Case reports

Listeria monocytogenes empyema in an HIV infected patient

Anna Marron, Beatriz Rosón, Jordi Mascaró, Jordi Carratalà

Abstract

Listeriosis in HIV infected patients is uncommon and usually presents as meningitis or bacteraemia. Pleural fluid infections caused by this organism are extremely rare. A case is described of empyema caused by *Listeria monocytogenes* in an HIV infected patient that was successfully treated with medical treatment only.

(*Thorax* 1997;**52**:745–746)

Keywords: *Listeria monocytogenes*, empyema, HIV.

Listeria monocytogenes is a Gram positive motile bacillus that mainly affects subjects with defects in cell mediated immunity, pregnant women, or neonates. Listeriosis usually presents as meningitis or bacteraemia, although other manifestations such as septic arthritis, brain abscess, infective endocarditis, endophthalmitis, and hepatitis have been described.[1] Pleural fluid infections caused by *L monocytogenes* are extremely rare.[2 3] We report a case of *L monocytogenes* empyema in an HIV infected patient.

Case report

A 33 year old woman, a former intravenous drug abuser who was HIV infected and a heavy drinker, presented to our hospital with a 48 hour history of fever, non-productive cough, pleuritic left pain, and shortness of breath. Two years before admission biopsy proven liver cirrhosis was documented. She had had several previous admissions for ascites, spontaneous bacterial peritonitis, or spontaneous bacteraemia. No HIV related pathology was known. She was receiving zidovudine 500 mg daily and prophylaxis with aerosolised pentamidine. On examination she appeared acutely ill, with a respiratory rate of 32/min and a temperature of 38.5°C. The liver and the spleen were enlarged but no ascites was detected. The findings of the chest examination were consistent with a left pleural effusion. Physical examination was otherwise normal. Initial laboratory tests revealed a white cell count of 5.2×10^3/ml (with 82% neutrophils, 10% lymphocytes, 6% monocytes, and 2% band forms), a CD4 T lymphocyte count of 41/mm^3 (8%, normal range 39–52%), haemoglobin of 8 mmol/l, and platelets of 84×10^3/µl. An arterial blood sample while breathing room air had a pH of 7.43, Pco_2 of 4.4 kPa, and Po_2 of 12.8 kPa. Chest radiography demonstrated a left pleural effusion. Thoracocentesis was performed yielding a cloudy yellowish fluid. Pleural fluid analysis revealed an exudate with a pH of 7.3, 35 g/l protein, 4.9 mmol/l glucose, 840 IU/l lactate dehydrogenase (LDH) (plasma glucose 5.6 mmol/l; LDH 420 IU/l, normal range 60–250 IU/l), and 1400 cells (83% polymorphonuclear leucocytes). Gram staining showed abundant polymorphonuclear cells but no bacteria. *Listeria monocytogenes* serotype IV was isolated from pleural fluid and blood.

The initial treatment with intravenous ceftriaxone 1 g daily was then discontinued and intravenous ampicillin 2 g every four hours plus gentamicin 200 mg daily were administered for 14 days. After four days of antibiotic therapy both blood and pleural fluid cultures were negative and she was discharged on oral trimethoprim-sulphamethoxazole. One year later the patient remains alive and no other listerial infections have been documented.

Discussion

A population based study has shown that patients with AIDS have an increased risk of developing invasive listeriosis of up to 145 times that of the general population.[4] However, listeriosis has rarely been described in HIV infected patients, and most of the reported cases presented as meningitis and bacteraemia.[5–7] To our knowledge only one case of pleural fluid infection with *L monocytogenes* in an HIV infected patient has been previously reported.[3] It should be noted that, as in our case, that patient also had chronic liver disease, a condition that could have been an additional predisposing factor for invasive listeriosis. In addition, our patient was receiving inhaled pentamidine instead of trimethoprim-sulphamethoxazole which may also have contributed to development of infection. Haematogenous spread and secondary seeding of the pleura seems to be the route of infection in most cases of pleural fluid infection caused by *L monocytogenes*, as we think occurred in our patient.[2] Conversely, the HIV infected patient previously described[3] had pneumonia and developed an effusion probably as a parapneumonic process since blood cultures yielded no bacteria. Neither ascites nor abdominal pain was present, although the association of listerial empyema with spontaneous bacterial peritonitis has also been recognised.[8] High doses of ampicillin or penicillin, together with an aminoglycoside, is the treatment of choice for listeriosis, principally when poor prognostic indicators are present.[9] Trimethoprim-sulphamethoxazole, commonly given to AIDS patients for prophylaxis of *Pneumocystis carinii* infection, is also an effective therapy for listeria

Infectious Disease Service, Ciutat Sanitària i Universitària de Bellvitge, University of Barcelona, Spain
A Marron
B Rosón
J Mascarò
J Carratalà

Correspondence to:
Dr Anna Marron, Infectious Disease Service, Hospital de Bellvitge, C/Feixa Llarga sn, 08907 L'Hospitalet, Barcelona, Spain.

Received 21 March 1996
Returned to authors 20 May 1996
Revised version received 10 July 1996
Accepted for publication 9 August 1996

infections.[9] Whether or not chest tube drainage in *L monocytogenes* empyema is necessary remains unclear. However, a complete resolution of the empyema was obtained in both previously mentioned cases with medical treatment only.

We therefore believe that *L monocytogenes* should be considered in the differential diagnosis of empyema in patients with HIV infection.

