

Serratia marcescens: Biochemical, Serological, and Epidemiological Characteristics and Antibiotic Susceptibility of Strains Isolated at Boston City Hospital

JAMES N. WILFERT,¹ FRED F. BARRETT,² W. H. EWING, MAXWELL FINLAND,
AND EDWARD H. KASS

Channing Laboratory, Thorndike Memorial Laboratory, Harvard Medical Unit, and Department of Medical Microbiology, Boston City Hospital, the Department of Medicine, Harvard Medical School, Boston, Massachusetts 02118, and the Enteric Bacteriology Laboratory, National Communicable Disease Center, Atlanta, Georgia 30333

Received for publication 17 November 1969

The biochemical, serological, and epidemiological characteristics of 95 strains of *Serratia marcescens* isolated at the Boston City Hospital were examined. Several strains were shown to be endemic, and the majority of isolates were cultured from urine or respiratory secretions. *Serratia* species were highly resistant to polymyxin B and the cephalosporins, and various proportions were also resistant to other antibiotics including kanamycin, but all of the isolates were sensitive to gentamicin. The appearance of resistance to kanamycin and nalidixic acid among endemic strains was demonstrated. The nosocomial nature of *Serratia* infections, particularly those involving the urinary tract, was confirmed. Many clinical bacteriology laboratories currently fail to identify the nonpigmented strains.

The problem of hospital-acquired infections continues to be a major one. Gram-negative bacilli, particularly *Klebsiella pneumoniae* and *Pseudomonas aeruginosa*, have been the most significant recently, but other species are also increasing in importance. In 1967 at the Boston City Hospital, an increasing incidence of isolations of *Serratia marcescens* in the clinical bacteriology laboratory was noted and led to the present study.

S. marcescens Bizio is a gram-negative bacterium of the family *Enterobacteriaceae* and the tribe *Klebsielleae*. Some strains produce a red pigment, prodigiosin, and episodes dating back to antiquity of the "miraculous" appearance of "blood" on bread, communion wafers, and other starchy foods were probably from such *Serratia* strains (15). In recent years, the organism has been recognized as causing "pseudohemoptysis" (14, 26) and the "red diaper syndrome" (35). *Serratia* species were long considered a non-pathogen for humans, and pigment-producing strains were used in the past as markers in ex-

perimental work. However, in 1913, *Serratia* was first reported as causing a pulmonary infection (38), and since then it was shown to be capable of causing suppurative infections of wounds, pneumonia, lung abscess, empyema, meningitis, urinary tract infections, endocarditis, septic arthritis, osteomyelitis, peritonitis, sinusitis, and septicemia. The literature on *Serratia* infections was reviewed recently by Ringrose et al. (25).

It has also been observed that nonpigmented strains of *Serratia* were more common than pigmented ones (5, 10) and that many clinical bacteriology laboratories were unable to identify nonpigmented strains correctly (13). In 1962, Ewing et al. (11) pointed out the nosocomial nature of many *Serratia* infections. Several hospital outbreaks involving urinary tract infections (1, 4, 18, 22, 31, 36) and respiratory tract infections (2, 3, 25) and two epidemics in nurseries for newborn infants (21, 30) have been described. Infections also have been noted to occur at the site of indwelling intravenous catheters (7, 23, 37) and after lumbar punctures (16, 32) or peritoneal dialysis (21). Previous antibiotic therapy and underlying chronic debilitating disease may also predispose to serious *Serratia* infection (7,

¹ Present address: Division of Infectious Diseases, Department of Medicine, University of Utah College of Medicine, Salt Lake City, Utah.

² Present address: Department of Pediatrics, Baylor College of Medicine, Houston, Tex.

37). Treatment of these infections may be a difficult problem because many recently isolated strains of this organism have been resistant to many antibiotics, although most isolates have been sensitive to kanamycin and all to gentamicin (27, 33, 37).

The present study deals with the isolation, identification, and some of the epidemiological features of the *Serratia* strains isolated at the Boston City Hospital.

MATERIALS AND METHODS

During the 5 months, September 1967 through January 1968, all isolates of *S. marcescens* obtained from cultures of patients in the outpatient department, accident floor, and hospital wards were collected. Multiple isolates from the same source in any patient were usually not collected, but an effort was made to obtain isolates from various sites in the same patient.

Initial identification was made in the Bacteriology Laboratory under the supervision of A. Kathleen Daly and Alice McDonald. *Serratia* strains were identified by previously described methods used in this laboratory (8, 12) and confirmed at the Enteric Bacteriology Laboratory of the Communicable Disease Center which also carried out serological identification (8).

