A hospital outbreak of Serratia marcescens in neurosurgical patients

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SUMMARY

We report an outbreak of serious infections with *Serratia marcescens* in patients on a neurosurgery ward. The epidemiological investigations undertaken are described. Features of outbreaks of infection with serratia and control measures are discussed.

INTRODUCTION

Outside hospital, healthy individuals rarely become infected with Serratia marcescens. An exception occurs with intravenous drug abusers (Daschner, 1980). However, nosocomial colonization and infection, mostly of the urinary and respiratory tract occur (Daschner, 1980; Tabaqchali, Chambers & Brooks, 1977). Predisposing factors include severe underlying disease, the post-operative state, and antibiotic therapy (Daschner, 1980).

S. marcescens has been implicated in outbreaks of nosocomial infection, both in neonates (Stamm et al. 1976; McCormack & Kunin, 1966), and adults (Schaberg et al. 1976). The most likely hospital sources of the organism in common-source outbreaks are hands of nursing personnel (Maki et al. 1973; Stamm et al. 1976), equipment such as ventilators (Sanders et al. 1970) and nebulizers (Ringrose et al. 1968), fluids (Grimont & Grimont, 1978; Palmer & McCracken, 1970; Stamm et al. 1976) and intravenous solutions (Daschner, 1980). Less common sources include sponges (Grimont & Grimont, 1978) and shaving brushes used during facial depilation (Grimont & Grimont, 1978; Whitby, Blair & Rampling, 1972).

In this hospital, we recently had a series of infections with *S. marcescens*, including post-operative meningitis and wound infection, among patients in a neurosurgical ward. In this paper, we report our investigations to determine the source of the infections and the control measures instituted.

CASE HISTORIES

Patient 1, JW

A 62-year-old woman was admitted on 18 December 1987, following a subarachnoid haemorrhage which required clipping of an aneurysm of the left anterior communicating artery. On 2 January 1988, 2 weeks after surgery, the

patient developed focal neurological signs and a clinical diagnosis of a subdural collection was confirmed by computed tomography. At operation, cloudy cerebrospinal fluid (CSF) was drained and an external ventricular drain was inserted. Examination of the specimen showed 9600 white cells per μ l (95% polymorphonuclear cells) and culture yielded S. marcescens, resistant to ampicillin and cefuroxime, but susceptible to gentamicin, cefotaxime, ceftazidime and ciprofloxacin by disk testing using Stokes's method (Stokes & Waterworth, 1972). Intravenous therapy was started with ceftazidime (2 g tds), gentamicin, chloramphenicol and flucloxacillin, with the latter two drugs discontinued after 1 day. However, the patient's condition deteriorated and, despite sterile CSF cultures, she died 18 days later whilst still on treatment.

Patient 2, BT

A 19-year-old man sustained a depressed skull fracture and bilateral extra- and subdural haematomata after falling 30 feet from a building on 14 November 1987. The blood clots were evacuated and a bone flap elevated with ampicillin and flucloxacillin cover. Six weeks after operation, on 30 December 1987, a clinical wound infection prompted removal of the bone flap and drainage of pus. Culture of an operative specimen yielded *Enterococcus faecalis* and *S. marcescens* with the same antibiogram as that of the previous patient. Intravenous therapy was started with ampicillin, cefotaxime and gentamicin for 2 weeks and oral ciprofloxacin was given subsequently for 4 weeks. There was good recovery and the wound healed well. Further cultures did not grow *S. marcescens*.

Patient 3, AS

A 25-year-old man was admitted unconscious on 3 October 1987, following a road traffic accident with a fracture of the right temporal bone and a right-sided intracerebral haematoma. The blood clot was evacuated immediately under antibiotic cover (penicillin and sulphadimidine). Eleven weeks later, he returned from a rehabilitation centre for insertion of a ventriculo-peritoneal shunt to drain a communicating hydrocephalus. Two weeks afterward, on 6 January 1988, he became pyrexial with clinical signs of meningitis. Ventricular and lumbar specimens of CSF contained 850 and 585 white cells per μ l (80% polymorphonuclear cells) and grew S. marcescens with the same antibiogram as previous isolates. The organism was also cultured from the shunt valve after removal and from a swab of the burr-hole. Intravenous therapy was started with ceftazidime 2 g tds and gentamicin 120 mg tds and was continued for 3 weeks. The CSF became sterile with 24 h and the patient recovered from the meningitis and was discharged to a long-term care unit.

Patient 4, AG

A 36-year-old man, treated for acute lymphoblastic leukaemia, underwent exploration and biospy of a cerebellar mass, diagnosed histologically as a grade III/IV astrocytoma. A screen of unhealed surgical incisions on the ward yielded a growth of *S. marcescens* from the patient's craniotomy wound on 9 January 1988. This patient had a poor prognosis and died of his underlying disease 6 days later. Antibiotic therapy was not given.

The first three of the *S. marcescens* infections described occurred within 5 days in patients on the same neurosurgery ward. Immediate action was necessary to determine the source of the organism and therefore an epidemiological survey was undertaken.

