Vibrio vulnificus Diarrhea in a Child with Respiratory Infection

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ABSTRACT

Vibrio vulnificus is a rare cause of disease and it is often unrecognized and underreported. It is a lactose-fermenting, halophilic *vibrio* causing severe disease in immunocompromised patients, but causing a mild type of gastroenteritis in healthy people, usually associated with consumption of seafood. We report here a case of diarrhea due to *V. vulnificus* in a male child who was admitted for fever, loose motions and productive cough. There was no history of consumption of any seafood; so, the source of infection could not be traced. As *V. vulnificus* is a rare entity, clinicians should have a high index of suspicion for the bacteria, when patients present with gastrointestinal illness, fever or shock, with or without ingestion of raw seafood. Pediatricians should also be alert as the bacterium causes a potentially fatal disease in children.

Key words: Diarrhea, *Vibrio vulnificus,* Child

INTRODUCTION

Vibrio vulnificus, formerly known as CDC Gr. EF-3, was recognized as an agent of disease in 1976.[1] The first documented case caused by *V. vulnificus* was in 1979, in which septic shock occurred due to ingested seafood and the organism was recovered from blood.[2] *V. vulnificus* can cause disease in those who eat contaminated seafood or have an open wound that is exposed to sea water. In healthy persons, only mild form of gastroenteritis occurs. In immunocompromised persons, particularly those with chronic liver disease, cancer, AIDS, chronic kidney disease and diabetes, it can cause a severe and life-threatening infection characterized by fever and chills, septic shock and blistering skin lesions (in 70% cases).[3] High fatality rate of 40–60% is associated with septic infections. From 1988 to 2006, CDC reported 900 *V. vulnificus* infections from Gulf Coast states.[3] Though *V. vulnificus* is a rare cause of disease, it is often unrecognized and underreported. Since 2007, infections caused by *V. vulnificus* and other *Vibrio* species have become nationally notifiable,[3] but reports from India are lacking. We report here a case of *V. vulnificus* diarrhea.

Access this article online

Quick Response Code:

Website: www.jgid.org

DOI: 10.4103/0974-777X.83544

CASE REPORT

A 1½ year-old male child was admitted to the Pediatric Intensive Care Unit of Lokmanya Tilak Municipal Medical College and Hospital in June 2007, with complaints of fever and loose motions for 15 days and cough and cold for 10 days. Frequency of motions was seven to eight per day and he had productive cough with post-tussive vomiting and breathlessness. There was no history of ingestion of raw seafood. He had measles when he was 8½ months old. His immunization was incomplete, as he received only OPV and BCG vaccines. On examination, he was lethargic, pale and dusky with shallow respiration. Pulse rate was 130/minute with bounding pulse, blood pressure was 100/65 mm of Hg and anterior fontanel was depressed with dry tongue. Pallor was present, but no icterus, edema, cyanosis, clubbing, lymphadenopathy or skin rashes were seen. He had altered sensorium, generalized hypotonia, reflexes were sluggish and plantar was flexed. Skin turgor was lost. He was malnourished, suffering from protein energy malnutrition (PEM). No abnormality was detected in other systems. He was diagnosed as a case of diarrhea with severe dehydration with respiratory infection and pallor. His hemoglobin was 11g% with leukocytosis and thrombocytopenia. Peripheral smear for malarial parasite was negative. His liver function and renal function tests were within normal limits and Widal test was negative. X-ray chest showed no abnormality.

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Blood and stool were sent for culture. Macroscopically, stool was greenish yellow liquid with mucus, no blood and no parasitic elements. Microscopically, there were 25–30 pus cells/high power field and no red blood cells, ova or cysts of parasites were seen. Blood culture and stool culture showed no growth. On admission, he was given IV fluids, Inj. Cefotaxime, Inj. Amikacin, Inj. Calcium gluconate and IV IgG for 5 days. After 4 days, Inj. Metronidazole was added.