Antibiotic sensitivity tests were done by the inocula-replicating method of Steers et al. (29). The 111 strains tested included the original isolates plus the isolates from different sites in these same patients. An overnight culture in Brain Heart Infusion Broth (Difco) was diluted 10^{-4} and replicated on Beef Heart Infusion Agar (Difco) containing serial twofold dilutions of the antibiotics. The antibiotics used and their suppliers were: streptomycin sulfate (E. R. Squibb & Sons); sodium cephalothin, cephaloridine, cephalixin, and cephaloglycin (Eli Lilly & Co.); sodium ampicillin and kanamycin sulfate (Bristol Laboratories Div.); tetracycline hydrochloride (Lederle Laboratories); chloramphenicol (Parke, Davis & Co.); polymyxin B sulfate (Burroughs Wellcome & Co.); gentamicin sulfate (Schering Corp.); rifampin (Ciba Corp.); and carbenicillin (Beecham Products Inc.).

Clinical and epidemiological data were obtained from the patients' hospital records. The information sought included temporal sequences in relation to hospitalization, prior antibiotic therapy, results of all bacteriological cultures, severity of the illness, underlying diseases and all prior surgical procedures, or manipulations of the genitourinary or respiratory tract. An organism was considered to be hospital-acquired when previous cultures were negative for *Serratia*, and the clinical significance of the isolates was based on the presence of signs of clinical infection when *Serratia* was the only or predominant organism. Bacteriuria was considered to be significant only when the colony counts of urine cultures were $> 10^5$ /ml (17). Isolates were streaked and stabbed on Trypticase Soy Agar slants in tubes (13 by 100 mm), and then sealed with paraffin-impregnated corks and stored at room temperature in the dark.

TABLE 1. Biochemical reactions of 95 isolates of *Serratia marcescens*

Test or substrate	Per cent positive		Per cent negative
	1-2 Days	3-14 Days	
Hydrogen sulfide (TSI)			100
Voges-Proskauer (37 C)	99		1
Indole	1 (week)		99
Citrate (Simmon's)	100		
Motility	100		
Gelatin (22 C)	90.5	9.5	
Lysine decarboxylase	100		
Ornithine decarboxylase	100		
Glucose: acid	100		
Glucose: gas ^a	55.8		44.2
Lactose			100
Dulcitol			100
Adonitol: acid	83.1	7.4	9.5
Adonitol: gas			100
Inositol: acid	94.7	2.1	3.2
			100
Arabinose			100
Raffinose			100
Rhamnose			100
Lipase (corn oil)	97.9	2.1	
Cellobiose: acid	9.5	16.9	73.6
Cellobiose: gas			100
Glycerol: acid	99	1	
Glycerol: gas			100
Deoxyribonuclease	92.4		7.6
Pigment	7.4		92.6

^a Less than 10% volume.

RESULTS

Biochemical reactions. The biochemical reactions of *S. marcescens* and its differentiation from other organisms have been reported in detail elsewhere (6, 8, 9). The reactions of the 95 primary isolates from this study are given in Table 1. It will be seen that these strains were highly consistent in most of their reactions, and reactions were complete in 1 to 2 days in most instances. At this hospital, the reactions most helpful for identifying *Serratia* species have been the consistent inability to ferment lactose, positive lysine decarboxylase, and the inability of *Serratia* to form more than small amounts ($< 10\%$ in a Durham tube) of gas with glucose as a substrate. The only subspecies, *S. marcescens* var. *kiliensis*, differs from the type species *S. marcescens* only in that it is Voges-Proskauer negative. Only one such strain was found. Pigment was produced by only 7.4% of strains isolated.

Serological studies. To date, 15 somatic ("O") and 13 flagellar ("H") antigens have been established for *Serratia*. Difficulty in serotyping because of "roughness" has not been a major problem; the majority of strains from a variety of sources and localities are typable. There were 23

TABLE 2. *Distribution of serotypes of Serratia marcescens*

O antigens ^a	H antigens ^b											Total	
	1	2	3	4	5	7	8	11	12	13	H undetermined		
1					1								1
2	1			21				1			1		25
4	17			1						1			19
5	3	1								4	1		9
6			1	2									3
7						1							1
9								2					2
11				10						5	3		18
12								1					1
13				1									1
14		1		1					3			1	6
O undetermined							2	1				5	8
Rough				1									1
Total	21	2	1	37	1	1	2	5	3	11	11		95

^a There were no serotypes of O antigens 3, 8, 10, and 15.