Epidemiological survey

Screening of medical, nursing and ancillary staff on the neurosurgical ward was carried out. Fifty-one swabs were taken from the nose, throat and hands of 23 people. Environmental swabs of equipment and solutions were taken from 28 sites in the operating theatre and from 14 sites in the ward. The unhealed surgical wounds of 3 patients were also swabbed.

MATERIALS AND METHODS

Swabs and aliquots of $10\,\mu l$ of fluids were plated on to cysteine lactose electrolyte-deficient medium (CLED Oxoid CM423) on to which disks containing $10\,\mu g$ of ampicillin were placed. After 18 h incubation at 37 °C, non-lactose-fermenting Gram-negative bacilli resistant to ampicillin were identified by API 20E. Isolates of S. marcescens were investigated further.

Typing

Serotyping, bacteriocin and phage typing of isolates of *S. marcescens* were performed at the Central Public Health Laboratory, Colindale. We also carried out polyacrylamide gel electrophoresis of ³⁵S-methionine-labelled whole-cell proteins, following the method described previously (Tabaqchali *et al.* 1984; Stephenson & Tabaqchali, 1986).

RESULTS

All screening specimens taken during the epidemiological survey were negative for S. marcescens except for patient 4, AG, and a brush used to apply povidone-iodine solution to the scalp before pre-operative shaving in theatre.

A computer search for all *S. marcescens* isolates in the preceding 6 months revealed another case of post-operative meningitis in a patient who had been on the same ward 2 months previously in November 1987. The 73-year-old woman with a ventricular catheter *in situ* died on intravenous cefotaxime treatment.

Unfortunately, the culture of serratia from this final patient was not available for further study. However, the antibiogram was identical to that of the other isolates. The serratia from the other four patients and the shaving brush were indistinguishable by typing with sera (type 014), bacteriocins (6177) and phages (reactions with phages 3, 4, 5, 8, 9, 10, 11). The polyacrylamide gel electrophoresis of [35S]-methionine-labelled whole-cell proteins also showed that the radiolabelled protein patterns of all the S. marcescens strains isolated from the patients and from the shaving brush were indistinguishable.

Control measures

The brush was removed and shaving continued with single-use sponges and scalpels. Ceftazidime was given to patients requiring antibiotic prophylaxis for

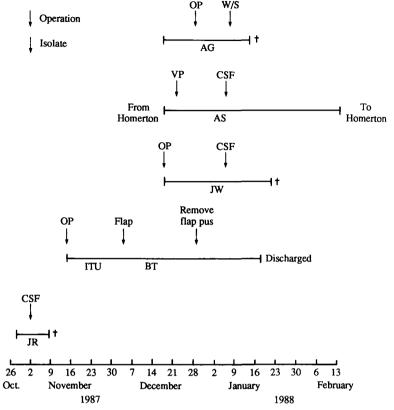


Fig. 1. Serratia infections on a neurosurgical ward.

surgery, but cancellation of operating schedules during holidays limited the need for its use. The infected patients were isolated in a four-bedded side room with cohort nursing. Attendants were disposable gloves and aprons, and disinfected hands with alcohol rub after leaving and between patients. Rescreening did not reveal any positive results.

The time course of the outbreak is illustrated in Fig. 1.

DISCUSSION

Cases of *S. marcescens* meningitis described in the literature have been associated with neonatal septicaemia, neurosurgery, head injury, or followed lumbar puncture (Rogers & Gittens, 1974; Von Graevenitz, 1980) and the patients reported here are included in these categories. Typing of the organisms provided evidence that the same strain was causing the outbreak and the use of ³⁵S-methionine-labelled proteins provided a rapid method for the fingerprinting of the strains. The outbreak strain was serotype 014 and the high prevalence of this strain in sporadic and epidemic *S. marcescens* infections might indicate enhanced virulence among these strains (Acar, 1986; Tabaqchali, Chambers & Brooks, 1977).

Following cardiac surgery, a series of wound infections caused by a pigmented

strain of S. marcescens was traced to contamination of the hands and equipment of barbers, including shaving brushes (Wilhelmi et al. 1987). The latter have also been implicated in an outbreak of serratia infection in an Intensive Care Unit, but the organism did not cause meningitis (Whitby, Blair & Rampling, 1972). However, a series of post-neurosurgery wound infections and meningitis due to Pseudomonas aeruginosa was traced to a shaving brush used for pre-operative scalp depilation (Ayliffe et al. 1965).

In our outbreak the stages by which the shaving brush became contaminated with S. marcescens are unknown, but the organism probably originated from a patient with skin colonization. The practised procedure of rinsing the brush with hot water after use and allowing it to dry in air was inadequate.

This outbreak illustrates previously documented features of the epidemiology of infection with *S. marcescens*. The organism can survive in close contact with disinfectants or antiseptics (Sautter, Mattman & Legaspi, 1984). In our outbreak, it survived repeated contact with povidone-iodine solutions and washing of the brush after use. This underlines the need for disposable equipment when appropriate.

