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Lactose-Fermenting, Multiple Drug-Resistant *Salmonella typhi* Strains Isolated from a Patient with Postoperative Typhoid Fever

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Two lactose-fermenting *Salmonella typhi* strains were isolated from bile and blood specimens of a typhoid fever patient who underwent a cholecystectomy due to cholelithiasis. One lactose-fermenting *S. typhi* strain was also isolated from a pus specimen which was obtained at the tip of the T-shaped tube withdrawn from the operative wound of the common bile duct of the patient. These three lactose-fermenting isolates: GIFU 11924 from bile, GIFU 11926 from pus, and GIFU 11927 from blood, were phenotypically identical to the type strain (GIFU 11801 = ATCC 19430 = NCTC 8385) of *S. typhi*, except that the three strains fermented lactose and failed to blacken the butt of Kligler iron agar or triple sugar iron agar medium. All three lactose-fermenting strains were resistant to chloramphenicol, ampicillin, sulfomethoxazole, trimethoprim, gentamicin, cephaloridine, and four other antimicrobial agents. The type strain was uniformly susceptible to these 10 drugs. The strain GIFU 11925, a lactose-negative dissociant from strain GIFU 11926, was also susceptible to these drugs, with the sole exception of chloramphenicol (minimal inhibitory concentration, 100 μg/ml).

The lack of lactose-fermenting ability of *Salmonella* species has long been regarded as one of the important attributes of salmonellae that differentiate salmonellae from members of several other genera of *Enterobacteriaceae*. Since 1959, frequent incidences of salmonellosis due to lactose-fermenting strains have been reported from the United States, Brazil, Canada, and Japan (1, 9, 14, 24). In these cases, including two outbreaks in Canada and Brazil, the isolates belonged to serogroups B, C_1, C_2, D, E_1, or E_4. Lactose-fermenting ability in salmonellae usually causes delay in the proper identification of such strains.

Among the lactose-fermenting isolates, strain ST-2 was the only strain of naturally occurring *Salmonella typhi* hitherto reported in the literature. Strain ST-2 was first reported by Baron et al. (4) in 1959 as a high frequency recombinant strain compatible with many strains of *Salmonella, Shigella*, and *Escherichia* species. However, Baron et al. did not mention its source of isolation. Falkow and Baron (8) later reported that ST-2 was isolated from the stool of a patient with gastroenteritis. In 1965, Massachusetts General Hospital reported a case of classical typhoid fever in a laboratory glassware washer due to a lactose-fermenting *S. typhi* strain (11). Since the isolates from the blood and urine of the patient did not differ from the laboratory strain labeled as 643 lac^+, the infection was thought to be laboratory acquired. Strain 643 lac^+ was derived from typical *S. typhi* 643 treated with the episome of strain ST-2 during the course of a genetic experiment.

Recently, we encountered an adult patient with typhoid fever due to a lactose-fermenting and multiple drug-resistant *S. typhi* strain, manifested 27 days after cholecystectomy. This report describes the clinical course of the illness and characterizes the isolates from the bile, blood, and pus of the patient for their biochemical attributes and antimicrobial susceptibility pattern.

CASE REPORT

Clinical course of the illness. In February 1982, a 45-year-old male developed sudden onset of fever (40°C), headache, nausea, and watery diarrhea lasting for 17 days (18 February to 6 March). The onset of symptoms occurred 27 days after the patient underwent cholecystectomy. Three days after the onset, the patient was treated with cephalothin (4 g) and tobramycin (120 mg) per day. Cephalexin (4 g) and cefmetazole (4 g) were later administered. Despite the treatment with these four drugs, he was still febrile with headache and nausea at each peak of the fever. He had proteinuria and also suffered from watery diarrhea one to five times each day during this febrile period.

The cultures of the bile and pus of the patient yielded two kinds of organisms: *Enterobacter agglomerans* and a lactose-fermenting, gram-negative rod. The bile was cultured on 23 February, and the pus on the tip of the T-shaped tube withdrawn from his common bile was cultured on 24 February. Enrichment culture of blood (25 February) also yielded a pure culture of lactose-fermenting, gram-negative rod.