^b There were no serotypes of H antigens 6, 9, and 10.

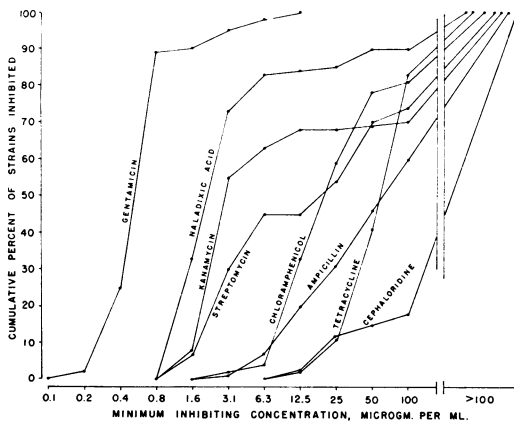


FIG. 1. *Susceptibility of Serratia marcescens to eight antibiotics: 111 strains isolated at Boston City Hospital, September 1967 through January 1968. All strains were resistant to 100 µg/ml of cephalothin and polymyxin B (not depicted in this figure).*

different serotypes among the present isolates (Table 2). The O antigens of 8 strains were not identifiable with available antisera, and the H antigens of 11 were undetermined. For nine strains only the O or the H antigen was identifiable; nine strains were completely untypable, but only one strain was rough. The four most frequent serotypes were O2:H4 (22%), O4:H1 (18%), O11:H4 (11%), and O11:H13 (5%).

In most cases, multiple isolates from the same source or isolates from different sources in the same patient were not serotyped separately. However, in those cases in which multiple isolates were

serotyped, the type was the same; these cases are not included in the analysis. In one culture from the stool of an infant in the outpatient department, two serotypes were identified. Three of the eight pigmented strains were of the same serotype.

Antibiotic susceptibility. In addition to the antibiotics shown in Fig. 1, all strains were tested with cephalothin and polymyxin B, but none was inhibited by a concentration of 100 µg/ml of either antibiotic. Nine strains were tested against rifampin, cephalixin, cephaloglycin, and carbenicillin. All were resistant to 100 µg/ml of cephalixin and cephaloglycin. Rifampin inhibited six strains at a concentration of 50 µg/ml and three at 100 µg/ml; carbenicillin inhibited one strain at 25 µg/ml and one at 200 µg/ml, but the other seven strains grew well in the latter concentration. By using a tube-dilution method, Thornton and Andriole (33) found that 8 of 35 isolates of *Serratia* had a minimal bactericidal concentration of 50 µg/ml or less against carbenicillin and suggested that this antibiotic might be of use in the treatment of urinary tract infections. Nitrofurantoin was not tested against the present strains because standard discs containing 100 µg have consistently shown no zone of inhibition.

As previously noted (37), nearly all *Serratia* strains are susceptible to gentamicin; all but 2 of the 111 isolates tested in this study were inhibited by 6.3 µg/ml or less (within the safe and nontoxic range for gentamicin serum levels). Nalidixic acid was inhibitory at 3.1 µg/ml for about 80% of the strains, and the rest required much higher concentrations. Thirty-two per cent of the isolates were resistant to 25 µg of kanamycin per ml, and

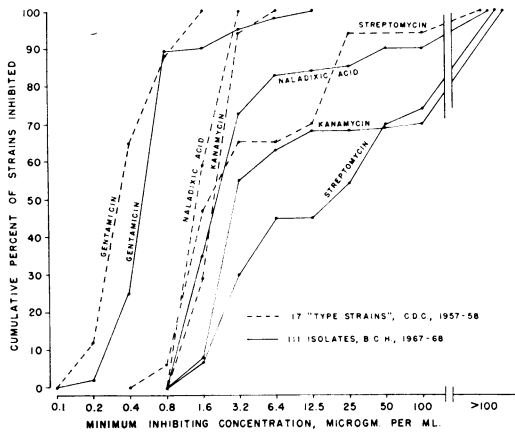


FIG. 2. Susceptibility of two groups of strains of *Serratia*. Comparison of the 111 isolates in this study with 17 strains received 10 years earlier at the Communicable Disease Center.

55% were resistant to this concentration of streptomycin. As can be seen from Fig. 1, the other antibiotics were even less active.

