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Successful Treatment of Neutropenic Enterocolitis from *Vibrio fluvialis* in Acute Myeloid Leukemia: A Case Report

Debei C. Cargando[1], John S. Delgado[2,3]*

[1]Department of Internal Medicine, Jose R. Reyes Memorial Medical Center, Manila, Philippines
[2]Section of Infectious Diseases, Jose R. Reyes Memorial Medical Center, Manila, Philippines
[3]Faculty of Medicine and Surgery, University of Santo Tomas, Manila, Philippines
Email: *dcargando@gmail.com

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Abstract

Introduction: Neutropenic enterocolitis (NEC) is a life-threatening gastrointestinal syndrome characterized by fever, abdominal pain, and neutropenia. While most commonly associated with opportunistic bacteria like *Pseudomonas spp.*, the full spectrum of its etiological agents is not completely understood. This report presents the first documented case of NEC where *Vibrio fluvialis* is identified as the etiologic agent in a profoundly immunocompromised patient. This finding expands the differential diagnosis for NEC and highlights the potential for environmental organisms to cause severe infections in susceptible hosts. **Case Presentation**: A 37-year-old male with acute myeloid leukemia (AML) developed profound neutropenia following induction chemotherapy. He presented with high-grade fever, right lower quadrant abdominal pain, and loose, mucoid stools. A computed tomography scan revealed circumferential wall thickening of the ascending colon and cecum, consistent with NEC. Both blood and fecal cultures confirmed the presence of pansensitive *V. fluvialis*. The patient's condition significantly improved with broad-spectrum antibiotics, bowel rest, and supportive care. He was successfully treated with a 14-day course of meropenem and was discharged in a stable condition without requiring surgical intervention. **Conclusion**: This case report highlights *V. fluvialis* as a rare but significant cause of NEC in an adult with AML. The patient's positive outcome underscores the importance of a high index of clinical suspicion and a comprehensive microbiological workup in immunocompromised patients presenting with symptoms of NEC. Early diagnosis and targeted antimicrobial therapy are crucial to prevent progression to transmural necrosis and avoid invasive surgical procedures. This report serves as a reminder for clinicians to consider atypical, environmental patho-

gens in the differential diagnosis of NEC, particularly in patients with a history of travel or dietary exposure to marine products.

Keywords

Vibrio fluvialis, Necrotizing Enterocolitis, Febrile Neutropenia

1. Introduction

Neutropenic enterocolitis (NEC), a severe and life-threatening gastrointestinal syndrome, primarily affects immunocompromised patients. It is most commonly seen in those with hematologic malignancies, acquired immune deficiency syndrome (AIDS), and following solid organ or hematopoietic stem cell transplantation [1]. The condition is clinically defined by the triad of fever, abdominal pain, and profound neutropenia, accompanied by radiological evidence of bowel wall thickening. This thickening most often affects the terminal ileum, cecum, and ascending colon [2] [3]. The pathophysiology of NEC is complex and multifactorial. It involves three key elements: mucosal damage from cytotoxic chemotherapy or other insults, severe neutropenia, and an impaired immune response that leaves the host vulnerable to microbial invasion. A diverse range of pathogens has been implicated, including both bacterial and fungal species. Common culprits include gram-negative bacilli, gram-positive cocci, and anaerobic bacteria like Clostridium species [4].

Vibrio fluvialis is a halophilic, Gram-negative bacterium frequently isolated from aquatic environments, including water, sewage, and seafood, as well as from animal and human feces. Since its initial description by Furniss *et al*. in 1977, *V. fluvialis* has emerged as a significant, albeit unusual, enteric pathogen. Its role in both sporadic cases and outbreaks of diarrheal disease has become a growing public health concern over the past two decades [5].

Infection with *V. fluvialis* typically presents as a gastroenteritis syndrome. The clinical manifestations include a constellation of gastrointestinal symptoms such as nausea, anorexia, vomiting, abdominal cramps, and watery, often bloody, diarrhea, along with a significant fever. While *V. fluvialis* can cause self-limiting illness in healthy individuals, certain host factors predispose patients to more severe, systemic infections. These known risk factors include chronic liver disease, various immunocompromised states like AIDS, iron overload, and diabetes mellitus [5].

To our knowledge, based on an extensive literature search, *V. fluvialis* has not been previously documented as an etiological agent of NEC. This case report describes a rare and clinically significant instance of infection with this organism in a profoundly immunocompromised patient. It highlights the potential for environmental organisms to cause severe opportunistic infections in highly susceptible hosts. This unique case expands the known spectrum of pathogens associated

with NEC and emphasizes the need for a broader differential diagnosis in neutropenic patients.

