



## CHOLERA BEYOND THE GUT: A RARE CASE OF PULMONARY INFECTION BY VIBRIO CHOLERAE

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### ABSTRACT

Pulmonary infections caused by *Vibrio cholerae* are exceedingly rare and remain underreported in clinical literature. While *Vibrio cholerae* is classically associated with acute watery diarrhea and cholera outbreaks, extraintestinal manifestations—including respiratory infections—are uncommon and poorly understood. We report a rare case of a 46 year old female from a rural background presenting with acute febrile illness and respiratory symptoms, ultimately diagnosed with right lower lobe pneumonia caused by non-O1 *Vibrio cholerae*. The patient presented with intermittent fever, chills, myalgia, anorexia, and bilateral chest and upper back discomfort of four days duration. Radiological imaging revealed right lower lobe consolidation. Laboratory investigations demonstrated leukocytosis with neutrophilia. Extensive infectious workup including Widal test, viral markers, and scrub typhus serology was negative. Sputum culture yielded *Vibrio cholerae*, confirmed by characteristic colony morphology, darting motility, and biochemical features, and further identified as a non-O1 strain by serotyping. This case highlights an unusual pulmonary manifestation of *Vibrio cholerae* infection in an immunocompetent individual with occupational exposure risk. The report underscores the importance of considering atypical pathogens in community-acquired pneumonia, especially in endemic regions. Early microbiological diagnosis is critical for targeted therapy and improved outcomes.

**Keywords:** *Vibrio Cholerae*, Non-O1 *Vibrio Cholerae*, Pulmonary Infection, Atypical Pneumonia, Extraintestinal Cholera, Case Report, India.

### INTRODUCTION

*Vibrio cholerae* is a Gram-negative, comma-shaped bacillus primarily known for causing cholera, an acute diarrheal illness characterized by profuse watery diarrhea and dehydration. The organism is typically transmitted through contaminated water or food and is strongly associated with poor sanitation and hygiene conditions. Classical cholera is caused by toxigenic strains belonging to serogroups O1 and O139, which produce cholera toxin leading to secretory diarrhea.

However, non-O1 and non-O139 strains of *Vibrio cholerae* have increasingly been recognized as causes of extraintestinal infections.

These include bacteremia, wound infections, urinary tract infections, and rarely, respiratory infections. Pulmonary involvement due to *Vibrio cholerae* is extremely uncommon, with only a handful of cases reported globally. Such infections are often associated with immunocompromised states, chronic liver disease, or exposure to aquatic environments.

The pathogenesis of pulmonary infection by *Vibrio cholerae* is not well understood. Possible mechanisms include aspiration of contaminated material, hematogenous spread, or direct inhalation of aerosolized organisms. Occupational exposure, particularly in environments involving food waste or contaminated surfaces, may play a significant role.

In this report, we describe a rare case of pneumonia caused by non-O1 *Vibrio cholerae* in an immunocompetent middle-aged female with no gastrointestinal symptoms. This case is notable for its atypical presentation, absence of classical risk factors, and microbiological confirmation from sputum culture. The report aims to contribute to the



www.ajmrhs.com  
eISSN: 2583-7761

Date of Received: 25-03-2026  
Date Acceptance: 05-04-2026  
Date of Publication: 01-05-2026

limited literature on respiratory infections caused by *Vibrio cholerae* and emphasizes the need for heightened clinical suspicion in unusual presentations.

