

***Vibrio vulnificus* Septicemia: Report of Four Cases**

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Vibrio vulnificus causes very severe infections. The organism is isolated, for the most part, from the blood, and skin lesions. Isolation from other sources, including the urine, is very rare. Four cases of *V. vulnificus* septicemia were bacteriologically diagnosed in 1984 and 1985 at Severance Hospital. All of the patients were men, 42 years and older, with preexisting liver disease. All of them showed hypotension and secondary skin lesions, and all expired. The organism was isolated from the blood in all patients, from the peritoneal fluid in one, and from skin lesions in two. From one patient, isolation from a urine specimen was also accomplished. All of the isolates were typical in their characteristics such as in their forming green colonies on Thiosulfate citrate bile sucrose (TCBS) agar, delayed acid production from lactose, and growth in broth with 6% NaCl.

Key Words: *V. vulnificus*, septicemia, urine culture.

Vibrios cause intestinal as well as extraintestinal infections. *Vibrio vulnificus* extraintestinal infection is notorious for its clinical severity and its extremely high fatality rate (Blake *et al.* 1979). The organism is mostly isolated from blood and skin lesions, but rarely from stool or urine specimens (Farmer *et al.* 1985). The organism is identified by cultural and biochemical characteristics (Baumann *et al.* 1984). Shimada and Sakazaki (1984) established 7 serovars according to somatic (O) antigens.

V. vulnificus infection appears to be relatively common in Korea. Since the first published report of bacteriologically proven infections in 1982 (Goo *et al.* 1982), at least 44 cases have been reported (Chong *et al.* 1982; Kim *et al.* 1984; Kim *et al.* 1985a; Kim *et al.* 1985b; Lee *et al.* 1985a; Lee *et al.* 1985b; Park *et al.* 1985a; Park *et al.* 1985b; Yoon *et al.* 1985; Hur 1986; Joh 1986). The organisms were isolated mostly from blood or skin lesions. Kim *et al.* (1985a) reported an isolation from a stool specimen. The serovar of the isolates from Korean patients has not been reported yet.

In 1984 and 1985, we isolated *V. vulnificus* from

4 septicemic patients. Specimens of blood and peritoneal fluid or skin lesion yielded the organism. From one urine specimen the growth was also obtained. Serovars of three isolates were determined. We are reporting here the clinical and bacteriological findings of these cases.

CASE REPORTS

Case 1.

A 42-year-old man (unit no. A1551671) was transferred from Kangwha Hospital to Severance Hospital on July 5, 1984, with the chief complaints of fever, diarrhea, and pain in both legs. He was an inhabitant of Kangwha Island and had a 2-year history of jaundice. Two days prior to admission he experienced abdominal pain, diarrhea, and vomiting. By the next day, a purple rash had appeared on both legs and the legs had become swollen and tender.

Physical examination on admission revealed an acutely ill appearing man in a confused mental state. His abdomen was distended, but not tender. His liver and spleen were not palpable. erythematous vesicular rash was observed on the back, left arm, and both legs. The blood pressure was 140/90 mm Hg and the heart beats 132/min. His body temperature was 38.5°C.

Laboratory findings were hemoglobin 13.1 g/dl; hematocrit 37%; and WBC count 2,400/ μ l with 74%

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segmented neutrophils, 10% bands, 14% lymphocytes, and 2% monocytes. The platelet count was 20,000/ μ l. Partial thromboplastin time was prolonged to 67 seconds. Electrolytes were normal with the exception of 15 mmol/L of CO_2 . Glucose, blood urea nitrogen, creatinine, amylase were normal. Three blood and one skin vesicle samples were cultured and all of them yielded *V. vulnificus* serovar 4. The stool culture yielded no pathogens.

Despite antimicrobial therapy and other supportive measures, the patient's condition deteriorated rapidly. Eight hours after admission, he became comatose and his blood pressure dropped to 100/60 mm Hg. Urine output was absent during his hospital stay. Skin became denuded with multiple bullae. The hemoglobin dropped to 7.6 g/dl. After a 12-hour stay he was discharged in a very grave condition.

Case 2.