He was put on ventilator on admission and taken off 2 weeks after the respiratory symptoms subsided. After 3 weeks, his total protein was 5.1g% and albumin was 2.6 g%. His hemoglobin reduced to 8.7g% and leukocytosis persisted. Another stool sample was sent for culture after 7 days to rule out *Campylobacter* species. On Gram stain, no spiral bacillus was seen. It was directly plated onto Campylobacter Charcoal Differential Agar and kept in incubator at 42°C under microaerophilic conditions for 48 hours.[4] *Campylobacter* species did not grow. But direct plating on MacConkey agar grew lactose-fermenting, oxidase-positive colonies, which were identified as *V. vulnificus* by standard biochemical tests.[4] It was a halophilic *vibrio*. It grew in nutrient broth containing 1% NaCl and fermented lactose (L + *vibrio*). It decarboxylated lysine and ornithine, but did not dihydrolyze arginine. It was sensitive to amikacin and resistant to amoxicillin–clavulanic acid, cefotaxime, nalidixic acid, co-trimoxazole, tetracycline and norfloxacin. Another stool culture sent after 4 days also grew the same bacteria. His endotracheal (ET) secretions grew *Klebsiella pneumoniae* sensitive to imipenem only and *Acinetobacter* species sensitive to imipenem and netilmycin.

He was started on Inj. Imipenem and Inj. Ciprofloxacin. Inj. Promethazine was given every 6 hours and nebulization (with ipratropium bromide) was done for 14 days. After 1 month, he was transferred to the ward. In the ward, he received Inj. Amikacin for 13 days after which he was discharged. At discharge, he had no complaints and his condition was stable.

DISCUSSION

Most *V. vulnificus* infections are reported from USA, Japan and Taiwan.[5] Chuang *et al.* have reported 28 cases of *V. vulnificus* infections in 27 patients from 1985 to 1990.[6] In a PubMed Search with "Diarrhea due to *Vibrio vulnificus* in India", no item was found. To the best of our knowledge, there is no other documented report on *V. vulnificus* diarrhea from India.

Gastroenteritis due to *V. vulnificus* is usually seen 16–38 hours after eating contaminated food, especially raw oysters, and is characterized by vomiting, diarrhea and abdominal pain. In this child, fever, diarrhea and vomiting were present along with respiratory symptoms. From ET secretion, *Klebsiella* and *Acinetobacter* species were grown, both resistant to first-line antibiotics. First stool culture was sent on the day of admission, when the bacterial load of *V. vulnificus* in the stool sample might have been very less; so, it failed to show growth of the same in the culture media.

Johnston *et al.* have reported three patients with diarrhea where *V. vulnificus* was the sole pathogen recovered from stool specimens. All three consumed raw oysters within past 1 week.[7] A case of fatal *V. vulnificus* septicemia has been reported in a thalassemic child, where there was no history of ingestion of raw shell fish.[8] In this case also, there was no history of consumption of any seafood like raw oysters. Therefore, source of infection could not be traced. The child was malnourished. PEM may be a predisposing factor in this case as *V. vulnificus* causes infection in immunocompromised patients.

The drug of choice for *V. vulnificus* is doxycycline/tetracycline and a third-generation cephalosporin. Initially, the child was given amikacin and cefotaxime. But as his condition deteriorated after 3 weeks, which may probably be due to growth of *Klebsiella* and *Acinetobacter* species from ET secretion, imipenem and ciprofloxacin were given for 14 days. He gradually improved, and after 1 month of ICU admission, he was shifted to the ward where he was given amikacin. Children in whom doxycycline and fluoroquinolone are contraindicated can be treated with trimethoprim-sulphamethoxazole along with an aminoglycoside. Respiratory infection may be an additional finding in this case.

Blood culture was also done in this case to rule out sepsis. It did not reveal any organism. Blood cultures are recommended if the patient is febrile or has any sign of sepsis. It is an acute illness and those who recover do not have any long-term consequence.

As *V. vulnificus* is rarely reported from India, it might be missed out or the laboratories are failing to diagnose it by not putting up the battery of biochemical tests that are required to identify this organism.

CONCLUSION

V. vulnificus is a rare cause of disease, but is also underreported. As it is a rare entity, clinicians should have a high index of suspicion for the bacteria when patients present with gastrointestinal illness, fever or shock, with

or without history of ingestion of raw seafood; or present with a wound infection after exposure to sea water. Pediatricians should also be alert as it is a potentially fatal disease, especially in immunocompromised children, so that prompt recognition would ensure immediate management, which has better prognostic implications.

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How to cite this article: De A, Mathur M. *Vibrio vulnificus* diarrhea in a child with respiratory infection. J Global Infect Dis 2011;3:300-2.

Source of Support: Nil. **Conflict of Interest:** None declared.