References

1. Langgand, H. & Smith, W.O. Self-induced water intoxication without predisposing illness. *N Engl J Med* 1962, **266**: 378–383.
2. Kennedy, R.M. & Earley, L.E. Profound hyponatraemia resulting from a thiazide induced decrease in urinary diluting capacity in a patient with primary polydipsia. *N Engl J Med* 1970, **282**: 1185–1186.
3. Beresford, H.R. Polydipsia, hydrochlorothiazide and water intoxication. *JAMA* 1970, **214**: 879–883.
4. Raskind, M. Psychosis, polydipsia and water intoxication. *Arch Gen Psychiatry* 1974, **30**: 112–114.
5. Gossain, V.V., Hagen, G.A. & Sugawara, M. Drug-induced hyponatraemia in psychogenic polydipsia. *Postgrad Med J* 1976, **52**: 720–722.
6. Fowler, R.C., Kronfol, Z.A. & Perry, P.J. Water intoxication, psychosis, and inappropriate secretion of antidiuretic hormone. *Arch Gen Psychiatry* 1977, **34**: 1097–1099.
7. Smith, W.O. & Clark, M.L. Self-induced water intoxication in schizophrenic patients. *Am J Psychiatry* 1980, **137**: 1055–1060.
8. Emsley, R.A. & Gledhill, R.F. Thiazides, compulsive water drinking and hyponatremic encephalopathy. *J Neurol Neurosurg Psychiatry* 1984, **47**: 886–887.
9. Illowsky, B.P. & Kirch, D.G. Polydipsia and hyponatremia in psychiatric patients. *Am J Psychiatry* 1988, **145**: 675–682.

Pulmonary geotrichosis

Sir,

Saprophytic fungi are known to produce opportunistic infections in immuno-suppressed individuals. Geotrichosis is a mycotic infection with oral, intestinal, bronchial or pulmonary lesions, caused by the ubiquitous fungus *Geotrichum candidum*.¹ Pulmonary involvement simulating tuberculosis is frequently reported.² A case of geotrichosis in an old tuberculous lung cavity occurring in an immunocompetent individual is reported.

A 45 year old male presented with cough and streaky haemoptysis of one week duration. He had been treated six years previously for pulmonary tuberculosis for a year. Examination revealed an ill looking male with extensive physical signs in the right chest. Chest X-ray showed a fibrocavitary lesion in the right upper zone and apicogram revealed a large cavity containing a solid density mass in the right upper lobe.

Geotrichum candidum was isolated in all 10 freshly expectorated sputum specimens. The particular features of *G. candidum* were absence of urea utilization, assimilation of glucose and galactose but not maltose, sucrose, salicon, inositol or raffinose, thus differentiating from genus *Trichosporon*. The patient responded to oral administration of a supersaturated solution of potassium iodide for 3 months.

Geotrichum candidum, which belongs to the class Fungi imperfecti, is an opportunistic human pathogen.¹ The repeated isolation of *G. candidum* with characteristic arthrospores and hyphae in freshly expectorated sputum samples and the absence of other pathogenic fungi or bacilli either by direct microscopy or culture of sputum confirms the diagnosis of geotrichosis.^{2,3} Radiologically, there may be patchy or fluffy infiltrates with a predilection for the upper lobes and, occasionally, cavity lesions (as in our patient). Although use of neomycin and colistin is anecdotal, nystatin and iodide preparations have been used commonly.^{3,4} Miconazole, clotrimazole, amphotericin B and 5-fluorocytosine have been shown to have *in vitro* activity at attainable concentrations.⁴

ricin B and 5-fluorocytosine have been shown to have *in vitro* activity at attainable concentrations.⁴

Rama Ramani
P. Vittal Rao¹
Girija R. Kumari
P.G. Shivananda

Departments of Microbiology & ¹Medicine,
Kasturba Medical College,
Manipal 576 119,
India.

