

Enteric Group 15 (*Enterobacteriaceae*) Associated with Pneumonia

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A gram-negative, oxidase-negative, fermentative rod belonging to enteric group 15 of *Enterobacteriaceae* was isolated in mixed culture from two patients with pneumonia. Both were elderly patients with chronic heart disease.

In 1980, Farmer et al. (J. J. Farmer III, P. A. D. Grimont, F. Grimont, and M. A. Asbury, Abstr. Annu. Meet. Am. Soc. Microbiol. 1980, C123, p. 295) reported a new member of the *Enterobacteriaceae* family enteric group 15, which was defined in 1977 by the Enteric Section, Centers for Disease Control, to provide an identity for a group of organisms which did not fit any of the named genera of *Enterobacteriaceae*. It was divided into two subgroups based on ornithine decarboxylase reaction and fermentation of sucrose and D-xylose (subgroup 1 was positive for all three and subgroup 2 was negative for all three). Enteric group 15 resembles *Serratia* spp. in its positive lipase (corn oil) reaction and resistance to colistin and cephalothin, but it differs in being negative for deoxyribonuclease production and gelatin hydrolysis. The new group is thus distinct from all named groups of *Enterobacteriaceae*. This report describes the isolation of enteric group 15 from sputum obtained from two patients with pneumonia.

Patient 1. A 65-year-old female was admitted on 4 August 1980 for weakness, abdominal pain, and labored breathing. She was a known diabetic and had hypertension with congestive heart failure. Physical examination revealed hepatomegaly and rales at both lung bases. She was afebrile on the day of admission.

The patient developed a temperature of 101°F (38.3°C) on the fourth day of hospitalization. The leukocyte count and chest X ray were normal. Direct Gram stain of her sputum did not reveal a preponderance of a bacterial species, and blood cultures done at that time were negative.

On 4 September 1980, the peripheral leukocyte count was 12,600/mm³, with a marked left shift, and a chest X ray showed right lower lobe pneumonia with minimal pleural effusion. A direct Gram stain of sputum and cultures taken at

that time contained numerous gram-negative rods with a few polymorphonuclear leukocytes and heavy growth of enteric group 15 (isolate 1), in addition to scant growth of *Klebsiella oxytoca* on sheep blood agar and MacConkey agar plates (BBL Microbiology Systems), respectively. On the basis of laboratory data, cefazolin therapy was initiated on 5 September. On 16 September, the patient became afebrile, and 2 days later, cefazolin was discontinued. A follow-up chest X ray taken on 23 September 1980 showed minimal residual infiltration in the right lower lobe.

Patient 2. A 76-year-old male was admitted on 7 August 1980 for increasing weakness on the right side. He had hypertension and arteriosclerotic heart disease.

Clinical examination revealed paralysis of the right side of the body along with signs of consolidation over the right lower lobe, with a temperature of 101°F. A chest X ray confirmed the clinical diagnosis of right lower lobe pneumonia. Leukocyte count on admission was 10,900/mm³, with a left shift.

Direct Gram stain of sputum on the day of admission indicated a few polymorphonuclear leukocytes, a few gram-negative rods, and rare gram-positive cocci in chains. Sputum cultures taken at that time grew out a few alpha-hemolytic streptococci; blood and urine cultures were negative. The patient was started on penicillin G, 600,000 U intramuscularly, twice a day.

In the initial 3 days, the patient appeared to be improving, but thereafter, he remained febrile and showed signs and symptoms of pneumonia. Sputum cultures taken on the fourth day of hospitalization yielded mixed growth of enteric group 15 (isolate 2), which was predominant, and scant growth of *Neisseria* species and alpha-hemolytic streptococci. Blood and urine cultures taken at that time were negative.

On 8 August, penicillin G therapy was dropped and cephalexin therapy was started. On 15 Au-

gust, the patient became afebrile, and the antibiotic treatment was discontinued 2 weeks later (on 29 August). A follow-up chest X ray taken 2 weeks after the first one revealed a significant improvement in the right lower lobe pneumonia, although infiltration still persisted.

