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Severe Acute Pancreatitis Complicated by Delirium Tremens and Ventricular Tachycardia: A Case Report

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Abstract

Acute pancreatitis is an acute inflammatory process of the pancreas that can initiate a systemic inflammatory response syndrome resulting in acute respiratory distress syndrome and multi-organ dysfunction. Alcohol is one of the common etiology of acute pancreatitis. Sudden cessation of alcohol consumption can result in acute alcohol withdrawal, and a few subsets of these patients can develop severe forms of withdrawal like delirium tremens. Cardiac arrhythmias like ventricular tachycardia, further increase risk of mortality in these patients. Severe acute pancreatitis, delirium tremens, and ventricular tachycardia manifesting all together in a single patient are rare and poses therapeutic challenge.

Case Description: *Here, we report a 36-year-old male who presented to the non-cardiac centre, a case of severe acute pancreatitis (alcohol-related) who developed delirium tremens on day 4 of admission and Ventricular tachycardia on day 5. He was managed with benzodiazepines, amiodarone infusion and synchronised cardioversion. Sinus rhythm was achieved after 6 hours of amiodarone infusion. Amiodarone infusion was continued for 48 hours, followed by maintenance doses. He again had Sustained Ventricular tachycardia. Restarted on amiodarone infusion along with lignocaine infusion. Rhythm was restored to Sinus rhythm after 03 hours. Benzodiazepines were discontinued on day 16 of admission after gradual tapering. His course in the hospital was complicated by respiratory distress requiring intubation, increased peripancreatic collection, which was managed with pigtail drainage. Pigtail was removed after 4 weeks of drainage. He was discharged after 12 weeks of in hospital management. Conclusion: This case highlights the importance of an aggressive approach in the management of ventricular tachycardia, as a severe complication of delirium tremens. Clinicians should maintain a high index of suspicion for cardiac complications in patients with severe acute pancreatitis and alcohol withdrawal, as timely diagnosis and management can be lifesaving.*

Keywords: Severe Acute Pancreatitis, Delirium Tremens, Ventricular Tachycardia and Pigtail Drainage

Key Clinical Message

- Multisystem complications can occur in severe acute pancreatitis, especially in patients with a history of chronic consumption of alcohol, posing a therapeutic challenge and increased morbidity.
- Alcohol withdrawal can result in autonomic dysfunction, resulting in life-threatening arrhythmias.
- Early recognition and aggressive management of cardiac arrhythmias in Delirium tremens is crucial for improved survival and requires a multidisciplinary approach.

1. Introduction

Acute pancreatitis (AP) is an acute inflammatory process of the pancreas that can initiate a systemic inflammatory

response syndrome (SIRS), resulting in acute respiratory distress syndrome (ARDS) and multi-organ dysfunction. The most common etiologies include alcohol, gallbladder stones, hypertriglyceridemia, endoscopic retrograde cholangiopancreatography, drugs, trauma, and post-operative. Uncommon etiologies include hypercalcemia, infections, cystic fibrosis, autoimmune, and hereditary causes. Delirium Tremens is the severe form of alcohol withdrawal. Ventricular Tachycardia is a known complication of delirium tremens. However, severe acute pancreatitis, delirium tremens, and ventricular tachycardia manifesting all together in a single patient is a rare scenario.

1.1. Case Description

A 36-year-old male presented on 30 Dec 2024 with a history of pain over the left hypochondrium and epigastric region of 01-day duration, which improved on bending forward and was associated with 3-4 episodes of vomiting and obstipation. He gave a history of consumption of alcohol of around 60-80 grams of alcohol/day for the last 5-6 years, and the last consumption of alcohol was 01 day before the onset of the pain. At the time of presentation, he gave no history of fever, breathlessness, tremors, or palpitations.

On admission, his vitals included a pulse of 88 per minute, blood pressure of 100/70 mm of Hg, temperature of 98.4o F, respiratory rate of 18 per minute, and oxygen saturation of 98% at room air. No pallor, icterus, clubbing, lymphadenopathy, edema, or resting tremors were observed. Tenderness was observed over the left hypochondrium; the rest of the general and systemic examination was normal.

