

Abstract

Introduction

Ankylosing spondylitis is an inflammatory disorder, which mainly involves spine and peripheral large joints of young adults. The disease modifying treatment of ankylosing spondylitis could be complicated with adverse reactions of medication. Here, I present a case of ankylosing spondylitis complicating drug induced liver injury requiring modification of highly effective treatment strategies.

Case presentation

A 17-year-old boy developed severe inflammatory back pain associated with peripheral large joint arthritis for 4 months duration. His Erythrocyte sedimentation rate was 64mm/1st hour and magnetic resonance imaging findings were compatible with bilateral sacroiliitis. HLA-B27 testing was positive and Anti-nuclear antibody was negative. His clinical presentation and supportive investigation findings, and in the absence of other features to suggest alternative cause of sero-negative spondyloarthritis, we made the diagnosis of ankylosing spondylitis. First line therapy of indomethacin in combination with sulfasalazine was started. However, this was complicated by the development of drug-induced hepatitis. His Mantoux test was reactive in the absence of active tuberculosis necessitating Isoniazid therapy prior to Anti-TNF alfa treatment. However, INAH could not be administered as patient had deranged liver enzymes. Therefore, an alternative approach of azathioprine and tramadol as required were commenced with a good response.

Conclusion

Axial and peripheral arthritis of ankylosing spondylitis well respond to non-steroidal anti-inflammatory drugs and non-biological disease modifying agents. Biological treatment with Anti-TNF alfa agents, which are associated with risk of reactivation of latent tuberculous infection, are used in selected cases. The clinician has to have alternative therapeutic approach where the biological agents cannot be used due to risk of reactivation tuberculosis outweigh the benefit of such agent.

Keywords: Ankylosing spondylitis, sero-negative spondyloarthritis, drug induced liver injury, Anti TNF alfa agents.