TUBERCULOSIS: A CASE REPORT

K Jain, Staff Surgeon, H Adhikary, Consultant, Ophthalmology
Royal Preston Hospital

INTRODUCTION

In recent years tuberculosis has re-emerged as a serious public health problem[1]. In the western world this is due in large part to an influx of immigrants from countries where the prevalence of tuberculosis remains high[2,3] and also to the HIV pandemic. Tuberculosis has been described as a systemic disease with protean manifestations. The predominant route by which tubercle bacilli reach the eye is through the bloodstream, after infecting the lungs. The pulmonary involvement may not be evident clinically or radiographically. Tuberculosis develops in approximately 10% of all infected persons[4]. It is thought to result from altered T cell or macrophage function or both. Miliary tuberculosis is a fulminating multisystem disease resulting from widespread haematogenous dissemination of tuberculous organisms. It may occur either at the time of primary infection or with reactivation of latent tuberculosis.

CASE REPORT

A 40-year-old Asian male of Indian origin, a goldsmith by occupation, presented with a history of blurred vision of two weeks' duration in the left eye. On examination the best-corrected visual acuity was 6/6 in the right eye and 6/18 in the left. Slit lamp bio-microscopy showed anterior chamber flare 1+, cells 1+ and an exudative membrane at the pupil. There were no keratic precipitates and the fundus examination was normal. He was diagnosed and treated for iritis with topical dexamethasone (0.1%) four times a day and cyclopentolate (1%) twice a day. Two weeks later the signs and symptoms subsided and vision returned to 6/6 in the left eye. A week later, with the same treatment, the left eye became quiet and at this stage treatment was tapered off. He was advised to return if he developed symptoms of redness, pain, photophobia or blurred vision.

Six months later he presented with a history of floaters, photophobia and blurred vision (left eye more than right). On examination the best-corrected visual acuity was 6/6 in the right eye and 6/12 in the left eye. The near vision was N6 for each eye. Mild flare and cells were noted in both eyes (left more than right) with no keratic precipitates and freely mobile pupils. Fundus examination of the left eye showed vitreous haze with white exudative snowballs inferiorly around the equatorial region. There were no snowballs at the *pars plana* on scleral depression. Blood vessels and maculae were entirely normal.

A detailed past history revealed that he had multiple symptomatology. He had had intermittent backache for a year, mild chest pain on and off for eight years, pins and needles in the hands on and off for 18 months and sexual impotency for a year. For all these symptoms he attended various departments with no specific diagnosis and was treated symptomatically. He also gave a history of lost appetite and occasional night sweats for the last two years. He had a BCG vaccination scar on his left arm. His father had tuberculosis and was treated in India in the past.

Full blood count, ESR, FTA-ABS, VDRL, auto antibody screen, toxoplasmosis titre (ELISA), serum angiotensin converting enzyme, serum lysozyme, liver function tests, HLAB27, urine culture, throat swab culture, chest X-ray and radiographs of sacro-iliac joints were all found to be within normal limits. The tuberculin skin test on the right forearm was positive (grade IV after 48 hours). The patient refused an aqueous tap to detect acid fast bacilli. In view of the multiple symptoms and positive tuberculin skin test, it was decided to put him on a therapuetic trial of antituberculous drug treatment. He was given triple therapy (pyrazinamide 2g, rifampicin 600mg, isoniazide 300mg, pyridoxine 10mg) for the first three months followed by a single drug (Rifampicin 600mg daily) for another nine months and was monitored by a chest physician and followed up at the eye clinic.

Six weeks after initiation of triple therapy, his visual symptoms started regressing. At three months all his ocular symptoms and signs disappeared except for an insignificant floater due to vitreous changes in the left eye. At six months all his systemic symptoms had disappeared. His appetite increased and his sexual life returned to normal. He was followed up for another five years and has not had any recurrence of ocular or systemic symptoms.

DISCUSSION

Tuberculosis[5] should be considered in the differential diagnosis of all cases of uveitis. The diagnosis is usually a presumptive one as bacteriological and histological samples are rarely available to ophthalmologists. The clinical picture can take many forms and the inflammation does not have to be granulomatous in nature[6]. The tuberculin skin test[7] plays an integral part in the diagnosis and its proper interpretation is important. The therapeutic trial as suggested by Schlagel[8] in cases of presumed uveitis of tuberculous origin plays an important role in further management of this type of case. A missed early diagnosis can lead to disastrous complications and the loss of an eye[9,10]. Monotherapy[11] may fail to treat the signs and symptoms in cases of resistant strain; hence a combination of three drugs is more helpful in deciding the effectiveness of the initial therapeutic trial, as in this case. The diagnosis of presumed tuberculosus uveitis in this case was suggested by the exclusion of other causes of uveitis by laboratory investigation, the recurrent nature of the inflammation, the history of lost appetite and night sweats, the family history of tuberculosis and the multiple symptoms. This case had vitritis and non-granulomatous iritis and did not show any sign of choroidal inflammation or periphlebitis, commonly seen in tuberculous patients. Thus it is important to recognise this rare sign of its presentation as in this case. The involvement of multiple body systems suggested that the

patient harboured tuberculosis in sub-clinical form. This was further supported by effective antituberculous treatment which resulted in the disappearance of his symptoms. This patient had a BCG vaccination scar yet still developed a tuberculous unveitis. BCG vaccination[12] is effective in preventing tuberculosis in 70% of cases, provided that it has been given to tuberculin skin test negative patients. If a patient has already contracted the disease BCG vaccination may not be beneficial.

