Postfracture spinal pain

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The treatment of fractures in the thoracic and lumbar spine has improved a lot during the last 25 years. Nowadays surgical management makes it possible to reduce and stabilize most fractures even after extensive laminectomies. The patients can be mobilized early, often with an increased chance of a better anatomical and neurological outcome.

However, the frequency of persisting back and leg pain after different kinds of fractures has only been analyzed in a few studies.

Clinical studies

Soreff reported on patients with wedge compression fractures 1977. 90 percent of all patients had dorsal insufficiency and 40 percent had chronic low back pain at follow-up 8 years after the injury. Residual severe back pain was noted in every second patient with an anterior vertebral body compression exceeding 25 percent. The pain was mostly located in the area of injury. Among these patients only 50 percent returned to their previous work. Only 10 percent of the 147 patients in his material were asymptomatic at follow-up.

54 burst fractures in level T12 and L1, treated conservatively at the Spinal Injury Centre in Perth W.A, were followed up by myself and assistants 1987–88 (Willén 1990). Patients with burst fractures including a rotational dislocation had severe low back pain and radiculopathy in 40 percent of all cases. The same was true, if the anterior vertebral body was compressed more than 50 percent and if the spinal canal was narrowed more than 50 percent. Burst fractures without these signs had severe low back pain and radiculopathy in only 15 percent.

At Sahlgren Hospital, Göteborg, a nonselective comparative study was performed on 50 patients with unstable fractures T11-L2 (Willén 1985). There were equal numbers of patients treated conservatively and with Harrington instrumentation. At the follow-up after 2 years no difference was found regarding thoracolumbar fatigue (40% and 60%, respectively), recurring thoraco-lumbar pain (60% and 40%, respectively) and pain on direct pressure at the fracture site (20% and 30%, respectively). However, the 8 patients in the conservative group with pain at direct pressure over the fracture site all had a gibbus angle exceeding 30 degrees.

Unstable fractures are better reduced and stabilized with transpedicular instrumentations such as the internal fixator. It preserves better mobility than is the case when using the Harrington instrumentation (Dick 1987). However, advantages regarding back and leg complaints compared to other treatment methods are not convincing (Olerud 1988).

There are a lot of possible causes of residual back pain after spinal fractures. McAfee minted the concept "late instability" 1982. Wedge fractures and burst fractures with signs of compression of the anterior vertebral body of more than 50 percent may give rise to an increasing gibbus deformation and pain, if the fracture and the posterior ligaments do not heal. The problem is to estimate the degree of injury in the initial period after the injury. Late instability may lead to post-traumatic kyphosis, which is generally recognized as a failure of the initial treatment. It occasionally results in severe pain, and neural compromise is not uncommon.

Neuropathic arthropathy with extensive degeneration of disc and facet joints at one or more levels after traumatic paraplegia is a relatively rare problem. But it must be considered in paraplegic patients with ill-defined, burning pain in a diffuse distribution around the level of the spine involved (Brown 1992).

There is every reason to assume that vertebral compression fractures and even so called "stable" burst fractures constitute a more complex injury than can be verified radiologically. Often the major fracture is correlated to a spinal fracture in the vicinity or some levels above or below. A compression fracture, which to a large extent is the result of an axial force to the spine should be considered as a joint injury and not mainly as an injury to the bone. In burst fractures there is always a severe injury to the disc above and below the fractured vertebra and even injuries to the facet joints. This has been shown experimentally (Willén 1984) and cliniconeuropathologically (Willén 1989).

Taylor (1990) presented a histological study on lumbar facet joints from spines of subjects dying from serious trauma. Injuries not visible on standard radiography, were shown in a high percentage of joints. They included chondral as well as bone injuries. It was suggested that such injuries may lead to early onset facetal arthrosis in survivors of major trauma.

In addition to the above mentioned possible causes of persisting pain after spinal fractures, muscle and tendon injuries are difficult to diagnose objectively. In the cervical spine MR investigations after whip-lash trauma has shown signs of hemorrhage in tendons and muscles as the result of ruptures.

Conclusion

The frequency of back pain is high after thoracolumbar fractures. 40–50 percent of all patients have pronounced complaints and only 10 percent are pain free in the long run.

The symptoms increase with the severity of the fracture. In wedge fractures and burst fractures with an anterior vertebral body compression exceeding 50 percent, a late instability may develop if the treatment has been inadequate. Compression fractures should be considered as joint injuries, as much as bone injuries.

No study, so far, has shown that surgical treatment of wedge fractures with a compression of the anterior vertebral body of less than 50 percent and stable burst fractures with narrowing of the spinal canal of less than 50 percent is superior to conservative treatment as far as persistent back is pain concerned. However, the indications for decompression and/or stabilization in thoracolumbar unstable injuries (flexion-rotation, translation, distraction and burst-rotations fractures) seems to be obvious to diminish residual spinal pain and radiculopathy.

References

Brown C W, Jones B, Donaldson D H, Akmakjian J, Brugman J L. Neuropathic (Charcot) Arthropathy of the spine after traumatic spinal paraplegia. Spine 1992; 17: 103–108.

Chakera T m H, Bedbrook G, Bradley C M. Sponteneous resolution of spinal canal deformity after burst dispersion fracture. AJ N R 1988; 9: 779–85.

Dick W. "Fixateur Interne" as a versatile implant for spinal surgery. Spine 1987; 12: 882–900.

McAfee P C, Yuan H A, Lasda N A. The unstable burst fracture. Spine 1982; 7: 365–73.

Olerud S, Karlström G, Sjöström L. Transpedicular fixation of thoracolumbar fractures. Clin Orthop 1988; 227: 44–51.

Soreff J. Assessment of the late results of traumatic compression fractures of the thoraco-lumbar vertebral bodies. A clinical, radiological and medio-social computer conducted study. Thesis 1977.

Taylor J R, Twomey L T, Corker M. Bone and soft tissue injuries of post-mortem lllmbar spines. Paraplegia 1990; 28: 119–29.

Willén J, Lindahl S, Irstam L, Aldman B, Nordwall A. The thoracolumbar crush fracture. An experimental study on instant axial dynamic loading. The resulting fracture type and its stability. Spine 1984; 9: 624–31.

Willén J, Lindahl S, Nordwall A. Unstable thoracolumbar fractures. A comparative clinical study of conservative treatment and Harrington instrumentation. Spine 1985; 10: 111–22.

Willén J, Gaekwad U H, Kakulas B A. Burst fractures in the thoracic and lumbar spine. A cliniconeuropathologic study. Spine 1989; 14: 1316–23.

Willén J, Anderson J, Tomooka K, Singer K. The natural history of burst fractures at the thoracolumbar junction. J Spinal Disorders 1990; 3: 39–46.