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Case Report

Microbiology Section

A Rare Report of Carbapenem Resistant *Vibrio fluvialis* Isolated from a Case of Walled off Necrosis: A Sequelae of Acute Necrotising Pancreatitis

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ABSTRACT

Vibrio fluvialis (V. fluvialis) is commonly found in coastal environments and is an emerging pathogen encountered in diarrhoeal outbreaks and sporadic extraintestinal cases. *V. fluvialis* infections usually manifest as watery bloody diarrhoea, nausea, vomiting, gastroenteritis and peritonitis. In this case report, an unusual presentation of Multidrug Resistant (MDR) *V. fluvialis* along with *Klebsiella pneumoniae* co-infection from a case of Walled Off Necrosis (WON)- A complication of Acute Necrotising Pancreatitis (ANP) is discussed. The patient presented with the acute abdomen and had peripancreatic collection for which Ultrasound (USG) guided 12F pigtail drain placed over the right lower quadrant of the abdomen and conservatively managed. Routine cultures were done and the growth on Sheep Blood Agar (SBA) plate and MacConkey (MAC) agar plates were subjected for automated phenotypic identification by Matrix Assisted Laser Desorption Ionisation Time of Flight-Mass Spectrometry (MALDITOF-MS) and Vitek-2 systems and was identified as *V. fluvialis*. Also, molecular tests like 16S rRNA Polymerase Chain Reaction (PCR) followed by Sanger's sequencing was done and the identification was further confirmed. Phenotypic RESIST-5 TRURAPID O.K.N.V.I. commercial lateral flow immunochromatographic assay for detection of clinically relevant and common carbapenemase genes like NDM, OXA-48, VIM, IMP and KPC was done which revealed this *V. fluvialis* isolate as New Delhi Metallobetalactamase (NDM) type of Carbapenemase producer. Patient was managed with levofloxacin and doxycycline and patient condition improved and was discharged.

Keywords: Bacterial, Carbapenem-resistant enterobacteriaceae, Communicable diseases, Drug resistance, Emerging

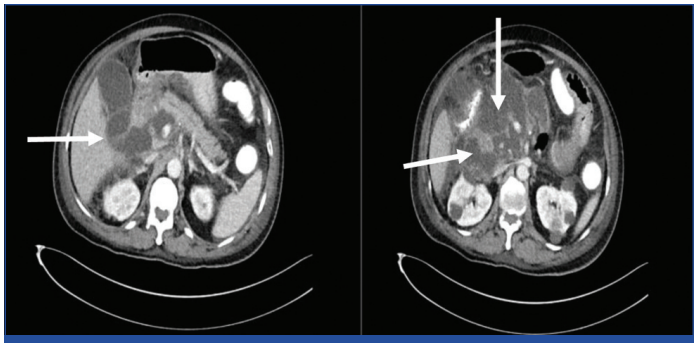
CASE REPORT

A 64-year-old non alcoholic and non smoker male, a farmer by occupation, presented to gastroenterology outpatient department with complains of severe abdominal pain, vomiting, decreased urine output for one day and inability to tolerate solid food for a week. Patient was a known case of secondary hyperparathyroidism, controlled systemic hypertension and dyslipidaemia. Abdominal pain was dull aching, located in the periumbilical region and radiating to back. He also complained of greenish bilious vomiting for past one week, about two to three episodes per day. Patient denied history of fever, chest pain, haematemesis, loose stools and cardiac respiratory symptoms. On physical examination: conscious and oriented. Moderately, built and nourished. Mild pallor and icterus present. Vital were stable: Blood Pressure (BP)-120/80 mmHg, Pulse Rate (PR)-80 BPM. Temperature (T)°C: Normal. Per abdomen examination showed diffuse distension and tenderness over right hypochondrium, and no evidence of radiation of pain to the back. No flapping tremors. Ascites grade II was present as per USG abdomen.

