LETTER

Ankylosing spondylitis complicated with traumatic thoracic fracture dislocation with old spinal cord injury

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A patient in their 50s with ankylosing spondylitis (AS) presented with paralysis attended to our department for evaluation and management. The patient was diagnosed with AS in their mid-20s, but the patient had never received standard medical therapy since then and the patient only received non-steroid anti-inflammatory drug when needed. The patient’s paralysis developed after a car accident 1 year ago, which caused cervical and thoracic fractures of the spine (figure 1A,B). Then, the patient underwent cervical surgery with open reduction and internal fixation by anterior stabilisation in the local hospital.

Figure 1 Images of the patient with ankylosing spondylitis. (A,B) CT performed after the accident revealed unstable fracture at the level of C5/6 and fracture dislocation at the level of T11/T12. (C,D) Postoperative positive and lateral position radiograph of the spine showing reduction and fixation of the cervical fracture. (E) CT images of the spine performed 1 year after the accident showed diffuse ligamentous fusion with bone destruction of the body of T11. (F) Three-dimensional CT performed 1 year after the accident revealed the fracture through body of T11 with forward displacement. T1-weighted scan (G), T2-weighted gradient echo scan (H) and T2 ‘fat-sat’-weighted scan (I) of the spine performed 1 year after the accident showed an altered signal intensity of the spinal cord of an old fracture dislocation of the thoracic spine, of which the spinal cord was mildly hypointense on T2-weighted images and mildly hyperintense on T1-weighted images. (J,K) Postoperative positive and lateral position radiograph of the spine showing fixation and fusion of the fracture dislocation with posterior spinal rods and screw.
(figure 1C,D). However, the patient develops severe pneumonia after the primary surgery. Therefore, the patient’s unstable situation could not enable a timely thoracic spine reduction and fixation. The patient selected conservative treatment for thoracic fracture dislocation. Until now, the patient’s neurological impairments of bilateral upper limb recovered to some extent but remained paraplegia.

To date, the patient has experienced paralysis and prolonged flat lying in bed for more than 1 year due to an unstable spine. The patient reported new fractures in the calf during the postoperative rehabilitation period of initial surgery. After admission to our department, CT of the spine revealed a displaced T11 fracture and complete spinal ankylosis (figure 1E,F) with extensively ossified ligaments, articular ossification and severe osteolysis of the vertebra at T11. MRI of the spine revealed bone destruction of the vertebra at T11 with a severely deformed spinal cord (figure 1H–J,G-I). Because decompression did not seem to be effective in such circumstances, the patient was only treated with stabilisation and fusion from the posterior direction (figure 1K,L,J,K).

AS is an immuno-mediated disease which is featured by osteoporosis and increased risk of fractures, and osteoimmunology-mediated increased bone resorption may play a vital role in this chronic process. Immune cells, including T cells and macrophages, can also interact with the skeletal system or bone cells to negatively promote bone healing and regeneration. Bone loss in the vertebrae occurs early in AS; therefore, vertebral fractures may easily occur in AS even with minor traumas and minimal force impact due to osteoporosis. Furthermore, prolonged bed rest and immobilisation could also have a huge adverse impact on bone quality, which may facilitate the loss of bone mineral and lead to disuse osteoporosis. Therefore, the fracture can hardly be healed or regenerated under this abnormal osteoimmunology status and osteoporosis. Furthermore, inflammation-mediated peri-inflammatory bone formation had shown to be increased in AS, resulting in healing of erosions, ossifying enthesitis and intervertebral connections. In contrast to this abnormal osteogenesis around enthesitis, we demonstrated the combined results of inflammatory bone remodelling processes of AS and mechanical effects of altered biomechanics after injury, which showed impaired bone healing and accelerated bone resorption in the fractured vertebra of AS and highlighted the importance of spinal stabilisation and fusion (figure 1A and E).

Injuries to the lower cervical spine are predominantly reported in AS; however, systematic imaging manifestations of traumatic thoracic fracture dislocation accompanied by old spinal cord injury in AS have been rarely reported. For a patient with fracture dislocation of the thoracic spine, it is crucial to protect neurological function by decompressing the spinal elements spontaneously to spare the spinal cord. In order to aid surgeons in therapeutic decision-making, an Arbeitsgemeinschaft für Osteosynthesefragen Spine Classification systems for the spine injuries had been generated based on the injury morphology patterns, which include compression injuries, tension band injuries and translational injuries. In our case, a timely surgery for reduction of the spinal cord and realignment of the spine should be conducted to prevent secondary injury to the spinal cord. However, the patient’s physical state could not enable such a surgery after the car accident. Therefore, we only treated the patient with stabilisation and fusion of the spine without spinal cord reduction. Therefore, our case was also unique in that we showed the systematic imaging manifestations of old traumatic thoracic fracture dislocation with old spinal cord injury in AS.

REFERENCES


