Review article

Management of thoracolumbar spine fractures with neurologic disorder

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ABSTRACT

Thoracic and lumbar fractures represent approximately 50% of neurologic spinal trauma. They lead to paraplegia or cauda equina syndrome depending on the level injured. In the acute phase, the extension of spinal cord lesions should be limited by immediately treating secondary systemic injury factors. Quick recovery of hemodynamic stability, with mean arterial blood pressure > 85 mmHg, appears essential. There is no clinical evidence in favor of high-dose corticosteroid protocols. Their effect on neurologic recovery is unproven, whereas they lead to a higher rate of secondary septic and pulmonary complications. Incomplete deficits (ASIA B-D) require urgent surgery. There is no consensus with regard to complete paraplegia (ASIA A), but early surgery can enable neurologic recovery in some cases. The principle of surgical treatment is based on spinal cord decompression, instrumentation and fracture reduction. Early stabilization of the spine improves respiratory function and shortens the duration of mechanical ventilation and thus intensive care unit stay. Depending on the severity of associated lesions, early surgery within 48 hours is beneficial in polytrauma patients. Percutaneous instrumentation combined with mini-open posterior decompression stabilizes the spine, limiting approach-related morbidity.

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1. Epidemiology of spinal cord trauma

Spinal trauma is frequent and shows neurologic complications in 15% to 30% of cases. The incidence of spinal cord trauma in France was estimated at 19.4 per million inhabitants [1]. In 2007, the French health authority (Haute Autorité de santé [HAS]) reported incidence of 1200 new cases per year and prevalence of about 50,000.

There is male predominance of 3:1, with frequency peaks between 16 and 30 years of age and, to a lesser extent, after 50 years [2].

Road traffic accidents represent 40% to 45% of etiologies, followed by voluntary or involuntary falls (15% to 30%), sport/leisure accidents (15% to 25%), work accidents and aggressions [2]. Young patients are mainly male with high-energy trauma, whereas falls are mainly implicated for older victims. Spinal cord trauma shows high rates of morbidity and mortality, increasing with age and number of associated injuries, especially cranial, thoracic or abdominal trauma [3].

Fractures with associated neurologic impairment mainly involve the cervical spine (> 50% of case), which is the most mobile and unstable segment.

The thoracic spine, involved in 20% to 30% of cases, is stabilized by the thorax and thus less exposed, although thoracic spinal cord vascularization is especially vulnerable.

The thoracolumbar junction (T12-L1) is involved in 15% of cases. It is a fragile area, concerned by half of all thoracic and lumbar spine fractures, at the junction between the relatively immobile thoracic kyphosis and the more mobile lumbar lordosis.

Lumbar-sacral junction fractures are rarer, but potentially unstable, thus representing a cause of neurologic impairment.

In 25% of vertebral fractures with neurologic deficit, there is another associated vertebral lesion [4,5].

In a study of 577 cases of neurologic spinal trauma, deficits were tetraplegia (43.3%), paraplegia (46.6%) or cauda equina syndrome (10.1%) [1].

2. Pathophysiology

2.1. Lesion mechanisms

Thoracolumbar spine fractures can damage the thoracic spinal cord, medullary cone and/or nerve roots.
Traumatic kyphosis with posterior wall displacement or dislocation of the spine can cause 4 different types of lesion, resulting from the initial impact, leading to neural or vascular tear:

- spinal cord concussion, which resolves in a matter of hours;
- spinal cord compression, inducing persistent spinal cord ischemia;
- spinal cord contusion, with axonal destruction and hemorrhagic infiltration;
- spinal cord sectioning.

There may be neurologic recovery in some cases of spinal cord compression, but little in contusion and none in sectioning [5].

2.2. Secondary lesion cascade

In the minutes following initial impact, a cascade of ischemic and enzymatic reactions gives rise to secondary spinal cord lesions. These evolve over several days to weeks, in 3 phases: inflammation, cicatization, and regeneration.

The initial hypoperfusion of the inflammatory edematous phase extends in a few hours to the gray and then white matter. Local consequences comprise blood-spinal cord barrier involvement and loss of the self-regulation of spinal cord perfusion; general consequences consist in systemic hemodynamic disorder, aggravating the spinal cord lesions.