During the first admission which lasted for two weeks (week 1 and week 2) diagnosis of ANP was made on basis of clinical symptomatology, laboratory and radiological findings and the patient was admitted on the same day as an inpatient in the medical gastroenterology ward for further evaluation and conservative management. Laboratory and radiological examination revealed the following: Prothrombin Time (PT) and INR was raised. Viral serology for Hepatitis B virus (HBV), Hepatitis C virus (HCV), Human Immunodeficiency Virus (HIV) negative [Table/Fig-1]. Urine routine-Pus cells seen; No organisms seen; Urine culture sterile; USG Neck: Normal. Plain Computed Tomography (CT) followed by Intravenous Contrast Spiral CT of Whole abdomen revealed radiological features suggestive of ANP with large walled off collection with head and uncinate process of pancreas showed necrosis >30% and replaced by ill-defined hypodense collection- 15×10×17 cm (Transverse (TR)×Anteroposterior (AP)×Craniocaudal (CC) extension). Modified CT severity score 10/10 as depicted in [Table/Fig-2].

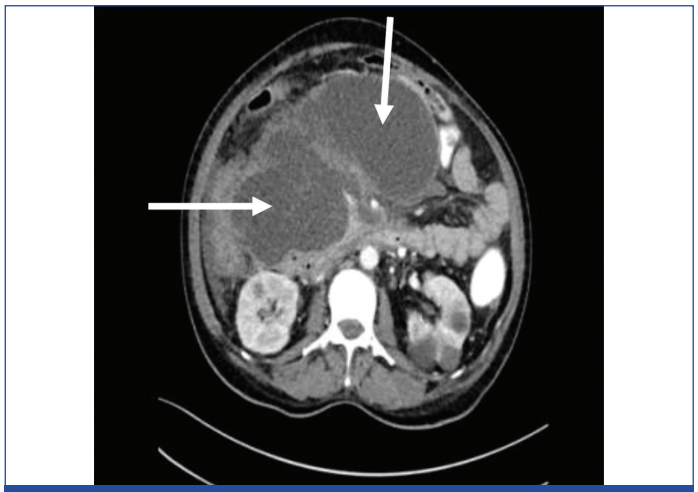
Investigation	Value obtained	Normal value
Haemoglobin (g/dL)	8.7	13-17
Total Leukocyte Count (TLC) (WBCs/10^3 cells/µL)	13.85	4-11
Platelet (10^3/mL)	611	150-350
Total bilirubin (mg/dL)	2.3	0.8-1.2
Alanine Transaminase (ALT) (IU/L)	89	0-50
Aspartate Transaminase (AST) (IU/L)	227	0-50
Alkaline Phosphatase (ALP) (IU/L)	1770	0-50
Serum Parathormone (PTH) (pg/mL)	99.7	10-65
Serum calcium (mg/dL)	8.18	9-11
Phosphorous (mg/dL)	2.06	2.8-4.5
Thyroid Stimulating Hormone (TSH) (IU/mL)	0.67	0.4-4.5
Erythrocyte Sedimentation Rate (ESR)	120	0-15
Creatinine (mg/dL)	0.61	0.8-1.2
Prothrombin time (sec)	11	11-13.5
Protein (g/dL)	6	6-8.3
Albumin (g/dL)	2.4	3.5-5.5
Serum amylase/Lipase (IU/mL)	91/45	Sr. Amylase: 30-110 Sr. Lipase: 24-151
Triglyceride (mg/dL)	242	<150
Low Density Lipoprotein (LDL) (mg/dL)	16	<100

[Table/Fig-1]: Laboratory investigations.



[Table/Fig-2]: Week 1: Contrast CT Abdomen: Large walled off collection (White arrow) with head and uncinate process of pancreas showed necrosis >30% and replaced by ill-defined hypodense collection- 15×10×17 cm (TR×AP×CC).