In an effort to ascertain whether drug resistance had developed or increased in *Serratia* species over the past 10 years, tests were done with 17 strains which were sent to the Communicable Disease Center in 1957-58 from various laboratories and used there as the type strains for preparing the original antisera for typing. The susceptibility of these 17 strains to those antibiotics for which changes were noted is compared with that of the strains isolated in the present study (Fig. 2). All of the earlier strains were susceptible to kanamycin and nalidixic acid, neither of which was in use in this country at that time. Furthermore, 18 of the 25 recent strains that were found to be resistant to kanamycin were of the 4 serotypes that are endemic in this hospital. In the case of nalidixic acid, 11 of the 14 strains that were not inhibited by 25 $\mu\text{g}/\text{ml}$ were of the same 4 serotypes; all but one of these strains were isolated from urine. Kanamycin was used frequently in this hospital, and the appearance of resistance was not unexpected in endemic strains. Nalidixic acid, on the other hand, was infrequently used here from 1964 through 1967, and resistance to it cannot be explained on this basis.

Epidemiologic observations. The sources of the 94 cultures that yielded *Serratia* species and the other sites with positive cultures in the same patients are shown in Table 3, and Table 4 gives the incidence of hospital acquisition and the estimate of the clinical significance of the strains according to their source. The most common sources were urine and sputum (including specimens obtained

TABLE 3. Sources of *Serratia* isolates

Source	No. of isolates	Other sources in same patients			
		Urine	Sputum ^a	Wound	Blood
Urine	34		1	4	3
Sputum	48	3		1	
Wound	7				
Blood	3	1	1	1	
Feces	2		1 ^a		

^a Includes one isolate from nasopharynx.

TABLE 4. Acquisition and clinical significance of *Serratia* isolates

Source	No. of isolates ^a	Hospital-acquired		Clinically significant	
		No.	Per cent	No.	Per cent
Urine	38	25	66	26	68
Sputum	52	24	47	3	6
Wounds ^b	13	11	85	4	36
Blood	6	6	100	5	
Feces	2	0		0	

^a Includes secondary sources.

^b Includes venous catheters.

by endotracheal suction, via a tracheostomy tube, or nasopharyngeal swabs). Patients with urinary isolates rarely had the organism in their sputum and vice versa. *Serratia* species were rarely isolated from stool specimens; the two strains in the present study were from infants. One of these two strains was pigmented, and the same serotype was also present in the nasopharynx of the same infant.

Bacteremia resulting from *S. marcescens* in patients at this hospital has previously been reported (37). In the present survey, there are three primary and three secondary isolations of *Serratia* species from blood cultures. In five of these six cases, the portal of entry was the urinary tract (four cases) and an indwelling venous catheter in one; in the sixth, blood cultures were drawn through an indwelling venous catheter and yielded three different organisms in addition to *Serratia* strains, so that contamination may have accounted for that isolation.

Of the 13 isolations of *S. marcescens* from wound cultures, 3 were cultured directly from indwelling venous catheters, and one was from a "scalp vein needle" withdrawn from a vein around which there was a cellulitis. The wounds from which three other isolates were grown followed abdominal or genitourinary surgery, and cultures of the urine in these cases also yielded

TABLE 5. *Distribution of the more frequent serotypes according to source of specimen and location of patient**

Serotype	Source				Location						
	Sputum	Urine	Wound	Blood	Genitourinary ward	Outpatient department	Medical ward			Neurology	Others
							A	B	C		
O2:H4	1	18		2	16	1	2				2
O4:H1	12	1	3	1				4		4	9
O11:H4	8	2					3		3		4
O11:H13		4	1		1	3					1

* Values indicate number of patients.

TABLE 6. *Factors associated with isolation of Serratia strains*

Source	No. of strains	Associated conditions	No. of strains	Per cent of source
Urine	38	Indwelling catheter	28	74
		Urological surgery	15	39
		Cystoscopy	13	13
		One or more of the above	32	84
		None of the above	6	16
Sputum	51	Intermittent positive pressure breathing	12	24
		Nebulizer	16	31
		Intubation	15	29
		One or more of the above	26	51
		None of the above	25	49

Serratia species. The other wounds followed surgical procedures except one from a case of cellulitis acquired outside the hospital.

The data in Table 5 suggest that strains of the four most frequently isolated serotypes were nosocomial. Of the 21 isolates of O2:H4, 8 were from urine; three of the patients with positive blood cultures also had this serotype in their urine. Each of these 21 patients had acquired the organism in the hospital or outpatient department; 16 patients were on the male genitourinary ward, and one patient, who was hospitalized here prior to the present study, was in the urological outpatient department. Serotype O11:H13 was isolated four times from urine of urological patients and once from a case of nonhospital-acquired cellulitis.