At cell level, neuronal involvement is ischemic, with impaired microperfusion leading to hypoxia, increased glucose use, reduced ATP production and metabolic acidosis. The resultant complex biochemical cascade leads to excitotoxic amino acid (glutamate) release and ATP transport dysregulation, inducing intra- and extracellular calcium ion concentration imbalance. This in turn induces a release of inflammation mediators (prostaglandin) and free radicals by astrocytes and macrophages of the microglia, causing lipid peroxidation, protein oxidation and DNA degradation, leading to neuronal apoptosis [5,6].

These ischemic and biochemical phenomena have three fundamental implications:

- there is an ischemic “penumbra” of perinecrotic tissue that may evolve toward apoptosis or toward recovery;
- spinal cord (cerebral) injury factors (secondary cerebral stress of systemic origin [SCS-O]) such as hypotension, hypoxia, hypercapnia, anemia, hypothermia, acidosis, and hypoxo- and hyperglycemia induce lesion extension;
- there is a therapeutic window within which treatment can limit this extension.

Cicatization, involving astrocytes, occurs later, followed by neuron regeneration.

2.3. Spinal cord vascularization and perfusion

Spinal cord vascularization relies on three anastomosed longitudinal arterial axes: the anterior spinal and two posterolateral arteries. In the thoracolumbar region, the anterior spinal artery derives from the intercostal arteries, including the artery of Adamkiewicz (variable origin within T8-L3), which provides the principal irrigation and may be supplemented by an ascending radiculomedullary artery. Thoracic vascularization is precarious in comparison to that of the medullary cone [7].

In healthy spinal cord, self-regulation maintains constant blood-flow by means of vasodilation and vasoconstriction, with varying perfusion pressures of between 50 and 150 mmHg (spinal cord perfusion pressure = mean arterial pressure – intramedullary pressure).

With spinal cord lesions, intramedullary pressure increases due to edema, and the impairment of sympathetic innervation caused by the spinal shock reduces mean arterial pressure. Consequently, spinal cord perfusion pressure diminishes, inducing neuronal ischemia. There is here a fundamental implication for patient management, that mean arterial pressure should be kept above 85 mmHg for the first week [6,8].

3. Clinical examination and initial management

Management at the accident site is the same as in any severe injury or polytrauma.

If the victim is conscious, paralysis is easy to diagnose, although sometimes associated lesions may mask neurologic deficit.

Coma makes diagnosis tricky. When early intubation is required, only the physician of the emergency medical ambulance team (in France, SAMU) can assess neurologic status.

While freeing the victim and during transport to a spine center, the cranio-caudal axis must be immobilized so as not to worsen the displacement caused by the fracture and the neurologic lesions: the cervical spine is held in a rigid collar, and the trunk is moved en bloc and secured in a vacuum mattress.

Hypotension correction by solute perfusion should be initiated in the ambulance. The ambulance team alerts the specialized center to prepare admission.

In hospital, management is coordinated between intensive care physicians, surgeons and radiologists. Care in the resuscitation room begins by looking for vital organ lesions. Cerebral neurologic (Glasgow score), thoracic and abdominal examination is performed. The patient is placed under continuous hemodynamic and electrocardiographic surveillance.

Chest and pelvis X-ray and especially full-body CT scan are performed, plus biological assessment.

Neurologic examination can now be undertaken. The level of the sensory lesion is located by testing the dermatomes proximal and distal to the spinal lesion. The motor level may or may not be the same, depending on spinal cord involvement. All lower-limb muscles are tested. Motor examination above the lesion level is important, even when imaging is available, as there may be secondary extension of the spinal cord lesion. Various spinal cord syndromes may be encountered.

3.1. Central spinal cord syndrome

Central spinal cord lesions cause paraplegia. The sublesional syndrome comprises loss of muscle tone and flaccid paralysis, loss of all modes of sensitivity and osteotendinous reflexes, anal sphincter atonia, urinary retention and, in males, priapism. The Babinski sign appears only secondarily: it may be found during the acute phase in case of partial spinal cord compression, with incomplete sublesional syndrome.