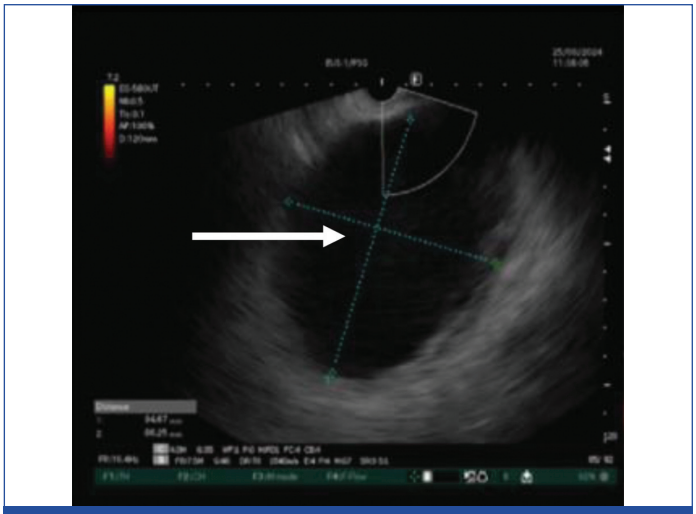
Aetiology of ANP was unknown even after the first line workup for microbial infections at the end of one week. Patient was managed empirically with Inj. ciprofloxacin 500 mg Q12H intravenously for seven days along with supportive and symptomatic therapy like enteric nutritional measures etc., (Week 2). Patient recovered well and was discharged at end of two weeks. Patient presented again with abdominal distension and poor appetite for one week and admitted for the second time for two more weeks (week 4 and week 5). Patient had diffuse abdominal pain and was hospitalised in the ward. Repeat Contrast Enhanced CT (CECT) abdomen-done revealed a significant walled-off fluid collection extending in the porta hepatis superiorly, omentum anteriorly, right anterior pararenal space inferiorly and abutting the abdominal wall anteriorly 13.7×18.6×22 cm (TR×AP×CC) in lieu of necrosis in the pancreatic head and body area as shown in [Table/Fig-3].



[Table/Fig-3]: Week 5: Repeat Contrast Enhanced CT Abdomen- CECT done revealed Walled Off Necrosis (WON) of pancreas with significant walled-off fluid collection (White arrow) extending in the porta hepatis superiorly, omentum anteriorly, right anterior pararenal space inferiorly and abutting the abdominal wall anteriorly 13.7×18.6×22 cm (TR×AP×CC) in lieu of necrosis in the pancreatic head and body area.

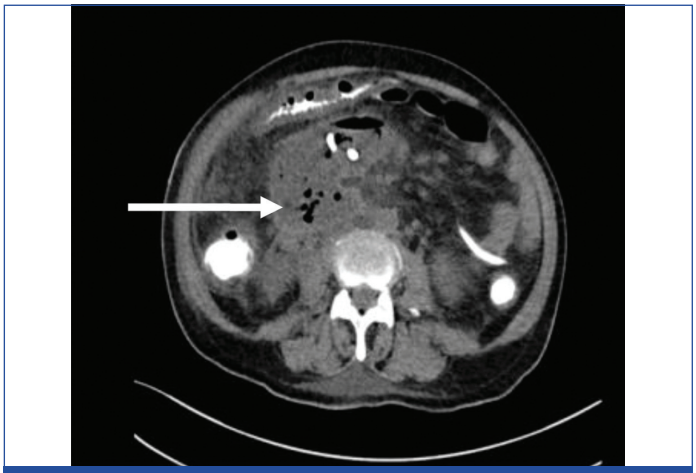
The working diagnosis of WON made and Culture negative sepsis made during second admission which is probably a sequelae of ANP. Percutaneous pig tail insertion was scheduled to empty the fluid after which Endoscopic Ultrasound (EUS) was performed (Week 5) as a diagnostic technique to evaluate peripancreatic fluid collection as depicted in [Table/Fig-4].

After EUS screening, it was decided that percutaneous drainage would be more beneficial than cystogastrostomy. Under local anaesthesia and aseptic precautions, a 12F pig tail catheter was inserted under USG guidance in the lower right quadrant of the abdomen. Serous fluid from pig tail site, blood and midstream urine specimens were sent to diagnostic microbiology for culture and sensitivity sent to rule out sepsis. Blood Culture was sterile after 48 hours and five days of aerobic incubation. Direct Gram stain revealed only pus cells. Aerobic bacterial culture from pig tail site showed no growth on aerobic incubation. Urine culture grew



[Table/Fig-4]: Week 5: Endoscopic Ultrasound (EUS) was performed as a diagnostic technique to evaluate peripancreatic fluid collection (White arrow).