According to the results of antimicrobial susceptibility testing, moxalactam (latamoxef, World Health Organization-approved generic name) treatment at 4 g per day began on 4 March. At the same time, fosfomycin (120 mg per day) was also administered, although its efficacy against the isolates was not determined. Within 48 h, his temperature and stool became normal. Eight bile cultures and four blood cultures during the period from 26 February to 24 March were uniformly negative for *S. typhi* strain and other bacteria.

The lactose-fermenting strains consisted of motile, oxidase-negative, gram-negative rods. They produced red colonies on MacConkey agar and turned triple sugar iron agar yellow (butt and slant) without gas formation. No indole production was observed on suitable media, and Voges-Proskauer reaction was negative. There was no growth on Simmons citrate agar. The mini-system for identification, Minitek Enterics 1977 (BBL Microbiology Systems, Cockeysville, Md.), suggested these bacteria were *S. typhi*. However, slide agglutination tests of the cultures with polyvalent *Salmonella* O and Vi antisera (Denka-Seiken, Niigata, Japan) were negative. The three isolates on lysine-iron agar medium revealed positive lysine decarboxylase activity and production of a small quantity of hydrogen sulfide. When replated on a MacConkey agar medium, the strain from pus yielded a few lactose-negative colonies. These colonies exhibited biochemically and serologically typical attributes of *S. typhi*. After three successive transfers to triple sugar iron agar slant, the lactose-fermenting strains were agglutinated by Vi antiserum and flagellar d antiserum; the heat-treated cells reacted with *Salmonella* O group D serum. Thus, all lactose-fermenting isolates were identified as strains of *S. typhi*, and the febrile illness was diagnosed as typhoid fever caused by lactose-fermenting *S. typhi*. It took 10 days to reach this final identification.

MATERIALS AND METHODS

Bacteriology. The histories and corresponding strain numbers of the type strain of *S. typhi* are listed in Table 1.

Flagellar morphology was determined by the Leifson flagella stain (13). Heart infusion broth (BBL) cultures were incubated at 20°C for 18 h. Acid production from carbohydrates was tested in purple broth base (Difco Laboratories, Detroit, Mich.) supplemented with 0.1 volume of a sterile 10% solution of each carbohydrate. Lysine iron agar medium (BBL) was used to confirm H_2S production. Decarboxylase medium base (Difco) containing 0.5% L-lysine hydrochloride, L-arginine hydrochloride, or L-ornithine hydrochloride was used.

In addition, five commercial identification systems were tested: API 20E (API System S. A., Montalieu Vercieu); Enterotube II (Hoffmann-La Roche, Basel, Switzerland); Biotest I (Eiken, Tokyo); ID test (Nissui, Tokyo); Minitek Enterics and Minitek Enterobacteriaceae II revised (BBL).

Susceptibility to antimicrobial agents. The susceptibility of the three lactose-fermenting isolates and one lactose-nonfermenting colonial dissociant against 10 antimicrobial agents was determined by means of disk diffusion method, using Tri-Discs (Eiken) (15).

Minimal inhibitory concentrations (MICs) of the following 18 drugs were determined by the agar dilution method, using Mueller-Hinton agar (Difco): ampicillin (ABPC), amoxicillin (AMPC), cephaloridine (CER), cephalothin (CET), cephalexin (CEX), cefazolin (CEZ), cefmetazole (CMZ), moxalactam (LMOX), tobramycin (TOB), gentamicin (GM), kanamycin (KM), streptomycin (SM), tetracycline (TC), chloramphenicol (CP), trimethoprim (TMP), sulfamethoxazole (SMX), and fosfomycin (FOM).

The organism was grown in Mueller-Hinton broth (Difco) at 37°C for 18 h, and 100-fold dilutions of the broth cultures were spot-inoculated by a multiple inoculator (Toyo Sokuki MIT-P) onto agar medium containing graded concentrations of the test antimicrobial agents. MICs were read after incubation at 30°C for 20 h.