3.2. Anterior syndrome

Anterior syndrome results from pure anterior spinal cord con- fusion, with ischemia in the territory of the anterior spinal artery. It comprises lower-limb paralysis (pyramidal tract lesion) and thermoalgesic anesthesia (anterolateral spinthalamic tract lesion) with conserved tactile and epicritic sensitivity (conserved posterior thalamic tract).

3.3. Posterior syndrome

Posterior syndrome is very rare in traumatology, involving proprioceptive, tactile and epicritic sensitivity.
Table 1
Test results for muscles and characteristic reflexes according to level of spinal cord involvement.

<table>
<thead>
<tr>
<th>Level</th>
<th>Muscle action</th>
<th>Reflex</th>
</tr>
</thead>
<tbody>
<tr>
<td>C5</td>
<td>Elbow flexion</td>
<td>Bicipital</td>
</tr>
<tr>
<td>C6</td>
<td>Wrist extension</td>
<td>Styloradial</td>
</tr>
<tr>
<td>C7</td>
<td>Elbow extension</td>
<td>Tricipital</td>
</tr>
<tr>
<td>C8</td>
<td>3rd phalanx flexion</td>
<td></td>
</tr>
<tr>
<td>T1</td>
<td>5th digit abduction</td>
<td></td>
</tr>
<tr>
<td>L2</td>
<td>Hip flexion</td>
<td></td>
</tr>
<tr>
<td>L3</td>
<td>Knee extension</td>
<td>Patellar</td>
</tr>
<tr>
<td>L4</td>
<td>Ankle extension</td>
<td></td>
</tr>
<tr>
<td>L5</td>
<td>Hallux extension</td>
<td></td>
</tr>
<tr>
<td>S1</td>
<td>Plantar flexion</td>
<td>Achilles</td>
</tr>
</tbody>
</table>

3.4. Brown-Séquard syndrome

Brown-Séquard syndrome consists of hemi-sectioning of the spinal cord, causing motor paralysis and ipsilateral tactile and epicritic sensitivity deficit with loss of contralateral thermoalgesic sensitivity, and is rarely typical in traumatology.

3.5. Assessment scores

The Frankel [9] and American Spinal Injury Association (ASIA) scores are superimposable, and indispensable for assessing lesion severity and monitoring evolution.

Motor scoring, from 0 to 5, is performed symmetrically according to the spinal cord level (Table 1) and sensitivity is assessed, by prickling or touching, as absent, reduced, normal or non-assessable, according to dermatome (Fig. 1).

This detailed examination determines the level and complete or incomplete nature of the spinal cord lesion, with a score between A and E (Table 2). Rectal examination is mandatory for differentiating between grades A and B.

Fig. 1. Dermatomes enabling assessment of involved spinal cord level for sensitivity.

Table 2
American Spinal Injury Association (ASIA)/Frankel score.

<table>
<thead>
<tr>
<th>Score: neurologic deficit</th>
<th>Examination results</th>
</tr>
</thead>
<tbody>
<tr>
<td>A: complete</td>
<td>No conserved sublesion motor or sensory function, notably in segments S4–S5</td>
</tr>
<tr>
<td>B: incomplete</td>
<td>Only sensory function conserved below neurologic level, sometimes in sacral segments S4–S5</td>
</tr>
<tr>
<td>C: incomplete</td>
<td>Motor function conserved below neurologic level and most key muscles below show motor scores &lt;3</td>
</tr>
<tr>
<td>D: incomplete</td>
<td>Motor function conserved below neurologic level and most key muscles below show motor scores ≥3</td>
</tr>
<tr>
<td>E: absent</td>
<td>Normal motor and sensory function</td>
</tr>
</tbody>
</table>

4. Imaging and fracture classification

After the clinical assessment and preliminary treatment (hemodynamic fractures), remote imaging of associated lesions, and specific spinal cord lesion imaging should be performed as quickly as possible.

4.1. CT

Full-body CT detects any intracranial, thoracic, abdominal or pelvic lesions that might be life-threatening.

Spinal bone reconstruction analyzes fracture type, displacement and degree of instability and any intracranial fragments.

4.2. MRI

Emergency MRI is recommended in spinal cord lesions, and is mandatory when the patient is unconscious, to assess the spinal cord lesion and the epi- or intra-dural hematoma.