Carbapenem-resistant *Escherichia coli* was reported, and was susceptible to ceftazidime-avibactam, amikacin, ciprofloxacin, fosfomycin and tigecycline. Patient was treated with Inj. ciprofloxacin 500 mg i.v. Q12H for seven days as per Hospital Antibiotic policy and culture and sensitivity results. Repeat USG done to assess collection and was found to be reduced than before (3.2×4×5 cm). Patient responded well with standard care namely supportive care and administration of antibiotics given as per antibiotic policy and/or Culture and sensitivity report and was discharged with catheter in-situ on (Week 6). The patient presented to Emergency Room (ER) once again during week eight with complaints of pain abdomen, decreased drainage output and low BP for one day. Patient was continued on Inj. ciprofloxacin. CT guided drainage tube placement: Upsizing to 14F and reposition done). After the patient's symptoms gradually got better, the pigtail catheter was left in place and they were released. Patient was admitted for the fourth time during week 9 as he had decreased drain flow, vomiting, fever and soreness at the catheter insertion site. He was admitted to the hospital once more in hypotensive shock, for which the normal procedure called for treating the patient with hypovolemic shock. Repeat CT Imaging (Week 9) revealed necrotic collection in head and body region, with multiple air pockets (6×11×13 cm), encased distal common bile duct as illustrated in [Table/Fig-5].

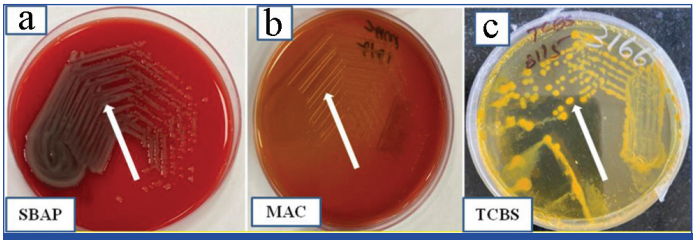


[Table/Fig-5]: Week 9: Repeat CT Imaging revealed necrotic collection of 6×11×13 cm in the head and body region of Pancreas with multiple air pockets (White arrow).

Under sterile aseptic precautions, the drain fluid was aspirated was transported to a diagnostic microbiology laboratory for aerobic culture and sensitivity testing using normal procedures. *Klebsiella pneumoniae* and *Vibrio fluvialis* NDM Carbapenemase producer were isolated by conventional culture and both the isolates were found to be resistant to carbapenems. The same microorganisms were grown in repeat drain samples submitted the following day, and the patient received

Inj. levofloxacin 500 mg i.v. Q24H and Inj. doxycycline 100 mg i.v. Q12H in accordance with the culture and sensitivity report.

Microbiological investigations: Drain fluid macroscopy was serous and odourless. Gram stain microscopy revealed moderate pus cells (++) and many curved Gram-negative bacilli ≈1×1.5-2 µm×0.5 µm size. Colony morphology on SBAP and MAC plate revealed two types of colonies were grown in culture after 24 hours of aerobic incubation. Colony 1: Sheep Blood agar: Non haemolytic gray moist colonies and MAC showed lactose fermenting colonies-oxidase negative, catalase positive, Non motile. Colony 2: Sheep Blood Agar: Beta haemolytic grey moist colonies and MAC: Non lactose fermenting colonies. Both oxidase and catalase positive. Colonies on MAC was colourless non lactose fermenter. Hanging drop preparation for fresh motility showed Darting type of motility. Colonies were inoculated in Thiosulphate Citrate Bile Salt Sucrose (TCBS) agar which grew yellow-coloured colonies due to sucrose fermentation colony morphology in routine and special media is depicted in [Table/Fig-6].

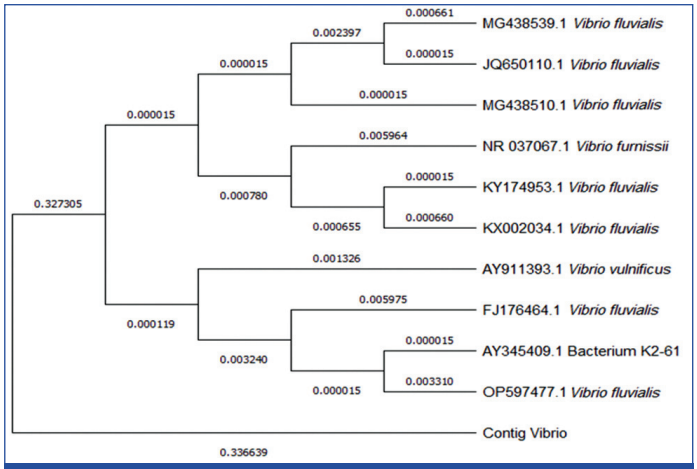


[Table/Fig-6]: Colony morphology of *Vibrio fluvialis* on routine culture media: a) Sheep Blood agar: Gray haemolytic colonies; b) MacConkey agar: Non lactose fermenting colonies and selective media; c) Thiosulfate citrate bile salt sucrose agar: Yellow colonies.