The strains with serotypes O4:H1 and O11:H4 were primarily from respiratory sources; at least 70% of O4:H1 isolates were hospital-acquired. Almost half of the O4:H1 strains were from two wards, one medical and the other neurological-neurosurgical. Fewer of the O11:H4 strains were hospital-acquired and two medical wards were each the source of three of them.

Table 6 lists the procedures associated with colonization or infection in patients who yielded

Serratia species from urine and sputum. Of the patients with *Serratia* strains in their urine, 84% had some form of urological manipulation, indwelling catheterization being the most common, and 80% had received antibiotics. In three cases in which there was no previous manipulation, the *Serratia* strains were probably contaminants.

In two previously described hospital outbreaks of *Serratia* species from respiratory sources, the common source was found to be contaminated intermittent positive pressure breathing machines (IPPB; 3) or ultrasonic nebulizers (25). In the present study, 26 of 52 patients with respiratory isolates of *Serratia* had been subjected to IPPB, nebulization, or some form of endotracheal manipulation; this occurred in 91% of those harboring serotype O4:H1. Antibiotics had been given to 64% of these patients. The seven pigmented strains all were isolated from respiratory sources and were also cultured from feces in one patient and from urine in another.

Clinical observations. The mean age of patients in this study was 61 years, excluding one newborn infant and two infants under 1 year old; 79% were males. Possible factors associated with *Serratia* colonization or infection were sought. None of the patients had received corticosteroids or anti-

metabolites before the first positive culture for *Serratia*, but 7 had diabetes, 23 were chronic alcoholics, 14 had some malignancy, and 17 were in the early postoperative period. Most of the postoperative isolates followed prostatic surgery.

DISCUSSION

The urinary tract has been the most frequent site of *Serratia* infections. The epidemiology of hospital outbreaks of *Serratia* infections of the urinary tract is still unclear. In an outbreak involving 135 cases of urinary tract infections resulting from *Serratia* on the "service" wards (in contrast to wards for private patients), Allen and Conger (1) confirmed the high incidence of indwelling catheterization and urinary tract abnormality noted by others. Their epidemiological study yielded negative results, except for the presence of *Serratia* species on the floor beneath the beds of patients with persistently positive urine cultures. Serotyping of strains was not done, but the uniformity of the antibiotic sensitivity data suggests that a single strain or a small number of strains were involved. Overcrowding was a significant factor, and the suggestion was made that closed drainage systems for all catheters, twice daily mopping of the floor with germicidal solutions, and isolation of infected patients might be useful in preventing or combating such outbreaks.

Wheat et al. (36) reported 11 patients with urinary infections from pigmented strains and noted that all had undergone instrumentation of the urinary tract. An attempt to determine the source of the organism was unrevealing; serotyping was not done. Magnuson and Elston (22) reported seven cases of *Serratia* infection, five of which were in the urinary tract. Two of the urinary strains were typed as O5:H1, and a wound infection also yielded this serotype. In another case, *Serratia* O5:H10 was isolated from sputum and urine and also from floor dust adjacent to the patient's bed.

In a series of 104 patients from whom *Serratia* was isolated from various sites over one year, Clayton and von Graevenitz (4) noted six cases, including four urinary tract infections, which occurred within 5 days on one ward. In the survey reported by Ewing et al. (11) of the serotypes of *Serratia* from three hospitals, 19 strains in one hospital were isolated from urine and three serotypes predominated, O4:H4, O13:H7, and O14:H4. Lancaster (18) reported 20 cases of *Serratia* urinary tract infections which occurred in 10 months; eight of the strains were serotyped but were heterogenous. Detailed epidemiological data were not reported; all but one of the patients had been catheterized. Taylor and Keane (31) described an outbreak of seven cases on a uro-

logical ward from a pigmented strain. All patients had indwelling catheters, and the outbreak was ended by stricter attention to care of the drainage systems. Environmental cultures were negative and serotyping was not done.

In the present study, strains of serotypes O2:H4 and O11:H13 were associated with urinary tract infections acquired mainly in the male urological wards and outpatient clinic. Interestingly, O2:H4 strains were frequently resistant to kanamycin, whereas O11:H13 strains were not. The expected high incidence of urinary tract instrumentation was found, but a point source was never identified. Environmental cultures were not done.