Investigation revealed hemoglobin of 18.1 grams/dl, hematocrit of 51.4, total leucocyte counts of 9060, raised GGT (275 U/L), amylase (257 U/L), and lipase (848 U/L). HbsAg, anti-HCV, and HIV were negative. He underwent a CT abdomen, the findings of which were suggestive of acute pancreatitis, moderate ascites, and no evidence of pleural effusion. He was initially managed conservatively as a case of acute pancreatitis (B0I0S0A0P0) with IV fluids, proton pump inhibitors, prokinetics, and other supportive care.

On day 3 of admission, he became febrile and started desaturating (saturation of 90% at room air). Chest X-ray was suggestive of bilateral pleural effusion, ABG- pH of 7.4, pCO2 of 30, HCO3- of 20, PO2 of 62, P/F ratio of 295 (suggestive of mild ARDS). The following day, he became disoriented, had agitated behaviour and body tremors, was profusely sweating, had sinus tachycardia and disturbance in attention and sleep. ECG was suggestive of sinus tachycardia (figure 1).

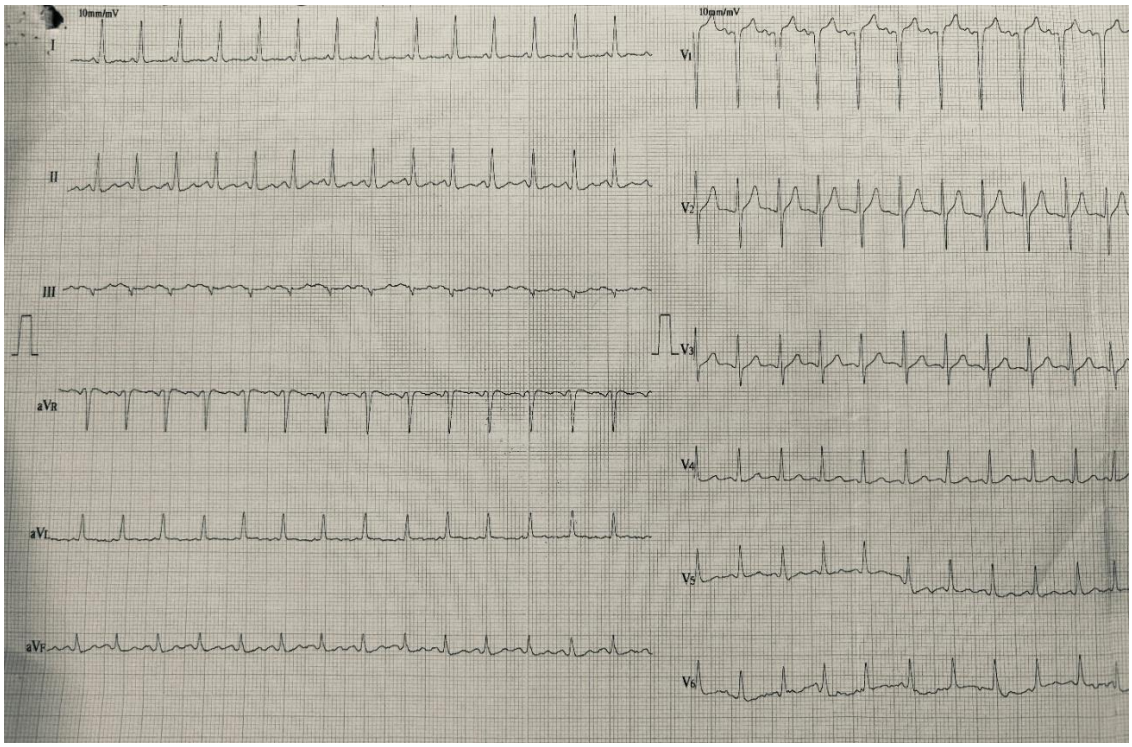

Figure 1: ECG at The Time of Delirium Tremens: Sinus Tachycardia with Hr Of 145 Per Minute

He was diagnosed as a case of Delirium tremens, shifted to the ICU, and was started on benzodiazepines, haloperidol, and promethazine. Doses of lorazepam were adjusted to maintain the state of somnolence.