A 43-year-old man (unit no. 1536500) was admitted to Severance Hospital on September 17, 1984, with the chief complaints of fever, nausea, abdominal pain, and general weakness for a day. He was an inhabitant of Seoul. Three months previously he had been treated for cirrhosis of the liver and esophageal varices.

On physical examination, the patient appeared to be acutely ill, but mentally alert. His blood pressure was 90/60 mm Hg, his heart rate 92/minute, and his body temperature 37.4°C. His sclera was icteric, and skin lesions were noted on the anterior chest wall. His abdomen was distended and a fluid wave with shifting dullness was noted. Direct and indirect tenderness was present over the entire abdomen.

The laboratory findings were hemoglobin 10.8 g/dl, hematocrit 33%, WBC count 7,600/ μ l with 88% segmented neutrophils and 13% lymphocytes. The platelet count was 232,000/ μ l. Partial thromboplastin time was prolonged to 110 seconds and fibrinogen degradation products was positive up to 1:80 dilution. Some of the abnormal blood chemistries were creatinine 5.4 mg/dl, protein 5.3 g/dl with 2.2 g albumin, total bilirubin 4.0 mg/dl, alkaline phosphatase 212 IU/L, ammonia 281 μ g/dl, and CO_2 3 mmol/L. Abnormal urinalysis findings were protein ++, blood ++, and bilirubin ++. Two blood and a peritoneal fluid samples were cultured and all of them yielded *V. vulnificus* serovar 1.

Ampicillin and tobramycin were administered. He was anuric and the blood pressure became unmeasurable. He became comatose and expired 17 hours after admission.

Case 3.

A 51-year-old man (unit no. 1575987) was admitted to Severance Hospital on September 17, 1984, with the chief complaints of dyspnea and diarrhea of one day's duration. He was a heavy drinker suffering from cirrhosis of the liver and diabetes mellitus. He was an inhabitant of Seoul.

Physical examination revealed an acutely ill-appearing man in an alert mental state. His blood pressure was 100/80 mm Hg and body temperature 36.8°C. His abdomen was distended with ascites. His liver was palpable 3-finger breadths. Bullae were noted on the right lower leg, which was swollen.

Laboratory findings were hemoglobin 12.4 g/dl; hematocrit 37%; and WBC count 3,400/ μ l with 72% segmented neutrophils, 10% bands, 21% lymphocytes, and 6% monocytes. The platelet count was 49,000/ μ l. Prothrombin time was prolonged to 14.6 seconds (55.8% of normal) and partial thromboplastin time to 70.8 seconds. Fibrinogen degradation product was positive up to 1:20 dilution. Abnormal blood chemistries were fasting glucose 153 mg/dl, aspartate aminotransferase 545 IU/L, alanine aminotransferase 119 IU/L, and total protein 5.6 g/dl with albumin 1.9 g. Urinalysis showed blood ++, protein ++, and ketone \pm . Three blood and a wound specimens were cultured and from all of them *V. vulnificus* serovar 4 was isolated.

Despite all efforts made to treat him including the administration of gentamicin, cefazolin, and fosfomycin, he became moribund and was discharged 28 hours after admission.

Case 4.

A 44-year-old man (unit no. A1683185) was transferred from a private clinic to Severance Hospital on August 16, 1985, with the chief complaints of fever, dyspnea, rash, edema, and pain on both legs of 12 hours duration. He was a resident of Seoul and a heavy drinker. He had alcoholic liver cirrhosis. His present illness had begun with a bout of heavy drinking a week previously.

Physical examination revealed an acutely ill-appearing man, with fingertip-sized purplish, erythematous wheals over the entire body. His legs were edematous, and both hip joints were painful. His body temperature was 38.3°C, blood pressure 90/70 mm Hg, and heart rate 92/minute.