Secondary reservoirs of serratia were not detected and removal of the brush terminated the outbreak. Disposable sponges soaked in povidone-iodine solution have now been recommended and are use to lather the scalp before shaving. Further control measures suggested include the revision of policies allowing widespread use of broad-spectrum antibiotics, particulary in hospitals with endemic problems with antibiotic-resistant serratia (Farmer et al. 1976).

No new infections with the epidemic strain have been discovered in the 5 months since the source was eliminated.

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REFERENCES

- Acar, J. F. (1986). Serratia marcescens infections. Infection Control 7, 273-278.
- AYLIFFE, G. A. J., LOWBURY, E. J. L., HAMILTON, J. G., SMALL, J. M., ASHESHOV, E. A. & PARKER, M. T. (1965). Hospital infection with *Pseudomonas aeruginosa* in neurosurgery. *Lancet* 2, 365-369.
- DASCHNER, F. D. (1980). The epidemiology of Serratia marcescens. In The Genus Serratia (ed. A. Von Graevenitz and S. J. Rubin), pp. 187-196. Boca Raton: CRC Press.
- FARMER, J. J. III, DAVIES, B. R., HICKMAN, F. W., PRESLEY, D. B., BODEY, G. P., NEGUT, M. & BOBO, R. A. (1976). Detection of Serratia outbreaks in hospital. *Lancet* ii, 73-83.
- GRIMONT, P. A. D. & GRIMONT, F. (1978). Biotyping of Serratia marcescens and its use in epidemiological studies. Journal of Clinical Microbiology 8, 73-83.
- McCormack, R. C. & Kunin, C. M. (1966). Control of a single source nursery epidemic due to Serratia marcescens. Pediatrics 37, 750-755.
- Maki, D. G., Hennekens, C. G., Phillips, C. W., Shaw, W. V. & Bennett, J. V. (1973). Nosocomial urinary tract infection with *Serratia marcescens*: an epidemiologic study. *Journal of Infectious Diseases* 128, 579–587.
- PALMER, P. H. & McCracken, L. M. (1970). Contaminated antiseptic solutions. Lancet ii, 776-777.

- RINGROSE, R. E., McKown, B., Felton, F. G., Barclay, B. O., Muchmore, H. G. & Rhoades, E. R. (1968). A hospital outbreak of *Serratia marcescens* associated with ultrasonic nebulizers. *Annals of Internal Medicine* **69**, 719–729.
- ROGERS, K. B. & GITTENS, B. (1974). An epidemic due to Serratia marcescens in a Neurosurgical Unit. Journal of Clinical Pathology 27, 930.
- Sanders, C. V., Luby, J. P., Johanson, W. G., Barnett, J. A. & Sanford, J. P. (1970). Serratia marcescens infections from inhalational therapy medications: nosomial outbreak. Annals of Internal Medicine 73, 15-21.
- SAUTTER, R. L., MATTMAN, L. H. & LEGASPI, R. C. (1984). Serratia marcescens meningitis associated with a contaminated benzalkonium chloride solution. Infection Control 5, 223.
- Schaberg, D. R., Alford, R. H., Anderson, R., Farmer, J. J. III, Melly M. A. & Schaffner, W. (1976). An outbreak of nosocomial infection due to multiply resistant Serratia marcescens: evidence of inter-hospital spread. Journal of Infectious Diseases 134, 181–188.
- Stamm, W. E., Kolff, C. A., Dones, E. M., Javariz, R., Anderson, R. L., Farmer, J. J. III & De Quinones, H. R. (1976). A nursery outbreak caused by Serratia marcescens scalp vein needles as a portal of entry. *Journal of Pediatrics* 89, 96–99.
- STEPHENSON, J. R. & Tabaqchali, S. (1986). New method for typing coagulase negative staphylococci. *Journal of Clinical Pathology* 39, 1271-1275.
- STOKES, E. J. & WATERWORTH, P. M. (1972). Antibiotic Sensitivity Tests. Association of Clinical Pathologists Broadsheet No. 55 (revised).
- Tabaqchali, S., Chambers, T. J. & Brooks, H. J. L. (1977). Serratia marcescens in hospital practice. Lancet i, 306-307.
- Tabaqchali, S., Holland, D., O'Farrell, S. & Silman, R. (1984). Typing scheme for Clostridium difficile: its application in clinical and epidemiological studies. Lancet i, 935-938.
- Von Graevenitz, A. (1980). Infection and colonisation with serratia. In *The Genus Serratia* (ed, A. Von Graevnitz and S. J. Rubin), pp. 167–186. Boca Raton, CRC Press.
- WHITBY, J. L., BLAIR, J. N. & RAMPLING, A. (1972). Cross-infection with Serratia marcescens in an intensive therapy unit. Lancet ii, 127-129.
- WILHELMI, I., BERNALDO DE QUIROS, J. C. L., ROMERO-VIVAS, J., DUARTE, J., ROJO, E. & BOUZA, E. (1987). Epidemic outbreak of Serratia marcescens infection in a cardiac surgery unit. Journal of Clinical Microbiology 25, 1298-1300.