On day 5 of admission, at around 2230, he was noticed to have a change in the morphology of tachycardia on the cardiac monitor. Urgent ECG (figure 2)

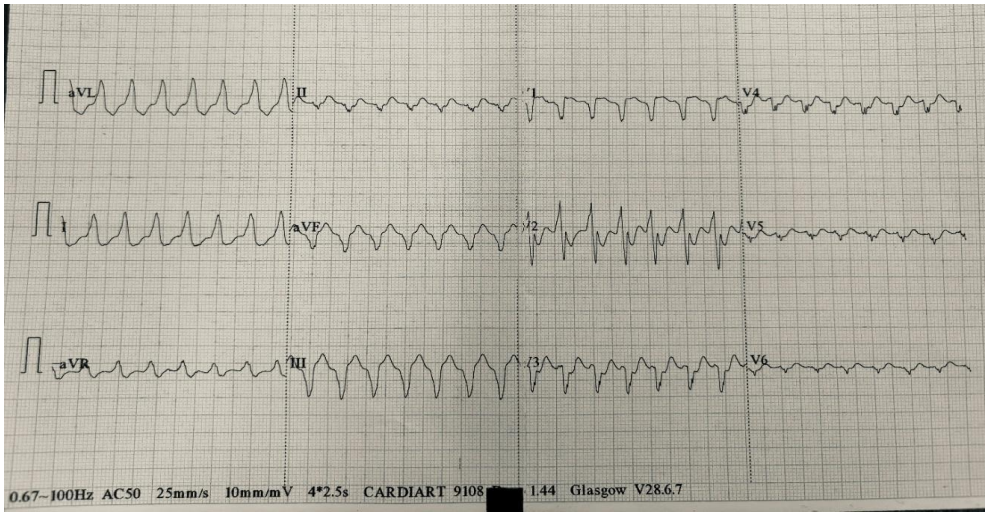

Figure 2: Ventricular Tachycardia With HR of 175 Per Minute

was done, which showed Ventricular tachycardia (Heart rate- 175/minute, QRS – 140 milliseconds, with duration of RS more than 100 milliseconds, QTc- 506 milliseconds). Pulse was palpable on examination. Corrected calcium was 7.6 mg/dl; the rest of the electrolytes were normal. He was initially managed with a calcium infusion and an amiodarone bolus of 150 mg. Post amiodarone bolus, the patient developed hypotension and continued to have ventricular tachycardia; an attempt was made to cardiovert the patient electrically. Considering sustained ventricular tachycardia, he was started on amiodarone infusion once the mean arterial pressure of more than 65 mm of mercury was achieved with vasopressors. Restoration of normal sinus rhythm was achieved after 06 hours of amiodarone infusion. He was continued on amiodarone infusion for 48 hours. Post discontinuation of amiodarone infusion, he was managed with an oral maintenance dose of amiodarone. He again had ventricular tachycardia. Amiodarone infusion was restarted. Considering the refractory nature of Ventricular tachycardia to amiodarone, lignocaine infusion (2 mg/minute was started after bolus dosing of 75 mg and 50 mg). Sinus rhythm was achieved after 3 hours of infusion.

On day 8 of admission, he was intubated, considering increased respiratory distress with respiratory acidosis (ABG – pH of 7.24, pCO^2 of 61, HCO^3- 25.5). He continued to be febrile on day 10. Evaluation revealed raised procalcitonin (4.03 ng/mL). He was empirically started on parenteral antibiotics (imipenem/cilastatin 500 mg every 6 hours and metronidazole 500 mg every 6 hours), considering the possibility of an infected collection of the pancreas. Around day 11, sinus tachycardia started settling, with improvement in sensorium. Amiodarone and lignocaine infusion were discontinued at day 11, and patient was started on maintenance doses of amiodarone and benzodiazepines. At day 12, he was extubated and placed on NIV support. At day 16, Benzodiazepines were discontinued after gradual tapering.

He continued to be febrile in the third week of admission and had increased abdominal distension. Repeat CECT abdomen was suggestive of acute necrotising pancreatitis with moderate ascites with CTSI score of 10/10 (figures 3,4,5,6,7) [1-3].