Serological examination and phage typing. Slide agglutination tests were performed by using O-antisera.

TABLE 1. Histories and corresponding numbers of five *S. typhi* strains used

Strain no.[a]					Status	Source	Source of isolation	Date (mo/day/yr) of isolation
GIFU	KM	SN	ATCC	NCTC				
11801	2922		19430	8385	Type	NCTC	Not recorded	1918
11924	3086	187				M. Takahashi	Bile[b]	2/23/82
11925	3087	189				M. Takahashi	Colonial dissociation from strain 11926	2/26/82
11926	3088	188				M. Takahashi	Pus[b]; T-tube tip	2/24/82
11927	3089	190				M. Takahashi	Blood[b]	2/25/82

[a] Abbreviations: ATCC, American Type Culture Collection, Rockville, Md.; GIFU, Department of Microbiology, Gifu University School of Medicine, Gifu, Japan; KM, Department of Microbiology, Kansai Medical University, Osaka, Japan; NCTC, National Collection of Type Cultures, London, England; SN, Saiseikai-Nakatsu Hospital, Osaka, Japan.

[b] From a 45-year-old male patient with typhoid fever after cholecystectomy.

TABLE 2. Dissimilar characteristics of three lactose-positive isolates and one lactose-negative dissociant compared with those of the type strain of *S. typhi*

GIFU strain no.	Substrate or test result					
	KI agar[a]		Black butt, KI agar within 2 days	ONPG production	Acid production in purple broth base from:	
	Slant	Butt			Lactose	Xylose
11924, 11926, 11927	A	A	−	+	+	−
11925	K	A	+	−	−	−
11801[b]	K	A	+	−	−	+

[a] A, Acid reaction; K, alkaline reaction.
[b] Type strain.

H and Vi antisera were purchased from Denka-Seiken, Niigata, Japan. Phage typing was done at the National Institute of Health, Japan (3).

RESULTS

The three lactose-fermenting strains (GIFU 11924, 11926, and 11927) and one lactose-nonfermenting dissociant (GIFU 11925) exhibited the same characteristics as the type strain of *S. typhi*, except for the five reactions listed in Table 2. The three lactose-fermenting strains acidified Kligler iron (KI) agar slant and butt (yellow) without blackening the butt. These strains produced acid from lactose in purple broth base and were positive in *O*-nitrophenyl-beta-D-galactosidase (ONPG) test in the Minitek system. Strain 11925 showed an acid butt and alkaline slant on KI agar, with a small quantity of hydrogen sulfide visible in the butt. This strain produced no acid in purple broth base supplemented with lactose and gave a negative reaction in the ONPG test.

The two lactose-nonfermenting strains were identified correctly with all six systems. The profile numbers obtained from the three lactose-fermenting strains were not included in the profile number code book of API-20E, Biotest-I, ID test, and Minitek Enterobacteriaceae II revised (Table 3). Moreover the profile number of strain 11926 in Enterotube II system corresponded to *Escherichia coli*. The Minitek Enterics (1977) alone identified the three lactose-fermenting *S. typhi* strains correctly.

The three lactose-positive isolates were resistant to six drugs, whereas the lactose-negative dissociant was resistant only to two drugs (Table 4).

The MICs of 18 antimicrobial agents for the five strains are listed in Table 5. The three lactose-fermenting strains were significantly more resistant to CP, ABPC, AMPC, SMX, TMP, TOB, GM, and KM. In contrast, the lactose-negative dissociant strain 11925 was resistant only to CP.

Serological examination revealed that the three lactose-positive and one lactose-negative strains possessed the antigenic formula of *S. typhi*, 9, 12, Vi:d:-.

