Recurring febrile illness in a slaughterhouse worker

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Abstract
A slaughterhouse worker presented with fever and a pleuropericardial effusion. Conventional microbiology failed to identify the responsible organism. However, DNA sequencing definitively identified Campylobacter fetus ssp fetus, which is rare in immunocompetent individuals. Prolonged treatment was required to eradicate the infection.

Keywords: pyrexia of unknown origin; Campylobacter fetus ssp fetus; pericarditis

Patients may be exposed to various infective agents in their working environment and when travelling. Thus a full occupational and travel history should be sought, particularly in patients presenting with a febrile illness. In view of the wide spectrum of organisms involved, microbiological identification may be difficult and specialist help with new techniques such as DNA sequencing may be required.

Case report
A previously fit 54 year old male slaughterhouse worker presented with pleuritic chest pain following a week of lethargy and fever. He had no past medical problems and was not on any treatment. A detailed occupational history was elicited. His job involved hosing out animal carcasses. He had to rip out the neural tissue (spinal cord) using gloved hands. He admitted that accidental damage had occurred to a glove. There was no history of foreign travel. He was a non-smoker and drank very little alcohol.

Clinical examination on admission was unremarkable but he developed a swinging pyrexia of 38°C and a pericardial rub two days after admission. An ECG showed upwardly concave ST elevation in lead II and V4–V6 (fig 1), indicating pericarditis. An echocardiogram showed pericardial and pleural effusions (fig 2).

Routine haematology and biochemistry results were normal, apart from raised serum C reactive protein (156 mg/l). Blood culture grew a Gram negative spiral organism could not be identified by standard biochemical methods but was identified by DNA sequencing as Campylobacter fetus ssp fetus.

The patient was treated with amoxycillin, metronidazole, and tetracycline and the fever and effusions resolved after seven days of treatment. He was discharged with advice to continue treatment with tetracycline for a further three weeks.

Three weeks later, after stopping antimicrobials, he was readmitted with fever and breathlessness. Clinical examination and blood investigations were again unremarkable except for a raised C reactive protein concentration (211 mg/l). He was treated with ciprofloxacin 500 mg twice daily for one week. Symptoms resolved and he was discharged home taking oral ciprofloxacin for a further four weeks. At the end of this time he was reviewed in the clinic and was found to be well. Follow up at three months showed no recurrence of the infection.

Figure 1  ECG showing upwardly concave ST elevation in lead II and V4–V6.
Reiter’s syndrome after BCG immunotherapy

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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, isoniazid, 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.


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. 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. 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
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. Mycobacterium tuberculosis in Freund’s adjuvant have been shown to elicit arthritis in susceptible species of rat. 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. 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. 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. 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.

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, 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. 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, although this has not been confirmed in man. 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.

We would like to thank Professor R Shaw for his help in the management of this patient.

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.
Telesales neuropathy

M B Lewis

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.

(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 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 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.

References
holding the telephone, probably both contributed to the rapid development of bilateral ulnar neuropathies.

Ulnar neuropathies at the elbow and other anatomical sites have been described from many causes,¹ some rather unusual,²³ but this is the first description of the syndrome occurring because of overuse of the telephone. Despite the increasingly sedentary lives of people today, occupational neuropathies previously associated with manual work continue to occur. The introduction of new technologies has simply expanded the spectrum of causative occupations.¹⁴

This case highlights the fact that people involved in the rapidly expanding area of telesales need proper training and should be provided with the most appropriate equipment to do their job. A simple headset device would have prevented the injuries occurring at all.

Reiter's syndrome following intravesical BCG immunotherapy

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