Laboratory findings were WBC count 5,100/ μ l with 76% segmented neutrophils, 2% eosinophils and 22% lymphocytes. The platelet count was 116,000/ μ l. His prothrombin time was 15.9 seconds (53% of normal),

and partial thromboplastin time 101 seconds. Fibrinogen degradation product was positive up to 1:80 dilution. Abnormal blood chemistries were protein 4.5 g/dl with 2.7 g albumin, aspartate aminotransferase 796 IU/L, alanine aminotransferase 745 IU/L, gamma-glutamyl transferase 225 IU/L, lactate dehydrogenase 547 IU/L, creatinine kinase over 59,850 IU/L with MB fraction over 490 IU, chloride 95 mmol and CO₂ 5 mmol/L. Other tests, including amylase, were normal. The abnormal urinalysis findings were protein ++, blood +++, bilirubin +, and many finely granular casts. Two blood and one urine specimens were cultured, and from each of them *V. vulnificus* was isolated. A stool culture was negative for any pathogens.

The patient was treated with cefamandole and tobramycin and given supportive care, but he took a downhill course, and expired 10 hours after admission.

For the bacteriological cultures, our routine methods were used. Briefly, 10 ml of blood samples were inoculated into 50 ml each of tryptic soy broth (TSB, Difco) and Brewer thioglycollate medium (BTM, Difco). Specimens from skin lesions and peritoneum were inoculated onto blood agar, MacConkey agar, and thioglycollate broth. Urine specimens were inoculated onto blood agar and MacConkey agar plates, using a 1/1000 ml standard loop. Incubations were

done at 35°C. Identification of the isolates was done by both the conventional method (Farmer *et al.* 1985) and by the API 20E system (Analytab Products, Plainview, N.Y.). Serovar determinations were done by the National Institute of Health, Japan. Antimicrobial susceptibility was tested by the standardized disk diffusion method (NCCLS 1984).

All of the TSB and BTM blood culture bottles showed turbidity after overnight incubation. Large greenish hemolytic colonies developed on the blood agar plates inoculated with specimens of skin lesion and peritoneal fluid. The colonies on the MacConkey agar plates were colorless. Plates inoculated with a urine specimen of a patient showed over 100 colonies, i.e., over 100,000 colony-forming units of *V. vulnificus* per ml. Subcultures of the isolates onto TCBS agar showed large green colonies. The cultural and biochemical reactions were typical, but some tests required a few days' incubation before positive results were obtained (Table 2). The growth in broth with 6% NaCl was often difficult to determine, but all of them were positive. API 20E system also identified all of the isolates as *V. vulnificus*. Three of the 4 isolates were determined for their serovars. One was serovar 1 and the other 2 were serovar 4. Antimicrobial susceptibility tests showed that none of the isolates was resistant to any of the drugs tested (Table 3).

Table 1. Clinical and laboratory features of *V. vulnificus* septicemia patients

Feature	Case 1 42/M	Case 2 43/M	Case 3 51/M	Case 4 44/M
Fever	+	+	-	+
Blood pressure (mm Hg)	100/60	90/60	100/80	90/70
Vomiting	+	-	-	-
Diarrhea	+	-	+	-
Preexisting wound	-	-	-	-
Secondary cutaneous lesion	+	+	+	+
hepatic disease	Jaundice	Cirrhosis	Cirrhosis	Cirrhosis
Diabetes mellitus	-	-	+	-
Alcoholism	-	-	+	+
WBC (per µl)	1,400	7,600	3,400	5,100
Platelet (per µl)	10,000	232,000	49,000	116,000
FDP, at dilution	NT	1:80	1:20	1:80
PTT (sec)	67	110	71	101
Blood culture	+	+	+	+
Peritoneal culture	NT	+	NT	NT
Skin lesion culture	+	NT	+	NT
Urine culture	NT	NT	NT	+

* FDP, fibrinogen degradation product; PTT, partial thromboplastin time; NT, not tested.

