Figure 2 Echocardiographic parasternal long axis view of the heart showing pleural and pericardial (arrowed) effusions. LV, left ventricle; MV, mitral valve; PE, pericardial effusion; PL EFF, pleural effusion.

Discussion

Campylobacter fetus ssp *fetus* infection is rare. It commonly presents with non-specific flu-like symptoms and bacteraemia.¹ It should be considered in the differential diagnosis of pyrexia of unknown origin. The organism is transmitted to humans through undercooked meat products or by direct contact with infected animals,² as in this case. Relapsing fever may occur.^{1,2} Pericarditis caused by *Campylobacter fetus* ssp *fetus* is rarely described in people with normal immunity.¹ In more than half the cases the bacteraemia is secondary to localised infections such as septic arthritis, pelvic inflammatory disease, meningi-

tis, endocarditis, pneumonia, thrombophlebitis, or mycotic aneurysm.³ Blood cultures may take from 3 to 25 days to grow the organism. In this unusual case of relapsing fever, a long course of treatment with ciprofloxacin was necessary. Mortality in such cases ranges from 17% to 43%.¹ Erythromycin and ciprofloxacin are the agents of choice for treatment.

The case also shows how a molecular approach can aid in the rapid identification of "difficult" bacterial isolates, using direct sequence analysis of the gene encoding the 16S RNA species of the ribosome.^{4,5} This bacterium's entire 16S gene was successfully amplified by polymerase chain reaction, sequenced, and compared with sequences on the database. It was unequivocally shown to share 100% identity with *Campylobacter fetus* ssp *fetus* (Genebank accession number M65012). This technique can be used in the diagnosis of organisms that are hard to identify by routine microbiological testing.

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Reiter's syndrome following intravesical BCG immunotherapy

M B Hogarth, S Thomas, M H Seifert, S M Tariq

Abstract

A 71 year old woman developed conjunctivitis, asymmetrical oligoarthritis, and cystitis (Reiter's syndrome) secondary to intravesical BCG treatment for transitional cell carcinoma of the bladder. She received oral prednisolone, izoniazid, and pyridoxine and made a full recovery. Increasing use of BCG as immunotherapy will lead to an increase in the incidence of BCG associated reactive arthritis. Prompt recognition and early diagnosis will facilitate treatment and recovery.

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Keywords: arthritis; BCG immunotherapy; Reiter's syndrome

BCG immunotherapy is widely used in the treatment of malignancy, and intravesical BCG is an effective agent for superficial bladder carcinoma. Tumour regression following BCG immunotherapy correlates with conversion of the purified protein derivative (PPD) skin test from negative to positive and the development

of antibodies to BCG, suggesting that its effectiveness results from an enhancement of cell mediated immunity.¹ In a recent review of 1278 cases, intravesical BCG was well tolerated by most patients, cystitis being the commonest side effect, occurring in up to 90% of cases.² Arthralgia or migratory arthritis occurred in 0.5% of cases. We describe a case in which a 71 year old woman developed Reiter's syndrome following intravesical BCG immunotherapy.

Case report

A 71 year old white woman was started on weekly intravesical BCG treatment (Evans Medical, Medeva Ltd, Leatherhead, Surrey, UK) for relapsed transitional cell carcinoma of the bladder which had developed three years previously. During week 4 of a six week course she developed pain and swelling affecting the right middle finger, left thumb (fig 1), and left knee. Two days later she developed red and itchy eyes (fig 2). She also complained of urinary frequency and dysuria. She had no rash and no preceding flu-like or diarrhoeal illness.

On examination she was pyrexial (37.8°C) and had bilateral conjunctivitis. She had right

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Figure 1 Swelling of hands after intravesical BCG.

Figure 2 Conjunctival inflammation after intravesical BCG. (Reproduced with the patient's permission.)

middle finger dactylitis and synovitis affecting the metacarpophalangeal joint of the left thumb. She also had synovitis of the left knee with an associated effusion. Ophthalmological review confirmed conjunctivitis. Laboratory studies revealed a raised erythrocyte sedimentation rate (88 mm/h) and C reactive protein concentration (374 mg/l), with normal blood count and biochemical profile. Autoantibodies were negative and complement normal. Urine microscopy showed 50–100 leucocytes and scanty red blood cells. Urine immunofluorescence for *Chlamydia trachomatis* was negative. Urine and blood cultures were sterile, and stool culture was negative (salmonella, shigella, campylobacter, yersinia); serological tests (antistreptolysin O, parvovirus, chlamydia) were also negative. Synovial fluid aspirate was turbid, with 2+ leucocytes and a sterile culture (including mycobacteria). She was HLA-B27, -DR4 positive. Radiology of the affected joints was normal.

She was started on diclofenac (50 mg three times daily) and intravesical BCG was discontinued. Her symptoms persisted and after one week she was started on prednisolone (20 mg once daily) plus isoniazid (300 mg/d), with pyridoxine prophylaxis (10 mg/d). Her arthritis and conjunctivitis resolved over 14 days. Steroids were gradually withdrawn and isoniazid was discontinued. She remains asymptomatic one year later.