2. Case Presentation

A 37-year-old Filipino male with a confirmed diagnosis of acute myeloid leukemia (AML) was admitted for induction chemotherapy. The planned regimen consisted of the standard "3 + 7" protocol, utilizing doxorubicin and cytarabine. Specifically, the patient was scheduled to receive doxorubicin at a dose of 60 mg on days 1 through 3, along with cytarabine at an initial loading dose of 300 mg/m^2, followed by a continuous infusion of 200 mg/m^2 from days 2 through 7. Upon admission, the patient's baseline laboratory values were as follows: a white blood cell (WBC) count of 3.4 × 10^9/μL, with an absolute neutrophil count (ANC) of 420/μL, a hemoglobin level of 82 g/L, and a platelet count of 32 × 10^9/μL. He had no other comorbid illness.

The patient's clinical course remained stable until day 4 of his cytarabine treatment, which corresponded to his 7th day of hospitalization. At this point, he developed a low-grade fever. He had no other associated symptoms, such as cough, diarrhea, or abdominal pain. A physical examination was unremarkable except for signs of localized inflammation at a previous intravenous doxorubicin infusion site on his left hand. Due to the febrile episode and his immunocompromised state, the Infectious Disease service was consulted for co-management. Laboratory tests at this time revealed a state of severe neutropenia, with a WBC count of 1.4 × 10^9/μL and an ANC of 163/μL. A chest radiograph showed no evidence of pulmonary infiltrates, and both urinalysis and fecalysis were unremarkable. Blood cultures were collected, and empirical broad-spectrum antibiotic therapy was initiated with cefepime and vancomycin to cover potential bacterial pathogens.

On the 8th day of hospitalization (day 5 of cytarabine treatment), the patient experienced recurrent febrile episodes. Despite this, he remained asymptomatic from other common signs of infection, reporting no dyspnea, cough, diarrhea, or abdominal pain. Notably, the erythema and inflammation at the site of his prior phlebitis showed signs of improvement. Due to the persistent fever, the cytarabine infusion was temporarily discontinued to evaluate the cause of the patient's condition.

On the 9th day of hospitalization, the patient presented with persistent high-grade fever and developed localized right lower quadrant abdominal pain. Physical examination was significant for both direct and rebound tenderness in this region, raising concern for an acute abdomen. Further laboratory analysis revealed a worsening of his myelosuppression, with the WBC count dropping to 0.7 × 10^9/μL and the ANC to 122/μL. His platelet count also fell to a critically low level of 17 × 10^9/μL. Fecalysis revealed the presence of 5 - 10 red blood cells (RBC)/hpf and 10 - 20 WBC/hpf, along with a significant amount of mucus (3+). A stool culture was requested for further pathogen identification. A rapid enzyme immunoassay for Clostridioides difficile toxin was performed, with a negative result. To

investigate the cause of his abdominal pain and rule out a surgical emergency, a whole-abdominal computed tomography scan was requested, and a surgical consultation was obtained. Later that same evening, the patient experienced four episodes of loose, watery, mucoid stools without any visible blood (Figure 1). Given the possibility of an acute surgical process, the patient was placed on parenteral feeding. Moreover, given the clinical suspicion for NEC, the empirical antimicrobial regimen was broadened to cover a wider spectrum of potential pathogens. Cefepime was discontinued and replaced with meropenem, while vancomycin was continued. Metronidazole and fluconazole were also added to the regimen.

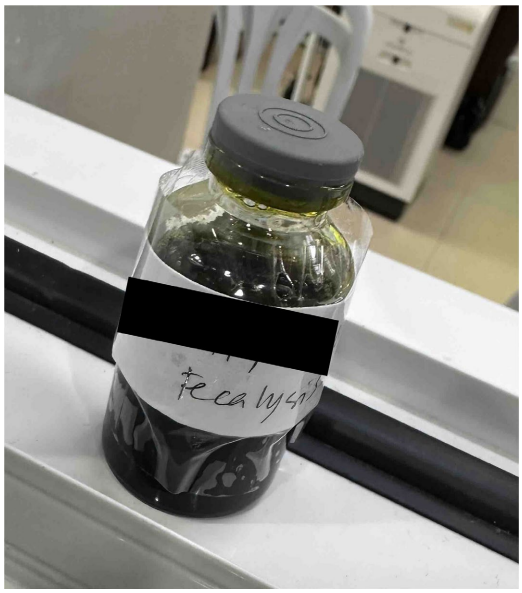

Figure 1. Stool specimen showing loose, watery consistency with green coloration and mucoid material.

