Correspondence

Bacteraemia caused by *Campylobacter* spp

We were interested to read the paper of Ladrón de Guevara *et al*¹ describing seven cases of bacteraemia caused by *Campylobacter* spp and would like to comment with a similar unusual case.

A 21 year old man with acute lymphoblastic leukaemia diagnosed six months previously was admitted with a temperature of 39°C. In the previous two weeks he had received chemotherapy with thioguanine, cytosine arabinoside and cyclophosphamide and had a white cell count of $0.8 \times 10^9/l$. He had received the chemotherapy through a Hickman line which was still in situ. Blood cultures were taken at this time but remained negative after one week of culture using 6B and 7A bottles in the BACTEC 460 system. He was treated empirically with intravenous antibiotics, flucloxacillin 500 mg four times daily, netilmicin 50 mg three times daily and azlocillin 5 g three times daily for five days with no resolution of his pyrexia. He was then changed to teicoplanin 400 mg/day and imipenem 500 mg four times daily. His Hickman line was removed the following day. The site was noted to be inflamed. His temperature dropped over the next three days from 39°C to 37.8°C.

The Hickman catheter tip was plated out by rolling it on blood agar plates and these were then incubated aerobically and anaerobically at 37°C overnight. A faint greenish coloration was noted in two areas of the anaerobic plate and it was reincubated. After 48 hours, a fine growth was noted and Gram stain of this revealed curved Gram negative bacillus. This growth was subcultured onto Columbia agar and incubated aerobically in 5% CO₂ and anaerobically in an atmosphere of 80% N₂, 10% H₂ and 10% CO₂ at 37°C.

It was also subcultured onto Columbia agar and Skirrow's medium and incubated in a microaerophilic atmosphere (85% N₂, 10% CO₂, 5% O₂) at 42°C. While the plates incubated at 37°C had only a very thin growth, there was a very good growth on both plates incubated at 42°C. The organism was identified by Api Camp as *Campylobacter jejuni* and antibiotic sensitivity testing on DST agar incubated aerobically at 37°C gave results as follows: the organism was sensitive to ampicillin, erythromycin, netilmicin, and ciprofloxacin and resistant to flucloxacillin.

On the basis of this finding, the patient was changed to ciprofloxacin 500 mg twice daily taken orally and started to feel better almost immediately. He was discharged but was readmitted three days later with a recurrence of the pyrexia (37.8°C) and diarrhoea. Blood and faecal cultures taken at this time remained negative. Teicoplanin was added to the ciprofloxacin. His condition improved again and he was discharged finally five days later.

This is the only case in which we have found the isolation of *C. jejuni* from the tip of an intravascular catheter. It is also unusual in that the patient developed diarrhoea several days after the organism was isolated which contrasts with previous reports of bacteraemia following on from diarrhoea caused by *Campylobacter* gastroenteritis.² The colonisation of the catheter tip was either due to an occult bacteraemia from an asymptomatic gastrointestinal source or less likely due to an ascending infection via the catheter. The initial failure to resolve the

fever was probably related to the persistent presence of the colonised Hickman catheter, which is substantiated by the resolution of fever only after catheter removal. In addition, the symptomatic improvement was dramatic but only followed catheter removal and ciprofloxacin therapy, which we would recommend for any bacteraemic patient with an intravascular catheter.

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1 Ladrón de Guevara C, Gonzalez J, Peña P. Bacteraemia caused by *Campylobacter* spp. *J Clin Pathol* 1994;47:174-5.

2 Butzler JP, Skirrow MB. *Campylobacter* enteritis [review]. *Clin Gastroenterol* 1979;8:737-65.

Uterine stromal sarcoma following tamoxifen treatment

Adverse reactions to tamoxifen are being increasingly reported and the recent article by Ismail¹ succinctly highlights the endometrial response to tamoxifen administration.

In this laboratory we have seen a similar range of endometrial reactions to tamoxifen treatment, but have recently encountered a low grade endometrial stromal sarcoma with a sex cord-like pattern following tamoxifen ingestion.

The patient, aged 61 years, presented with post-menopausal bleeding associated with atypical endometrial hyperplasia. She had received a total cumulative dose of 36.5 g tamoxifen administered over five years since the excision of a stage 1 infiltrating duct carcinoma of the breast. She had taken no other hormonal therapy or medication.

Examination of the hysterectomy specimen showed, in addition to the generalised endometrial hyperplasia, focal well differentiated endometrioid adenocarcinoma and a myometrial tumour $4 \times 3 \times 2.5$ cm. Histologically, this showed features of a low grade endometrial stromal sarcoma with a prominent sex cord-like pattern. There was conspicuous vascular invasion but a mitotic count of up to two mitoses per 10 high power fields ($\times 40$ objective, field area 0.196 mm^2). Immunohistochemical staining showed positive staining with vimentin but not with low molecular weight cytokeratin (Cam 5.2), epithelial membrane antigen, or desmin.

Endometrial stromal sarcomas have been well documented in the literature^{2,3} and a minority show a sex cord-like pattern.^{4,5} We are not aware of any established association with exposure to tamoxifen. In a recent large study of 30 597 women with breast carcinoma,⁶ 271 developed malignant uterine tumours, 118 of them after treatment of the breast carcinoma. Only 15 of these women had received tamoxifen and there were no cases of stromal sarcoma in either group.

In this case we propose that the endometrial pathology is a direct consequence of tamoxifen ingestion, but the role of tamoxifen in the aetiology of the uterine stromal sarcoma remains speculative.

Continued surveillance of patients receiving tamoxifen treatment is indicated and we endorse Ismail's suggestion¹ for a register of patients receiving tamoxifen.

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1 Ismail SM. Pathology of endometrium treated with tamoxifen. *J Clin Pathol* 1994;47:827-33.

- 2 Tinkler SD, Cowie VJ. Uterine sarcomas: a review of the Edinburgh experience from 1974 to 1992. *Br J Radiol* 1993;66:998-1001.
- 3 Chang KL, Crabtree GS, Lim-Tan SK, Kempson RL, Hendrickson MR. Primary uterine endometrial stromal neoplasms. A clinicopathological study of 117 cases. *Am J Surg Pathol* 1990;14:415-38.
- 4 Jiang ZM. Sarcomas of the uterus: immunohistochemical characterisation and diagnosis. *Chin J Pathol* 1992;21:336-8.
- 5 Lillemoe TJ, Perrone T, Norris HJ, Dehner LP. Myogenous phenotype of epithelial-like areas in endometrial stromal sarcomas. *Arch Pathol Lab Med* 1991;115:215-19.
- 6 Silva EG, Tornos CS, Follen-Mitchell M. Malignant neoplasms of the uterine corpus in patients treated for breast carcinoma: the effects of tamoxifen. *Int J Gynecol Pathol* 1994; 13:248-58.

Notices

The Royal College of Pathologists

presents

An Update on Gut Infections

Wednesday 5th July 1995

and

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Thursday 21 September 1995

Both courses will be held at the Royal College of Pathologists, 2 Carlton House Terrace, London SW1, and are open to both members and non-members of the College.

For further details and application forms, please contact: Scientific Meetings Officer, RCPATH, 2 Carlton House Terrace, London SW1Y 5AF (tel: 0171 930 5862 ext. 24/25).

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For further information, please contact: Madame A Alcaraz, Laboratoire de Biochimie C, CHURG, B.P. 217, F-38043 Grenoble Cedex 9, France (tel: (33) 76 76 54 84; fax: (33) 76 76 56 64).

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