

Coeliac Plexus Block for Pain Management in a Case of Intractable Pain of Chronic Pancreatitis: A Case Report

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ABSTRACT

Chronic pancreatitis is a progressive inflammatory disease of the pancreas, frequently seen in middle-aged chronic alcohol consumers. It is characterised by irreversible morphological changes, such as parenchymal atrophy, ductal dilatation, and calcifications, often resulting in persistent abdominal pain. While initial management is primarily conservative with analgesics and enzyme replacement, pain in some patients remains refractory, necessitating interventional approaches. One such technique is the coeliac plexus block, which disrupts the visceral afferent pain pathways. We report a case of a 50-year-old chronic alcoholic male from a resource-limited setting who presented with intractable epigastric pain radiating to the back, severely impacting his quality of life. Imaging revealed classic features of chronic pancreatitis, including pancreatic calcifications and ductal dilatation. Laboratory investigations confirmed elevated pancreatic enzymes and inflammatory markers. Despite optimal medical management, his pain remained uncontrolled. A fluoroscopy-guided posterior para-aortic coeliac plexus block was administered using a combination of 0.25% bupivacaine and triamcinolone. The patient experienced significant and sustained pain relief, with a Numerical Rating Scale (NRS) score reduction from 8/10 to 2/10. He was discharged in a stable condition and reported improved appetite and daily functioning during follow-up. This case emphasises the utility of fluoroscopic coeliac plexus block as a viable, safe, and effective intervention in the management of chronic pancreatitis-related pain, particularly when conventional therapy fails. The report also underscores the importance of selecting an appropriate technique based on clinical scenario, resource availability, and patient suitability.

Keywords: Anaesthesia, Bupivacaine, Fluoroscopy, Steroids, Triamcinolone

CASE REPORT

A 50-year-old male from a third-tier city in India presented to the medicine outpatient department with complaints of chronic dull aching epigastric pain radiating to the back for the past one year. The pain worsened after meals and was partially relieved with over-the-counter analgesics. It significantly impaired his daily activities, with a NRS pain score of 8/10 on arrival. The patient had been a chronic alcoholic for the past 20 years, consuming approximately 180–240 mL of country-made liquor daily. He reported multiple hospitalisations for episodes of acute pancreatitis over the last five years.

Previous treatment included pancreatic enzyme supplementation, proton pump inhibitors, opioids, and lifestyle modifications, but symptoms remained refractory. He had no history of gallstones, hypertriglyceridaemia, trauma, or autoimmune conditions. On abdominal examination, deep tenderness was noted in the epigastric region without palpable mass or guarding.

Laboratory investigations revealed elevated serum amylase (701 U/L) and lipase (692 U/L), leukocytosis (WBC: 18,100 cells/ μ L), and mildly elevated bilirubin (2.8 mg/dL) and alkaline phosphatase (265 IU/L), consistent with active inflammation [Table/Fig-1]. Haemoglobin was low (8.3 g/dL), suggesting anaemia of chronic disease. Liver transaminases, blood glucose, and Glycosylated Haemoglobin (HbA1c) were within normal limits. Contrast-enhanced Computed Tomography (CT) of the abdomen demonstrated an atrophic pancreas with parenchymal calcifications and a dilated pancreatic duct, confirming chronic pancreatitis [Table/Fig-2].

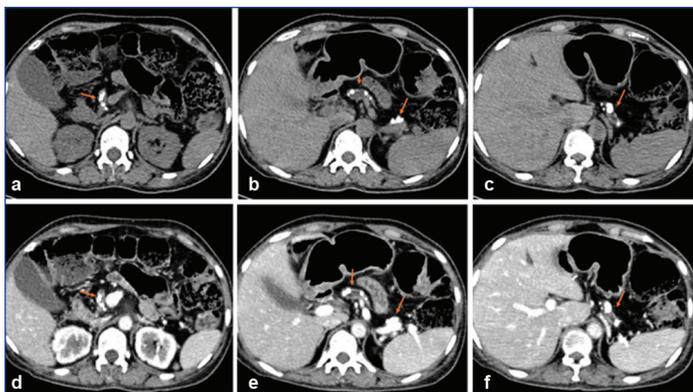
Due to poor response to medical management, a decision was made to perform a fluoroscopy-guided coeliac plexus block. After explaining the procedure, informed written consent was obtained. The patient was placed prone on a fluoroscopy table. Using a posterior para-aortic approach, a spinal needle was inserted

Investigation	Normal range	Result
Blood tests		
Serum amylase (U/L)	30-110	701
Serum lipase (U/L)	0-160	692
Complete Blood Count (CBC)		
Haemoglobin (g/dL)	M: 13.8-17.2 F: 12.1-15.1	8.3
White blood cell count (cells/ μ L)	4,500-11,000	18,100
Liver Function Tests (LFTs)		
Bilirubin (mg/dL)	0.1-1.2	2.8
Alkaline phosphatase (IU/L)	44-147	265
Alanine aminotransferase (ALT) (IU/L)	7-56	21
Aspartate aminotransferase (AST) (IU/L)	10-40	30
Blood glucose levels		
Fasting blood glucose (mg/dL)	70-100	89
HbA1c	<5.7%	5.3%
Random blood glucose (mg/dL)	>125	129

