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Association of *Chlamydia trachomatis* and HLA B27 in ankylosing spondylitis in an unmarried sexually active young adult

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Abstract

This case report highlights the association of *Chlamydia trachomatis* infection leading to development of ankylosing spondylitis. A young promiscuous adult presented with a bubo in left groin for 10 days in STI clinic in Government Medical College Hospital, Nagpur. He gave history of urethral discharge thrice in a year. He also had low backache for 3-4 months and recently developed pain in the left knee. Nonspecific investigations revealed raised inflammatory markers and IgM and IgG antibodies against *C trachomatis*. *C trachomatis* antigen was demonstrated by direct fluorescent antibody test. The infection responded to 21-day course of doxycycline. Joint pain responded to non-steroidal anti-inflammatory drugs.

Keywords: Ankylosing spondylitis *Chlamydia trachomatis*, HLA B27

Introduction

Chlamydia trachomatis antigens or antibodies in patients with ankylosing spondylitis (AS) could show its aetiological role in this condition [1]. The prevalence of HLA B27 varies between populations, from nil in Australian aborigines to 50% in Haida Indians [2]. The frequency among North Indians is more than 5% compared to less than 5% in South Indians [3]. This gene makes the person vulnerable to AS, but many do not develop the condition. HLA B27 is found in 89% of patients of AS, but the reverse is not true [4]. The condition is triggered by one or more environmental factors like injury to bowel or bacterial infections, though all may not be known [5].

We report a case of lymphogranuloma venereum caused by distinct serovars of *C trachomatis* with ankylosing spondylitis.

Case Report

A 20-year-old male presented in a clinic for Sexually Transmitted Infection (STI) in the Department of Dermatology, Venerology & Leprology, Government Medical College, Nagpur with a painful swelling in groin for about 10 days. He complained of low back pain for 3-4 months and felt the pain in left knee also, the duration of which was incomprehensible.

Past History

The patient gave history of urethral discharge on three occasions within a period of one year for which no treatment had been taken. Each time the discharge subsided gradually. The patient had unprotected and multiple sexual exposures with multiple known and unknown female partners in the past 2-3 years. He did not have sex with men.

There was no history of fever, oral ulcer, conjunctivitis or diarrhoea. There was no history of fall or trauma. Patient had taken pain killers for low backache prescribed by local practitioner.

Examination

The patient was afebrile, was not distressed but also was not at ease. He was mildly anaemic, there was no ocular signs or symptoms. Pulse rate and respiratory rate was within normal limits.

Respiratory, cardiovascular, nervous and gastrointestinal systems were not contributory. Genitourinary system showed an enlarged lymph node (bubo) in the left groin of size 2 cm X 1 cm, soft and tender. Urethral discharge was not present. There was no genital papule, ulcer or wart.

The musculoskeletal system showed tenderness over sacroiliac region with no restriction in mobility. There was mild inflammation of left knee.

Investigations

In blood examination, RBCs showed mild microcytosis and mild hypochromia, haemoglobin was 8.4 gm%. WBC count was 8400/cm. Platelets were adequate. ESR was 44 mm/ 1 hour. CRP was raised. HLA typing was positive for HLA B27.

Gram stain of pus from bubo was full of polymorphs with no organisms. Direct fluorescent antibody (DFA) test was positive for *C trachomatis* antigen (Fig 1).

Serum was positive for both *C trachomatis* IgM and IgG antibody.

Tests to rule out other STIs i.e. urine culture for *Neisseria gonorrhoeae* was negative, VDRL and HIV antibody tests were both nonreactive.

X ray was not done.



Fig 1: Direct fluorescent antibody test demonstrating bubo pus studded with unusually large no. of equally sized and equally intense elementary bodies of *Chlamydia trachomatis*

Diagnosis

A case of lymphogranuloma venereum caused by distinct serovars of *C trachomatis* with ankylosing spondylitis.

Treatment

Treatment given was Tablet Doxycycline 200 mg BD for 21 days and non-steroidal anti-inflammatory analgesics.

Patient was counselled for abstinence during treatment, to practice safe sex and was educated about progress and complications of his current STI and also backache. He was advised life-long exercise and regular follow-up.

Outcome

The patient returned after 15 days with markedly resolved bubo with relief in backache and joint-pain. Thereafter he did not return.

Discussion

The diagnosis of *C trachomatis* infection was based on typical clinical presentation, a positive DFA test and serology. DFA is known to give false positive results and serological diagnosis is not specific. However in the present case bubo pus was studded with unusually large numbers of the fluorescent bodies (Fig 1) which cannot be taken as false positive. Specific diagnosis for genotyping by NAT is recommended but this was our limitation^[6].

The differential diagnosis of AS in early stages is lumbar disc lesion. This was ruled out as there was no history of fall or trauma. However, the age and sex of the patient favoured AS together with *C trachomatis* infection prior to development of low backache in the patient.

The triggering factor for development of AS in the patient was *C trachomatis* infection^[7], which was acquired because of patient's promiscuous behaviour. To link *C trachomatis* infection with AS, it is important to demonstrate sequence homology between HLA B27 and serovar L1, L2, L3 and also characterise monoclonal antibody reacting with these antigens. Though these tests were outside the scope of our laboratory, if performed, they could have proved molecular mimicry as the mechanism of pathogenesis of AS.

The structural basis of AS is the glycosylated pattern of foreign antigens, that lingers as non-biodegradable fragments in the joints inducing alkalosis that is the stiffening of joints.

Conclusion

To reduce the frequency and intensity of the symptoms of AS, in a person who is positive for HLA B27, the triggering factor for HLA B27 should be elicited by history taking, examination, and investigations. The management of such triggering factors is an essential component of the management of AS.

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