Discussion

The association between mycobacterial infection and sterile joint inflammation is well recognised. In 1897, Poncet described sterile joint inflammation in association with *Mycobacterium tuberculosis* infection. Arthralgia and arthritis are rare but well recognised complications of BCG treatment, occurring in 0.5% of patients receiving intravesical BCG.² Mycobac-

Summary points

- BCG immunotherapy may precipitate reactive arthritis (including Reiter's syndrome).
- Increasing use of BCG as immunotherapy will lead to an increase in the incidence of reactive arthritis.
- Prompt recognition and early diagnosis will facilitate treatment and recovery.

teria in Freund's adjuvant have been shown to elicit arthritis in susceptible species of rat.³ The demonstration that arthritogenic T cell derived clones from these rats recognise a mycobacterial epitope that cross reacts with a self antigen in joint cartilage has led to suggestions that structural mimicry may be responsible for the inflammation.⁴ However, the low incidence of reactive arthritis following mycobacterial infection suggests that structural mimicry is unlikely to be the sole explanation.

This patient developed Reiter's syndrome during intravesical BCG immunotherapy. Reiter's syndrome following BCG immunotherapy has been described in only three patients (two female and one male) in English language reports.^{5–7} All developed asymmetrical arthritis and bilateral conjunctivitis within nine days of the fourth dose of immunotherapy, and all made a good recovery, although treatment varied between non-steroidal anti-inflammatory drugs (NSAID) alone, NSAID plus isoniazid, and NSAID plus isoniazid and rifampicin. Only one patient was tissue typed (for HLA-B27 only), and that patient was HLA-B27 positive, like our patient.⁵ Our patient was HLA-DR4 positive as well. This may be important in the pathogenesis, as it is recognised that the intradermal reaction to the tubercle antigen is influenced by the HLA phenotype, and HLA-DR4 has been shown to be associated with high responsiveness to antigens specific for *M tuberculosis*.⁸

The increasing use of BCG immunotherapy may lead to an increase in the incidence of BCG related arthritis. Such arthritis has a good prognosis and may respond to treatment with NSAID alone,⁶ although it has been suggested that all patients should be given isoniazid, and rifampicin may be added if there is poor response or severe symptoms indicating systemic infection.² The use of prophylactic isoniazid may prevent the development of arthritis following BCG immunotherapy in susceptible patients. However, animal studies suggest that such prophylaxis could reduce the antitumour effect,⁹ although this has not been confirmed in man.¹⁰ Furthermore, the low incidence of arthritis following BCG immunotherapy precludes the use of isoniazid prophylaxis in all patients. Tissue typing before treatment may enable high risk patients to be targeted with prophylactic isoniazid, although the effectiveness of this strategy needs to be assessed by a formal controlled trial.

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Telesales neuropathy

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Abstract

A case of bilateral ulnar neuropathies caused by overuse of the telephone is described in a 17 year old double glazing salesman. The importance of taking a good occupational history is emphasised and the need for correct staff training and appropriate equipment highlighted.

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Keywords: ulnar neuropathy; telephone use

The last few years have seen an explosion in the number of business that are abandoning the expense of operating from branches in favour of doing business at a distance, either over the internet or, more commonly, by telephone. Telesales is a major growth area and it brings with it its own spectrum of occupational diseases.

Case report

A 17 year old right handed Asian man was referred by his general practitioner with a progressive six week history of tingling and weakness affecting both hands, particularly the left. He had also noticed that the little finger on his right hand and the little and ring fingers on his left hand were becoming permanently bent and he could only straighten them by using his other hand.

He was still at college and three months earlier he had started an evening job as a telesales operator for a double glazing company. On an

Summary points

- Occupations with repetitive elbow flexion/extension can result in ulnar nerve dysfunction.
- Simple education and provision of appropriate equipment is important in minimising the risk of occupational neuropathies.
- A good occupational history is important in the medical diagnosis.
- New technologies bring with them their own spectrum of occupational diseases.

average night he would make around 100 telephone calls. Although he was right handed, he usually used the telephone in his left hand so as to leave his right hand free for writing, and would lean his elbow on the desk. There was nothing else of any note in the history.

General examination was normal and on neurological examination the abnormalities were restricted to the hands. He had wasting and weakness of the ulnar innervated small muscles of both hands, much worse on the left. There was clawing of the little and ring fingers on the left (fig 1) and of the little finger on the right. The median and radial nerve innervated muscles were normal. Sensory testing revealed decreased sensation over the little fingers and the medial half of the ring fingers bilaterally. He had a normal male carrying angle at both elbows and no abnormality was palpable over the cubital tunnels or ulnar grooves.

Nerve conduction studies confirmed the clinical diagnosis of bilateral ulnar entrapment neuropathies at the elbow. The remaining investigations to look for an underlying cause, including genetic testing for hereditary neuropathy with liability to pressure palsies (HNPP), were negative.

Discussion

In this patient the use of an ordinary hand held telephone to make so many calls, and the patient's habit of leaning on his elbow while

Figure 1 The clawing of the little and ring fingers on the left hand is clearly seen in this image.

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