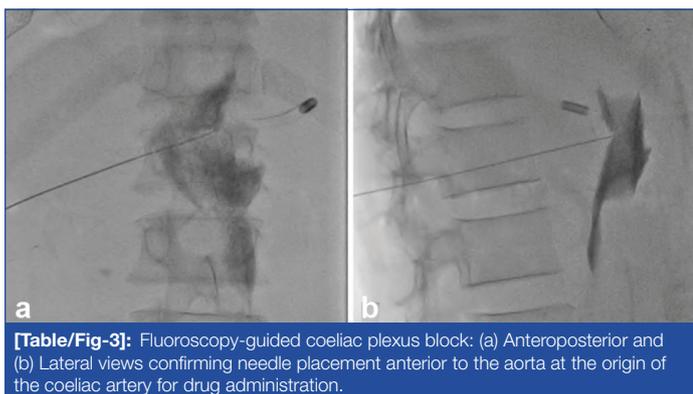
[Table/Fig-1]: Laboratory parameters of the patient showing elevated serum amylase and lipase levels, leukocytosis, and abnormal liver function tests, consistent with chronic pancreatitis.

bilaterally at the level of L1, just anterior to the aorta near the origin of the coeliac artery. Proper positioning in the coeliac region was confirmed with contrast injection.

A total of 10 mL of 0.25% bupivacaine mixed with 40 mg (1 mL) of triamcinolone acetate, diluted with 3 mL of non-ionic contrast medium, was injected under fluoroscopic guidance [Table/Fig-3]. No immediate complications occurred. Post-procedure, the patient reported significant relief, with an NRS score of 2/10, was able to tolerate oral feeds, and showed marked improvement in appetite and activity.



[Table/Fig-2]: Contrast-enhanced Computed Tomography (CECT) axial images of the abdomen: (a-c) Non-contrast axial sections; (d-f) Portal venous phase images showing atrophic pancreatic parenchyma, dilated main pancreatic duct, and coarse parenchymal calcifications (orange arrows), suggestive of chronic pancreatitis.



[Table/Fig-3]: Fluoroscopy-guided coeliac plexus block: (a) Anteroposterior and (b) Lateral views confirming needle placement anterior to the aorta at the origin of the coeliac artery for drug administration.

He was observed for 24 hours and discharged on the second day with instructions for outpatient follow-up. At 15-day follow-up, the patient reported sustained pain relief and improved quality of life, with no recurrence of severe pain.

DISCUSSION

Chronic pancreatitis is a long-standing inflammatory condition that results in irreversible damage to the pancreatic parenchyma, often leading to persistent abdominal pain. This pain, particularly when resistant to standard analgesic regimens, significantly affects a patient's quality of life and may necessitate interventional pain management strategies. Among these, the Coeliac Plexus Block (CPB) has emerged as an effective minimally invasive procedure aimed at interrupting visceral pain transmission pathways [1,2].

The pancreas, a retroperitoneal organ with both endocrine and exocrine functions, is highly susceptible to injury from recurrent inflammation. Alcohol-induced pancreatitis is one of the most common causes in middle-aged males, with chronic alcohol use contributing to progressive enzymatic damage, glandular atrophy, ductal dilatation, and calcifications over time [3]. On imaging, chronic pancreatitis characteristically presents with a shrunken, calcified pancreas and a dilated main pancreatic duct. While conservative management remains the first-line approach, surgical intervention is usually reserved for complications like pseudocysts or abscesses [4,5].

In cases where medical treatment fails, CPB offers an excellent alternative for pain relief. The coeliac plexus, also known as the solar plexus, is a complex network of interconnected ganglia and autonomic fibres located around the coeliac artery and the superior mesenteric artery at the level of T12 to L1 vertebrae [6,7]. It provides sympathetic and parasympathetic innervation to most upper abdominal viscera, including the pancreas, liver, spleen, kidneys, small intestine, and proximal large intestine [8,9]. Interrupting these pain-transmitting pathways via CPB reduces visceral nociception and may enhance functional status.

The coeliac ganglia serve as major relay centres for visceral afferent signals. Along with the aortico-renal and superior mesenteric ganglia, they contribute to the formation of the broader coeliac plexus. This intricate network plays a key role in mediating visceral pain, and its disruption forms the basis of CPB in chronic pancreatitis and other benign or malignant intra-abdominal conditions [9].

The block can be administered through anterior or posterior para-aortic approaches using imaging guidance. In our case, a fluoroscopy-guided posterior para-aortic approach was selected due to its wider availability and familiarity in resource-constrained settings. While Endoscopic Ultrasound (EUS)-guided techniques allow real-time imaging and may avoid radiation exposure, they are often limited by the need for specialised equipment and trained personnel [10].

The choice of technique must be individualised based on clinical context, available expertise, and patient-specific anatomy. Complications of CPB are rare but must be considered, including hypotension, transient diarrhea, bleeding, infection, paresthesia, and inadvertent vascular injection [11,12]. When performed carefully, especially in benign settings using local anaesthetics and steroids rather than neurolytic agents, the procedure is generally safe and highly effective. This case underscores the practical utility of fluoroscopy-guided CPB as a feasible, cost-effective, and impactful pain management option in chronic pancreatitis, especially where access to advanced endoscopic facilities is limited.

CONCLUSION

Chronic pancreatitis often leads to severe, treatment-resistant abdominal pain that significantly affects a patient's quality of life. In such cases, fluoroscopy-guided coeliac plexus block is a safe, accessible, and effective interventional technique for long-term pain relief, especially in resource-limited settings. This case highlights its clinical utility as a valuable adjunct when conservative therapy fails.

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