Table 2. Cultural and biochemical characteristics of *V. vulnificus* isolates

Property	<i>V. vulnificus</i> ^a	Case 1 84-7-5216	Case 2 84-9-5880	Case 3 84-9-5914	Case 4 85-8-5656
Indole production	97	+	+	+	+
Voges-Proskauer	0	-	-	-	-
Citrate, Simmons'	75	+	+	+	+
H ₂ S on TSI	0	-	-	-	-
Urea hydrolysis	1	-	-	-	-
Phenylalanine deaminase	35	-	-	-	-
Arginine dihydrolase	0	-	-	-	-
Lysine decarboxylase	99	+	+	+	+
Ornithine decarboxylase	55	+	+	+	+
Motility	99	+	+	+	+
Gelatine hydrolysis	75	+	+	+	+
Gas from glucose	0	-	-	-	-
Acid from:					
Adonitol	0	-	-	-	-
Arabinose	0	-	-	-	-
Cellobiose	99	+	+	+	+
Dulcitol	0	-	-	-	NT
Erythritol	0	-	-	-	NT
Galactose	96	+	+	+	+
Glycerol	1	-	-	+	NT
Inositol	0	-	-	-	-
Lactose	85	+ (72 h)	+ (48 h)	+ (48 h)	+ (48 h)
Maltose	100	+	+	+	+
Mannitol	45	+	+	+	+
Mannose	98	+	+	+	+
Melibiose	40	-	-	-	-
Raffinose	0	-	-	-	-
Rhamnose	0	-	-	-	-
Salicin	95	+	+	+	+
Sorbitol	0	-	-	-	-
Sucrose	15	-	-	-	-
Trehalose	100	+	+	+	NT
Xylose	0	-	-	-	NT
Esculin hydrolysis	40	+	+	+	NT
Nitrate reduction	100	+	+	+	+
Oxidase	100	+	+	+	+
DNase	50	+	+	+	NT
Tween 80 hydrolysis	92 ^c	+	+	+	NT
ONPG test	75	+	+	+	+
Growth in broth with:					
0% NaCl	0	-	-	-	-
6% NaCl	65	+	+	+	+
8% NaCl	0	-	-	-	-
API 20E code		5146105	5346105	5146105	5346105

^a Farmer *et al.*, 1985. Figures show percentage of strains positive.^b +, positive; -, negative; NT, not tested.^c Lipase.

Table 3. Antimicrobial susceptibility of *V. vulnificus* isolates

Antimicrobial agent	<i>V. vulnificus</i> ^a	Susceptibility of isolate no.			
		84-7-5216	84-9-5880	84-9-5914	85-8-5656
Penicillin G	2	R ^b	R	R	NT
Ampicillin	99	S	S	S	S
Carbenicillin	54	S	S	S	S
Cephalothin	65	S	S	S	S
Colistin	2	R	R	R	R
Tetracycline	99	S	S	S	S
Chloramphenicol	100	S	S	S	S
Kanamycin	53	I	S	S	NT
Amikacin		S	S	S	S
Gentamicin	100	S	S	S	S
Tobramycin		S	S	S	S
Cotrimoxazole		S	S	S	S

^a Farmer *et al.* 1985. Figures show percentage of strains susceptible.

^b R, resistant; S, susceptible; I, intermediate. Penicillin G disk produced 17 mm of inhibition diameters and carbenicillin disk, 21-24 mm of inhibition diameters.

DISCUSSION

Clinical isolates of *V. vulnificus* had been often mistaken for *V. parahaemolyticus* or other species (Weaver and Ehrenkranz 1975; Blake *et al.* 1979) until the organism was recognized as a distinct species. We ourselves had an experience in which two isolates were misidentified in 1978 (Kim *et al.* 1984). Nowadays in Korea, it appears that the clinical laboratories are well aware of the characteristics of the organism and most clinicians easily recognize the infection. The infection seems to be more prevalent in Korea than in other countries such as Japan and America. This may be due to the frequent eating of raw sea food even in inland cities, and the prevalence of underlying liver disease. There are at least 48 reported cases in Korean literature.

The infection was originally reported to have two forms, i.e., primary septicemia following the ingestion of raw sea food and primary wound infection after receiving a wound (Blake *et al.* 1979). Now it is known that the organism can cause pneumonia (Kelly and Avery 1980), endometritis (Tison and Kelly 1984), keratitis and other infections (Wickboldt and Sanders 1983). All of our present cases were primary septicemia. Histories of eating sea food was not available, but it could be assumed that the men indulged in eating raw fish along with drinking alcoholic beverages. Joh (1986) reported that his patients had ingested quite variety of salt water fish

and shellfish before they became infected. In the United States it is oyster that most often cause the infection.

