

Thoracic Epidural Analgesia in the Management of Acute Pancreatitis : A Narrative Review

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Abstract: *Acute pancreatitis, a condition with high morbidity and mortality, often presents with severe abdominal pain, necessitating effective analgesic strategies. Thoracic epidural analgesia has emerged as a promising option due to its ability to reduce pain intensity and improve pancreatic perfusion by enhancing splanchnic circulation. This review synthesizes clinical and experimental findings supporting the use of thoracic epidural analgesia in acute pancreatitis. The approach not only alleviates pain but also minimizes systemic opioid use and may prevent the progression of pancreatic necrosis. While generally safe, careful hemodynamic monitoring is essential to mitigate risks such as hypotension and infection.*

Keywords: acute pancreatitis, thoracic epidural analgesia, pain management, pancreatic perfusion, critical care

1. Introduction

Acute pancreatitis is a medical condition associated with high morbidity and mortality, particularly in moderate to severe cases. The global incidence ranges from 5 to 30 per 100,000 population per year.¹ The increasing incidence of acute pancreatitis is partly attributed to the rising prevalence of obesity, which contributes to gallstone formation. Gallstones are the leading cause of acute pancreatitis, accounting for approximately 40–70% of cases.²

According to the Revised Atlanta Classification (RAC), acute pancreatitis is categorized into mild acute pancreatitis (MAP), moderately severe acute pancreatitis (MSAP), and severe acute pancreatitis (SAP). Patients with MSAP and SAP exhibit higher morbidity and mortality due to multiorgan dysfunction, including acute respiratory distress syndrome (ARDS), renal failure, prolonged intensive care unit (ICU) stay, and increased healthcare costs.¹

Although the exact pathophysiology of acute pancreatitis is not fully understood, disturbances in pancreatic microcirculation are known to play a significant role. Reduced pancreatic blood flow during the early phase of the disease contributes to the development of edema and necrosis. Microcirculatory dysfunction, including hypoperfusion, ischemia–reperfusion injury, and venous outflow obstruction, are among the mechanisms involved.³

The main clinical manifestation of acute pancreatitis is abdominal pain. Thoracic epidural analgesia is a key modality used by anesthesiologists for pain management, particularly in postoperative thoracic and abdominal procedures.⁴ It is one of the most widely used analgesic techniques during the perioperative period and is also employed in obstetric labor, trauma cases, and in the management of acute, chronic, and cancer-related pain. In addition to blocking afferent stimuli, epidural analgesia induces selective bilateral thoracic sympathetic blockade.⁵

The effects of thoracic epidural analgesia on splanchnic blood flow have become a subject of increasing interest in recent years. Several recent studies have evaluated its role in both animal models and human cases of acute pancreatitis. These

studies propose that selective segmental sympathectomy induced by thoracic epidural analgesia may enhance splanchnic perfusion and improve microcirculatory function, thereby increasing pancreatic perfusion.² Improved pancreatic microcirculation may also prevent progression from mild pancreatic edema to severe necrotizing pancreatitis.⁶

Based on these considerations, the authors are interested in conducting a literature review on the efficacy and safety of thoracic epidural analgesia in the management of acute pancreatitis. The potential of thoracic epidural analgesia to improve patient outcomes represents an important and promising area for further investigation. This review is significant because it consolidates evidence on a technique that may improve outcomes in a high-risk patient population and offers a potential shift in acute pain and perfusion management strategies in pancreatitis care.

2. Discussion

This narrative review is based on a comprehensive search of peer-reviewed articles from PubMed, ScienceDirect, and Google Scholar using terms such as ‘acute pancreatitis,’ ‘thoracic epidural analgesia,’ and ‘pain management.’ Studies included randomized controlled trials, animal studies, and observational cohorts published between 2003 and 2023.

Acute pancreatitis is characterized by the sudden onset of severe abdominal pain. Alcohol consumption and gallstones account for 75–80% of acute pancreatitis cases in industrialized countries, although the prevalence varies across different regions worldwide.⁷ Hypertriglyceridemia is the third most common cause globally, responsible for approximately 10% of all cases, and may induce acute pancreatitis when triglyceride levels exceed 1000 mg/dL.⁸

The Revised Atlanta Classification (RAC) categorizes acute pancreatitis into three groups based on organ dysfunction and the presence of local or systemic complications: mild acute pancreatitis (MAP), moderately severe acute pancreatitis (MSAP), and severe acute pancreatitis (SAP). Severe acute pancreatitis is characterized by a persistent systemic inflammatory response lasting more than 48 hours after the

onset of inflammation. In SAP, inadequate pancreatic microvascular perfusion and tissue hypoxia contribute significantly to disease progression.^{9,10}

Accurate diagnosis and early assessment of disease severity are essential during the initial phase of acute pancreatitis. Active supportive therapy—including organ function monitoring and early identification of complications—plays a critical role in the management of acute pancreatitis. Early diagnosis and timely intervention are key to reducing morbidity and mortality associated with the condition. Severe acute pancreatitis has a substantially higher mortality rate, ranging from 15% to 30%, compared with only 0% to 1% in mild acute pancreatitis.^{9,11}