**Case Presentation**

A 46 year old female from a rural village presented to the outpatient department of Government Medical College, Ramanathapuram, Tamil Nadu, India, with complaints of fever of four days duration. The fever was intermittent in nature and associated with chills and rigors. She also reported generalized myalgia and loss of appetite over the same duration. In addition, she complained of discomfort in the chest and upper back bilaterally, which was persistent and non-radiating. There was no history of cough with expectoration, hemoptysis, breathlessness, or pleuritic chest pain. Importantly, there was no history of diarrhea, vomiting, or abdominal pain, thereby excluding classical gastrointestinal manifestations of cholera. The patient had no known comorbidities such as diabetes mellitus, hypertension, chronic liver disease, or immunosuppressive conditions. She had no history of prior hospitalization and denied any addictive behaviors including alcohol consumption or smoking. Occupational history revealed that she was employed in a hotel, where her responsibilities included cleaning tables and handling food waste disposal. There was no direct history of exposure to sewage or contact with cattle. On physical examination, the patient was conscious, oriented, and hemodynamically stable. Vital signs were within normal limits. Systemic examination revealed no abnormalities in the cardiovascular or abdominal systems. Respiratory examination did not reveal significant findings such as crepitations or bronchial breath sounds at presentation. Given the clinical picture of acute febrile illness, a provisional diagnosis of “fever for evaluation” with a differential of enteric fever was considered.

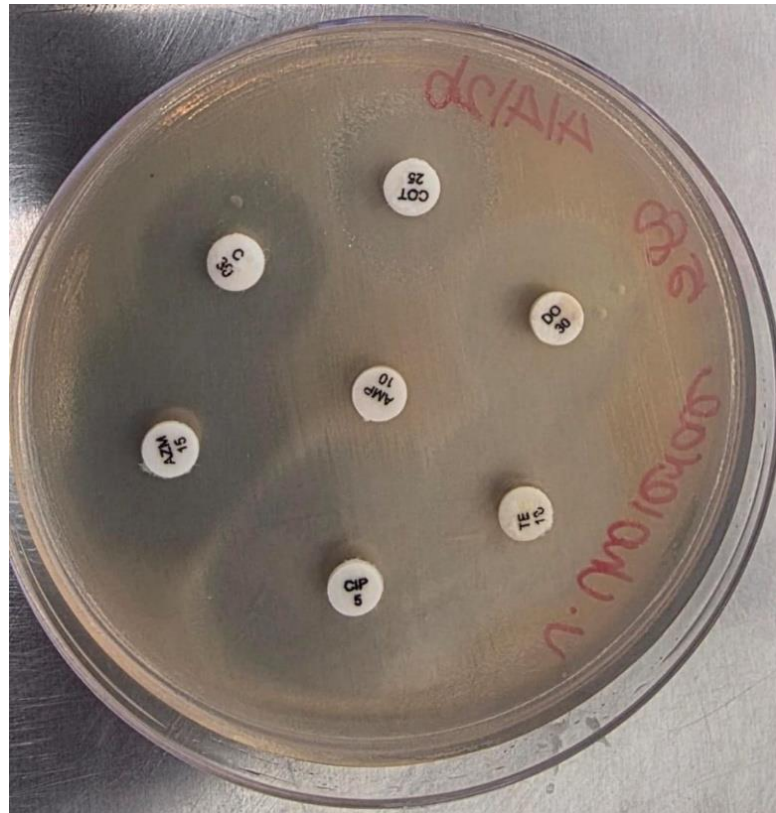
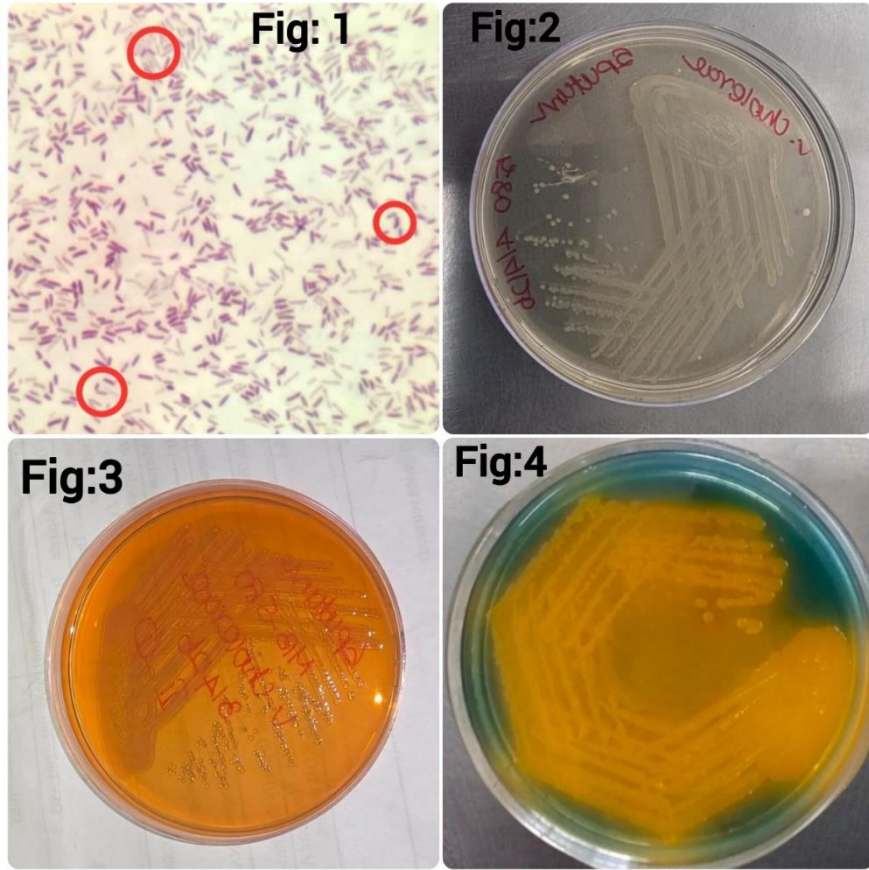
**Investigations**