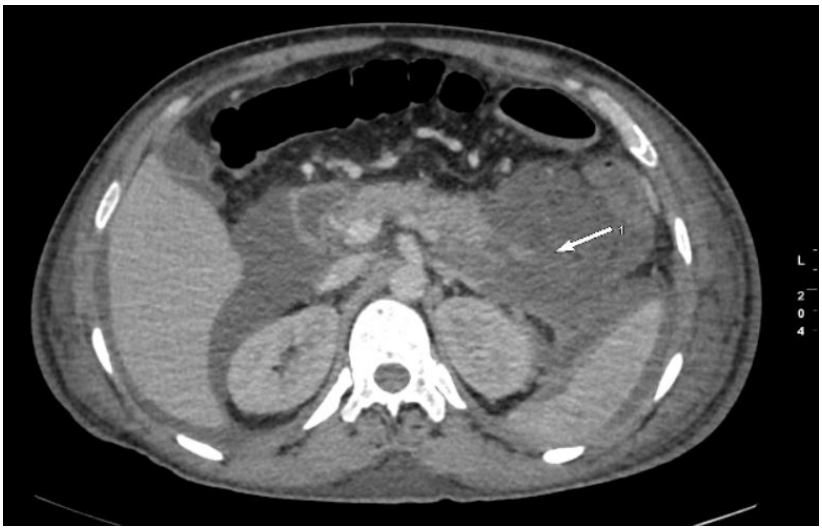

Figure 3: CECT Abdomen (Axial Section at the Level of The Pancreas) Revealing Non Enhancing Necrotic Areas in The Region of the Body & Tail of The Pancreas with Surrounding Hypodense Acute Necrotizing Collections

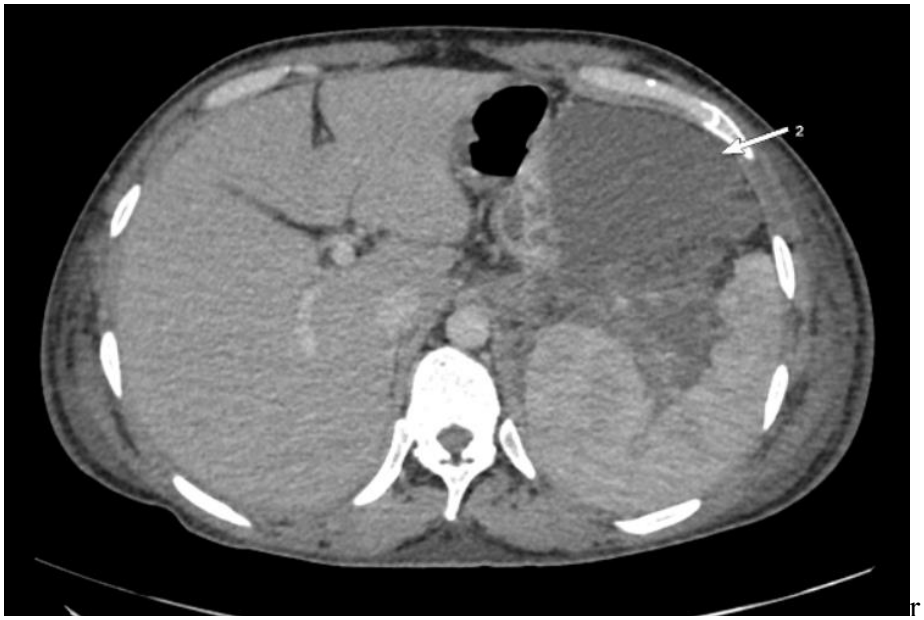

Figure 4: Acute Necrotizing Collections Seen Extending Along the Greater Curvature of The Stomach and Splenic Hilum