Even so, we find less than 6 hours’ spinal cord decompression favorable to neurologic recovery in some patients. Therefore, if on CT the spinal lesion is clear and concordant with the neurological findings, in our opinion MRI can be dispensed with as wasting time to surgery.

MRI is useful in unstable disco-ligamentous lesions, which can be difficult to diagnose on CT, and is indispensable to screen for medullary hypersignal on T2-weighted sequences in trauma that appears normal on X-ray (spinal cord injury without radiological abnormality [SCIWORA]) or CT (spinal cord injury without obvious radiological evidence of trauma [SCIWORET]). These may be lesions caused by stretching, ischemia (vasospasm) or, in children, invagination of the yellow ligament or of cartilage fragments. In adults, trauma in hyperextension with canal stenosis or thoracic discal hernia may induce spinal cord compression (Schneider’s syndrome) [10].

4.3. Denis classification

The Denis classification [11] is based on the concept of the spine comprising three columns in the sagittal plane: anterior, middle and posterior (Fig. 2). The anterior column (anterior two-thirds of the vertebral body) is involved in compression fracture. The middle column (posterior third of the vertebral body and posterior longitudinal ligament) includes the posterior wall, involvement of which may lead to spinal cord compression by bone fragment migration into the canal. When the posterior column (pedicles and posterior arch) is involved, the articular and spinous processes and the ligaments are ruptured, creating an unstable lesion.
4.4. Magerl classification

The Magerl [12] or Arbeitsgemeinschaft für Osteosynthesefragen (AO) classification is the most widely used in Europe, and the classification we use. It is based on imaging analysis and takes lesion mechanism into account in three main groups:

- type A: vertebral body lesions caused by axial compression. The fractures liable to induce spinal cord compression are the so-called “split” type A2 (Fig. 3) and incomplete (A3.2) or complete (A3.3) “burst” fractures (Fig. 4);
- type B: lesions in flexion-distraction caused by posterior traction and anterior compression, mainly ligamentous (B1) or osseous (B2) (Fig. 5). Discal-ligamentous ruptures caused by hyperextension (B3) are difficult to diagnose on CT (Fig. 6) and require MRI;
- type C: lesions caused by torsion (Fig. 7). Type C1 also involves body compression, type C2 anterior flexion and posterior distraction, and type C3 an oblique fracture line. Neurologic complications are frequent.

4.5. Thoracolumbar Injury Severity Score (TLISS) classification

The TLISS classification [13] is based on a score from 1 to 10, leading to a recommendation for treatment. It takes account of fracture severity in terms of morphologic features (1 to 4 points), lesions of the ligamentous complex on MRI (0 to 3 points) and neurologic lesions (0 to 3 points).

5. Systemic treatment

5.1. Control of secondary injury factors

Control of secondary cerebral stress of systemic origin (SCSSO) seeks to limit exacerbation of spinal cord lesions.

Fig. 2. Denis classification: anterior, middle and posterior columns.

Fig. 3. Split (type A2) fracture with posterior displacement of the posterior wall.

Fig. 4. Burst fractures (types A3.2 and A3.3) with spinal cord compression.
Fig. 5. Chance fracture (type B2.1) by flexion-distraction: pure bone lesion in vertebral body (arrows), lamina and spinous process (star).

Fig. 6. Disco-ligamentous lesion in extension (type B3), evidence of gas in the canal and around facet joint lesion on CT (arrows); secondary displacement following discal tear (star) on fluoroscopy.

Fig. 7. T12-L1 dislocation fracture (type C2) with 2 vertebral bodies on axial view.
Volemic expansion by filling solutes, guided by the associated hemorrhagic lesions, is fundamental. Vasoconstrictors (noradrenaline) enable systolic pressure to be held at > 120 mmHg with a mean > 85 mmHg for 7 days. Even so, 75% of spinal cord trauma victims experience at least 1 episode of systolic pressure < 90 mmHg, inducing neuronal ischemia [6,8].

