ANKYLOSING SPONDYLITIS

A SUMMARY AND REVIEW

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Abstract: A review of the etiology, clinical, radiological and laboratory presentation, differential diagnosis and management.

Index Terms: Ankylosing spondylitis (AS), Marie-Strumpell spondylitis, Von Bechterew's disease, chiropractic.

Summary: Ankylosing spondylitis is a relatively rare disease affecting 0.1% to 0.2% of the white population (1)(2).

It is a disease characterised by inflammatory stiffening of the spine, affecting in particular the cartilaginous joints of the spine and the sacro-iliac joints. In the advanced form there is ossification of ligaments and spinal fusion with a characteristic stooped posture (3).

Ankylosing spondylitis occurs most commonly in white men in their twenties with the male:female ratio at 3:1 (2). There is a strong familial occurrence (8), and it is less common in American Negroes (4).

Ankylosing spondylitis is not a form of rheumatoid arthritis, although they do have similarities. Ankylosing spondylitis exists as a spectrum of clinical presentations both axial and appendicular (3).

Ankylosing spondylitis shares many of its features with other related seronegative (for rheumatoid factor) spondyloarthopathies, such as Reiter's Syndrome, psoriatic arthritis, reactive arthritis, enteropathic arthropaties (associated with ulcerative colitis, Crohn's disease and Whipple's disease) and juvenile chronic arthritis (5).

Approximately 90% of patients with ankylosing spondylitis have an association with the HLA antigen with B27 specificity, whereas only 6% - 8% of the population have the gene (6).

According to Ebringer (7) ankylosing spondylitis is probably produced by repeated episodes of Klebsiella-reactive arthritis, usually in HLA-B27 positive individuals.

He suggests a pathogenetic pathway commencing with gut wall infection produced by Klebsiella. Followed by antibody production against Klebsiella produced by local lymph nodes, related to the gut, sacro-iliac joints and the lumbar spine.

Then inflammation is produced because anti-Klebsiella antibodies bind to HLA-B27 positive cells in the spine, entheses (ligament and tendon attachments to bone) and lymph nodes thereby activating the so called "complement cascade".

This is followed by Klebsiella reactive arthritis which is produced by reoccurent waves of infection due to commensal Klebsiella, probably evoked by a high starch diet.

Finally, ankylosing spondylitis appears to be the end stage of repeated Klebsiella-reactive arthritis, occurring almost exclusively in HLA-B27 positive individuals (7).

The clinical picture of ankylosing spondylitis commonly presents as dull pain of insidious onset in the sacro-iliac region, lumbar region, greater trochanter region or even down the proximal part of the posterior thigh. Pain is usually aggravated by sudden movements and coughing and sneezing.

As the condition worsens back pain and stiffness occur at rest particularly in the morning. This may last several hours and is often relieved by heat and exercise (2.3,6,8).

Enthesitis causing extra-articular bony tenderness can be a common complaint particularly at the costo-sternal junctions, spinous processes, iliac crests, greater trochanters, ischial tubercles or heels (8).

Hip and shoulder joints are involved in one third of patients. Hip involvement as the presenting manifestation is more common in childhood or adolescence (9). Involvement of the peripheral joints is relatively infrequent (8).

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About 25% of all patients with ankylosing spondylitis develop at least one episode of acute iritis (3). Cardiovascular complications such as aortitis, aortic valve incompetence, pericarditis and cardiomegaly can occur but are rare. Pulmonary function remains normal generally due to an increased diaphragmatic contribution (3,6,8).

As ankylosing spondylitis progresses the patient becomes stooped into flexion and with cervical involvement the head is held in an anterior position and neck movements are restricted. Finally, bony ankylosis of the entire spine can occur resulting in profound stiffness and the so-called "bamboo spine" appearance as seen on radiographs (1,3,10).

Trauma to the ankylosing spondylitis patient may result in serious fractures and can result in severe neurological deficits (12). The fractures can occur even with minimal trauma and are due to spinal immobility and osteoporosis (12,13).

Differential Diagnosis: A differential diagnosis list includes rheumatoid arthritis, diffuse idiopathic skeletal hyperostosis (DISH), psoriatic arthritis, Reiter's syndrome, enteropathic arthritides, reactive arthritis, septic sacro-iliitis, Scheuermann's disease, congenital kyphoscoliosis, osteitis condensans ilii, hyperparathyroidism, osteoarthritis, gouty arthritis and infection (1,2,3,5,6,8,11).

Usual and Customary Examination Procedures:

1. Physical examination: This reveals loss of mobility of the lumbar spine. Note here that flexion may not be a reliable guide to a decreased lumbar range of motion due to hip movements (8).

   A decrease in chest expansion and a stooped kyphotic posture as the disease progresses are tell-tale signs(2,8). There is often tenderness over one or both sacro-iliac joints and the entheses (2).