References

1. Rippon, J.W. *Medical Mycology; The pathogenic fungi and the pathogenic actinomycetes*, 2nd ed. W.B. Saunders, Philadelphia, 1982, pp. 642–645.
2. Pahwa, R.K., Yadav, V.K., Singh, M.M. & Chaturvedi, V.P. Pulmonary geotrichosis. *Indian J Chest Dis Allied Sci* 1983, **25**: 54–59.
3. Emmons, C.W., Bindord, C.H., Utz, J.P. & Kwon-Chung, K.J. *Medical Mycology*, 3rd ed. Lea and Febiger, Philadelphia, 1977, pp. 202–205.
4. Fishback, R.S., White, M.L. & Finegold, S.M. Bronchopulmonary geotrichosis. *Am Rev Respir Dis* 1973, **108**: 1388–1392.

Urinary bladder carcinoma initially manifested as brain metastases

Sir,

Only 1% of transitional cell carcinomas (TCC) of the bladder give rise to brain metastasis throughout their natural history.^{1,2} Similarly, a bladder origin has been discovered in only approximately 1% of patients with cerebral metastases.³ We describe two cases of bladder carcinoma, the clinical presentation of which was solely due to the presence of brain metastases.

Case 1

A 67 year old male developed complete homonymous hemianopia, visual agnosia and a brief history of disorientation. Physical examination showed left supraclavicular adenopathy and tomographic (CT) scan confirmed a left occipital mass. Gland biopsy showed undifferentiated carcinoma. Further investigations revealed infiltrating bladder cancer with local and distant lymphadenopathy. There were no previous urological symptoms and haematuria only developed later. Autopsy gave histological confirmation for both TCC of the bladder and cerebral metastases.

Case 2

A 54 year old male, with a perineal urethral orifice following urethroplasty, complained of multiple urinary stones and urinary tract infection. He complained of frequent bifrontal headaches that awoke him during the night, and emotional lability and undue irritability. Generalized seizures and weakness of his left arm developed. Papilloedema was discovered and a small right periventricular mass that enhanced with contrast

was demonstrated on CT scan. Macroscopic haematuria was later noted. Intravenous urography confirmed the suspicion of bladder carcinoma and biopsy proved it to be a poorly differentiated TCC. CT scan of the pelvis revealed infiltrating bladder tumour with enlarged bilateral external iliac glands. Bone scintigram proved negative. Pelvic and cranial external beam irradiation, and steroid therapy were started. However, the patient developed massive metastatic pulmonary lesion and died 9 weeks later.

We have only been able to find one other published case in which a brain metastasis presented as the first manifestation of a bladder carcinoma.⁴ Similarly, we have already reported another unexpected presentation of urinary bladder carcinoma in the form of a retro-orbital mass.⁵ We therefore stress the importance of wide differential diagnoses regarding metastatic disease of the brain. Physicians should be aware of and investigate occult urological symptoms in a patient with neurological signs of an intracranial mass.

Javier C. Angulo

Jose I. Lopez¹

Miguel Unda-Urzaiz

Nicolas Flores

Servicios de Urología y

¹Anatomía Patológica,

Santo Hospital Civil de Bilbao,

Avd Montevideo 18,

48013 Bilbao, Spain.

References

- Steinfeld, A.D. & Zelefsky, M. Brain metastases from carcinoma of bladder. *Urology* 1987, **29**: 375–376.
- Kabalin, J.N., Freiha, F.S. & Torti, F.M. Brain metastases from transitional cell carcinoma of the bladder. *J Urol* 1988, **140**: 820–824.
- Graf, A.H., Buchberger, W., Langmayr, H. & Schmid, K.W. Site preference of metastatic tumors of the brain. *Virchow's Arch [A]* 1988, **412**: 493–498.
- Le Chevalier, T., Smith, F.P., Caille, P. & Rouesse, J.G. Sites of primary malignancies in patients presenting with cerebral metastases. A review of 120 cases. *Cancer* 1985, **56**: 880–882.
- Angulo, J.C., Lopez, J.I., Larrinaga, J.R. & Flores, N. Metastasising carcinoma of the urinary bladder presenting as a retro-orbital mass. Case report. *Scand J Urol Nephrol* 1991, **25**: 83–84.