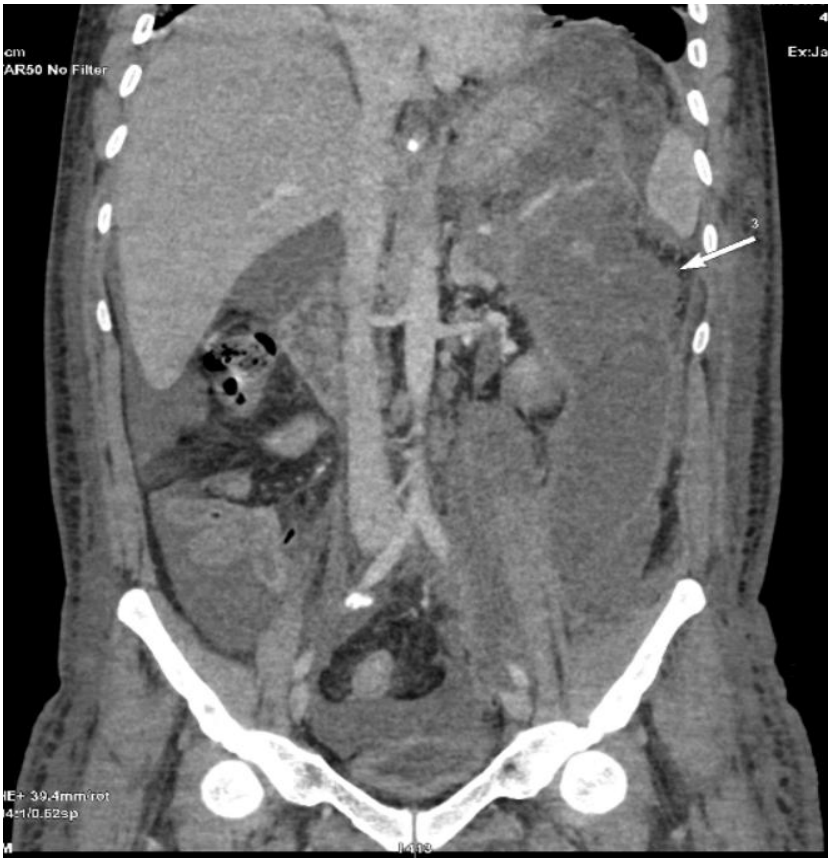

Figure 5: Coronal Section Revealing Acute Necrotizing Collections Extending Along the Left Anterior Pararenal Space and Paracolic Gutter

