complete destruction and loss of cord parenchyma. Klüver-Barrera stain.

laceration on the top of the head with complete tetraplegic C6. He could move the deltoid muscle and the M. biceps brachii, and anaesthesia was observed at and below C6. Voluntary breathing was possible using the diaphragm. Roentgenograms revealed a vertical compression fracture of C6 body with wide separation of the fragments (Fig 12a). Skull traction was performed. Bulbo-cavernous and anal reflexes were present on the following day. Neurological deficits gradually deteriorated and hypesthesia

Figure 7 Lateral radiographs, showing conservative reduction of fracture-dislocation of C5 on C6 (a) At time of initial examination, (b) during skull traction, and (c) release of facets locking.

ascended to C4 on the 4th day. Tracheotomy had to be performed due to dyspnoea on the 5th day. Hypesthesia of the face was observed and he could not move his head and neck on

Figure 8 (a) Hemisected cervical spine, showing anterior and posterior longitudinal ligaments and intervertebral disc rupture. The spinal cord is extremely swollen with wrinkled leptomeninges. (b) Sagittal section of the spinal cord, showing complete transverse haemorrhagic necrosis below C3.

Figure 9 Lateral radiograph of the cervical spine, showing subluxation of C4 on C5 with slight displacement.

the 7th day. He died from renal and cardiac failure 15 days after injury. He was conscious until the day before death.

Autopsy revealed extensive haemorrhage around the C6 vertebra and rupture of the anterior longitudinal ligament at C5-6. Aside from relaxation of the posterior ligament complex of C5-6 and C6-7, neither fracture or fracture-dislocations nor rupture of the capsular ligaments could be observed. Lateral roentgenograms of the removed cervical spine showed normal alignment on manual extension (Fig 12b), but excessive ante flexion of C5-6 and C6-7 segments on flexion (Fig 12c). Sagittal sections through the cervical spine and the spinal cord in situ showed that cancellous bones of the C6 vertebral body were completely crushed into fine sand-like fragments and projected into the spinal canal.

The anterior and posterior longitudinal ligaments and dura were disrupted and in tatters. The spinal cord and the anterior spinal vessels were damaged by the fragments. Colliquative necrosis of the spinal cord with *cafe au lait* discolouration was observed 3 cm in length.

Figure 10 Sagittal section of the spine and spinal cord with lamina and dura resection. Left: Without spinal cord. Right: With spinal cord and nerve root. The entire length of the spinal cord is soft and reduced in size due to necrosis.

Figure 11 Cross sections of the spinal cord at each segment with the cord parenchyma being amorphous due to colliquative necrosis. The anterior spinal artery and vein and minute vessels in the spinal cord at each segment were occluded with coagulated blood cells. Hematoxylin-eosin stain.

Furthermore, colliquative necrosis extending up and down the whole length of the cord with yellow discolouration was observed (Fig 13).

A brief description of cases 10 and 11 are presented in Table I.

Figure 12 Lateral radiographs of case 7. (a) Shows vertical compression fracture of C6 at the time of initial examination. (b) Shows normal alignment by extension of the removed cervical spine. (c) Shows diastasis of the spinous processes of C5, 6 and C6, 7 by flexion.

Figure 13 Sagittal section of the spine and spinal cord, showing crushed fragments of C6 projected into the spinal canal and colliquative necrosis of the entire spinal cord.

Discussion

Dislocation in case 1 could be reproduced by applying anteflexion with rotation of the removed cervical spine. Lateral roentgenograms of the cervical spine demonstrated the same findings as those taken at the initial examination. This movement study suggests that the spinal cord was forcibly stretched at the state of maximum dislocation. The mechanism involved was crushing of the spinal cord between the lamina of the upper vertebra and posterior superior border of the vertebral body below.^{2,3} Haemorrhage of the grey matter of C7-T1 appeared as longitudinal traction of the cord due to the direct stretching force.⁴

The autopsy of case 2 demonstrated no abnormal mobility of the vertebral body at the site of bone injury even though the fracture-dislocation remained unreduced. The cause of central cavitation is not known. The bilateral vertebral arteries were occluded. Severe dizziness in raising the head suggests circulatory insufficiency of the brain due to occlusion of the vertebral arteries. Hughes⁵ reported two cases of acute central infarct of the spinal cord due to insufficiency of the vertebral artery.

Case 3 was tetraplegic below C4 due to severe fracture-dislocation of C5 on C6. If resuscitation had not been administered, he would have died from respiratory failure a few hours after the injury. He survived for 6 weeks with the aid of a respirator. During this period, the pathological changes of the damaged spinal cord progressed and showed complete transverse haemorrhagic necrosis. The leptomeninges did not rupture but wrinkled due to shrinkage of the spinal cord. The leptomeninges, the inelastic membrane closely surrounding the spinal cord, inhibited the swelling of the spinal cord and disturbed circulation in the minute vessels in the cord to promote necrosis. This is considered to have given rise to severe damage of the cord. The mechanism of the cord damage appeared to be compression and shearing of the cord by the dislocated vertebrae.