CONCLUSION

This case emphasises the important role of the physician and ophthalmologist in the diagnosis and management of sub-clinical cases of tuberculosis. Any unexplained symptoms involving multiple body systems in an Asian immigrant should be fully investigated for tuberculosis and when in doubt one should consider a therapeutic trial of an antituberculous treatment in the presence of positive tuberculin skin test. It is difficult to arrive at tuberculous aetiology in sub-clinical cases and the antituberculous therapeutic trial plays an important role in such cases. It is not known how many people have exposure to or contract tuberculosis prior to their BCG vaccination. The presence of a BCG vaccination scar does not rule out tuberculosis in its dormant or sub-clinical form. The non-granulomous uveitis and vitritis are rare occular presentations in tuberculosis and an ophthalmologist should consider this sign in the differential diagnosis of tuberculosis, especially in immigrants from countries where the prevalence of tuberculosis remains high.

REFERENCES

1 Bundney K, Dobkin J. Resurgent tuberculosis in New York City. Am Rev Respir Dis 1991;144:745-749.
2 Abbott CJ. Tuberculosis uveitis (letter). Am J Ophthalmol 1983;95(1):126-127.
3 Abrahams IV, Jiang Y. Ophthalmology in China: endogenous uveitis in a Chinese ophthalmological clinic. Arch Ophthalmol 1986;105:444-446.
4 American Thoracic Society. Diagnostic standards and classification of tuberculosis. Am Rev Respir Dis 1990;142:725-735.
5 Helm CJ, Holland GN. Ocular tuberculosis. Survey of ophthalmology 1993;38:229-256.
6 Hagerstron MD, Harper and Row. Bacterial uveitis (tuberculous and syphilitic) in duane text (ed) Clin Ophthalmol 1980;4:1-9.
7 Abrahams J, Schlagel TF Jr. The tuberculin skin test in the diagnosis of tuberculous uveitis. Am J Ophthalmol 1983;96:295-298.
8 Abrahams J, Schlagel TF Jr. The role of isoniazid therapeutic test in tuberculous uveitis. Am J Ophthalmol 1982;94:511-515.
9 Donahue HC. Ophthalmic experience in a tuberculosis sanitorium. Am J Ophthalmol 1967;64:742-8.
10 Moli MC, Mordi TE, Grane VP *et al.* Tuberculous panophthalmitis. J Pediatr Ophthalmol 1978;15:383.
11 Iseman MD, Madsen LA. Drug-resistant tuberculosis. Clinics in Chest Medicine 1989;10(3):341-53.
12 Barclay WR. Effectiveness of BCG vaccination in prevention of tuberculosis. J Am Med Assoc 1975;232(7):755.

COMMENT

S Partridge
Consultant Microbiologist, Furness General Hospital

TUBERCULOSIS IN MORECAMBE BAY

Tuberculosis in its various forms, including scrofula and consumption, has been a worldwide scourge for thousands of years. Palaeoarchaeology has found evidence of the disease in Egyptian mummies.

The incidence of tuberculosis in the UK has been declining since the early twentieth century due to improvements in living conditions and nutrition. The introduction of effective drug treatment in the 1950s consolidated that decline.

In recent years there has been a levelling out of the trend. The incidence of TB has risen in populations originating in areas where it is endemic, and in socially deprived groups.

So what is the situation in Morecambe Bay? The disease is relatively uncommon. In the years 1990-1998 the annual number of notifications was between 5 and 15; the national figures were between five and six thousand. In London, the area of the UK with the highest incidence, the rate of infection in 1998 was 31.6 per 100,000 population!

This is not an indication for complacency, however. A vital prerequisite of making the diagnosis is to consider the possibility of that diagnosis in the first place. This allows both treatment of the individual and the public health aspects to be managed.

What developments have there been in the laboratory diagnosis of tuberculosis? In most cases the fundamental elements are still microscopy and culture. A recent alternative to the Ziehl-Neelsen stain is the use of auramine staining, in which the bacilli fluoresce under UV light.

Culture can be semi-automated by the use of 'liquid culture' where the specimen is inoculated into a liquid medium and growth monitored continuously by a machine. This can allow earlier detection than conventional solid culture. The technology also allows for sensitivity testing to be performed more quickly than was previously possible.

The use of 'molecular' methods, notably the Polymerase Chain Reaction (PCR) has also entered the world of mycobacteriology. In this way cultures can be rapidly tested to distinguish *Mycobacterium tuberculosis complex (M tuberculosis, M bovis* and *M africanum*) from atypical mycobacteria. It is also possible to screen for the presence of the gene for rifampicin resistance, a marker of MDR TB.

Occasionally a specimen is examined by PCR to detect the presence of TB; the technique does not offer great advantage over microscopy in terms of sensitivity and is therefore not used routinely.

In short, therefore, laboratory methods are being refined to offer more rapid and sensitive diagnosis. The onus remains with the clinician to consider the diagnosis in order that these techniques may be brought into play.