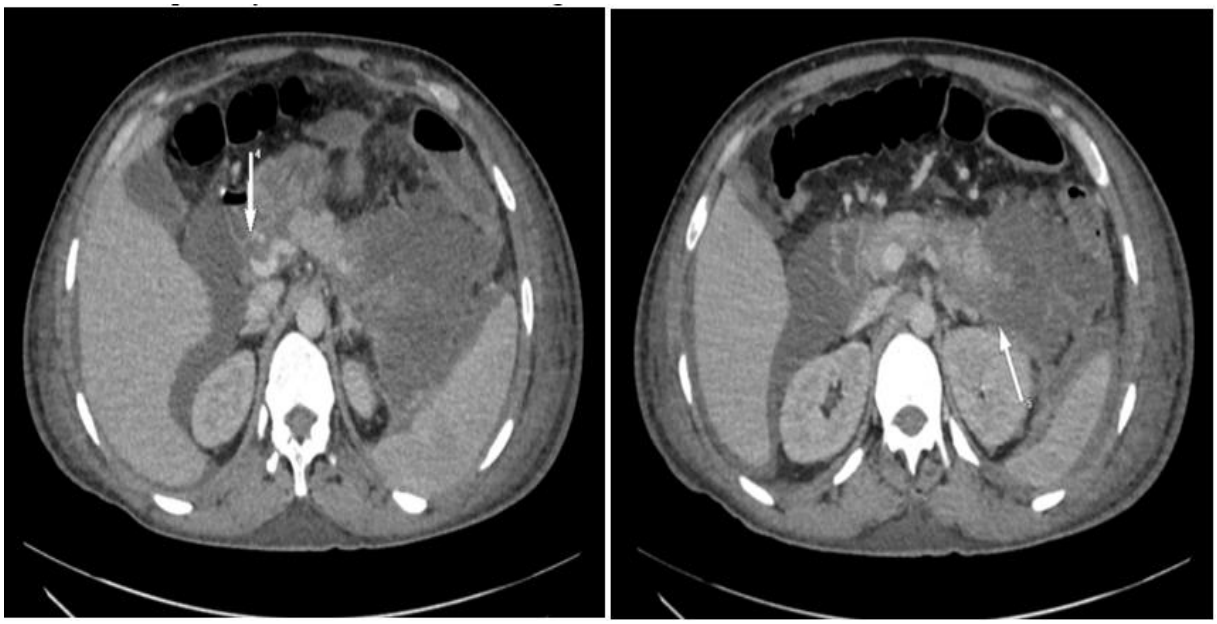

Figure 6: CECT Abdomen at the Level of the Portal Vein in the Portal Venous Phase Revealing a Hypodense Eccentric Filling Defect Within the Portal Vein, Suggestive of Partial Portal Vein Thrombosis. Splenic Vein is Not Opacified by Contrast, Suggestive of Thrombosis with Few Mesenteric Porto Systemic Collaterals Draining into the SMV

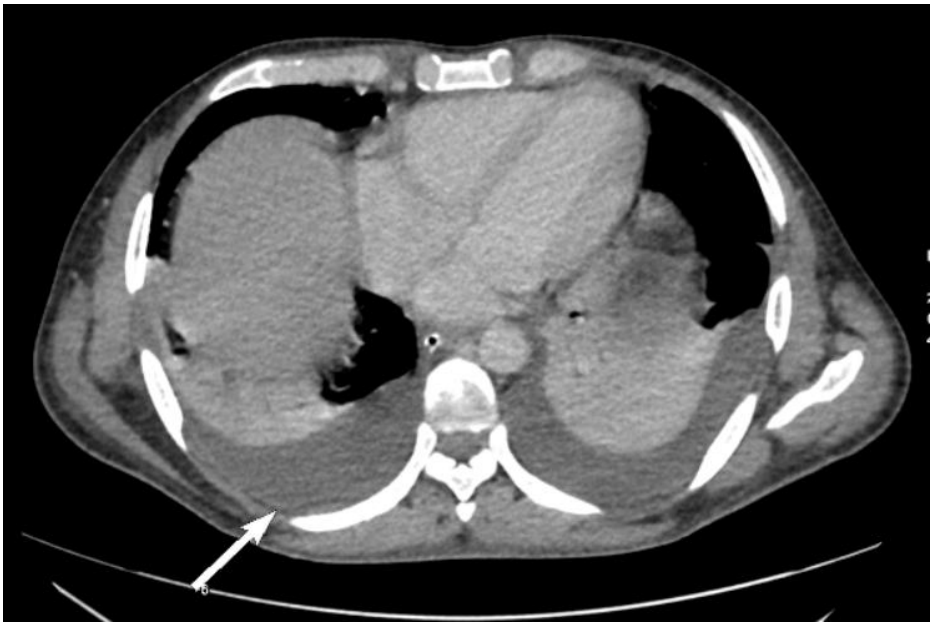

Figure 7: Axial Section at the Level of Basal Segments of Lungs Revealing Bilateral Pleural Effusion with Subjacent Collapse Consolidation

Antibiotics were changed to cefepime/tazobactam (1gram IV every 8 hours), teicoplanin (400 mg IV every 12 hours for 03 doses followed by 400 mg every 24 hours), amikacin (750 mg IV every 24 hours), and caspofungin (70 mg on day 1 followed by 50 mg every 24 hours). He continued to be febrile in the fourth week. Bedside ultrasound of the abdomen revealed an extensive thick-walled collection (8x11 cm) in the left lumbar/iliac region surrounded by bowel loops. He underwent CT-guided pigtail insertion, and 03 litres of fluid were drained (figure 8).

Figure 8: Pigtail Drainage

Serial cultures of fluid showed no growth. B-d-glucan (51.83 pg/ml, normal <70 pg/ml) and galactomannan (<0.1 ug/L, normal <0.25) were normal, so anti-fungals were discontinued. After 4 weeks of pigtail drainage, the pigtail was removed, and antibiotics were discontinued. Lab parameters have been summarised in table 1.