Radiological evaluation was performed to assess the cause of chest discomfort. Chest X-ray revealed consolidation in the right lower lobe. This finding

was further confirmed by computed tomography (CT) of the chest, which demonstrated localized consolidation in the same region, consistent with lobar pneumonia. Hematological investigations showed significant leukocytosis with a total leukocyte count of 20,500 cells/mm<sup>3</sup>. Differential count revealed neutrophilia (82%), lymphocytes 12%, and monocytes 6%. Hemoglobin was 11.0 g/dL, and platelet count was within normal limits at 1.99 lakh/mm<sup>3</sup>. Renal function tests were within normal limits, with urea at 25 mg/dL and creatinine at 0.8 mg/dL. Liver function tests showed mild elevation of transaminases (SGOT: 47 IU/L, SGPT: 42 IU/L), with normal bilirubin levels. Serum electrolytes were within normal limits. Serological investigations including Widal test, HIV, VDRL, HBsAg, and anti-HCV were negative. Scrub typhus IgM ELISA was also negative. ECG showed normal findings. Sputum sample was collected and sent for bacterial culture and sensitivity testing. Culture on nutrient agar showed typical colonies. On MacConkey agar, non-lactose fermenting colonies were observed. Thiosulfate-citrate-bile salts-sucrose (TCBS) agar showed characteristic yellow colonies suggestive of sucrose fermentation. Gram staining of the isolate revealed comma-shaped Gram-negative bacilli, often appearing as “gull-wing” shapes. Motility testing demonstrated characteristic darting motility. (Figure 1,2,3,4) Biochemical identification confirmed the organism as *Vibrio cholerae*. Serotyping revealed that the isolate did not agglutinate with O1 antisera, indicating a non-O1 strain. Antimicrobial susceptibility testing (AST) was performed using standard disk diffusion method as per CLSI M100 36<sup>th</sup> Edition 2026 guidelines. The isolate was found to be sensitive to doxycycline, azithromycin, chloramphenicol, ciprofloxacin, and tetracycline, while showing resistance to ampicillin. (Table 1) These findings are consistent with the known susceptibility patterns of non-O1 *Vibrio cholerae* strains. (Figure 5) The isolate was sent to the National Reference laboratory for further characterization, and the findings were consistent with *Vibrio cholerae*.

