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Review

Management of spinal fractures in patients with ankylosing spondylitis

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ABSTRACT

Ankylosing spondylitis is a seronegative spondyloarthropathy that primarily affects the spinal column and sacroiliac joints. With disease progression autofusion of the spinal column takes place. This combined with the brittle bone quality make patients prone to fractures and spinal cord injury. The typical fracture pattern is extension type and involves all three columns. These fractures and injuries may involve the craniovertebral junction, the subaxial cervical spine, and the thoracolumbar spine. While at times these fractures are challenging to manage especially when they affect the elderly, there is evidence that supports long segment fixation and fusion. This article presents a narrative review on managing spinal fractures in patients with ankylosing spondylitis.

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1. Introduction

Ankylosing spondylitis (AS) is a seronegative spondyloarthropathy that primarily involves the vertebral column and the sacroiliac joints [6]. The disease has a characteristic caudal to rostral progression and overtime alters the strength and biomechanical properties of the spine through extensive remodeling involving ligamentous ossifications, vertebral joint fusion, osteoporosis and kyphosis. These changes lead to a brittle spine that is more susceptible to fractures affecting the three spinal columns thus resulting in spinal cord injury (SCI). AS is a chronic disease that typically starts before the age of 30, it has a slow but steady progression [13]. Typically spinal fractures occur mostly in patients with advanced

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Table 1
The modified New York criteria for the diagnosis of AS.

Diagnosis
Definite AS: 1 radiologic criterion + at least one clinical criterion
Probable AS: radiological criterion without clinical criteria, or three clinical criteria without radiological criterion
Clinical criteria
Inflammatory back pain: Low back pain of more than 3 months, improved by exercise, not relieved by rest
Limitation of lumbar spine motion in both sagittal and frontal planes
Limitation of chest expansion relative to normal values for age and sex
Radiological criterion
Sacroiliitis ≥2 bilaterally or grades 3–4 unilaterally

Table 2
MR grading of sacroiliitis according to Bennett et al.

Grade	Radiographic findings
0	Normal
1	Suspicious changes
2	Minimal abnormality: Small localized areas with erosion or sclerosis, without alteration in the joint width
3	Unequivocal abnormality: Moderate or advanced sacroiliitis with one or more of the following: erosions, evidence of sclerosis, widening, narrowing, or partial ankylosis
4	Severe abnormality: total ankylosis

age and therefore have inherently a poor outcome. Treatment of spinal fractures in AS is controversial. While older studies have deemed non-surgical management safer due to the high morbidity and mortality associated with operative management, more recent studies have demonstrated much better outcomes in patients treated surgically. In this review paper, we present the factors that lead to increased risk of spinal fractures in AS patients; we also discuss the management options of these fractures. We look particularly at injuries of the craniovertebral junction, the subaxial cervical spine and the thoracolumbar spine. We finally discuss strategies in place to prevent spinal fractures in AS patients.

2. Epidemiology

AS affects between 0.5 and 14 per 100,000 new people every year. Its overall prevalence is estimated to be between 0.1% and 1.4% [5]. Males are affected twice as often as females and in general tend to have more pronounced symptoms [13]. Some authors have argued that the prevalence of the disease is roughly equal in both genders, and that milder forms of the disease in women has led to underreporting of the condition in females and therefore to an exaggeration of the estimation of male predominance [49]. The disease is most prevalent in Northern European countries and seen least in people of Afro-Caribbean descent [19].

AS is the major subtype of the seronegative rheumatic spondyloarthritides group, which also includes reactive spondyloarthritis, psoriatic spondyloarthritis, spondyloarthritis associated with inflammatory bowel disease and undifferentiated spondyloarthritis [6]. These spondyloarthritides share a common genetic predisposition related to the major histocompatibility complex class I molecule HLA-B27. Though only 5% of HLA-B27 positive individuals develop AS, while 95% of patients with AS are positive for HLA-B27 [8,27]. This suggest that there is still unknown genetic or environmental factors that contribute to the development of AS.

3. Diagnosis

The diagnosis of AS is based on clinical and radiographic factors initially proposed in 1984 by van der Linden et al. as a modification to the original New York Diagnostic Criteria (Table 1) [27,45]. The modified New York criteria have a higher sensitivity and specificity; however, they may not be helpful for early diagnosis of AS and involvement of the sacroiliac joint remains the sine qua non for definite diagnosis.

Central to the diagnosis of AS are the presence of sacroiliitis and inflammatory back pain:

1. Sacroiliitis is defined on MR imaging and graded from 0 to 4 corresponding to findings ranging from normal to full blown total ankylosis (Table 2) [3].
2. Inflammatory back pain was studied by Rudwaleit et al. who developed diagnostic criteria to distinguish back pain related to spondyloarthritides from other types of back pain [38].

Fulfillment of two or more of the criteria listed in Table 3 is diagnostic of inflammatory back pain.

4. Pathological changes in AS

From a pathologic standpoint, AS is characterized by inflammation and new bone formation. Inflammation, mainly involves ligamentous insertion points throughout the axial skeleton, this is known as enthesopathy. This process promotes ossification of the affected ligaments. Ossification also involves the intervertebral discs, the endplates and the apophyseal structures. Extensive ossification leads to the formation of syndesmophytes, which span the ossified nucleus pulposus at each level. Throughout the slow disease process, acute and chronic spondylitis lead to remodeling of the vertebral bodies. Square vertebrae form as a result of continuous destruction and rebuilding of the cortex and spongiosa [2]. The combination of syndesmophytosis and squared vertebral bodies results in the typical hyperkyphotic “bamboo” spine.

AS is paradoxically associated with osteoporosis and low bone density despite an increased propensity for ossification [17,24]. This is thought to be secondary to uncoupling of bone formation and bone resorption. While, ectopic bone formation is induced by the inflammatory response, increased osteoclastic activity leads to unregulated absorption of bone within the vertebrae and therefore to a weak spinal column [17].

Progressive ligamentous ossification and syndesmophytosis result in a fused rigid hyperkyphotic spine, with tremendously altered biomechanics. Jacobs et al. compared the fused spine to a long bone that acts as a rigid lever and is unable to appropriately dissipate the energy of a traumatic event [23]. Unfavorable biomechanics, in addition to osteoporotic bone make the AS spine particularly susceptible to vertebral column fractures [14,23,48].

Additional factors that exacerbate the susceptibility of AS patients to spinal fractures include significantly impaired mobility directly related to their rigid spine along with variable degrees of joint arthritis that might increase the susceptibility to falls. This is supported by multiple case series that have shown that falls are the most common causes of fractures in AS patients [31,37]. Cooper et al. found that the odds ratio for vertebral fractures in AS patients is 7.7 when compared to the general population. They also noted a cumulative incidence of spinal fracture of 17% three decades after the diagnosis [12]. The most common mechanism of fracture being hyperextension, which, again, reflects the vulnerability of patient with AS to falls. When a fracture does occur in

Table 3
Inflammatory back pain criteria by Rudwaleit et al. fulfillment of two or more criteria is suggestive of inflammatory back pain.

Morning stiffness lasting more than 30 min
Improvement of back pain with activity and not rest
Awakening due to back pain during the second half of the night
Alternating buttock pain