In Korea there seem to have been no proven cases of primary wound infection. Although a carpenter was reported to have received a penetrating wound prior to the development of a septic infection (Hur 1986), it was difficult to establish any relationship between the infection and the wound. There was a case of a septicemic patient who was reported to have immersed his legs in the sea (Park *et al.* 1985a). Infections have been known to occur following exposure to brackish water (Tacket *et al.* 1984a). A boy with a laceration and an old man with only a suspected insect bite were infected.

Blake *et al.* (1979) reported that the infection occurs most frequently in men of 40 years of age or over who have underlying liver disease. Only one of the 48 patients whose cases have been reported in the Korean literature was a woman (Joh 1986). Most of the patients were over 40 years of age and had either liver disease or habit of heavy drinking. Our present 4 cases were similar in these aspects.

Our patients showed typical clinical features. Blood pressures were low at the time of admission. As was noted in other patients, their WBC and platelet counts were generally low. Partial thromboplastin time was prolonged and fibrinogen degradation products were positive indicating a disseminated intravascular coagulation. Urinalysis was generally abnormal. All showed secondary skin manifestations such as bullae,

or swelling and pain in legs. Although Joh (1986) reported decreased mortality to 60% with the improved patient care, all of our patients either expired or were discharged in grave condition within 10 to 28 hours after admission.

V. vulnificus is susceptible, in vitro, to many antimicrobial agents. However, tetracycline has been shown to be more bactericidal, in vitro (Rhee and Chung 1985) and only tetracycline was effective in reducing mortality in experimental mice infection (Bowdre *et al.* 1983). Unfortunately, our 4 patients were not treated with tetracycline.

V. vulnificus was isolated from samples of sea food and sea water in Korea (Chong *et al.* 1984; Kim and Kim 1985; Song *et al.* 1985). As the organism is present in the normal marine ecosystem without fecal contamination, sea food collected from clean area may contain it. The risk of infection was considered to be great and those compromised to opportunistic infection were advised to eat their oysters well cooked (Johnston *et al.* 1983). In this country other sea foods also have caused the infection. We should cook all fish and shellfish especially during the summer months.

V. vulnificus have been isolated mainly from blood and wounds. Farmer *et al.* (1985) reported isolation from other specimens: 6 from stool, 2 from spinal fluid, and one each from urine and sputum. It was Pollack *et al.* (1983) who first isolated the organism from a stool specimen, and Tison and Kelly (1984) from the endocervix. In Korea, Kim *et al.* (1985a) reported an isolation from a stool specimen of a septicemic patient. We isolated the vibrio from a urine specimen of one patient. These results indicate that examination of specimens other than those of blood or wound may sometimes be helpful in the diagnosis of *V. vulnificus* infection.

The organism grew very rapidly in blood culture bottles of both TSB and BTM. After overnight incubation all of the bottles showed definite turbidity. Often the identification had been delayed because the organism was not suspected, and salt-containing media had not been used for biochemical tests. For API 20E, it was convenient to use saline instead of regular suspension fluid for all of the nonfastidious gram-negative bacilli identification.

The biochemical characteristics of *V. vulnificus* seemed at first quite homogeneous (Hollis *et al.* 1976; Blake *et al.* 1980). However, strains with more diverse reactions seemed to be present (Farmer *et al.* 1985); 35% were listed as being positive for phenylalanine deaminase, 15% for acid production from sucrose, and 1% for urease. Our present isolates showed typical

reactions, although acid production from lactose took 2-3 days, and 6% salt tolerance was difficult to determine. It may be necessary to be aware of the possibility of isolating biochemically atypical strains and of misidentifying other vibrios as *V. vulnificus* as Brady and Concannon (1984) suspected happened in some Australian cases.

Serovar determination may be a great aid in the identification of *V. vulnificus* and in epidemiology. Besides the serovars 1 and 4 of the present isolates, serovar 5 and 9 were isolated from man and 4 and 7 from shellfish in Korea.

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