On the 10th day of hospitalization, the patient's abdominal pain had improved, yet he continued to have a persistent fever and was passing loose, dark green stools. A whole abdominal computed tomography scan revealed a long, 9.7 cm segment of the ascending colon and cecum with circumferential and heterogeneous wall thickening (up to 1.0 cm). This thickening caused mild luminal narrowing and was associated with surrounding stranding and nodularity. The appendix was also prominent but did not show signs of frank inflammation (Figure 2).

By the 13th day of hospitalization, the patient's clinical status showed some improvement, with less frequent fever episodes and a reduction in abdominal pain. Despite this, he continued to have persistent loose, watery, and mucoid stools. A physical examination revealed slight tenderness remaining in the right lower quadrant. Laboratory tests still showed severe myelosuppression, with a WBC count of 0.47×10^9/μL, an absolute neutrophil count (ANC) of 82/μL, and significant thrombocytopenia with a platelet count of 26×10^9/μL. Blood and fecal cultures confirmed the presence of pansensitive *Vibrio fluvialis* (Table 1). This finding, combined with the clinical and radiological presentation, established the

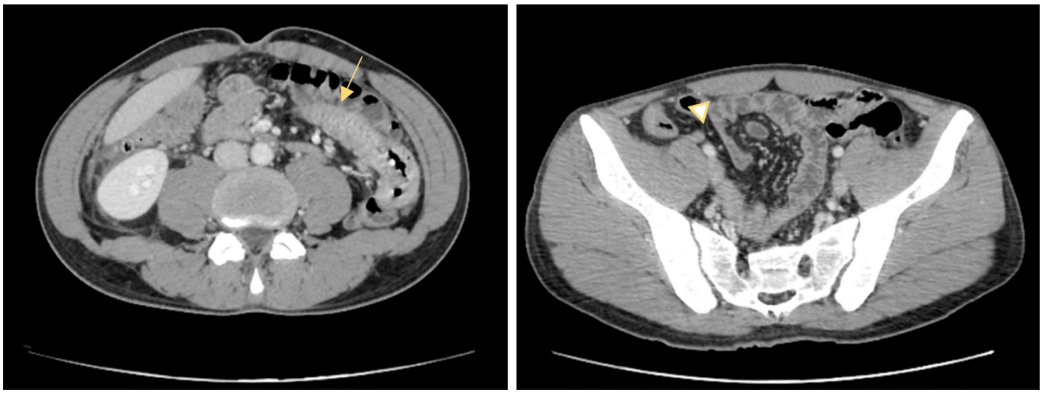

Figure 2. In axial computed tomography imaging of the anterior colon (thin arrow) and cecum (arrowhead) with heterogeneously-enhancing, long-segment bowel wall thickening causing mild luminal narrowing.

Table 1. Stool culture and antimicrobial susceptibility report.

Specimen type organism isolated	Stool moderate growth of *Vibrio fluvialis*				
Antimicrobial	MIC	Interpretation	Antimicrobial	MIC	Interpretation
Amikacin	4	S	Amoxicillin		
Co-Amoxiclav	≤2	S	Ampicillin	8	S
Benzylpenicillin			Cefepime	≤1	S
Cefoxitin	≤4	S	Ceftazidime	≤1	S
Ceftriaxone			Cefuroxime	4	S
Cefuroxime-Axetil			Ciprofloxacin	≤0.25	S
Clindamycin			Ertapenem		
Erythromycin			Gentamicin	≤1	S
Gentamicin-high			Imipenem	≤0.25	S
Levofloxacin			Linezolid		
Meropenem	≤0.25	S	Moxifloxacin		
Nitrofurantoin			Oxacilin		
Piperacillin/Tazobactam	≤4	S	Quinupristin/Dalfopristin		
Rifampin			Streptomycin high level		
Tetracycline			Tigecycline		
TMP/SMX	≤20	S	Vancomycin		
AES findings					

diagnosis of neutropenic enterocolitis secondary to this organism. With the diagnosis confirmed and the pathogen's susceptibility known, the antibiotic regimen was de-escalated from broad-spectrum agents to ciprofloxacin. The patient was maintained on parenteral nutrition, and surgical intervention was not deemed

necessary. Supportive care, including platelet apheresis and granulocyte colony-stimulating factor (G-CSF), was continued to manage his ongoing cytopenias.

