

Did you know that there may be a delay of up to 10 years between symptom onset and the diagnosis of ankylosing spondylitis?^{1,2}





A guide to the early identification of Ankylosing Spondylitis (AS) The development of this educational booklet was funded by

ABOUT THIS BOOKLET

This booklet has been designed to assist the primary healthcare provider to identify patients with possible ankylosing spondylitis (AS) early in their disease. The clinical outcome of this disease is variable, but its severity is defined by its course within the first 10 years of symptom onset and patients have been shown to benefit considerably from early rheumatological referral.^{3,4}

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WHAT IS ANKYLOSING SPONDYLITIS?

Ankylosing spondylitis (AS) is a form of chronic, painful arthritis that primarily affects the spine, although other joints may also be affected.^{5,6} AS and non-radiographic axial spondyloarthritis (SpA), an early presentation of AS, are together referred to as axial SpA.⁷ The spectrum of axial SpA, depicted in the diagram below, is delineated by the presence or absence of structural damage visible on x-ray.⁷

Axial spondyloarthritis (SpA)



It should be noted that not all patients evolve from non-radiographic axial SpA to AS; studies suggest 8-10% after 2 years and 59% after 10 years.⁷ Adapted from: Rudwaleit and Sieper 2012. *Nat Rev Rheumatol.* 8;262-68.

In non-radiographic axial SpA, acute inflammatory lesions may be visible on MRI only.^{8,9} In AS, structural changes in the sacroiliac joints are visible on x-ray and in advanced AS inflammation of the facet joints of the spine may also be present.¹⁰ **Such inflammation may lead to new bone formation, causing the spine to fuse in a fixed, immobile position, sometimes resulting in a forward-stooped posture.**^{3,11}

Axial SpA is associated with extra-articular manifestations, the presence of HLA-B27 and a positive family history of the disease.^{5,6,12}

Comorbidities associated with axial SpA include cardiovascular manifestations, osteoporosis and infrequently, impaired pulmonary function.¹³⁻¹⁵

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WHO IS AFFECTED BY ANKYLOSING SPONDYLITIS?

The prevalence of AS in New Zealand is unknown; however, based on prevalence data from Europe (0.238%), Asia (0.167%) and North America (0.319%),¹⁶ **local rheumatologists** estimate that between 7,000 and 12,000 New Zealanders suffer from AS.

Who is most commonly affected by ankylosing spondylitis?

- AS generally affects adults and the mean age of symptom onset is the mid-twenties.¹⁷
- AS is more common in men (65-80%).18
- Individuals positive for HLA-B27 are at increased risk, with between 82% and 95% of patients with AS carrying this gene.^{18,19}
- There is an increased incidence of AS in individuals with first-degree relatives with AS, or a personal or family history of psoriasis, acute uveitis, reactive arthritis or inflammatory bowel disease (IBD).²⁰





DIAGNOSING ANKYLOSING SPONDYLITIS

The onset of AS may be insidious and the clinical signs subtle, contributing to the frequent 5-10 year delay between the first appearance of symptoms and the diagnosis of this disease.^{1,2}

Over time, inflammation in the spinal joints may lead to bone formation and subsequent spinal fusion, resulting in reduced spinal mobility and a significant reduction in quality of life.^{11,21} It is therefore essential that AS is recognised and treated early.²¹

The most common and prominent clinical

symptom of AS and its early form (nonradiographic axial SpA) is chronic inflammatory back pain (IBP), and recognising this feature is key to early diagnosis.

IBP is present in up to 90% of patients with axial SpA.²⁰

Other clinical features include:6

- arthritis
- extra-articular manifestations (e.g. anterior uveitis, IBD, psoriasis, dactylitis and enthesitis)
- a good response to non-steroidal anti-inflammatory drugs (NSAIDs) and
- a family history of SpA.