String test with 0.5% sodium deoxycholate was negative. Cholera red reaction was negative. Indole test negative, Citrate utilised, Urease negative, Mannitol Fermented and motile, Triple sugar Iron test showed K/A with no gas and no H_2S, Phenyl pyruvic acid test negative, glucose fermented, lactose not fermented, Sucrose fermented, Arginine dihydrolased, Lysine and ornithine were Not decarboxylated- Control satisfactory and Voges Proskauer test negative as shown in [Table/Fig-7].

[Table/Fig-7]: Biochemical reactions for *Vibrio fluvialis*. Indole test negative, Citrate utilised, Urease negative, Mannitol fermented and motile, Triple sugar Iron test showed K/A with no gas and no H_2S, Phenyl pyruvic acid test negative, Glucose fermented, Lactose not fermented, Sucrose fermented, Arginine dihydrolased (+), Lysine and ornithine were not decarboxylated (-) and Control satisfactory.

No agglutination with poly 'O' antisera specific for *Vibrio cholerae* and controls satisfactory. This non Cholera Vibrios (NCV) species isolated was further subjected to automated identification. (MALDI-TOF MS: MALDI Sirius One Biotyper, Bruker Daltonics, Germany) identified *V. fluvialis* with the Score Value of 2.14. VITEK-2 compact ID and AST system (bioMérieux, Marcy l'Etoile, France) was identified it as *V. fluvialis* with 98% excellent identification by using N405 card. Automated identification and antibiotic susceptibility were done by bioMérieux VITEK 2 compact system which detected carbapenem-resistant *Klebsiella pneumoniae* by using N405 card. And 16S-23S intergenic spacer region amplification was done by conventional PCR in the Molecular Biology laboratory and the amplicons were sent for Sanger's sequencing done at Barcode Genetics, Bangalore. The identification was confirmed as *Vibrio fluvialis*, a NCV as illustrated in [Table/Fig-8].



[Table/Fig-8]: Phylogenetic tree: Hierarchical clustering of *Vibrio fluvialis* generated using unweighted pair grouping with mathematical averaging method.

Antimicrobial susceptibility testing done on Mueller Hinton agar plate by Kirby Bauer disc diffusion method. Disc diffusion and MIC breakpoint both were interpreted according to Clinical and Laboratory Standards Institute (CLSI) M45 guidelines 3rd Edition, October 2015 [1]. Susceptibility of *V. fluvialis* was done by Vitek-2 and was found to be susceptible to amikacin, gentamicin, ciprofloxacin and levofloxacin. Resistant to ceftazidime, cefepime, imipenem, cefoperazone-sulbactam. *Klebsiella pnemoniae* was found to be susceptible to gentamicin, doxycycline, tobramycin and netilmycin, resistant to ceftazidime, cefepime, ciprofloxacin, imipenem, and cefoperazone-sulbactam. Quality control with *Escherichia coli* ATCC 25922 was satisfactory. Both these isolates were subjected to Specific Carbapenemase Beta lactamase (bla) screening using Phenotypic Carbapenemase rapid card (RESIST- TRURAPID O.K.N.V.I) test. *Vibrio fluvialis* showed bla_{NDM} carbapenemase positive and KPC and OXA 48 were negative and *Klebsiella pneumoniae* as showed negative to NDM, KPC and OXA 48 carbapenemases enzymes, provided control satisfactory.

DISCUSSION

Vibrio fluvialis is a Non Agglutinating (NAG) halophilic *Vibrio* which is an oxidase-positive, curved Gram-negative bacilli typically found in seafood and coastal water [2]. In clinical settings, *V. fluvialis* is implicated in wound infections leading to complications like septicaemia in immune-compromised patients. Unusual case presentations of *V. fluvialis* are reported rarely causing diverse clinical syndromes like gastroenteritis, diarrhoea, bacteraemia, suppurative cholangitis, peritonitis, otitis, endolphthalmitis and urinary tract infections both in immunocompetent and immunosuppressed individuals [3]. Unusual case of *V. fluvialis* bacteraemia associated with severe watery diarrhoea was reported by Lai CH and one more case of *V. fluvialis* infection reported in haemorrhagic cellulitis and cerebritis have been documented thus far by Huang KC and Hsu RW [4,5]. Toxigenic strain of *V. cholerae*, *V. vulnificus* and *V. parahaemolyticus* are well-known pathogens of diarrhoea/cholera and extraintestinal infections respectively. *V. fluvialis* is considered as an emerging pathogen as it has been implicated in surge of outbreaks of diarrhoea and sporadic extraintestinal infections [6]. *V. fluvialis* is easily cultivable with routine methods and there is a chance of misdiagnosis as *V. cholerae* because of its phenotypic similarity with *V. cholerae* and *Aeromonas* species. Genotypic tools like PCR and Sequencing methods, Multi Locus Sequence Typing (MLST) are of robust importance for molecular confirmation and could serve as an epidemiological tool [7].