On the 14th day of hospitalization, the patient's clinical status had significantly improved, marked by the absence of fever and abdominal pain. He also reported fewer episodes of loose, watery stools. Given this positive response to treatment, his diet was advanced to general liquids as tolerated. Ciprofloxacin was continued as the definitive antimicrobial therapy.

On the 16th day of hospitalization, the patient's diet was advanced to soft foods. However, this was followed by the new onset of a maculopapular rash on his trunk and extremities (Figure 3). A hypersensitivity reaction to ciprofloxacin was suspected, leading to the immediate discontinuation of the antibiotic. The patient was then switched back to meropenem, a drug he had previously tolerated without adverse effects.

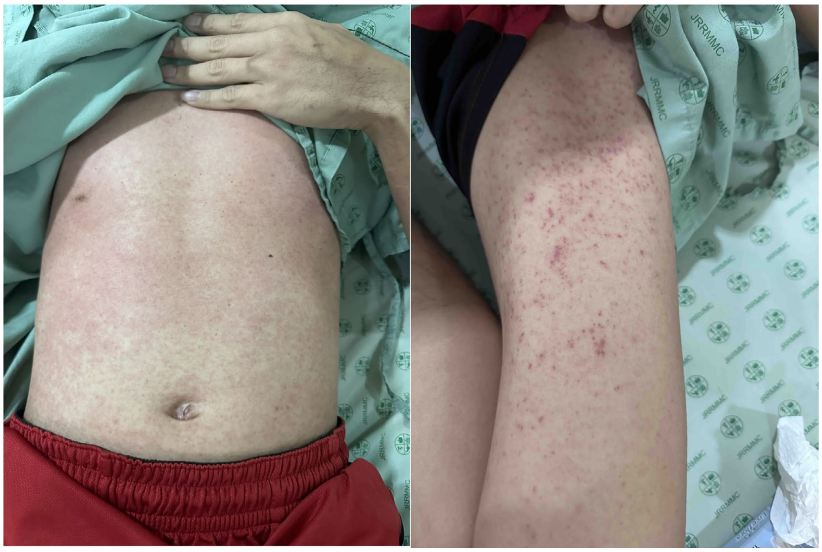

Figure 3. Maculopapular rash observed on the patient's trunk and extremities. Given its onset following the initiation of ciprofloxacin, a drug-induced hypersensitivity reaction was highly suspected.

On the 18th day of hospitalization, the patient's maculopapular rash had completely resolved. He remained afebrile, and his bowel movements had returned to a normal, formed, and non-mucoid consistency. He was tolerating a full diet without difficulty. A physical examination revealed a non-tender abdomen, and subsequent blood cultures were sterile, confirming the resolution of the bacteremia. Following the completion of a 14-day course of meropenem, the patient was discharged in a clinically improved and stable condition.

3. Discussion

Previously characterized as a disease of preterm neonates, NEC is now an increasingly recognized condition in adults, particularly in the context of profound immunosuppression [6] [7]. Adult-onset NEC has been reported among patients

with hematologic malignancies, those undergoing chemotherapy, organ transplantation, or prolonged critical illness. The pathophysiology in adults mirrors that in neonates, with intestinal mucosal injury, impaired host defenses, altered microbiota, a proinflammatory cascade culminating in ischemia, necrosis, and bacterial translocation [8].

In this case, NEC was documented in a patient with AML The clinical context was characterized by profound neutropenia and compromised mucosal barriers, both of which are common sequelae of cytotoxic chemotherapy. These factors collectively predispose the patient to bacterial translocation, leading to opportunistic and atypical enteric infections [9]. While NEC is commonly associated with bacterial isolates such as *Pseudomonas spp.*, *Escherichia coli*, *Klebsiella spp.*, *Staphylococcus aureus*, and *Streptococci* [10], the isolation of *V. fluvialis* in this patient represents an exceptionally rare etiology. To our knowledge, this is the first documented case of *V. fluvialis*-induced NEC in an adult with AML.