***Campylobacter jejuni* – an unusual cause of infectious arthritis**

Sir,

We wish to report what we believe to be the first published case of acute infective arthritis due to *Campylobacter jejuni*.

A 51 year old woman was admitted with acute arthritis of her left knee. She had rheumatoid arthritis and common variable hypogammaglobulinaemia, for which she was treated with corticosteroids and intravenous gammaglobulin respectively. She was afebrile, but the left knee was swollen, red and hot, and had been so for a few days prior to admission.

An arthrocentesis yielded 30 ml of purulent fluid. The Gram stain showed many polymorphonuclear cells and

no bacteria; culture was negative during the first 72 h of incubation. At that time the patient became febrile.

Blood and joint aspirate culture were taken again and intravenous ceftriaxone 2 g per day was started, and 20 mg of triamcinolone was injected in the affected joint. The temperature returned to normal within 2 days while the acute arthritis symptoms persisted.

Twelve days after admission *C. jejuni*¹ was identified in cultures from the joint fluid as well as from the blood. *In vitro* sensitivity tests showed that the strain was resistant to ceftriaxone, cephalothin, cotrimoxazole, penicillin, aztreonam and ceftazidime, and sensitive to ciprofloxacin, pefloxacin, nalidixic acid, norfloxacin, ampicillin and erythromycin. Ceftriaxone was discontinued and intravenous ciprofloxacin 200 mg three times a day was administered.

The patient showed rapid improvement of local signs with negative blood cultures 72 h after treatment with ciprofloxacin started, and negative joint aspirate 96 h after treatment with ciprofloxacin started. A 4 week course of intravenous ciprofloxacin was given.

C. jejuni is an uncommon cause of infective complications apart from acute enteritis. Bacteraemia has been reported in less than 1% of patients with *C. jejuni* infections; it appears to be more common at extremes of age. Meningitis and endocarditis have been rarely reported and acute cholecystitis, pancreatitis, and cystitis caused by *C. jejuni* appear to be the result of local spread of the micro-organisms from enteric foci rather than from haematogenous seeding.^{2–4}

A reactive arthritis has been reported after *C. jejuni* enteritis (2% of HLA B 27 positive antigen patients)⁵ but acute infective arthritis has not been documented before to our knowledge.

In this particular patient the infectious event could have been favoured by gamma-globulin deficiency, steroid therapy, and moderately impaired renal function. The gastrointestinal tract might have been the primary site of the infection though symptoms of enteric disease were not reported by the patient even after specific questioning. Stool culture proved negative for *C. jejuni*, but it was taken 72 h after ciprofloxacin was started.

M.B. Pasticcini

E. Baratta¹

A. Del Favero¹

A. Gillio²

F. Baldelli

S. Pauluzzi

Institute of Infectious Diseases,

¹Institute of Internal Medicine,

and ²Department of Orthopaedics,

Policlinico Monteluce,

University of Perugia,

06100 Perugia, Italy.

References

- Morris, G.K. & Patton, C.M. *Campylobacter*. In: Lennette, H., Balows, A., Hausler, W.J.Jr. & Shadomy, H.J. (eds) *The Manual of Clinical Microbiology*, 4th ed, 1985, pp. 302–308.
- Blaser, M.J. & Reller, L.B. *Campylobacter enteritis*. *N Engl J Med* 1981, **305**: 1444–1452.
- Spelman, D.W., Davison, N., Buckmaster, N.D., Spicer, W.J. & Ryan, P. *Campylobacter bacteremia: a report of 10 cases*. *Med J Aust* 1986, **144**: 503–505.