Bacteriology. Isolates 1 and 2 grew well on MacConkey and sheep blood agar plates (BBL) at 37°C. The colonial morphology of both isolates resembled that of lactose-negative members of *Enterobacteriaceae* on MacConkey agar (BBL) and showed grayish-white colonies on sheep blood agar (BBL).

Conventional biochemical tests (1) revealed that both isolates were identical and exhibited the characteristics shown in Table 1. The two isolates were arginine dihydrolase positive only after 48 h of incubation at 37°C in Moeller decarboxylase broth (BBL). This feature accounted for the two API 20E (Analytab Products, Div. of Ayerst Laboratories) profile numbers, 1104121 and 3104121, which were obtained for the organism after 24 and 48 h of incubation, respectively.

Both isolates were susceptible to amikacin, carbenicillin, cefamandole, chloramphenicol, gentamicin, kanamycin, tetracycline, tobramycin, and trimethoprim-sulfamethoxazole, and both were resistant to cephalothin, as determined by the standard disk diffusion method.

The isolates, referred to the Centers for Dis-

ease Control, Atlanta, Ga., were identified as members of enteric group 15 (isolate 1 = CDC no. 0111-81; isolate 2 = CDC no. 0110-81) and had essentially the same biochemical characteristics as those observed in our laboratory (Table 1). Both isolates were positive for ornithine decarboxylase reaction and fermented sucrose and D-xylose and therefore belonged to enteric group 15, subgroup 1 (the Davis subgroup) (J. J. Farmer III et al., Abstr. Annu. Meet. Am. Soc. Microbiol. 1980, C123, p. 295).

There had been no isolates of enteric group 15 in the hospital until the two cases described in this report. Since patients 1 and 2 were hospitalized one room apart from each other at the time the organism was isolated, a possible common source or cross infection of the organism was suspected. However, no additional isolates of enteric group 15 have been encountered since these isolations.

The data presented have not adequately shown that enteric group 15 was the cause of pneumonia in the two patients. The organism was not isolated from the blood, and both patients responded to a cephalosporin to which the two strains of enteric group 15 were resistant in the in vitro test. On the other hand, enteric group 15 was the predominant organism recovered from both patients, and no significant bacterial species (other than scant growth of *K. oxytoca* from the sputum of patient 1) was isolated. Based on the clinical and radiological findings, it is apparent that patient 2 acquired pneumonia in the community. What is not certain, however, is whether he acquired enteric group 15 during the first 4 days of hospitalization or he harbored the organism at the time of admission. Despite this uncertainty, the organism appeared to have spread from one patient to the second in the hospital.

It is noteworthy that both patients first seen in our institution with enteric group 15 were elderly and had preexisting chronic heart disease. It is tempting to speculate that enteric group 15 might have caused pneumonia in these patients, but further study obviously is needed to clarify the etiological role of these organisms.

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LITERATURE CITED

1. Lennette, E. H., A. Balows, W. J. Hausler, Jr., and J. P. Truant (ed.). 1980. Manual of clinical microbiology, 3rd ed. American Society for Microbiology, Washington, D.C.

TABLE 1. Characteristics of isolates 1 and 2, as determined by the Long Island College Hospital and the Centers for Disease Control

Test	Results for isolates 1 and 2
Voges-Proskauer, indole, H ₂ S (triple-sugar iron agar), urease (Christensen), gelatin, acetate, and deoxyribonuclease	-
Methyl red, citrate (Simmons), malonate, KCN, ONPG, and NO ₃ ⁻ → NO ₂ ⁻	+
Lysine decarboxylase	-
Arginine dihydrolase	+ ^a
Ornithine decarboxylase	+
Phenylalanine deaminase	-
Motility (at 22°C)	+
Glucose, sucrose, mannitol, salicin, and D-xylose	+
Lactose, dulcitol, adonitol, inositol, sorbitol, arabinose, raffinose, and rhamnose	-
Oxidase	-

^a At 2 to 6 days.

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