Cross-infection with *Serratia marcescens*

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SYNOPSIS  Cross-infection in a urological unit due to *Serratia marcescens* is reported. The bacteriology of the organism and its mode of spread are described. It is suggested that *Serratia marcescens* may be a more virulent organism than is generally believed, especially in situations in which there is an excess of mucus.

The normal habitat of *Serratia marcescens* (*Chromobacterium prodigiosum*) is in soil and water. It is usually considered not to be pathogenic to man, but on a number of occasions has been isolated from situations which suggest that it can sometimes behave as a human pathogen. Thus Woodward and Clarke (1913) described its isolation from a patient with bronchiectasis who produced red sputum, and Aronson and Alderman (1943) reported meningitis caused by *Serratia marcescens* septicaemia has followed superficial infection of an extensive burn (Graber, Tumbusch, Rudnicki, and Vogel, 1960), and mild acute illness has followed human exposure to *Serratia marcescens* aerosols (Paine, 1946). Urinary tract infections caused by a member of the chromobacterium group, probably *Serratia marcescens*, were reported by Wheat, Zuckerman, and Rantz (1951). They described 11 cases following genito-urinary manipulation; two developed bacteraemia, one subsequently died of *serratia* bacterial endocarditis.

None of the published reports give accounts of cross infection with this organism. The seven cases recorded here show that cross infection may occur, adding further evidence that *Serratia marcescens* is a potential human pathogen.

CASE REPORTS

CASE 1  A man aged 40 years had been known to have diabetes mellitus for the past 15 years. He was reasonably well until he was scratched by a cat in November 1960 and was given an injection of anti-tetanus serum. He developed severe swelling of the tissues surrounding the scratch and became pyrexial, had rigors, and vomited.

This pyrexial illness persisted intermittently for several weeks and he was admitted to the local hospital. Whatever the significance of the cat scratch, the symptoms were thought to be due to a urinary infection. He was treated with antibiotics, but relapsed when treatment was withdrawn. He was transferred to the Manchester Royal Infirmary.

Investigation revealed a tight stricture of the penile urethra. The urine was found to contain large numbers of pus cells, and culture yielded a heavy pure growth of *Serratia marcescens*. Blood cultures on three occasions were sterile. The Wassermann reaction, Price's precipitation reaction, and the gonococcal complement-fixation test were all negative, leaving the aetiology of the stricture in doubt.

He was treated by two-stage urethroplasty with closed bladder drainage from a suprapubic cystotomy wound. *Serratia marcescens* was isolated from his urine on seven occasions during a period of two months. The infection failed to respond to treatment either with chloramphenicol or with methenamine mandelate.

CASE 2  A man aged 80 years was admitted with acute retention of urine. Culture of the urine at this time was sterile. Investigation showed the patient to be suffering from carcinoma of the prostate, and he was treated by trans-urethral prostatectomy with postoperative closed bladder drainage. Urine examined six days after operation showed large numbers of pus cells, and cultures yielded a moderate growth of *Serratia marcescens* together with *Streptococcus faecalis*.

CASE 3  A man aged 64 years was admitted for investigation of haematuria. He was found to have a carcinoma of the bladder which was partially removed at cystoscopy. At this time his urine contained only occasional pus cells and was sterile. Post-operative closed bladder drainage was instituted, and urine examined six days later contained large numbers of pus cells and yielded a moderate mixed growth of *Serratia marcescens* and *Str. faecalis*. Treatment with methenamine mandelate resulted in the disappearance of the *Str. faecalis* but the *Serratia marcescens* persisted. It was still present together with large numbers

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of pus cells when he was transferred to another hospital for radiotherapy.

CASE 4 A boy aged 11 years was admitted for investigation of incontinence. He was found to have a diverticulum of the bladder and this was excised. Urine examined at the time of operation was sterile. After operation he was treated by closed bladder drainage. Six days later the urine contained large numbers of pus cells, and yielded a heavy mixed growth of *Serratia marcescens* and *Escherichia coli*. A later specimen produced a growth of *S. marcescens* only, although no antibiotic treatment had been given in the meantime.

CASE 5 A man aged 69 was admitted to hospital with acute retention of urine. He was known to have had a urethral stricture for many years, and he was now found to suffer from prostatic hypertrophy in addition. He was treated by dilatation of the urethra and continuous bladder drainage. Later a transurethral prostatectomy was carried out. Following operation a urinary tract infection due to *Esch. coli* developed. This responded to a course of sulphonamide resulting in a sterile urine. Later the pyuria recurred and urine culture produced a heavy growth of *S. marcescens* together with scanty colonies of a Proteus.

CASE 6 A man aged 73 was admitted complaining of urinary frequency and nocturia. He was found to have prostatic hypertrophy and a suprapubic prostatectomy was carried out. At the time of operation the urine was found to be sterile. Closed bladder drainage was instituted after operation. A severe haematemesis four days after the prostatectomy necessitated a partial gastrectomy. Examination of the urine six days after catheterization revealed moderate numbers of pus cells and a pure growth of *S. marcescens* was obtained on culture.

CASE 7 A man aged 75 was admitted to hospital in congestive heart failure and with benign prostatic enlargement. The congestive failure was treated and he was placed on continuous closed bladder drainage. At this time the urine was found to be sterile. Examination of the urine 12 days after admission showed large numbers of pus cells and culture produced a heavy mixed growth of *Serratia marcescens*, a coliform, and *Str. faecalis*. He was treated with chloramphenicol for six days when a specimen of urine still contained numerous pus cells and cultures yielded a heavy pure growth of *S. marcescens*.

