

TREATMENT AND RESULTS IN VERTEBRAL FRACTURES WITH NEUROLOGIC DEFICITS

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ABSTRACT:

The operations performed in order to maintain normal anatomy and stabilization of the spinal canal in vertebral injuries have frequently been a matter of discussion. The relationship between neurologic healing and correction of the spinal canal is not very clear. Neurologic findings of the cases at admission, preoperative and postoperative examination were grouped according to Frankel classification. Dislocation angulation of the bones and their pressure on the neurologic tissues were evaluated with AP and lateral X-rays and Computerized Tomography (CT) of spine.

Conservative management and internal stabilization with laminectomy and laminectomy plus posterior instrumentation were performed. Their neurologic state were followed up for one year minimum. We observed that the level of neurologic healing was not in correlation with the ratio of the correction in cases of preoperative advanced vertebral dislocation. Neurologic progress of our cases in which decompression was obtained with early laminectomy was better at the beginning, but later, pain occurred and neurodeficit increased with deformity

Key Words: Spinal trauma, Neurologic deficit, Spinal instrumentation, Spinal CT

INTRODUCTION

Modern and positive approach results in a long-term excellent life in cases of spinal trauma. Methods of treatment in cases of thoracolumbar trauma with instable vertebrae and neurologic deficit are controversial (1, 2, 3). Paraplegia occurred at site of accident is considered as permanent. In cases with instable fractures neurologic injury may occur during rescue and transfer. Prevention of these secondary injuries is important (1, 4).

Handling of stable vertebrae fractures is principally conservative (2, 5). Spinal stability is described according to the morphologic injury of vertebral column into three parts; anterior, middle and posterior; and reports that key of the stability is the middle part. Fracture of at least two parts means that spine is no more stable. It is widely accepted that combination of posterior instrumentation and fusion results in better anatomic stabilization and rehabilitation (1, 5, 6, 7). Therefore, Harrington instruments have been widely used since 1967 in stabilization of thoracic and lumbar vertebrae fractures to shorten hospitalization and for early mobilisation and rehabilitation (8, 9, 10, 11). Laminectomy whether combined with posterior fusion or not has some disadvantages in unstable thoracolumbar vertebra fractures. Spinal cord cannot be de-

compressed sufficiently and complications increase. Additional pain and increase of deformity destroy stability of spine (9, 12, 13). However, some authors admit that posterior stabilization should be combined with anterior vertebrectomy or posterolateral decompression in cases of burst fractures and posterior displacement of vertebral body (3, 14, 15, 16). Anterior approach is recommended especially in cases with lesions at L2 or below and whose posterior elements are not fully destroyed without paraplegia. Here, mortality and morbidity incidence is higher compared to the posterior approaches (3, 4, 17).

Purpose of surgery in spinal traumas are correction of deformity, stabilization of vertebral fracture, releasing of neural tissues by decompression of cord and early rehabilitation (1, 2, 3). Effects of decompression on neural tissues is not clear yet. Because the relationship between the diameter of spinal canal and function of neurologic transport is not fully determined. Many authors have reported that they could not explain the exact relation between the initial radiologic findings about posttraumatic spinal narrowing and neural injury (5, 6, 18).

As a summary, although the effect of spinal column reconstruction on neurologic healing is not fully known, the criteria for the most suitable surgical approach are not clear yet (5, 19, 20). In our study, we examined the relation between reconstruction of traumatic spinal deformities and postoperative progress in healing of preoperative neurologic status.

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MATERIAL AND METHOD

98 cases of spinal trauma who had neurologic deficit and was admitted between 1989 and 1993 were reviewed. Those without CT and died because of cerebral trauma were excluded.

Initial neurologic examination of the patients at the emergency based on Frankel classification. Considering this classification, all cases were examined with preoperative and postoperative X-rays and CT. By the help of these, compression, and preoperative and postoperative AP and lateral dislocation and angulation were evaluated. Traumatic regions were compared to intact parts. Whatever the method of treatment was, our purposes were decompression of the spinal cord and maintenance of the spinal column at the anatomic position.

FINDINGS

24.5% of the cases were transferred to emergency within the six hours after trauma while 75.5%, the majority, later. Details are given at Table 1. These cases were seven to 63 years old. 21 to 30 years were the most frequent group with a ratio 33.7% (Table 2). Most frequent cause was traffic accident seen in 33 cases (33.7%), 32 cases fell from high (32.6%), 19 cases were wounded by firearms was the third with an incidence of 19.4% (Table 3).

Table 1: Interval between incident and delivery.