The three lactose-fermenting and one lactose-nonfermenting dissociant strains were deter-

TABLE 3. Identification results of five *S. typhi* strains by six mini-systems for members of the family *Enterobacteriaceae*

Kit	GIFU strain no.	Code obtained	Identification[b]
API 20E (1980)	11801[a]	6404540	*S. typhi*
	11924, 11926, 11927	5404540	—
	11925	4004540	*S. typhi*
Enterotube-II (1981)	11801, 11925	52040	*S. typhi*
	11924, 11927	52240	—
	11926	50240	*E. coli*
Biotest-I (1982)	11801, 11925	0041415	*S. typhi*
	11924, 11926	2041415	—
	11927	6041415	—
ID test (1979)	11801, 11925	0010025	*S. typhi*
	11924, 11926, 11927	0011025	—
Minitek (1977)	11801, 11925	30200	*S. typhi*
	11924, 11926, 11927	30202	*S. typhi*
Minitek (1980)	11801, 11925	4011010	*S. typhi*
	11924, 11926, 11927	4051014	—

[a] Type strain.
[b] —, Not included in the profile number code book.

TABLE 4. Antimicrobial susceptibility of three lactose-positive and one lactose-negative *S. typhi* strains according to disk diffusion method[a]

Drug	Characteristic of GIFU strain no.[b]:	
	11924, 11926, 11927[c]	11925[d]
CP, SMX	R	R
ABPC, CER, GM, KM	R	S
CEZ, CEX, LMOX, TC	S	S

[a] Primary cultures of *S. typhi* 11924, 11925, 11926, and 11927 were used; the disk diffusion method was used with tri-disc (Eiken).
[b] R, Resistant; S, susceptible.
[c] Lactose-positive strains.
[d] Lactose-negative strain.

mined as phage type M1 by Akiko Nakamura, National Institute of Health, Japan.

DISCUSSION

Strain ST-2, the only lactose-fermenting *S. typhi* strain hitherto reported in the literature (3), was an isolate from the stool of a gastroenteritis patient (7). A lactose-fermenting *S. typhi* strain, obtained as the etiological agent of a case of laboratory-acquired typhoid fever, was thought to be identical to a strain 643 lac^+ harboring the (lac^+) plasmid from ST-2 strain (10). Thus, there has been no report of any case of typhoid fever due to a naturally occurring lactose-fermenting *S. typhi*, despite frequent reports of salmonellosis caused by lactose-fermenting strains from various countries (1, 9, 14, 24).

As with strain ST-2, the lactose-fermenting ability of a *S. newport* strain in Canada was plasmid mediated and transmissible (1). On the other hand, the lactose-fermenting characteristics of the Brazilian *S. typhimurium* and *S. oranienburg* could be transferred to *S. typhi* 643 only after mobilization (14). Spontaneous segregation of lactose-nonfermenting colonies suggests the instability and extrachromosomal nature of lactose-fermenting ability of our three isolates. Our preliminary experiments indicate that their lactose-fermenting and part of their drug resistance capabilities are transmissible.

Lactose-fermenting *S. typhi* strains may be overlooked when grown on plates of selective differential medium containing lactose. Even when their presence is suspected, proper identification may be delayed because of reactions elicited with either KI or triple sugar iron agars. Mini-systems for identification now in general use fail to identify such aberrant strains. In the case described here, only the oldest edition of Minitek profile number code book for Enterics (1977) suggested the possibility of *S. typhi*. However, the lack of proper reactions of the primary isolates with polyvalent *Salmonella* O antisera and Vi antisera also confused proper identification. The isolates became positive in slide agglutination test only after repeated transfer. Usually, repeated subculturing of *S. typhi* results in the loss of detectable Vi antigen. At present, the reason for the serological conversion of our lactose-fermenting strains is unknown.