Table 1: Antimicrobial Susceptibility Pattern of *Vibrio cholerae*

Antibiotic	Result
Doxycycline	Sensitive
Azithromycin	Sensitive
Chloramphenicol	Sensitive
Ciprofloxacin	Sensitive
Tetracycline	Sensitive
Ampicillin	Resistant



## DISCUSSION

The present case describes an exceedingly rare manifestation of *Vibrio cholerae* infection presenting as pulmonary consolidation in an immunocompetent individual without any gastrointestinal symptoms. While *Vibrio cholerae* is classically associated with cholera—a secretory diarrheal disease mediated by cholera toxin—its role in extraintestinal infections is increasingly recognized, particularly with non-O1 and non-O139 serogroups. This case contributes to the limited but growing body of literature describing respiratory involvement by *Vibrio cholerae*, highlighting both microbiological and clinical complexities.

From a microbiological perspective, *Vibrio cholerae* is a curved, comma-shaped, motile Gram-negative bacillus belonging to the family Vibrionaceae. Its hallmark feature is its polar flagellum, which imparts the characteristic “darting motility” observed under microscopy. The organism is oxidase positive and thrives in alkaline environments, with optimal growth at a pH of 8.0–9.5. Its ability to grow in thiosulfate-citrate-bile salts-sucrose (TCBS) agar, producing yellow colonies due to sucrose fermentation, remains a cornerstone of laboratory diagnosis. In this case, the isolate demonstrated classical microbiological characteristics including non-lactose fermenting colonies on MacConkey agar, yellow colonies on TCBS, and the presence of comma-shaped bacilli with darting motility, confirming the organism as *Vibrio cholerae*.

The classification of *Vibrio cholerae* into serogroups is based on the O antigen of lipopolysaccharide. More than 200 serogroups exist; however, only O1 and O139 are typically associated with epidemic cholera due to their ability to produce cholera toxin (CT). Non-O1/non-O139 strains, such as the one isolated in this case, generally lack the *ctxAB* genes encoding cholera toxin and therefore do not cause classical cholera. Instead, they are increasingly implicated in localized and systemic infections including wound infections, bacteremia, and, rarely, respiratory infections. The absence of agglutination with O1 antisera in this case confirms the isolate as a non-O1 strain, aligning with its extraintestinal presentation.

The pathogenesis of pulmonary infection by *Vibrio cholerae* remains poorly understood, largely due to the rarity of such cases. Several hypotheses can be proposed based on known virulence mechanisms of the organism. Unlike toxigenic strains, non-O1 *Vibrio cholerae* relies on a diverse array of virulence factors that facilitate colonization, invasion, and tissue damage. These include hemolysins, cytotoxins, metalloproteases, outer membrane proteins, and type VI secretion systems. Among these, the hemolysin (*hlyA*) has been shown to exert cytotoxic effects on epithelial cells and may play a role in pulmonary tissue damage. Similarly,

the RTX toxin (repeats-in-toxin) contributes to actin cytoskeleton disruption and cell rounding, potentially aiding in tissue invasion.

Another critical virulence determinant is the mannose-sensitive hemagglutinin (MSHA), a type IV pilus that facilitates adherence to epithelial surfaces and environmental substrates. In the respiratory tract, such adherence mechanisms may enable colonization of bronchial or alveolar epithelium, particularly in the presence of microaspiration. The organism’s ability to form biofilms further enhances its persistence in hostile environments, including the human host.

In the present case, the absence of gastrointestinal symptoms suggests that the route of infection was unlikely to be enteric. Instead, inhalation of contaminated aerosols or aspiration of contaminated material emerges as a plausible mechanism. The patient’s occupational exposure—handling food waste in a hotel environment—may have facilitated contact with contaminated organic matter harboring *Vibrio cholerae*. It is well established that *Vibrio* species can survive in moist environments and associate with organic debris, plankton, and biofilms. Aerosolization of such material during cleaning activities could result in inhalational exposure.