	No of cases	%
0 - 3 hours	10	10.2
4 - 6 hours	14	14.3
7 - 24 hours	49	50.0
more than one day	25	25.5
Total	98	100

Table 2: Age distribution of cases

Age	Male	Female	Total	%
0 - 10	2	1	3	3.1
11 - 20	9	6	15	15.3
21 - 30	27	6	33	33.7
31 - 40	12	2	14	14.3
41 - 50	15	-	14	15.3
51 - 60	12	1	13	13.2
61 - 70	2	3	5	5.1
Total	79	19	98	100

Table 3: Causes of injury in cases

Causes of injury	No of cases	%
Traffic accident	33	33.7
Fall from a height	32	32.6
Gun shot	19	19.4
Work accident	7	7.1
Hitting	3	3.1
Sport	1	1.0
Others	3	3.1
Total	98	100

According to the site, cervical spine was injured in 35 cases (35.7%), thoracic spine in 38 cases (38.8%) and lumbar spine in 25 cases (25.5%) (Table 4), 66.3% of cases were classified as Frankel A, 8.2% as Frankel B, 11.2% as Frankel C and 14.3% as Frankel D (Table 5). According to the initial CT findings of the cases transferred to emergency 65 cases had vertebral body compression (66.3%), 40 cases had dislocation (40.8%), 69 cases had spinal narrowing (70.4%), 18 cases had loose bone fragment within the spinal canal (18.4%), 10 cases had intramedullary haemorrhage or contusion (10.2%), and spinal canal was intact in eight cases (8.2%). No pathology was determined in radiologic examinations in 5 (5.1%) cases (Table 6).

Table 4: Level of traumatic pathology

Localisation	No of cases	%
Cervical	35	35.7
Thoracal	38	38.8
Lumbal	25	25.5
Total	98	100

Table 5: Initial neurologic evaluation of cases

	No of cases	%
Frankel A	65	66.3
Frankel B	8	8.2
Frankel C	11	11.2
Frankel D	14	14.3
Total	98	100

44.9% of these cases with neurologic deficit were managed conservatively; combination of laminectomy and internal stabilization were performed in 5.1%, and internal stabilization in 18.4% (Table 7). Cases were

grouped as follows at discharge: 58.2% of cases in Frankel A, 11.2% in Frankel B, 10.2% in Frankel C, 11.2% in Frankel D and 9.2% in Frankel E (Table 8).

Table 6: Early CT findings

	No of cases	%
Compression	65	66.3
Dislocation	40	40.8
Canal narrowing	69	70.4
Intraspinal loose fragment	18	18.4
Intramedullary contusion or heamatoma	10	10.2
Intact canal	8	8.2
Burst fracture	5	5.1
No pathology	5	5.1

Table 7: Performed method of treatment

	No of cases	%
Conservative	44	44.9
Laminectomy	31	31.6
Laminectomy + Internal stabilization	5	5.1
Internal stabilization	18	18.4
Total	98	100

Table 8: Neurological evaluation of cases at discharge

	No of cases	%
Frankel A	57	58.2
Frankel B	11	11.2
Frankel C	10	10.2
Frankel D	11	11.2
Frankel E	9	9.2
Total	98	100

The more preoperative dislocation towards spinal canal, more severe was the neurologic deficit (Fig 1). This increases at L1 or upper levels. However, a correlation could not be determined between postoperative anatomic correction of the spine and neurologic healing. Even separate evaluation of L1 and upper

level injuries did not result in a significant relation. Surgical method did not have a significant relation with healing of neurological deficit, too.

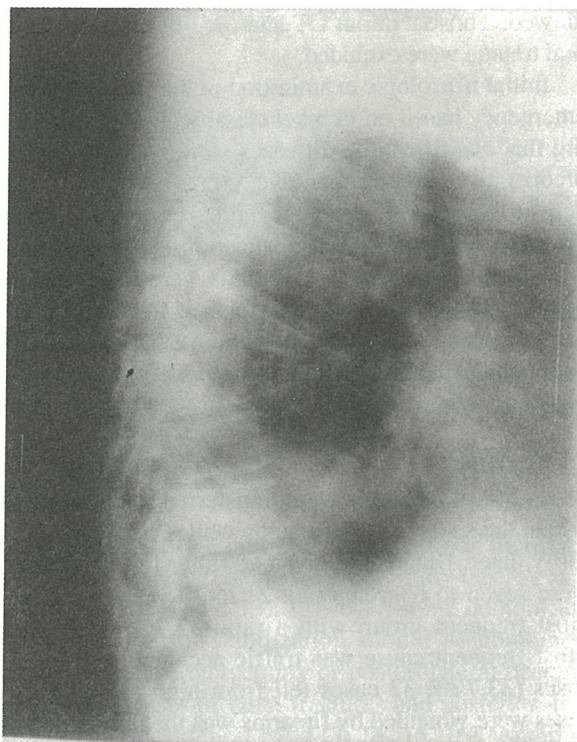


Fig 1: Dislocation towards spinal canal

DISCUSSION

Vertebral injuries can occur at any age since the most frequent cause is traffic accidents (1, 7). In our cases also, it had an incidence of 33.7%, 32.6% fell from high and 19.4% was injured with a gun fire. Higher incidence of the last two causes compared to literature is related to our region.