The virulence factors, toxin production, pathogenicity and diverse clinical presentations are less studied until now and to be evaluated in detail [2]. This is the first case report of *V. fluvialis* infection in an Indian patient with ANP and its sequelae WON. This is an unusual presentation, rare occurrence and identification of a NCV species

namely *V. fluvialis* from pig tail tube drain collection sent for aerobic bacterial culture. Subsequently one more specimen of drain fluid sent for culture and sensitivity on the next day also grew *V. fluvialis*. This isolate was distinguished from *Aeromonas* spp. by its growth in the presence of 6.5% salt. Yellow colonies on TCBS agar and darting motility misguided us and mimicked us as *V. cholerae*. But automated identification like MALDITOF MS and Vitek-2 helped us to phenotypically identify this clinical isolate as *V. cholerae*. Further 16S-23S rRNA PCR followed by sequencing helped us to confirm the isolate as *V. fluvialis* stressing the need to use sophisticated molecular tools to confirm the same and establish the phylogenetic tree which would in turn serve as epidemiological tool. The key virulence factors linked to *V. fluvialis* infections include cytolysin, heat-labile cytotoxin, haemolysin, mucinase, cell adhesion, cell vacuolation, lipase and protease [8]. Although microbial contamination of water remains the largest and most immediate health hazard, infection rates coupled with mortality rates are highest in areas with low or inadequate general standards of living, water supplies and sanitary conditions. *V. fluvialis* infections are transmitted by person-to-person contact, intake of raw or contaminated marine items, high levels of faecal contamination in water resources and food sources. Discharges of treated or untreated municipal wastewater into receiving waterways at a particular point-source may be the source of infection [9]. As a part of infection control and prevention, environmental surveillance of hospital environment and water analysis especially in Gastroenterology ward was done. Culture was sterile and the results were found to be satisfactory. No other patient in the same ward or other locations of the hospital was found to have the same microbial pathogen isolated. Travel history to endemic regions and consumption of raw municipal water resistant to chlorination could possibly be the source for the same.

Antimicrobial Resistance (AMR) is more common in *V. fluvialis* than other species of *Vibrio*. In present study, Carbapenemase producing *V. fluvialis* was found to be bla_{NDM} which is one of the important mechanism of Carbapenem resistance in this patient. This isolate was susceptible to levofloxacin and doxycycline. Similar susceptibility pattern was reported in a study by Chowdhury G et al., [10]. *Vibrio* species including *V. fluvialis* isolated from in Mediterranean fish farms were found to be resistant to the following antimicrobials like co-trimoxazole, ampicillin, carbenicillin, kanamycin and cefalothin [11]. The isoforms and transconjugants of *V. fluvialis* generally have the ability to easily acquire genes for antibiotic resistance via mobile genetic elements and by identical plasmids [12]. In a study published in 2011 blaNDM-1 positive *Vibrio fluvialis* was first reported in Kolkata. From 2011 to 2013, between 25% and 50% of *V. fluvialis* isolates had the NDM gene [13]. Neverthless, it is noteworthy that the patient had not been previously treated with carbapenems. According to published literature, *V. fluvialis* isolates carrying NDM respond well to azithromycin and doxycycline. Levofloxacin and doxycycline combination therapy proved to be an effective treatment for this mixed infection and the patient condition improved on close follow-up and was discharged [14]. Published literature review about patients who had *V. fluvialis* infection and salient features depicted in [Table/Fig-9] to compare diagnosis, treatment and recovery [4,5,15-22]. Step-up approach in the form of optimal use of culture and sensitivity and treatment with appropriate