The roentgenograms of case 7 showed subluxation of C5 on C6 with slight displacement. All ligaments and intervertebral discs

Table 1 Fracture and fracture-dislocations of the cervical spine

Case no.	Age	Sex	Cause of injury	Roentgeno-gram	Level and grade of paralysis	Survival time	Tissues involved				Macroscopic features	Extent of spinal cord damage
							All	PLL	ISL	Disc		
1	47	M	Fall of 3.5 m	C5-6 bil frac disl 40%, red	C4 complete	11 days	-	+	+	+	Transverse haemorrhagic necrosis C4-6 petechia in grey matter C7 T1	10.5 cm
2	23	M	Fall on to head	C4-5 bil frac disl 50%, unred	C4 complete	4 months	-	+	+	+	Occlusion of vertebral artery, central cavity, disappearance of parenchyma C4	5.0 cm
3	68	M	Fall of 2.0 m	C5-6 bil frac disl 100%, red	Respiratory quadriplegia	6 weeks	+	+	-	+	Swelling, complete transverse haemorrhagic necrosis C3-7	6.0 cm
4	16	M	Fall from motorbike	C5-6 bil frac disl 40%, unred	C6 complete	8 months	-	Unk	+	+	Loss of parenchyma, attenuation, firm adhesion of cord with dura	4.0 cm
5	57	M	Fall of 3.0 m	C4-5 bil frac disl 40%, red	C4 complete	11 month	+	Unk	+	+	Attenuation and scar tissue of cord	6.0 cm
6	59	M	Fall of 3.0 m	C6-7 subluxation red	C7 complete	55 month	+	Unk	+	+	Softening, atrophy, adhesion of C6 root	8.0 cm
7	54	M	Fall on 3.5 m	C4-5 subluxation red	C4→ tetraplegia	8 weeks	- Relax	+	+	+	Occlusion of all vessels in cord, parenchyma amorphous	Entire length
8	38	M	Fall of 5.0 m	C5-6 subluxation red	C6 complete	3 months	-	-	+	+	Softening, necrosis with cream-like appearance	5.0 cm
9	34	M	Dived into shallow pool	C5-6 burst frac	C6→ tetraplegia	16 days	+	+	+	+	Destruction of anterior spinal vessels, liquefactive necrosis, yellow pus	Entire length
10	19	M	Head struck by falling object	C5-6 burst frac	C4 complete	10 days	+	+	+	+	Complete severance of cord, oedema	3.0 cm
11	23	M	Fall of 30 m with vehicle	C5-6 burst frac	C4 complete	12 hours	+	+	+	+	Total disruption of cord, oedema	3.0 cm

All = anterior longitudinal ligament; PLL = posterior longitudinal ligament; ISL = interspinous ligament; M = Male. bil = bilateral; frac = fracture; disl = dislocation; red = reduction; unred = unreduction; Unk = unknown.

except the anterior longitudinal ligaments were ruptured. Manual flexion of the removed cervical spine demonstrated extreme flexion between C5 and C6. The observed position in the X-ray immediately after admission was always a partially reduced position.³ Burke⁶ has made the following comment: 'I think there is a tendency to underestimate the amount of displacement that the spine showed at the moment of impact. We don't see this. We see the X-ray taken even half an hour afterwards and I am sure that is a very different situation to that at the moment of impact. And therefore possibly the degree of damage to the cord is much greater than we expect from the radiological appearance because we didn't see it at that moment of impact.'

According to an eyewitness who saw the accident of case 7, his head and neck were flexed with the chin on the chest at the moment of impact. The anterior spinal artery and vein were crushed and completely occluded. The occlusion of the anterior spinal artery was observed only in this case among the 31 cases of this present series.

The aetiological factor of colliquative necrosis of this case may be circulatory disturbance.⁷ The cord suffers not only from direct injury to axon and blood vessels at the time of injury but also from a second chain of events which result in ischaemia, hypoxia, oedema and ultimate infarction of haemorrhagic necrosis of the cord. The histological appearance varies strikingly during the period of survival.

In the movement study of the removed cervical spine of case 9, the mechanism of cervical spine injury elucidated a combination of two factors, one being vertical compression of the cervical column and the other being forcible flexion which relaxed the posterior ligament complex. Tetraplegia of C6 deteriorated to paraplegia in 7 days. It is not known why the clinical course and the pathological changes of the spinal cord progressed to the whole length of the cord in spite of good conservative therapy. Extensive damage of the spinal cord and vessels was observed at autopsy. Why did it hap-

pen? In traumatic spinal cord damage, in addition to primary damage induced by external force, secondary damage due to circulatory impairment must be considered. In cases 7 and 9, necrosis of the central nervous system was localised only in the spinal cord because they were conscious during life.

Conclusion

- 1 The mechanisms of spinal cord injuries due to fracture and fracture-dislocations of the cervical spine were crushing and stretching, as well as the shearing stress of the dislocated vertebral bodies or the dispersed fragments.
- 2 The intervertebral disc injuries occurred in all cases in this series, but they never participated in the mechanism of cord injury.
- 3 Roentgenograms taken in a manually flexed and extended position of the removed cervical spine were useful in elucidating the mechanism of cervical spine injury.
- 4 The degree of spinal cord damage due to fracture and fracture-dislocations was far more severe than intervertebral disc injuries or hyperextension injuries of the cervical spine.
- 5 In complete transverse lesions in which flaccid paralysis remains unchanged for 48 h, it has usually been proved to be either complete crushing and destruction of the cord or at least very severe irreparable damage.
- 6 Neural damage was present in a certain section of the cord. The length of the damaged area explains the seriousness of the case in the early stage and the causes of death.
- 7 The cord suffers not only from direct injury to the axon and blood vessels at the time of injury but also from a second chain of events which results in ischaemia, hypoxia, oedema, and ultimate infarction of haemorrhagic necrosis of the cord. The histological appearance varies strikingly according to the period of survival.

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