Lab Parameters	On admission	Day 4	Day 05	Day 08	Day 10	Day 21	08 weeks
Hb g/dl	18.1	11.8	9.7	9.5	9.2	8.2	9.0
TLC /uL	9060	4200	8300	10,080	10, 200	22,900	3,150
DLC	N-75%, L- 16 %	N- 79 % L- 10%	N- 85 % L- 07 %	N- 87 % L - 07%	N- 83 % L - 13%	N- 92 % L – 03%	N- 62% L – 16%
Platelets /uL	2,81,000	1,30,000	90,000	1,25,000	1,54,000	3,56,000	2,06,000
PT INR aPTT	14.0 1.0 34						14 1.2 36
PBS					Haemolysis not seen		
Urea ng/dl Creatinine	47 1.3	86 1.8	23 1.0	38 1.0	43 1.0	23 0.9	13 0.61
Na^+ K^+ mEq/L	135 5.0	137 5.1	142 3.6	140 4.0	150 4.8	140 4.2	135 4.1
Ca^{2+} $Po4^{3-}$ mg/dl	8.4 5.2		7.6 1.5	10.0 2.1	8.5 2.1	9.1 4.1	9.3 3.7
Bilirubin (D/I) mg/dl	1.6/0.6	1.5/0.9			1.0/0.5		
SGOT SGPT IU/L	38/46	62/45			48 36		

ALP GGT U/L	79 275						
Protein gm/dl Albumin gm/dl	5.8 3.01	5.6 2.4	5.2 2.0	4.2 1.5	4.4 2.8	5.5 2.3	
Amylase U/L Lipase U/L	257/848						
Procalcitonin					4.03	1.44	
Urine C/S			No growth			No growth	
Blood C/s					Serratia Marcescens Sensitive to Imipenem	Enterococcus Gallinarium S to Amikacin	
Tracheal C/s					Chrysobacterium Indologenes S to Septran		
ABG pH pCo2 mm Hg, $HCO3^-$ mmol/L PO2 P/F ratio		 7.4 30 20 62 295		 7.24 61 25.5			
HbsAg Anti HCV HIV		Negative Negative Negative					

Table 1: Lab Parameters

He underwent de-addiction therapy with a psychiatrist. Repeat imaging of the abdomen at 12 weeks revealed a normal-sized pancreas with heteroechoic echotexture and edematous peri-pancreatic planes, normal pancreatic duct, few periportal and para-splenic portosystemic collaterals, likely due to splenic vein thrombosis (figure 9).