Laboratory features of axial SpA include:

- HLA-B27 positivity
- elevated CRP and ESR levels (although around 30-50% of patients with axial SpA have normal levels).^{18,19}

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Patients who present with inflammatory back pain and a history of any of the extra-articular manifestations of AS (either in themselves or family members) should raise the suspicion of AS.¹⁰

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INFLAMMATORY BACK PAIN – A KEY SYMPTOM OF ANKYLOSING SPONDYLITIS

IBP is present in up to 90% of patients with axial SpA.²⁰ Such pain, along with stiffness, usually develops gradually over time, is especially bad at night and improves with exercise.¹⁰

Differentiating between inflammatory and mechanical back pain:

Lower back pain is very common, with 60-80% of the population experiencing this condition at some stage in their life.²² In most cases the cause of the pain is mechanical.²³ However, in approximately 5% the cause is inflammatory.²⁴ Mechanical back pain is due to structural changes in the spinal joints, vertebrae or soft tissues, and while it may be chronic (lasting >3 months), it is usually acute in onset and self-limiting.²³ In contrast, IBP may arise from an underlying inflammatory rheumatic disease and lasts for >3 months.²⁵ IBP usually develops in the third decade of life and is unlikely to begin after age 45.²⁵

Chronic IBP is the leading symptom in patients with AS²⁵

During a physical examination the following key features may indicate IBP caused by AS or non-radiographic axial SpA:





INFLAMMATORY BACK PAIN

- Age of onset: <40 years
- Insidious onset: less likely to be acute
- Pain improves with
 exercise
- Pain does not improve with rest
- Pain at night which may wake patient during second half of the night
- Morning stiffness greater than 30 minutes

Adapted from NASS Module One. "Differentiating Inflammatory and Mechanical Back Pain," August 2012. (http://nass.co.uk/loose-leaf-pages/differentiatinginflammatory-and-mechanical-back-pain/)

MECHANICAL BACK PAIN

- •Age of onset: any age
- •Variable onset: may be acute
- Pain may worsen with movement
- Pain often improves with rest
- Morning stiffness less than 30 minutes

According to Assessment of SpondyloArthritis international Society (ASAS) experts' criteria, IBP in those with chronic back pain (>3 months) is likely if the patient exhibits at least four of the following: age at onset <40 years; insidious onset; improvement with exercise; no improvement with rest; pain at night (with improvement upon getting up).²⁵

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DO YOU SUSPECT ANKYLOSING SPONDYLITIS?

The following Ankylosing Spondylitis Action Pathway (ASAP) is a referral guide to help you decide whether to refer a patient to a rheumatologist.





ARTHRITIS NEW ZEALAND Adapted from the following: Sieper J et al. Ann Rheum Dis 2013;72:1621-7; Sieper J et al. Ann Rheum Dis 2009;68:784-8; AS Awareness Council (ASAC) Ankylosing Spondylitis Action Pathway (http://www.isrie/images/stories/ AS_Algorithm_form.pdf); by Dr Douglas White, Associate Professor Andrew Harrison, Dr Tracey Kain and Dr Rafi Raja.

TREATING ANKYLOSING SPONDYLITIS

NSAIDs and exercise while waiting for a specialist

While waiting for an appointment with a rheumatologist, it may be beneficial to start the patient on NSAID treatment unless contraindicated (and consider anti-ulcer treatment if required) and commence an exercise regime supervised by a physiotherapist²⁷ (see www.nass.co.uk/exercise).

How is ankylosing spondylitis treated?

Pharmacological therapy for AS should always be combined with non-pharmacological treatment (education, regular exercise/physiotherapy, rehabilitation, patient associations, self-help groups).⁴

The ASAS/EULAR recommendations for the treatment of AS are:

- NSAIDs + non-pharmacological therapy as first-line therapy²⁸
- TNF-antagonists as second-line therapy²⁸

TNF-antagonists for the treatment of AS are subsidised by PHARMAC on Special Authority when prescribed by a rheumatologist.

Getting a diagnosis from the rhenmatologist finally gave me something that explained my <u>condition.</u>*

> Alice, Wellington Diagnosed with AS at age 15. *This is the experience of an individual patient and may not apply to all patients.