When *Serratia* strains are found in the urine, it is usually of clinical significance. In all of the cases reported by Allen and Conger (1), colony counts were high; this was true in 68% of our cases. However, the organism may appear as a contaminant, and, when this is suspected, cultures should be repeated.

Four of the 26 patients with "significant" bacteriuria developed bacteremias. This high rate may be attributable to the fact that many of these patients had been recently subjected to prostatic surgery, the fresh wound forming an easy portal of entry.

The treatment of urinary tract infections in patients with indwelling catheters or after prostatic surgery is difficult. For *Serratia* species, the presumptive drug of choice currently is gentamicin, unless sensitivity tests show the organism is sensitive to a less toxic agent. For treatment of asymptomatic bacteriuria due to *Serratia* when all catheters have been removed and surgical wounds are healed, nalidixic acid may be useful and perhaps should be tried even when disc sensitivity studies show resistance, since drug levels in the urine may reach 50 to 200 $\mu\text{g/ml}$ (19).

Although the most frequent source of *Serratia* isolates in this study was the respiratory tract, these isolations were infrequently of clinical significance (Table 4). In neither of the *Serratia* outbreaks associated with respiratory equipment was the incidence of infection, as opposed to colonization, reported. However, *Serratia* species can cause pneumonia, empyema, and lung abscess. In the two largest series of *Serratia* bacteremias reported, roughly 15% had the respiratory tract as the portal of entry. Tillotson and Finland (34) reported 149 pneumonia cases in which serial cultures were made. *Serratia* strains were not isolated from the initial culture in any of these cases. However, this organism was subsequently isolated from 10 of the patients, in pure culture in 4 of them, but in no instance did superinfection (in contrast to colonization) by this organism ensue.

The findings in this study seem to confirm the role of respiratory equipment and procedures such as endotracheal suction in the spread of *Serratia* species in a hospital. Cabrera (3) showed how a respiratory strain could infect wounds and the urinary tract by demonstrating contamination of containers of "sterile" saline used for irrigation of wounds and catheters and findings that such containers were also used to fill intermittent positive pressure breathing machines which were probably contaminated in this manner. In the present study, intensive epidemiological studies of respiratory equipment were not done since the respiratory strains did not present a clinical problem. Both previous studies pointed out the difficulty in sterilizing respiratory equipment once contamination occurs. Ringrose et al. (25) had to discontinue the use of ultrasonic nebulizers to end the outbreak. Cabrera (3) found that only by discontinuing use of reservoir nebulizers in the IPPB machines and sterilizing the "medication nebulizer," manifold tubing, and mouthpiece between each patient usage could the outbreak be controlled.

The frequency with which the respiratory tract was the source of the pigmented strains is of interest. It is not known whether this represents some propensity on the part of pigmented strains to grow in the respiratory tract or merely reflects the higher number of serotypes and non-hospital acquired strains among the respiratory isolates.

Other types of hospital outbreaks and iatrogenic infections from *Serratia* species deserve comment. Rabinowitz and Schiffrin (24) described an outbreak of 11 cases of meningitis, wound infections, or arthritis on a pediatric ward. *Serratia* was isolated from a single bottle of 5% dextrose in saline used intravenously and topically in several of the cases. In an outbreak in a nursery of newborn infants reported by McCormack and Kunin (20), the reservoir of infection was found to be contaminated caps of "cord saline" bottles. *Serratia* species were isolated from 20 newborns, and in 22% of nursing mothers the breasts were colonized. Eight cases of clinical illness were associated with *S. marcescens* among these patients (five cases of urinary tract infections, and one each of balanitis, omphalitis, and respiratory tract infection). The strain was a pigmented one, O6:H2. Stenderup et al. (30) reported on a more serious epidemic in a maternity hospital affecting premature infants. There were six deaths due to septicemia, six cases of purulent conjunctivitis, and one case of arthritis with possible septicemia. The strain in these cases was nonpigmented, O14:H12. An epidemiological survey was unrevealing.

The iatrogenic nature of other types of *Serratia*

infection is illustrated in several reports. The case of meningitis in the outbreak reported by Rabinowitz and Schiffrin (24) was from intravenous injection of a contaminated solution. In two cases, meningitis followed lumbar puncture for diagnostic purposes (32) or spinal anesthesia (16). Both organisms were pigmented strains; one was serotype O5:H2. Shedden (28) described three cases of meningitis and septicemia which followed insertion of Holter valves for hydrocephalus associated with spina bifida. This cluster of three cases suggests some common source. McCracken and Lipscomb (21) reported three cases of peritonitis associated with peritoneal dialysis; the isolates were all pigmented, and two of the cases occurred simultaneously. The association of *Serratia* infections with indwelling venous catheters noted previously (37) was confirmed in the present study.