The possibility of hematogenous spread, although less likely in this case due to the absence of bacteremia, cannot be entirely excluded. In reported cases of *Vibrio cholerae* bacteremia, pulmonary involvement may occur as a secondary focus. However, in this patient, the localized consolidation and absence of systemic dissemination favor a primary pulmonary infection. While the pathogenesis of pulmonary involvement by non-O1 *Vibrio cholerae* remains speculative, microbiological evidence in the present case provides critical insights into the probable route of infection and localization of the organism.

An important and defining feature of the present case is the isolation of *Vibrio cholerae* exclusively from sputum, with concurrent negative blood and stool cultures. This microbiological pattern has significant pathogenetic, diagnostic, and clinical implications, reinforcing the hypothesis of a primary localized pulmonary infection rather than secondary dissemination from an intestinal or systemic source.

Classically, *Vibrio cholerae* infection is enteric, with stool cultures yielding the organism in large numbers during the acute diarrheal phase. In cases of extraintestinal involvement, particularly bacteremia, the organism is often isolated from blood, especially in immunocompromised hosts or those with chronic liver disease. However, in the present case, the absence of *Vibrio cholerae* in both stool and blood effectively excludes the two most common pathways of infection—intestinal colonization with secondary spread and primary bacteremia with metastatic seeding.

This finding strongly supports the concept of primary pulmonary acquisition, which is exceedingly rare. The most plausible mechanism in this scenario is direct inhalation or microaspiration of contaminated material, leading to localized infection in the lower respiratory tract. The involvement of the right lower lobe, as seen in this patient, further supports an aspiration-related pathogenesis, as this region is anatomically predisposed due to the more vertical orientation of the right main bronchus.

From a microbiological standpoint, the isolation of *Vibrio cholerae* in pure culture from sputum, combined with consistent Gram stain findings (comma-shaped Gram-negative bacilli) and absence of competing respiratory flora, significantly reduces the likelihood of contamination or colonization. In respiratory microbiology, distinguishing true pathogens from colonizers is often challenging; however, several criteria in this case support pathogenicity:

1. Correlation with radiological findings (lobar consolidation)
2. Presence of systemic inflammatory response (marked leukocytosis with neutrophilia)
3. Absence of alternative etiological agents after extensive workup
4. Growth of a single organism in culture with characteristic morphology

The negative stool culture is particularly noteworthy because it indicates that the organism did not colonize the gastrointestinal tract in detectable numbers. This may suggest either a low inoculum exposure limited to the respiratory tract or the involvement of a strain with reduced enteric colonization potential but enhanced extraintestinal virulence traits. Non-O1/non-O139 *Vibrio cholerae* strains are known to exhibit significant genetic diversity, including the presence of virulence factors that favor tissue invasion rather than toxin-mediated diarrhea.

Similarly, the absence of bacteremia (as evidenced by negative blood culture) indicates that the infection remained localized and did not progress to systemic dissemination. This is consistent with the patient's relatively stable clinical condition and lack of features suggestive of sepsis. It also suggests that host immune defenses were effective in containing the infection within the pulmonary compartment.

This microbiological pattern aligns with rare reports in literature where *Vibrio cholerae* has been isolated from unusual sites such as pleural fluid or respiratory secretions without concurrent intestinal involvement. Such cases challenge the traditional paradigm of *Vibrio cholerae* as an exclusively enteric pathogen and highlight its opportunistic potential in non-gastrointestinal niches.

The antimicrobial susceptibility profile observed in this case is consistent with previously reported patterns of non-O1 *Vibrio cholerae*, which generally

remain susceptible to tetracyclines, macrolides, and fluoroquinolones. Resistance to ampicillin, as noted in this isolate, has been increasingly documented and may be attributed to beta-lactamase production. The susceptibility to doxycycline and azithromycin is clinically significant, as these agents remain the mainstay of therapy in *Vibrio* infections. Continuous surveillance of antimicrobial resistance in *Vibrio cholerae* is essential, given the emerging trends of multidrug resistance.