V. fluvialis is a halophilic gram-negative bacillus typically associated with foodborne gastroenteritis in endemic coastal regions, acquired through the ingestion of raw or undercooked seafood or contaminated water [5]. Although sporadic cases of bacteremia, peritonitis, and wound infections have been reported, invasive disease remains uncommon and usually occurs in immunocompromised hosts [6]. Several virulence attributes plausibly connect *V. fluvialis* to the rapid mucosal injury characteristic of NEC. Strains can produce hemolysins, cytolysins, and enterotoxin-like factors, along with proteases and the ability for biofilm formation. Purified *V. fluvialis* hemolysin, for instance, exhibits cytotoxic activity that can amplify epithelial damage and facilitate bacterial translocation in a neutropenic gut [11]. These mechanisms provide biologic plausibility that *V. fluvialis* may act not merely as a bystander colonizer but as a trigger or accelerator of the inflammatory-ischemic cascade that characterizes NEC.

Thiosulfate-citrate-bile salts-sucrose (agar is the conventional selective medium for isolating clinically significant Vibrio species. On this medium, the colony morphology of *V. fluvialis* is phenotypically indistinguishable from that of *Vibrio cholerae*, as both appear as yellow colonies after direct plating or enrichment in alkaline peptone water. Consequently, a comprehensive battery of biochemical tests (including lysine decarboxylase, ornithine decarboxylase, arginine dihydrolase, and L-arabinose fermentation) is essential for accurate, species-specific identification. The failure to perform these minimal biochemical tests, particularly in resource-limited settings, can lead to the misidentification of *V. fluvialis* as *V. cholerae*, thereby underreporting its true prevalence [12]. In this specific case, our institution's adherence to a complete biochemical testing protocol was critical in correctly identifying the pathogen.

The isolation of *V. fluvialis* in this case report carries several significant implications. First, it underscores the critical need for a comprehensive microbiological workup (including blood cultures, stool/enteric panels, and selective tissue cultures with complete biochemical tests) in cases of suspected NEC in immunocom-

promised patients. Second, this finding raises important epidemiological considerations, such as dietary exposures to raw seafood, hospital water sources, and regional ecology, which can guide infection-prevention strategies and patient counseling during neutropenic periods. Finally, this case adds to the small but growing body of literature on invasive *V. fluvialis* disease, compelling clinicians to include Vibrio species in the differential diagnosis of NEC in profoundly immunosuppressed adults, particularly those in coastal or aquaculture-adjacent settings.

While reports of human *V. fluvialis* infections in the Philippines are scarce, related studies provide valuable insights into its potential environmental presence. For instance, a study in Peninsular Malaysia detected *V. fluvialis* at a low prevalence of 0.3% in aquacultured groupers [13]. Similarly, research conducted in South Korea isolated *V. fluvialis* from Manila clams (*Ruditapes philippinarum*), a species native to the Philippines, that were sold in Korean markets [14]. Although these studies were not conducted in the Philippines, the findings underscore the possibility of environmental reservoirs of *V. fluvialis* within the country's marine ecosystems or among its seafood exports. This evidence suggests a potential, albeit minor, source of contamination in Southeast Asian marine products that could have parallels in the regional aquatic environment, warranting heightened clinical suspicion.

Upon recognizing the high probability of NEC in this case, management was initiated with prompt administration of broad-spectrum antimicrobial therapy, bowel rest, aggressive supportive care, and surgical consultation. Initial empiric regimens commonly include antipseudomonal β-lactams such as piperacillin–tazobactam or carbapenems. While treatment for *V. fluvialis* varies among reported cases, combination therapy involving β-lactams with aminoglycosides, fluoroquinolones, or tetracyclines has proven effective. Notably, both carbapenems and quinolones have demonstrated reliable in vitro activity and clinical success in treating invasive *V. fluvialis* infections [5] [15]. Accordingly, the definitive therapy in this case appropriately included a carbapenem, with a fluoroquinolone considered as a step-down or adjunctive agent once antimicrobial susceptibilities were confirmed and clinical stability was achieved. This comprehensive approach ensured coverage for both typical enteric flora and this rare halophilic pathogen.

4. Conclusion

This case report significantly contributes to the limited body of literature documenting rare pathogens, such as *V. fluvialis*, as an etiology for NEC in adults. For clinicians managing immunocompromised patients, particularly those with hematologic malignancies, this case highlights the importance of including Vibrio species in the differential diagnosis of NEC. Early recognition and accurate microbiological confirmation are crucial for initiating targeted antimicrobial therapy, which can prevent progression to transmural necrosis and perforation and, as demonstrated in this case, improve outcomes by precluding the need for invasive surgical intervention. This successful outcome underscores the importance of a

high index of suspicion and rapid, definitive treatment for this vulnerable population.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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