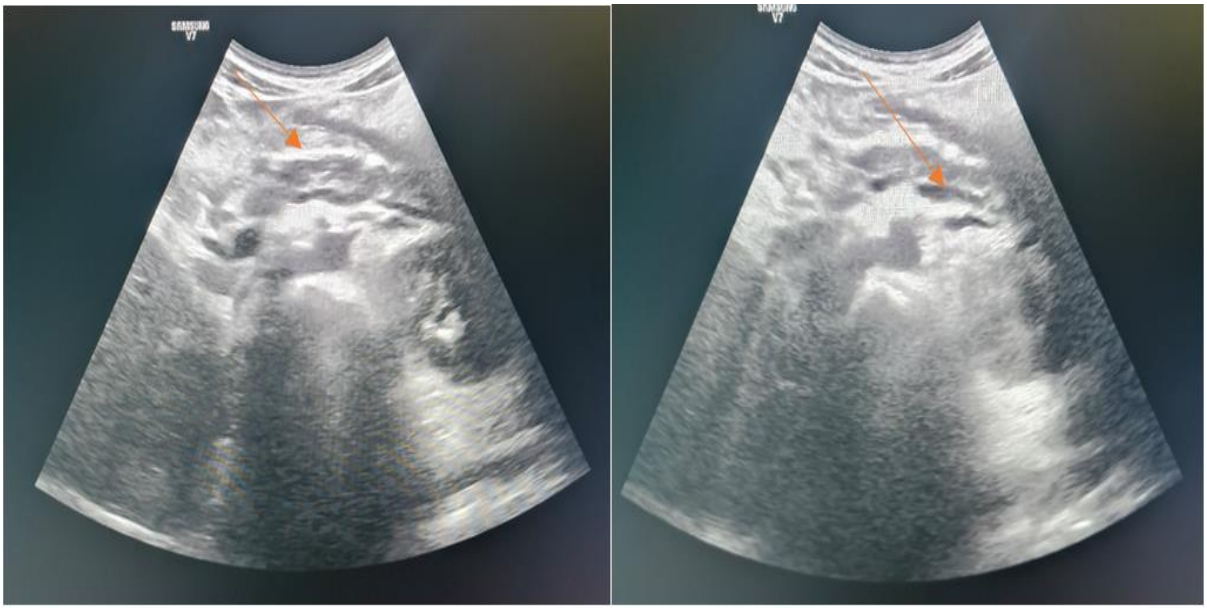

Figure 9: Follow-up Ultrasound of Abdomen: Arrow 1 Heteroechoic Echotexture of Pancreas with Mild Pancreatic Atrophy. Arrow 2 – no Significant Pancreatic/Peri-Pancreatic Collection. Echogenic Contents Seen within the Splenic Vein, Suggestive of Thrombosis

2D echocardiography before discharge revealed a normal study. He was discharged after 12 weeks of admission and is under follow-up.

2. Discussion

Delirium tremens is the severe form of alcohol withdrawal. Mortality has substantially reduced to 1 to 4% over the years, especially after the introduction of benzodiazepines. It is diagnosed based on DSM-5 criteria/ICD-10. Benzodiazepines are considered the mainstay among the treatment modalities. Adjunctive therapy includes antipsychotic drugs like haloperidol to control the agitated behaviour, thiamine supplementation, adequate hydration, correction of electrolyte imbalance, and other conservative measures. Death usually results from cardiac arrhythmias, hyperthermia, complications of withdrawal seizures, and other medical conditions [1-5].

Our case presented with features of acute pancreatitis at the time of presentation. Initially, he had no features of alcohol withdrawal and was managed conservatively. However, he was closely observed for alcohol withdrawal. On day 5, he developed features of delirium tremens and was shifted to the ICU and started on benzodiazepines. Doses of benzodiazepine were adjusted to maintain the state of somnolence. The following day, he developed ventricular tachycardia (diagnosis was based on Brugada criteria . The only electrolyte abnormality found was hypocalcaemia. He was initially managed with calcium infusion and an amiodarone bolus of 150 mg. Post amiodarone bolus, the patient developed hypotension and continued to have ventricular tachycardia. An attempt was made to cardiovert the patient electrically. Considering sustained ventricular tachycardia despite synchronised cardioversion, he was started on amiodarone infusion once the mean arterial pressure more than 65 mm of mercury was achieved with vasopressors. Normal sinus rhythm was restored after 06 hours of amiodarone infusion. He was continued on amiodarone infusion for 48 hours. Post discontinuation of amiodarone infusion, he was managed with an oral maintenance dose of amiodarone. He again had ventricular tachycardia. Amiodarone infusion was restarted. Considering the refractory nature of ventricular tachycardia to amiodarone, lignocaine infusion (2 mg/minute was started after bolus dosing of 75 mg and 50 mg. Sinus rhythm was achieved after 3 hours of infusion. His course in the hospital was complicated by respiratory distress requiring intubation, an infected pancreatic collection requiring antibiotics, and pigtail drainage. He was discharged after 12 weeks of admission [6,7].

This case highlights the importance of vigilant supervision of cardiac function, electrolytes and cognitive status in patients with alcohol related severe acute pancreatitis. This case was successfully managed in a non-cardiac centre. Early recognition and management are essential for positive outcomes in such critically ill patients. Clinicians should maintain a high index of suspicion for cardiac complications in patients with severe acute pancreatitis and alcohol withdrawal, as timely diagnosis and management can be lifesaving.

Authorship List: Inder Preet Singh Bhatia1*, Hitesh Sinduriyan2, Sanjay Singh Rawal3, Raj Narayan Mandal4

Author 1: Contributed to conceptualisation, literature search, data acquisition, data analysis and preparation, and editing of manuscript.
Author 2: Contributed to analysis, collection of investigations, supervision, and editing of manuscript.
Author 3: Contributed in conceptualisation, collection of investigations, preparation, and editing of manuscript.
Author 4: Contributed in supervision, visualisation, and editing of manuscript.

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Patient Consent: Written informed consent was obtained from the patient for publication of this case report and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

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