5.2. Corticosteroid-based protocols

The National Acute Spinal Cord Injury Studies corticosteroid-based protocols NASCIS I, II and III, intended for acute phase spinal cord trauma, are highly controversial. NASCIS III comprises an initial bolus of 30 mg/kg methylprednisolone sodium succinate, relayed by continuous infusion of 5.4 mg/kg/hr IV by electric syringe for 48 hours [14].

Anti-inflammatory action in the spinal cord and improvement in neurologic prognosis remain to be proven. On the other hand, the rates of septic complications, respiratory distress syndrome and pulmonary embolism are elevated following high-dose corticosteroids [6,15,16]. Moreover, corticosteroids can induce potentially neurotoxic peaks in hyperglycemia.

5.3. Neuroprotective treatment

Pharmaceutical research into inflammation following spinal cord trauma is ongoing.

Preliminary clinical studies of the neuroprotective action of certain drugs (riluzole, minocycline, anti-Nogo antibodies) are presently being conducted [6,16].

6. Surgical treatment

6.1. Decompression, reduction and stabilization techniques

Surgery for neurologic thoracolumbar fractures is based on 3 principles [17,18]:

- medullary or radicular decompression;
- fracture reduction by increasing lordosis;
- stabilization by posterior osteosynthesis (pedicle screws, rods and hooks) (Fig. 8).

Emergency posterior surgery requires the use of cell saver, as thoracolumbar fractures are very hemorrhagic (vertebral body bone bleeding and epidural venous bleeding).

Emergency anterior surgery allows removal of bone fragments from the dura, but the abundance of vertebral body vascularization can make surgery difficult and highly hemorrhagic – and thus dangerous in case of hemodynamic and respiratory fragility. We recommend the posterior approach, used in most centers.

6.1.1. Decompression

Canal decompression is performed by laminectomy of the fractured vertebra and overlying vertebra, the latter covering the compressed part of the canal. The laminae and spinous processes are oblique downward and backward, piling up like tiles in the thoracic level, then becoming more horizontal in the lumbar region. Extracting intracanal bone fragments requires wide exposure (resection of articular processes and sometimes of pedicles). Manipulation of the spinal cord is to be avoided, so as not to aggravate the spinal cord lesion. At the lumbar level, nerve roots may be mobilized to allow bone fragments to be removed.

6.1.2. Reduction

Positioning in lordosis enables reduction of some of the intracanal bone fragments by ligamentotaxis: the apex compressing the spinal cord is unfolded (Fig. 9). Prone positioning (taking care to avoid abdominal compression) allows partial fracture reduction by lengthening the anterior column. Cranial halo traction and lower-limb traction can enhance the ligamentotaxis effect. Reduction is completed by pedicular instrumentation. There are several techniques for lengthening the fractured segment and increasing lordosis.

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The AO reduction technique uses mono-axial Schanz screws. First, parallel screw distraction in the craniocaudal axis of the spine induces ligamentotaxis and reduction of posterior fragments. The screws are then inclined upward and downward, using a posterior center of rotation to restore lordosis.

In situ bending is based on a 90° angle between rods and mono-axial screws above and below the fracture. Rod lordosis is increased directly in situ using bending irons on either side. Reduction is achieved by simultaneously increasing anterior column length and lordosis.

“Persuaders” enable a rod pre-bent in lordosis to be introduced into the screw heads. Instrumentation over several levels, with slight hyperlordosis in the rod, pushes the spine anteriorly, inducing ligamentotaxis, distributing stress over all the pedicle screws.

6.1.3. Stabilization
In lumbar levels, mobile segments should be spared as far as possible without jeopardizing stability of instrumentation.

In lumbar levels, Magerl type A or B fractures are generally instrumented one level below and one above the fractured level. Type C fractures require a longer construct, sometimes instrumenting 2 lumbar levels (Fig. 8). Adding sub- and supra-laminar hooks or sublaminar banding increases instrumentation stability, reducing stress on the pedicle screws.

In T12 and L1 fracture, the thoracolumbar junction is bridged, with a cranial anchorage in T9 or T10. The caudal pedicle screws can be protected by sublaminar hooks.

In the thoracic region, which is relatively immobile, long instrumentation is recommended, over 3 levels over- and under-lying the fracture, to maintain reduction in the sagittal plane and prevent proximal junctional kyphosis.