Case	Author, publication year	Age/Sex	Country	Site of disease	Bacteraemia	Underlying disease	Route of acquisition	Treatment	Outcome
1	Chen PJ et al., [15], 2012	40 Y/ Female	Taiwan	Otitis externa	No	None	Seawater exposure	AMPC/CVA	Survived
2	Liu WL et al., [16], 2011	88 Y/ Female	Taiwan	Cholangitis	No	DM, ESRD, cirrhosis, intrahepatic ductal stones	None	PIPC/TAZ+transhepatic biliary drainage	Survived
3	Lee JY et al., [17], 2008	52 Y/ Female	South Korea	Peritonitis	No	Post abdominal blunt injury from traffic accident	None	Cephamycin+netilmycin, PIPC/TAZ+MNZ, cefoperazone+prepenem+ amikacin	Died
4	Koh EM et al., [18], 2007	70 Y/ Male	Korea	Gastroenteritis, gastric cancer	Yes	DM, hypertension	NA	NA SBT/CPZ+ISP	Survived
5	Lai CH et al., [4] 2006	65 Y/ Male	Taiwan	Gastroenteritis	Yes	DM, chronic liver disease	None	CXM to ST	Survived
6	Ratnaraja N et al., [19], 2005	55 Y/ Female	New Zealand	Peritonitis	No	ESRD (on CAPD), DM	Seafood	CTRX CPFX AMPC+GM (ip)	Survived
7	Huang KC and Hsu RW [5], 2005	45 Y/ Male	Taiwan	Haemorrhagic cellulitis cerebritis	Yes	Alcoholic liver disease	Fire ant bites, brackish water exposure	OXA+GM CAZ+oxytetracycline+fasciotomy+left transfemoral amputation	Died
8	Albert MJ et al., [20], 1991	5 months/ Male	Bangladesh	Gastroenteritis	Yes	Malnutrition	NA	ABPC+GM+amdinocillin pivoxil	Died
9	Usta J et al., [21], 2018	52 Y/ Female	Lebanon	UTI	No	Fibroid uterus, post-total abdominal hysterectomy and bilateral oophorectomy	Home tap water	CPFX	Survived
10	Kitaura S et al., [22], 2020	65 Y/ Male	Japan	Liver abscess	Yes	Malnutrition, pancreatic cancer, bladder cancer (surgically resected)	Sea food	PIP/TAZ+MINO, CLDM+MINO, biliary stent placement, PTAD	Survived
Patient characteristics reported in this case study									
11.	Present study, 2024	65 Y/ Male	India	Walled Off Necrosis (WON) - Sequeae of Acute Necrotising Pancreatitis (ANP)	No	Dyslipidaemia, hypertension	-	Levofloxacin (*Vibrio fluvialis* blaNDM)+Doxycycline (*Klebsiella pneumonia* Carbapenemase)	Survived

[Table/Fig-9]: *Vibrio fluvialis* infection and globally reported patient characteristics depicting Diagnosis, treatment and recovery [4,5,15-22].
Y: Years; ABPC: Ampicillin; AMPC: Amoxicillin; AMPC/CVA: Amoxicillin/clavulanate; CAPD: Continuous ambulatory peritoneal dialysis; CAZ: Ceftazidime; CLDM: Clindamycin; CPFX: Ciprofloxacin; CTRX: Ceftriaxone; CXM: Cefuroxime; DM: Diabetes mellitus; ESRD: End-stage renal disease; GM: Gentamicin; HD: Haemodialysis; ip: Intraperitoneal; ISP: Isepamycin; MINO: Minocycline; NA: Not available; OXA: Oxacillin; PIPC/TAZ: Piperacillin/tazobactam; PTAD: Percutaneous transhepatic abscess drainage; SBT/CPZ: Sulbactam-cefoperazone; ST: Trimethoprim-sulfamethoxazole; blaNDM: betalactamase New Delhi Metallobetalactamase

antibiotics is very crucial especially when patient is not responding to standard therapy. Source control in WON is primary, in addition to optimisation of General Nutritional Measures.

CONCLUSION(S)

V. fluvialis is an emerging pathogen implicated in diverse clinical infections. High degree of suspicion, use of automated systems and molecular detection methods would facilitate the differentiation of *V. fluvialis* from *V. cholera*e or *Aeromonas* species. Appropriate diagnostic stewardship and antimicrobial stewardship practices in case of *V. fluvialis* would aid in diagnosis and rational therapy with appropriate antibiotics for a good clinical outcome.

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