Contusion can occur in spinal cord as a result of vertebral trauma. Destruction of the vertebral body, traumatic disc hernia, spinal epidural haematoma and neural injury can occur if spinal deformity proceeds (14, 16, 21, 22, 23). Ischemia, haemorrhagic edema and necrosis also worsen the neurologic status. Purpose of surgery is to decompress the neural tissues; and to shorten the interval for rehabilitation (21, 22). We observed postoperative spinal deformity and pain in five cases with laminectomy. Compressive traumatic hernia and spinal epidural haematoma were seen in one each.

Both grade of spinal deformity and extension of neurologic injury changes with the different conduction of kinetic energy during the accident. Severity of the neurologic deficit depends on the rate of preoperative posterior dislocation into spinal canal. However, there is not any positive correlation between the reconstruction achieved either conservatively or surgically and neurologic healing. We observed that dislocation was more symptomatic in thoracic region. Experimental studies showed that irreversible necrosis could occur in spinal cord if the circulation was blocked with a mild edema or compression (4, 21, 22). This shows that a correlation cannot exist between neurologic deficit and vertebral dislocation or compression. 66.3% of our cases included into this study were transferred with complete paraplegia. Its rate was also as high as 58.2% at discharge. 36.7% of cases had paraparesis and 9.2% recovered completely. This shows that initial neurodeficit developed at the site of accident is irreversible. Thus, surgery shortens hospitalization and prompts rehabilitation. Only 10.2% of our cases were delivered to our emergency in three hours' time, 14.3% in six hours' time and the rest later. Many authors underline the importance of early beginning to the treatment (2, 4, 18). Hence, our results do not seem to be truly successful.

Influence of trauma magnitude on vertebra and its conduction on the neural tissues is related to the biomechanical of the injury. For example, the energy delivered by axial loading first effects vertebral body and intervertebral disc space via burst fracture (8, 14). Diameter of the spinal canal shows the initial status of the patient, so it cannot demonstrate the spinal deformity occurred just at the moment of accident. Elasticity of the spinal components and postural effect assist in reduction of initial spinal deformity (9, 13). Degree of spinal angulation may not be same at time of transfer and accident. It seems to be impossible to determine the exact duration of compression on spinal cord caused by spinal deformity and angulation; real magnitude of the collision on the cord. Spinal cord is not as resistant as cauda equina against trauma whatever its type (6, 8, 14, 19).

Some authors emphasized the importance of CT to determine the presence of a bone fragment or foreign material within the spinal canal accurately (5, 18, 22). In our study after the initial diagnosis with spine X-rays, spinal CT was performed on each case to confirm the diagnosis, surgical indication and planning. The most frequently injured level was thoracic region

with an incidence of 38.8% in our cases. This is similar to the literature. Successful results of surgical treatment are reported in cases with angulation of 15 degrees maximum and dislocation less than 10%. It is controversial whether spinal instrumentation can relieve the compression of vertebral body fracture alone or not (12, 14, 15, 19, 23). We performed laminectomy in 31.6% of our cases according to the CT results. Laminectomy is indicated in cases with intraspinal bone fragment or foreign material. We performed internal stabilization combined with laminectomy in 5.1% of our cases. Burst fractures and combinations fracture and dislocations are usually coexist with neurologic deficit. Injuries with fracture and dislocation typically decreases the diameter of spinal canal and causes kyphosis. In such cases spinal column can be reconstructed sufficiently with posterior instrumentation (5, 6, 11). Burst fracture resulted in neurologic deficit in 5% of our cases. Internal stabilization was performed on these cases.

In this study, we concluded that neither laminectomy nor posterior instrumentation had an advantage over the other in neurologic healing when performed alone. Although neurologic deficits of the cases with only laminectomy had a rapid healing at the beginning, postural disorders, related neurologic deficits and pain were observed later. As a result:

1. Degree and severity of the neurologic deficit in vertebral trauma is related to the magnitude of the strike on the cord at the moment of accident.
2. Although amount of dislocation or fracture seems to correspond to the degree of neurologic damage, it is actually related to the cord injury caused by the effect of initial strike.
3. CT should be performed in each case for adequate visualisation of the spinal cord.
4. Laminectomy alone is not sufficient for decompression in spinal traumas, and should be combined with internal stabilization.
5. There is no correlation between reconstruction of the spinal canal and recovery of the neurologic deficit.