In case of osteoporosis, vertebroplasty by cement injection via cannulated and laterally perforated pedicle screws is useful, increasing pullout resistance.

6.1.4. Fusion
The laminectomy product is systematically used for posterolateral bone grafting. The spinous processes are resected, and an iliac bone graft may be associated in case of fusion over several levels. It is essential to graft the unstable fractured levels and decompressed levels.

In the lumbar region, arthrodesis is limited to the unstable levels. In the others, the joint apophyses and their capsules should be preserved so as to maintain function when the instrumentation is removed. The mobility of each lumbar segment contributes to functional prognosis, whether the patient is walking or in a wheelchair.

At thoracic levels, arthrodesis is extended to the whole instrumented spine, as loss of mobility is negligible.

6.2. Dural tear repair
Intracanal bone fragments and trauma-related stretching can tear the dura mater.

The tear must be fully exposed for suturing with Prolene® 4.0 (Ethicon).

A thoracolumbar fascia patch can be used to cover dural defects and improve suture tightness.

Intracanal hematoma contains fibrin and promotes repair. Fibrin glue may also be used. The need for a cerebrospinal fluid drainage remains exceptional.

6.3. Anterior column reconstruction
Vertebral body lesion and discal tear may require secondary anterior reconstruction to ensure stability and maintain sagittal balance without loss of reduction. Untreated bone defects evolve toward non-union, with a risk of secondary rod breakage due to high stress levels in seated and standing positions.

A minimally invasive approach by thoracotomy, lumbotomy or video-assisted thoracophrenolombotomy enables anterior column reconstruction.
reconstruction with a cage containing bone harvested from the fractured vertebral body and the rib segment in the approach (Fig. 10). This technique avoids the need for a supplementary iliac graft [19].

Secondary anterior spinal cord decompression may be indicated if control CT shows dural sheath compression by large bone fragments. This indication is rare and limited to incomplete neurologic lesions in which recovery is possible.

When the cord has been released by laminectomy, a possible slight residual displacement of the posterior wall does not require anterior release, as the fragments held in place by the posterior longitudinal ligament undergo secondary remodeling.

6.4. Time to treatment and neurologic recovery

It is generally agreed that spinal trauma with incomplete spinal cord lesion (ASIA B-D) may evolve toward neurologic recovery if surgery is early, preferably within 6 hours of trauma [16–18].

In complete deficit (ASIA A), there is no consensus and opinions differ. The definition of “early” treatment varies from 8 to 24 hours in the literature. Some authors find no benefit for early surgery if paraplegia is complete [20–22]. McKinley et al. [23] consider early surgery to be without influence except to reduce postoperative complications and hospital stay. In contrast, Cengiz et al. [24] and Dendrinos et al. [25] consider early surgery to promote neurologic recovery.

A survey of time to surgery following spinal cord trauma, including 971 spine surgeons, found that more than 80% prefer operating within 24 hours [18] and consider that, with early surgery, incomplete neurologic lesions recover better than complete ones.

In our own experience, trauma-to-surgery time seems to be a fundamental factor in recovery [17]. It should be as short as possible if the patient’s condition and associated lesions permit. There is a race against time, in which all agents need to know their role so as to maximize the chances of recovery. The prognosis for recovery depends on the lesion mechanism. Emergency MRI enables the spinal cord lesions to be visualized [20] but changes the surgical indication only in case of spinal cord sectioning. Spinal cord compression and contusion cannot be definitely distinguished. Early surgery thus seems advisable to limit secondary extension to multiple spinal cord levels. Spinal cord lesions can be assessed on MRI in the first postoperative days.

Recovery prognosis also varies according to the region involved. The thoracolumbar junction seems to benefit from early surgery when trauma involves the medullary cone or cauda equina [15,17]. Thoracic fracture is often associated with other more serious thoracic lesions that may be life-threatening and require postponement of emergency decompression and stabilization. Thoracic segment involvement also seems to be a factor of poor prognosis, due to the fragility of spinal cord vascularization [7,21]. Finally, superior thoracic lesions impair sympathetic innervation and thus spinal cord hemodynamics and perfusion.

Patient age is predictive of neurologic recovery: younger patients recover better walking capacity following spinal cord trauma [26].