Another important implication of negative stool culture is in infection control and public health. Classical cholera cases require strict enteric precautions due to high transmissibility via fecal-oral route. In contrast, isolated pulmonary infection without gastrointestinal shedding may pose a lower risk of transmission, although environmental contamination cannot be entirely excluded.

From a diagnostic perspective, this case emphasizes the importance of site-specific sampling. Had sputum culture not been performed, the etiological agent would have remained unidentified, and the case might have been managed empirically as routine bacterial pneumonia. This underscores the critical role of microbiology laboratories in identifying atypical pathogens through comprehensive culture techniques.

Furthermore, the discordance between sputum positivity and stool negativity raises interesting questions regarding strain-specific tropism. It is conceivable that certain non-O1 strains possess genetic determinants that favor survival and replication in respiratory epithelium. Advanced molecular studies, including whole-genome sequencing of the isolate (currently pending at the ICMR National Institute of Research in Bacterial Infections), may provide valuable insights into such adaptations.

In summary, the exclusive isolation of *Vibrio cholerae* from sputum with negative blood and stool cultures is a pivotal finding in this case. It supports a diagnosis of primary pulmonary *Vibrio cholerae* infection, highlights an atypical route of transmission, and reinforces the need for heightened clinical suspicion and targeted microbiological investigation in unusual presentations of pneumonia. These findings further emphasize the importance of comprehensive microbiological evaluation in atypical respiratory infections and highlight the diagnostic value of site-specific cultures.

Radiologically, the presence of right lower lobe consolidation is consistent with bacterial pneumonia. The lower lobes are particularly susceptible to aspiration-related infections due to gravitational factors, lending further support to the hypothesis of aspiration as a route of infection. The absence of classical respiratory symptoms such as productive cough or dyspnea is notable and underscores the atypical presentation.

The hematological profile showing marked leukocytosis with neutrophilia is indicative of an acute bacterial infection. Mild anemia and slightly elevated transaminases may reflect systemic inflammatory response rather than specific organ involvement. Importantly, the extensive negative serological workup—including Widal test, HIV, viral hepatitis markers, and scrub typhus—helped exclude more common etiologies of febrile illness in this geographical region.

From a diagnostic microbiology standpoint, this case underscores the importance of routine culture techniques and careful interpretation of unusual isolates. In many laboratories, non-enteric isolates of *Vibrio cholerae* may be dismissed as contaminants, particularly in respiratory samples. However, the consistent growth of the organism in pure culture, along with supportive Gram stain findings and clinical correlation, strongly supports its etiological role in this case.

The use of selective media such as TCBS agar is critical in isolating *Vibrio* species, especially when they are not initially suspected. The yellow colonies observed on TCBS in this case are characteristic of sucrose-fermenting *Vibrio cholerae*, distinguishing it from other *Vibrio* species such as *Vibrio parahaemolyticus*, which typically produces green colonies. Motility testing demonstrating darting motility and oxidase positivity further aid in identification.

Serotyping remains an essential step in differentiating epidemic from non-epidemic strains. The lack of agglutination with O1 antisera confirms the isolate as non-O1, which correlates with its atypical presentation. Further molecular characterization, such as PCR for virulence genes, would provide deeper insights into the pathogenic potential of the isolate. The referral of the isolate to the ICMR National Institute of Research in Bacterial Infections is therefore a crucial step for advanced characterization, including whole-genome sequencing, which may reveal unique virulence determinants.

The ecological niche of *Vibrio cholerae* also plays a significant role in its pathogenicity. The organism is naturally found in aquatic environments, particularly in brackish water, where it associates with plankton and chitinous surfaces. Climate factors such as temperature, salinity, and nutrient availability influence its proliferation. In tropical regions like Tamil Nadu, environmental conditions are conducive to the survival and spread of *Vibrio* species. Although the patient did not report direct exposure to sewage or aquatic environments, indirect exposure through contaminated food waste remains plausible.