Serological identification of *Salmonella* is ap-

TABLE 5. Antimicrobial susceptibility of three lactose-positive and one lactose-negative *S. typhi* strains compared with the type strain of the species

Drug	MIC (μg/ml) of GIFU strain (lactose fermentation)				
	11924 (+)	11926 (+)	11927 (+)	11925 (−)	11801[a] (−)
Chloramphenicol	100	100	100	100	6.3
Ampicillin	>100	>100	>100	0.4	0.4
Amoxicillin	>100	>100	>100	0.4	0.4
Sulfamethoxazole	>100	>100	>100	3.2	3.2
Trimethoprim	100	>100	100	0.4	0.4
Tobramycin	100	100	100	0.4	0.4
Gentamicin	100	100	100	0.8	0.8
Kanamycin	>100	>100	>100	3.2	3.2
Cephaloridine	50	50	50	1.6	1.6
Cephalothin	25	12.5	25	3.2	1.6
Cefazolin	6.3	6.3	6.3	0.8	0.8
Moxalactam	0.1	0.1	0.1	0.1	0.1
Cefmetazole	0.4	0.4	0.4	0.4	0.2
Cephalexin	3.2	6.3	6.3	6.3	6.3
Amikacin	1.6	1.6	1.6	1.6	1.6
Tetracycline	1.6	1.6	1.6	1.6	1.6
Streptomycin	12.5	12.5	12.5	12.5	6.3
Fosfomycin	12.5	12.5	12.5	12.5	25

[a] Type strain.

plied in Japan usually after isolates have demonstrated metabolic characteristics compatible with *Salmonella*. In the case of aberrant strains, such as these lactose-fermenting strains of *S. typhi*, the serological identification may not be performed.

To overcome the difficulties in isolating and identifying lactose-fermenting *S. typhi* strains, a bismuth-sulfite agar (5, 22) and a lysine-iron agar (5, 11, 22) should be used in clinical bacteriology laboratories. Our strains would have been properly identified earlier had a presumptive clinical diagnosis of typhoid fever been available.

The fact that fermentation products of carbohydrates by salmonellae masked the iron sulfide indicator (6) was also confirmed in our lactose-fermenting *S. typhi* strain. The mechanism of this interesting phenomenon remains to be solved.

Chloramphenicol has long been known as the drug of choice for the treatment of typhoid fever with ABPC (18, 20), AMPC (17), and co-trimoxazole (SMX-TMP) (21) as alternatives. However, our three lactose-fermenting *S. typhi* strains were resistant in vitro to all four drugs. CEX and CMZ, though their MICs for the three isolates were 3.1 and 0.4 μg/ml, respectively, showed no appreciable clinical result. The MICs of LMOX and FOM were 0.1 and 25 μg/ml, respectively. Physicians in Japan feel that FOM is effective against salmonellae but that cephalosporin derivatives are not. Since the combination was used in this instance, it is not possible to ascribe effectiveness to either drug alone.

Chloramphenicol-resistant strains of *S. typhi* were isolated in Chile in 1966 and in Aden and Kuwait in 1967. A large outbreak of CP-resistant typhoid fever occurred first in Mexico in 1972 to 1973 and then in India, Vietnam, and Thailand. The isolates in Kuwait were resistant to CP and ABPC (2). The isolates in Mexico (10, 23), India (19), Vietnam (7), and Thailand (12) exhibited a four-drug resistance: to CP, TC, SU, and SM (2). In Japan, there have been no CP-resistant strains of *S. typhi* isolated from the blood (16).

This case of typhoid fever due to a lactose-fermenting and multiple drug-resistant strain of *S. typhi*, described here, developed 27 days after a successful cholecystectomy. Since the bile of this patient was not cultured until the onset of his febrile illness, it cannot be known whether the patient carried *S. typhi* in his biliary tract at the time of cholecystectomy. The patient and his family did not have any febrile illness or any overseas travel until this instance. Furthermore, the 28-day postoperative period for the appearance of fever seems too long for an infection of endogenous origin, suggesting the possibility of a nosocomial acquisition of typhoid fever. In December 1981, a female patient with cholelithiasis was admitted to the same ward. She was found to be an asymptomatic carrier of *S. typhi* by a positive culture of bile specimen made at the time of her operation. Although the isolate (SN172) was neither lactose fermenting nor drug resistant, it was identified as phage type M1, similar to the above-mentioned lactose-fermenting isolates in March 1982. After the case due to a lactose-fermenting strain occurred in February 1982, there has been no additional clinical case of typhoid fever in the hospital.

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