**BACTERIOLOGY**

The strain of the organism isolated from all seven patients gave identical reactions. It was identified as *Serratia marcescens* on the following criteria (Breed, Murray, and Smith, 1937). It was a Gram-negative motile bacillus growing equally well at room temperature and at 37°C on both nutrient agar and on McConkey agar. When grown at room temperature it produced a bright red, non-diffusible pigment which was only very slightly soluble in water at neutral pH but was readily soluble in ether, chloroform, or alcohol. An alcoholic solution became orange-yellow with the addition of alkali and bright red at acid pH.

The organism fermented glucose, maltose, mannitol, dulcitol, and salicin producing acid but no gas. Lactose became slightly fermented after 10 days' incubation. Other reactions were as follows:—Indole negative, M, R. negative, Voges-Proskauer positive and growth and positive in Koscer's citrate, catalase positive, coagulase negative, H2S negative, Möller's KCN positive. Litmus milk turned acid and clotted and when grown in gelatine it produced infundibuliform liquefaction and intense pigment.

The organism was tested for antibiotic sensitivity by the disc technique. It was resistant to penicillin, streptomycin, chloramphenicol, tetracycline, sulphonamide, and nitrofurantoin and sensitive to kanamycin, polymyxin, and methenamine mandelate.

**MODE OF CROSS-INFECTION**

All the *Serratia marcescens* urinary tract infections occurred during a period of one month and all the patients were treated in the same urological ward. With the exception of the first patient who was admitted already suffering from the infection, all the other patients developed urinary tract infections due to *S. marcescens* within a short time of catheterization. Before transfer to the surgical ward Case 1 was investigated in another ward without being catheterized. No serratia infections were recognized in this ward. These facts strongly suggest that catheterization played an important part in the spread of the organism. It was thought to be unlikely that catheterization in the operating theatre was the source of the infection as other patients treated in the same theatre by similar techniques but nursed in a different ward did not develop urinary tract infections due to *S. marcescens*, nor did patients on the same urological ward who were treated by means other than closed bladder drainage.

The possibility of aerial transfer of the organism within the ward was investigated, and attempts made to isolate it from the air, dust, bed linen, curtains, and pieces of ward equipment. All were unsuccessful. The method used to sterilize the catheters and collecting bottles was tested and found to be adequate. Thus faulty aseptic technique in the management of the bladder drainage seemed to be the most likely mode of spread. It was found that in all cases the closed drainage systems were being opened frequently in order to take catheter specimens of urine for examination. For part of the time Case 1 had samples of urine examined for sugar at four-hourly intervals because of his diabetes. The ward routine was to collect specimens of urine and empty the collecting bottles serially from patient to patient, so that should an unsatisfactory aseptic technique be employed cross-infection was inevitable. Careful attention to aseptic technique and the restriction of the number of times the closed drainage systems were opened has resulted in no further cases of *S. marcescens* infection in the ward.
DISCUSSION

The relative infrequency with which infections due to Serratia marcescens are reported would suggest either that this organism behaves as a very low-grade pathogen, and hence only causes infections in very favourable conditions, or that infections are more common but fail to be recognized. Many of the reported cases of infection due to Serratia marcescens have occurred in association with chronic disease. In these circumstances it would be expected that either local or general resistance to infection would be low. Thus Serratia marcescens infections have complicated established urinary tract disease (Wheat et al., 1951), chronic pulmonary disease (Woodward and Clarke, 1913; Robinson and Woolley, 1957; Gale and Lord, 1957), severe burns (Graber et al., 1960), and death due to recurrent peptic ulcer (Patterson et al., 1952). On the other hand, the occurrence of seven cases of Serratia marcescens infection in one surgical ward at the same time shows that in some circumstances this organism can behave in a much more communicable manner than has been previously recognized. Further, the finding of considerable pyuria, associated in five of the cases with a pure growth of Serratia marcescens, suggests that this organism must be considered to be a human pathogen. It has been found that the mouse virulence of a strain of Serratia marcescens was increased when the organisms were injected together with mucus (Gale and Lord, 1957), and perhaps the excessive mucus secreted around the indwelling catheters of the cases described played a part in increasing the virulence of the organism.

Serratia marcescens does not produce the characteristic pigment if it is grown at 37°C. Therefore, if media are not incubated at a temperature lower than 37°C, or non-lactose-fermenters growing on McConkey’s medium are not investigated in detail, then strains of Serratia marcescens may be reported as ‘atypical coliform organisms’. For this reason Serratia marcescens infections may be more common than is recognized. It should be noted, however, that a survey of the Enterobacteriaceae isolated from infective lesions in the Manchester Royal Infirmary over a period of four months did not reveal any strains of Serratia marcescens other than those described here.

In common with other strains of Serratia marcescens described in the literature the organism responsible for this outbreak proved to be resistant to most of the available antibiotics. Although sensitive by the disc technique to methenamine mandelate, treatment by this substance failed to cure the infection. Chloramphenicol was suggested by Robinson and Woolley (1957) and by Papapanagiotou and Aligizakis (1959) but this antibiotic failed to improve the three patients in this series in whom it was tried. The ideal form of treatment for Serratia marcescens infections is still to be found.

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