1 Niemen RE, Lorber B. Listeriosis in adults: a changing pattern. Report of eight cases and review of the literature, 1968–1978. *Rev Infect Dis* 1980;**2**:207–27.
2 Mazzulli T, Salid IE. Pleural fluid infection caused by *Listeria monocytogenes*: case report and review. *Rev Infect Dis* 1991; **13**:564–70.
3 Domingo P, Serra J, Sambeat MA, Ausina V. Pneumonia due to *Listeria monocytogenes*. *Clin Infect Dis* 1992;**14**:787–9.
4 Jurado RL, Farley MM, Pereira E, Harvey RC, Schuchat A, Wenger JD, *et al*. Increased risk of meningitis and bacteremia due to *Listeria monocytogenes* in patients with human inmunodeficiency virus infection. *Clin Infect Dis* 1993;**17**: 224–7.
5 Berenguer J, Solera J, Diaz MD, Moreno S, López-Herce JA, Bouza E. Listeriosis in patients infected with human immunodeficiency virus. *Rev Infect Dis* 1991;**13**:115–9.
6 Decker CF, Simon GL, DiGioia RA, Tuazon CV. *Listeria monocytogenes* in patients with AIDS: report of five cases and review. *Rev Infect Dis* 1991;**13**:413–7.
7 Mascola L, Lieb L, Chiu J, Fannin SL, Linnan MJ. Listeriosis: an uncommon opportunstic infection in patients with acquired immunodeficiency syndrome. A report of five cases and a review of the literature. *Am J Med* 1988;**84**: 162–4.
8 Simpson JF, Leddy JP, Hare JD. Listeriosis complicating lymphoma. Report of four cases and interpretative review of pathogenic factors. *Am J Med* 1967;**43**:39–49.
9 Jones EM, MacGowan AP. Antimicrobial chemotherapy of human infection due to *Listeria monocytogenes*. *Eur J Clin Microbiol Infect Dis* 1995;**14**:165–75.

Thorax 1997;**52**:746–748

Occupational asthma in an isothiazolinone manufacturing plant

S J Bourke, R P Convery, S C Stenton, R M Malcolm,* D J Hendrick

Abstract

A chemical plant operator developed asthma five months after starting work in an isothiazolinone manufacturing plant. He described symptoms of late asthmatic reactions after work with isothiazolinone. Airway responsiveness to methacholine improved tenfold when he was removed from the plant for 18 days. A workplace challenge study then resulted in a deterioration in airway responsiveness to its earlier level and in progressive falls in forced expiratory volume in one second (FEV_1) over three days at work compared with control days, indicating statistically significant late asthmatic reactions of increasing severity.

(*Thorax* 1997;**52**:746–748)

Keywords: occupational asthma, biocides, isothiazolinone.

Department of Respiratory Medicine and Regional Unit for Occupational Lung Disease, Newcastle General Hospital, University of Newcastle upon Tyne, UK
S J Bourke
R P Convery
S C Stenton
R M Malcolm
D J Hendrick

*Present appointment: Chief Medical Officer, Tayside Police.

Correspondence to: Dr S J Bourke, Department of Respiratory Medicine, Royal Victoria Infirmary NHS Trust, Newcastle General Hospital, Newcastle upon Tyne NE4 6BE, UK.

Received 4 April 1996
Returned to authors 18 July 1996
Revised version received 22 August 1996
Accepted for publication 26 September 1996

Isothiazolinones are being used increasingly as broad spectrum biocides in various water systems and in products such as paper, paints, and cosmetics.[1 2] In some circumstances they have been used as substitutes for formaldehyde and glutaraldehyde which are known to cause asthma.[3] There have been many reports of isothiazolinones causing occupational dermatitis but evidence of asthma due to isothiazolinones is lacking.[4 5] We describe the occurrence of asthma in an isothiazolinone manufacturing plant, and a workplace challenge study which suggested it was occupational in origin. We think it likely that this was the result of sensitisation to isothiazolinone.

Case report

A 53 year old chemical plant operator developed asthma five months after starting work in an isothiazolinone manufacturing plant. He had no previous history of asthma or atopic disease and he was an ex-smoker. His job involved the filling of containers with various formulations of isothiazolinones in aqueous solution, including a combination of 5-chloro-2-methyl-4-isothiazolin-3-one and 2-methyl-4-isothiazolin-3-one. This was performed in an enclosed booth fitted with an extractor, but as he frequently opened the door of the booth to replace containers there was potential exposure to low concentrations of airborne isothiazolinone, which is weakly volatile. He described symptoms of cough and wheeze, which occurred towards the end of his working shifts, persisted into the evenings after work, and often disturbed his sleep at night. He was diagnosed as having asthma by his general practitioner and was prescribed salbutamol and beclomethasone inhalers. He had noticed that his symptoms were related to his work and he had tried to reduce his exposure, but he remained at the same job over the next five years until he was referred for assessment. His forced expiratory volume in one second (FEV_1) was 3.91 litres (109% predicted) and his forced vital capacity (FVC) was 5.69 litres (127% predicted) while taking salbutamol and beclomethasone, and he had moderate airway responsiveness to methacholine with a provoking dose responsible for a 20% fall in FEV_1 (PD_{20}) of 230 μg. Skin tests for atopy gave negative results. His history was suggestive of occupationally provoked late asthmatic reactions and a workplace challenge study was undertaken.

WORKPLACE CHALLENGE STUDY

His asthma treatment was stopped, he was withdrawn from the workplace for 18 days, and then re-exposed to his normal work environment. Spirometric tests were performed hourly from 07.00 hours to 17.00 hours on three control days away from work, and hourly from 07.00 hours to 23.00 hours on a fourth control day. He then returned to his normal job, working a 07.00 hours to 15.00 hours shift,