The development of hospital-acquired antibiotic resistance is well illustrated in the present study. Although all of the 1957-58 strains were kanamycin-susceptible, 35% of the isolates in the present study, most of them of endemic serotypes, were resistant.

The true dimensions of the problem of nosocomial infections from *Serratia* species will not be known until most clinical bacteriology laboratories are able to identify the nonpigmented strains. In September 1968, the Diagnostic Laboratory of the Massachusetts Department of Public Health sent out a typical strain of *S. marcescens* to the 121 laboratories which participate in its Laboratory Approval Program; 60% of them were unable to identify the organism correctly. It was most frequently called *Enterobacter* or *Pseudomonas* species, but was also identified as *Shigella*, *Proteus*, *Providencia*, or *Escherichia coli*. Recent reports of series of clinical isolates show that many laboratories are still reporting species as "Paracolon," "intermediate coliforms," or "Paracolobactrum" species, and strains of *Serratia* may well be included in these groups. The therapeutical and epidemiological implications of a *Serratia* infection demand its prompt identification. It should be emphasized that *Serratia* strains can be readily identified 1 to 3 days after isolation by using commonly available media.

ACKNOWLEDGMENTS

The authors are grateful to the staff of the Bacteriology Laboratory of the Boston City Hospital for their cooperation, to Betty Davis for performing the serotyping, and to Clare Wilcox for assisting with the susceptibility tests.

This study was supported by grant HD 03693 from the National Institute of Child Health and Human Development and grants 5R1-AI-0023 and 2T1-AI-00068 from the National Institute of Allergy and Infectious Diseases. F. F. Barrett was Epidemic Intelligence Officer, Epidemiology Program, National Communicable Disease Center.