The concept of “viable but non-culturable” (VBNC) state is another important aspect of *Vibrio cholerae* biology. In adverse conditions, the organism can enter a dormant state where it remains metabolically

active but cannot be cultured by standard methods. Upon entering a favorable environment, such as the human host, it can resuscitate and regain virulence. This phenomenon may contribute to underdiagnosis of *Vibrio* infections and underscores the importance of molecular diagnostic tools.

Another intriguing aspect is the role of quorum sensing in regulating virulence gene expression in *Vibrio cholerae*. The organism uses chemical signaling molecules to coordinate gene expression based on population density. At low cell density, virulence genes are upregulated, facilitating colonization and infection. At high cell density, these genes are downregulated, promoting detachment and dissemination. Such regulatory mechanisms may influence the course of infection in non-enteric sites, including the lungs.

Antimicrobial susceptibility patterns of non-O1 *Vibrio cholerae* are variable and region-dependent. While the organism is generally susceptible to tetracyclines, fluoroquinolones, and third-generation cephalosporins, emerging resistance has been reported. In the context of pulmonary infection, appropriate antibiotic therapy guided by sensitivity testing is essential. Empirical therapy for community-acquired pneumonia may not adequately cover *Vibrio* species, leading to delayed clinical response.

The clinical implications of this case are significant. First, it highlights the need for clinicians to consider atypical pathogens in patients presenting with pneumonia, especially when initial investigations are inconclusive. Second, it underscores the importance of detailed occupational history, which may provide clues to unusual exposures. Third, it emphasizes the critical role of microbiology laboratories in identifying rare pathogens and guiding appropriate therapy.

From a public health perspective, the emergence of extraintestinal *Vibrio cholerae* infections raises important questions about environmental reservoirs and transmission dynamics. Surveillance systems traditionally focus on diarrheal disease, potentially overlooking non-enteric manifestations. Strengthening laboratory capacity and integrating environmental monitoring may help in early detection and prevention.

In comparison with previously reported cases, this case is unique in several aspects. The patient was immunocompetent, lacked classical risk factors, and presented without gastrointestinal symptoms. The isolation of *Vibrio cholerae* from sputum in pure culture, along with radiological evidence of pneumonia, strongly supports its role as a primary pathogen. Such cases challenge the conventional understanding of *Vibrio cholerae* as an exclusively enteric pathogen and expand its clinical spectrum.

In conclusion, this case underscores the remarkable adaptability of *Vibrio cholerae* and its ability to cause disease beyond the gastrointestinal tract. The

interplay of environmental exposure, microbial virulence, and host factors culminates in rare but clinically significant presentations such as pulmonary infection. Enhanced awareness, meticulous microbiological evaluation, and interdisciplinary collaboration are essential for the timely diagnosis and management of such unusual infections.

### CONCLUSION

This case highlights an unusual presentation of *Vibrio cholerae* infection as pneumonia in an immunocompetent individual without gastrointestinal involvement. It emphasizes the need for clinicians and microbiologists to be vigilant for atypical presentations of common pathogens. Routine microbiological evaluation, including culture and sensitivity, is essential in identifying rare etiological agents. This case also underscores the importance of occupational exposure as a potential risk factor. Further studies are needed to better understand the pathogenesis and clinical spectrum of extraintestinal *Vibrio cholerae* infections.

### Conflict of Interest

None declared.

### Funding

No external funding was received.

### Acknowledgment

We acknowledge the support of the microbiology laboratory staff and the ICMR National Institute of Research in Bacterial Infections for confirmatory testing.

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**How to cite this article:** Saheed Askar S I, Subitha B, Brindha J, Eunice S, Fatima Bathool Rani K, Rajeswaran S, Manjari K V, Rajathy Fathima S, Thamizhselvi S, Praveen Raj K, Ragani C M, Jayaraman K, CHOLERA BEYOND THE GUT: A RARE CASE OF PULMONARY INFECTION BY VIBRIO CHOLERAE, *Asian J. Med. Res. Health Sci.*, 2026; 4 (2):-1-8.

**Source of Support:** Nil, Conflicts of Interest: None declared.