2. Diagnostic Imaging: Plain radiographs are the imaging modality of choice although bone scans may be "hot" in affected joints when the disease is active (18).

   (a) Sacro-iliac joint involvement commences with pseudo-widening of joint spaces, then erosive and sclerotic changes particularly on the iliac side, and finally ankylosis of the joints which narrow and obliterate. Sclerosis dissipates and is replaced by generalised osteoporosis (14).

   (b) Spine: Syndesmophyte formation, which represents ligamentous ossification are the spinal hallmarks of ankylosing spondylitis, often commencing at the thoraco-lumbar junction but affecting eventually the entire spine to give the "Bamboo-spine" appearance (2,8,14).

   The apophyseal joints fuse, there is a loss of the lumbar lordosis and an increase in the thoracic kyphosis, eventually the spine becomes osteoporotic (2,3,4,8,13,14).

   (c) Peripheral Joints: Initially, the x-ray appearance of proximal joints in ankylosing spondylitis may resemble rheumatoid arthritis. However, there is a greater tendency in ankylosing spondylitis to central articular erosion's and proliferative new bone formation at the margins and whiskery spicules occur due to enthesitis in the pelvis at the sacrotuberious and sacrospinal ligament insertions, the greater trochanter of the femur, plantar fascia and achilles tendon (6).

3. Laboratory Tests:

   (a) ESR/CRP is raised in up to 75% of patients but it is not a reliable indicator of disease activity (6,8).

   (b) Mild normocytic, normochromic anaemia may be present in 15% of patients (8).

   (c) HLA-B27 is positive in over 90% of ankylosing spondylitis patients (2).

   (d) A positive rheumatoid factor is notably absent (4).

GOALS OF TREATMENT:

1. Control pain and inflammation.
2. Maintain maximum skeletal mobility.
3. Prevent deformities.
4. Maintain or enhance aerobic fitness, muscular strength and endurance.
5. Educate patients and family about the disease.

CHIROPRACTIC MANAGEMENT:

1. Analgesic and anti-inflammatory physical therapy modalities can be used for exacerbations.

2. An active exercise programme is the cornerstone of management (15). Rest should only be prescribed during acute exacerbations. Exercises should be aimed at spinal ranges of motion particularly extension. Swimming is ideal in this regard and it also is performed without weight bearing and enhances aerobic capacity.

Cycling and jogging are to be condoned only with reservation, cycling because of the flexed posture and jogging because of the jarring effect.
Breathing and chest expansion exercises are also important (2.3.4.5.6).
Heat applied before and cold applied after exercises may be useful (3).

3. The use of mobilisation and manipulation in ankylosing spondylitis is yet to be studied. However, two factors should be borne in mind:

   (a) Ankylosing spondylitis patients like any other patient may develop mechanical spinal dysfunction which may respond to mechanical treatment.

   (b) The main contraindications to mechanical treatment are: ankylosing spondylitis in the actively inflamed stage where treatment might exacerbate an already inflamed area, joint immobility or fusion and any attendant osteoporosis where vigorous treatment could cause bony damage particularly fracture and atlanto-axial instability from the disease process itself where treatment might conceivably result in disastrous sequelae.

   Certainly, gentle techniques in the quiescent phase of early ankylosing spondylitis would not seem to be contraindicated where a manipulable lesion exists.

4. Periodic review is important to gauge the progression of the disease. In this regard clinical and x-ray tests are used. Clinical indicators include measuring pain and inflammation: pain can be assessed using a visual analogue scale while inflammation can be gauged by the duration of morning stiffness (eg. 2 hours) (16).

   Mobility of the spinal column should be measured checking lateral flexions, extension and rotations. Flexion should be measured using the Schober method (8). Chest expansion should also be measure suing reproducible methods (8).

   Two other indices of disease progression are the enthesopathy index proposed by Mander et al (16) and the articular and functional indices proposed by Dougados et al (17).

   Serial radiographs are useful to monitor the disease progression but may not correlate to any favourable effect of treatment (8).

5. Anti-inflammatory drugs are the most useful medical therapy. They suppress the pain and stiffness and facilitate exercise programmes (3). However, drug therapy does not alter the progression of the disease (6). The aim of prescription is to find the smallest dose necessary for repeated symptomatic relief (5).

6. Counselling and education for patients and family regarding ankylosing spondylitis should be an integral part of management (3.15).

PROGNOSIS:

Although not curable, ankylosing spondylitis is one of the most rehabilitable of all the chronic rheumatic diseases (3). Less then 10% develop relentlessly crippling disease. Most longitudinal studies of ankylosing spondylitis survival curves approximate that of the general population (6). Death if it occurs is usually attributable to cardiac involvement, cervical spine fractures or rarely secondary amyloidosis (2).

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