LITERATURE CITED

1. Allen, S. D., and K. B. Conger. 1969. *Serratia marcescens* infection of the urinary tract: a nosocomial infection. *J. Urol.* 101:621-623.
2. Burdin, J. C., J. Lochard, F. Schaak, M. Edert, and J-C. Georges. 1967. Epidemic d'infections pleuro-pulmonaires dues a *Serratia marcescens* en milieu hospitalier. *Presse Med.* 75:466-468.
3. Cabrera, H. A. 1969. An outbreak of *Serratia marcescens* and its control. *Arch. Intern. Med.* 123:650-655.
4. Clayton, E., and A. von Graevenitz. 1966. Nonpigmented *Serratia marcescens*. *J. Amer. Med. Ass.* 197:1059-1064.
5. Davis, B. R., and W. H. Ewing. 1957. The biochemical reactions given by members of the Serratia group. *Int. Bull. Bacteriol. Nomencl. Taxon.* 7:151-160.
6. Davis, B. R., and W. H. Ewing. 1964. Lipolytic, pectinolytic, and alginate activities of Enterobacteriaceae. *J. Bacteriol.* 88:16-19.
7. Dodson, W. H. 1968. *Serratia marcescens* septicemia. *Arch. Intern. Med.* 121:145-150.
8. Edwards, P. R., and W. H. Ewing. 1962. Identification of *Enterobacteriaceae*, 2nd ed., Burgess Publishing Co., Minneapolis, Minn.
9. Ewing, W. H. 1966. Enterobacteriaceae taxonomy and nomenclature. Communicable Disease Center Publication, Communicable Disease Center, Atlanta, Georgia.
10. Ewing, W. H., B. R. Davis, and R. W. Reavis. 1959. Studies on the Serratia group. CDC Monogr., Communicable Disease Center, Atlanta, Georgia.
11. Ewing, W. H., J. G. Johnson, and B. R. Davis. 1962. The occurrence of *Serratia marcescens* in nosocomial infections. U.S. Dept. of Health, Education and Welfare, Washington, D.C.
12. Ewing, W. H., J. N. Wilfert, L. J. Kunz, M. Dumoff, and H. D. Isenberg. 1969. Round table: how far to go with Enterobacteriaceae? *J. Infec. Dis.* 119:197-213.
13. Fulton, M., C. E. Forney, and E. Leifson. 1959. Identification of *Serratia* occurring in man and animals. *Can. J. Microbiol.* 5:269-275.
14. Gale, D., and J. D. Lord. 1957. Overgrowth of *Serratia marcescens* in respiratory tract, simulating hemoptysis. *J. Amer. Med. Ass.* 164:1328-1330.
15. Gaughran, E. R. 1969. From superstition to science: the history of a bacterium. *Trans. N.Y. Acad. Sci.* 31:3-24.
16. Graber, C. D., L. S. Higgins, and J. S. Davis. 1965. Seldom-encountered agents of bacterial meningitis. *J. Amer. Med. Ass.* 192:956-960.
17. Kass, E. H. 1956. Asymptomatic infections of the urinary tract. *Trans. Ass. Amer. Phys.* 69:56-63.
18. Lancaster, L. J. 1962. Role of *Serratia* species in urinary tract infections. *Arch. Intern. Med.* 109:536-539.
19. Leshner, G. Y., E. J. Froelich, M. D. Gruett, J. H. Bailey, and R. P. Brundage. 1962. 1,8-Naphthylidene derivatives; a new class of chemotherapeutic agents. *J. Med. Pharm. Chem.* 5:1063-1065.
20. McCormack, R. C., and C. M. Kunin. 1966. Control of a single source nursery epidemic due to *Serratia marcescens*. *Pediatrics* 37:750-755.
21. McCracken, A. W., and F. E. Lipscomb. 1965. *Serratia marcescens* infection complicating peritoneal dialysis. *Brit. Med. J.* 1:1536-1537.
22. Magnuson, E. W., and H. R. Elston. 1966. Infections caused by non-pigmented *Serratia marcescens*. *Ann. Intern. Med.* 65:409-418.
23. Morse, L. J., and L. E. Schoenbeck. 1968. Hand lotions—potential nosocomial hazard. *N. Engl. J. Med.* 278:376-378.
24. Rabinowitz, K., and R. Schiffrin. 1952. A ward contamination by *Serratia marcescens*. *Acta Med. Orient.* 11:181-184.
25. Ringrose, R. E., B. McKown, F. G. Felton, B. O. Barclay, H. G. Muchmore, and E. R. Rhoades. 1968. A hospital outbreak of *Serratia marcescens* associated with ultrasonic nebulizers. *Ann. Intern. Med.* 69:719-729.
26. Robinson, W., and P. B. Woolley. 1957. Pseudohaemoptysis due to *Chromobacterium prodigiosum*. *Lancet* 1:819.
27. Sanford, J. P. 1969. Sensitivity tests of Klebsiella, Enterobacter and Serratia. *J. Infec. Dis.* 119:388-390.
28. Shedden, W. I. 1963. Meningitis due to *Serratia marcescens*. *Arch. Dis. Childhood* 38:97.
29. Steers, E., E. L. Foltz, and B. S. Graves. 1959. Inocula replicating apparatus for routine testing of bacterial susceptibility to antibiotics. *Antibiot. Chemotherap.* 9:307-311.
30. Stenderup, A., O. Faergeman, and M. Ingerslev. 1966. *Serratia marcescens* infections in premature infants. *Acta Pathol. Microbiol. Scand.* 68:157-160.
31. Taylor, G., and P. M. Keane. 1962. Cross-infection with *Serratia marcescens*. *J. Clin. Pathol.* 15:145-147.
32. Thompson, J. L. 1943. Meningitis caused by *Bacillus prodigiosus*. Report of a case. *Med. Ann. D.C.* 12:145-146.
33. Thornton, G. F., and V. T. Andriole. 1969. Antibiotic sensitivities of non-pigmented *Serratia marcescens* to gentamicin and carbenicillin. *J. Infec. Dis.* 119:393-394.
34. Tillotson, J. R., and M. Finland. 1969. Bacterial colonization and clinical superinfection of the respiratory tract complicating antibiotic treatment of pneumonia. *J. Infec. Dis.* 119:597-624.
35. Waisman, H. A., and W. H. Stone. 1958. The presence of *Serratia marcescens* as the predominating organism in the intestinal tract of the newborn. The occurrence of the "red diaper syndrome." *Pediatrics* 21:8-12.
36. Wheat, R. P., A. Zuckerman, and L. A. Rantz. 1951. Infection due to chromobacteria. Report of 11 cases. *Arch. Intern. Med.* 88:461-466.
37. Wilfert, J. N., F. F. Barrett, and E. H. Kass. 1968. Bacteremia due to *Serratia marcescens*. *N. Engl. J. Med.* 279:286-289.
38. Woodward, H. M., and K. B. Clarke. 1913. A case of infection in man by the *Bacterium prodigiosum*. *Lancet* 1:314.