In practice, it is difficult to predict potential neurologic recovery in emergency, and chances should be maximized. Complete ASIA grade A thoracolumbar junction paraplegia can partially or completely recover in a few isolated cases [17]. Even if not, however, early osteosynthesis at least improves respiratory function and eases nursing care and early wheelchair rehabilitation [16,24].

7. Features of polytrauma

In polytrauma patients, all the lesions need to be taken into account and those that might be life-threatening need assessment.
Thoracic spine fracture may be associated with hemotherax, pulmonary lesions or aortic dissection.

At the thoracolumbar junction, liver or spleen lesions may contraindicate prone positioning.

When general health status and associated lesions permit, the spine should be stabilized within 48 hours. Early management improves respiratory function, and reduces mechanical ventilation time, secondary pulmonary complications and mortality [3,27,28]. It facilitates nursing and intensive care and shortens hospital stay.

Percutaneous osteosynthesis is interesting for polytrauma patients, reducing the morbidity associated with the surgical approach and bleeding. Minimally invasive osteosynthesis may be associated to a mini-open approach in laminectomy, taking the best of both percutaneous instrumentation and open spinal cord decompression.

8. Fractures in ankylosing spondylitis

Spinal fractures are 4 times as frequent in ankylosing spondylitis as in the general population, with an incidence ranging from 5% to 15%.

Involvement is usually of all 3 spinal columns, making the fracture especially unstable, with rates of neurologic complications ranging from 33% to 58% in thoracic and lumbar locations.

Diagnosis is often late, which is a major problem. This is due to the difficulty of interpreting imaging. Rates of neglect range from 19% to 60%, in which case early adapted immobilization is not implemented, leading to rates of secondary neurologic aggravation before treatment of up to 15% [29].

CT is the reference diagnostic examination. Diagnosis is clear in case of calcified anterior longitudinal ligament tear following trauma in extension with a “bamboo spine”, or if the fracture line is visible in the vertebral body and posterior column (Fig. 11). Some non-displaced fractures can be invisible, in which case, if there is enduring pain, bone scan may help reveal a recent bone lesion.

In such low-energy trauma, MRI can identify the site and age of the fracture, and should be systematically associated to CT. In the acute phase, MRI reveals post-traumatic edema in the vertebral body and posterior bone structures. Hypersignal is easily detected on T2-weighted STIR sequences, while the T1-weighted sequence shows the fracture line [30].

In fracture on ankylosing spondylitis, even when not displaced, a conservative attitude is to be avoided due to the risk of displacement and secondary neurologic complication. Moreover, braces are difficult to fit in these patients with rigid kyphosis.

Management of these unstable fractures is therefore often surgical, based on long posterior instrumentation, with multiple bone anchorage due to the low mineral density. In case of neurologic disorder, laminectomy should be associated.

9. Conclusions

Vertebral neurologic trauma is responsible for major morbidity and mortality.

The thoracic and lumbar regions are involved in 50% of fractures, with neurologic deficit, paraplegia or cauda equina syndrome depending on the region.

When managing a patient with a traumatic medullary injury, it is essential to treat secondary systemic injury factors so as to limit extension of spinal cord lesions. Systolic blood pressure should be kept above 120 mmHg and mean blood pressure above 85 mmHg par by volemic expansion and vasoconstrictors.

Corticosteroid-based protocols do not seem justified: impact on neurologic recovery is unproven and they induce septic and pulmonary complications and hyperglycemia which is a factor of secondary injury in spinal cord lesions.

Fig. 11. CT scan showing T8–T9 fracture in ankylosing spondylitis: oblique fracture line through all 3 columns (arrows); typical cortical rupture (star).
Neurologic involvement should be treated as a surgical emergency when incomplete and general health status permits. In complete deficit, there is no consensus, but surgery seems logical to promote neurologic recovery and avoid decubitus-related complications. Early stabilization of the spine also improves respiratory function and reduces mechanical ventilation time.

Surgical strategy is based on spinal cord decompression, osteosynthesis and fracture reduction.

Associated lesions permitting, surgery within 48 hours is of proven benefit in case of polytrauma.

Disclosure of interest

The authors declare that they have no conflicts of interest concerning this article.

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