REFERENCES:

1. Lemons VR, Wagner FC and Montesano PX: Management of Thoracolumbar Fractures with Accompanying Neurological Injury. *Neurosurgery* 30(5): 667-671, 1992.
2. Willen J, Lindahl S and Nordwall A: Unstable Thoracolumbar Fractures. A Comparative Clinical Study of

- Conservative Treatment and Harrington Instrumentation. *Spine* 10 (2): 111-122, 1985.
3. Kostuik JP: Anterior Fixation for Burst Fractures of the Thoracic and Lumbar Spine with or without Neurological Involvement. *Spine* 13: 286-293, 1988.
 4. Dunn HK: Neurologic recovery following anterior spinal canal decompression in thoracic and lumbar injuries. *J Bone Joint Surg* 8: 160-165, 1984.
 5. Durward Q, Schweigel J, Harrison P: Management of fractures of the thoracolumbar and lumbar spine. *Neurosurgery* 8: 555-561, 1981.
 6. Denis F: Spinal Instability as Defined by Three-Column Spine Concept in Acute Spinal Trauma. *Clin Orthop* 189: 65-76, 1984.
 7. Pamir N, Benli K, Özcan OE, Özgen T, Erbeni A, Bertan V, Sağlam S, Gürçay Ö: Posterior Fusion upper Cervical Spine Fractures. *Cerrahpaşa Tıp Fak. Dergisi* 13: 353-360, 1982.
 8. Davies WE, Morris JH, Hill V: An analysis of conservative management of thoracolumbar fractures and fracture-dislocations with neural damage. *J Bone Joint Surg* 62A: 1324-1328, 1980.
 9. Osebold WR, Weinstein SL, Sprague BL: Thoracolumbar spine fractures. Results of treatment. *Spine* 6: 13-34, 1981.
 10. Willen J, Lindahl S and Nordwall A: Unstable Thoracolumbar Fractures. A Comparative Clinical Study Conservative Treatment and Harrington Instrumentation. *Spine* 10(27): 111-122, 1985.
 11. Alıcı E: Stabilization and reduction with Harrington rod in unstable fractures and dislocations with accompanying fractures of the dorsal and lumbar vertebrae. *Dokuz Eylül Tıp Fak. Dergisi* 3 (2): 12-30, 1988.
 12. Jacobs R, Asher M, Sneider R: Thoracolumbar spinal injuries-a comparative study of recumbent and operative treatment in 100 patients. *Spine* 5: 463-477, 1980.
 13. Clark WK: Spinal cord decompression in spinal cord injury. *Clin Orthop* 154: 9-13, 1981.
 14. Benzel E, Larson S: Functional recovery after decompressive operation for thoracic and lumbar spine fractures. *Neurosurgery* 19:772-778, 1986.
 15. Dunn HK: Anterior spine stabilization and decompression for thoracolumbar injuries. *Orthop Clin North Am* (1), 17: 113-119, 1986.
 16. Lifeso R, Arabie K, Kadhi MS: Fractures of the thoracolumbar spine. *Paraplegia* 23: 207-224, 1985.
 17. Kaneda K, Abumi K and Fujiya M: Burst fractures with neurologic deficits of the thoracolumbar spine. Results of anterior decompression and stabilization with anterior instrumentation. *Spine* 9: 788-795, 1984.
 18. Keene J, Fischer S, Vanderby R, Drummond D, Turski P: Significance of acute posttraumatic bony encroachment of the neural canal. *Spine* 14: 799-802, 1989.
 19. Herndon W, Galloway D: Neurologic return versus cross sectional canal area incomplete thoracolumbar spinal cord injuries. *J Trauma* 28: 680-683, 1988.
 20. Ağuş H, Araç Ş, Us R and Öztürk H: Conservative Treatment in Fractures of the Vertebrae. *Acta Orthop. Traum. Turcica* 22: 98-100, 1988.
 21. Ikata, Iwasa K, Morimoto K, Tonai T, Taoka Y: Clinical considerations and biochemical basis of prognosis of cervical spinal cord injury. *Spine* 14: 1096-1101, 1989.
 22. Koberne AI and Bernstein JJ: Experimental Spinal Cord Injury. Eds. Wilkins RH and Rengachary SS. *Neurosurgery*, Section B. Spinal Trauma. Mc Graw-Hill Book Company. New York, 1985 Pp. 1694-1700.
 23. Maiman D, Larson S, Benzel E: Neurological improvement associated with late decompression of the thoracolumbar spinal cord. *Neurosurgery* 14: 302-307, 1984.