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ASSESS YOUR KNOWLEDGE OF INFLAMMATORY BACK PAIN

(Answers are at the end of the survey.)



- a. 60 year old male with back pain for less than 3 months, which causes pain at night and sleep disturbances
- b. 34 year old male with back pain for 5 years, which is worst at night
- c. 25 year old female with chronic back pain, which is eased with rest
- d. 29 year old male with back pain for 3 months, which is worsened by exercise





Adapted from NASS Module One. "Differentiating Inflammatory and Mechanical Back Pain," August 2012. (http://nass.co.uk/loose-leaf-pages/differentiating-inflammatory-and-mechanical-back-pain/)

Answers to questions: 1:b; 2:a; 3:a,c,d; 4:b; 5:d; 6:b Further information on AS can be found at: <u>www.arthritis.org.nz</u> <u>www.nass.co.uk</u> <u>https://itunes.apple.com/nz/app/iankylosingspondylitis/id414586259?mt=8</u>

Brandt HC et al. Ann Rheum Dis. 2007;66(11):1479-84 2. Feldtkeller E et al. Rheumatol Int. 2003;23(2):61-6 3. Carette S et al. Arthritis Rheum. 1983;26(2):186-90
 Poddubnyy D. Ther Adv Musculoskelet Dis.2013;5(1):45-54 5. van der Heijde D et al. Arthritis Rheum. 2006;54(7):2136-46 6. Ehrenfeld M. Best Pract Res Clin Rheumatol. 2012;26(1):135-45 7. Rudwaleit M and Sieper J. Nat Rev Rheumatol. 2012;8(5):262-8 8. Bennett AN et al. Arthritis Rheum. 2008;58(11):3413-18 9. Sieper J and van der Heijde D. Arthritis Rheum. 2013;65(3):543-51 10. Sieper J et al. Ann Rheum Dis. 2009;68(Suppl II):ii1-ii44 11. Carter S et al. Ther Adv Musculoskelet Dis. 2012;4(4):293-9
 Khan MA. Ann Intern Med. 2002;136(12):896-907 13. van der Horst-Bruinsma IE and Nurmohamed MT. Ther Adv Musculoskelet Dis. 2012;4(6):413-22 14. Bessant R and Keat A. J Rheumatol. 2002;29(7):1511-9 15. Dincer U et al. J Exp Med. 2007;212(4):423-30 16. Dean LE et al. Rheumatology 2014;53(4):650-7
 Sieper J and Rudwaleit M. Ann Rheum Dis. 2005;64:659-63 18. Sieper J et al. Ann Rheum Dis. 2002;61:iii8-iii18 19. Rudwaleit M et al. Arthritis Rheum. 2009;60(3):717-27 20. Rudwaleit M et al. Ann Rheum Dis. 2009;68(6):777-83 21. Machado P et al. Ann Rheum Dis. 2010;69(8):1465-70 22. Waddell G and Burton AK. Occup Med (Lond).2001;51(2):124-35 23. Chien JJ and Bajwa ZH. Curr Pain Headache Rep. 2008;12(6):406-11 24. Weisman MH et al. Ann Rheum Dis. 2003;72(3):369-73
 Sieper J et al. Ann Rheum Dis. 2009;68(6):777-83 24. Machado P et al. Ann Rheum Dis. 2010;69(8):1465-70 22. Waddell G and Burton AK. Occup Med (Lond).2001;51(2):124-35 23. Chien JJ and Bajwa ZH. Curr Pain Headache Rep. 2008;12(6):406-11 24. Weisman MH et al. Ann Rheum Dis. 2013;72(3):369-73
 Sieper J et al. Ann Rheum Dis. 2009;68(6):784-8 26. Rudwaleit M et al. Arthritis Rheum. 2006;54(2):569-78 27. Pharmac Schedule (Section H) June 2014; page 136 Available from: http://www.pharmac.govt.nz/2014/06/01/HML.pdf 28. Braun J et al. Ann Rheum

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d. Tell patient to rest