Severe abdominal pain is one of the most frequently reported symptoms in patients with acute pancreatitis, occurring in up to 95% of cases. The classic presentation is persistent pain localized in the upper abdomen. The pain may radiate to the back and frequently presents with accompanying symptoms such as nausea and vomiting. Oral intake frequently exacerbates the pain, leading to anorexia in many patients. Pain in the epigastric and periumbilical regions results from the leakage of inflammatory fluids and enzymes into the retroperitoneal space. Acute pain in pancreatitis may additionally increase metabolic activity, which in turn enhances pancreatic enzyme production.^{8,11,12}

The conservative management of acute pancreatitis primarily consists of supportive therapy, including fluid management, rehydration, and analgesic administration. In the management of pain associated with acute pancreatitis, the WHO analgesic ladder may be applied, beginning with low-potency NSAIDs (such as paracetamol, indomethacin, and diclofenac) for mild to moderate disease. If NSAIDs fail to provide adequate pain relief, analgesic therapy may be escalated to weak opioids (including tramadol and codeine) or potent opioids (such as pentazocine, fentanyl, and buprenorphine). Abdominal pain is typically more severe during the early phase of the disease; therefore, a multimodal analgesic approach is often required.^{1,7,13,14}

Pain management in acute pancreatitis remains challenging for clinicians, and currently no guideline provides clear recommendations for optimal analgesic strategies in these patients.^{11,15} A 2022 systematic review and meta-analysis comprising 12 randomized controlled trials with a total of 542 participants evaluated pain management approaches in acute pancreatitis and demonstrated that epidural analgesia significantly reduced pain within the first 24 hours.¹¹ The use of epidural analgesia is particularly recommended for patients with severe acute pancreatitis (SAP) and critical acute pancreatitis (CAP) who require high-dose opioids for prolonged periods.¹⁴

Thoracic epidural analgesia is known to provide adequate pain control in approximately 87.5–100% of patients. Epidural analgesia offers potential benefits in individuals with acute pancreatitis by improving pancreatic perfusion.^{11,16} In addition to achieving more rapid pain reduction, thoracic epidural analgesia decreases opioid requirements, thereby reducing the incidence of systemic opioid-related adverse effects.¹⁷

It is well established that acute stress and pain in acute pancreatitis increase sympathetic nervous system activity, leading to intestinal vasoconstriction and reduced blood flow. Pancreatic blood flow also decreases during the early phase of acute pancreatitis, contributing to the development of edema and subsequent pancreatic necrosis.⁴ In septic conditions, splanchnic hypoperfusion further reduces intestinal blood flow, promoting bacterial translocation and predisposing patients to critical illness. Thoracic epidural analgesia induces segmental sympathetic blockade, which enhances neural supply to the splanchnic circulation and promotes vasodilation, thereby improving regional blood flow. Additionally, it increases local microcirculation, which may enhance splanchnic and pancreatic perfusion and reduce hepatic injury.¹

Several experimental studies and clinical data support the use of epidural analgesia in cases of acute pancreatitis. The main effects of epidural analgesia observed in animal experiments include improved survival, reduced lactic acidosis, decreased hepatocyte apoptosis and sinusoidal vasoconstriction, improved perfusion of the pancreas and ileum, enhanced intestinal barrier function and renal perfusion, improved pulmonary hypoxic vasoconstrictive response, and decreased plasma interleukin-6 levels.^{2,18}

Lauer et al. (2007) conducted an experimental study on the role of thoracic epidural analgesia in pulmonary vasoconstriction in rats. The study consisted of three groups: acute pancreatitis without intervention, acute pancreatitis with thoracic epidural analgesia intervention, and thoracic epidural analgesia intervention without acute pancreatitis. The results indicated that thoracic epidural analgesia improved the clinical condition of animals with pancreatitis by reducing pulmonary vasoreactivity. The study found that the use of thoracic epidural analgesia in rats with acute pancreatitis resulted in better oxygenation and lactate levels, as well as lower nitric oxide levels, thereby preserving hypoxic pulmonary vasoconstriction (HPV).¹⁹

Bachmann et al. (2013) also conducted an animal study involving 34 pigs induced with severe acute pancreatitis (SAP), which were subsequently divided into two groups: an intervention group receiving thoracic epidural analgesia (group 1) and a control group without thoracic epidural analgesia (group 2). The study demonstrated that the intervention group exhibited significantly superior microcirculation ($P < 0.05$) and tissue oxygenation ($P < 0.05$), which contributed to a marked reduction in histopathological tissue damage compared with group 2.⁶

These observations are consistent with findings reported by Sadowski et al. (2015). In this study, 35 patients with acute pancreatitis were divided into two groups: 13 patients who received thoracic epidural analgesia and 22 patients who received patient-controlled analgesia (PCA). The study reported a 43% increase in pancreatic arterial perfusion in the epidural group, whereas the PCA group showed only a 7% increase ($p = 0.0025$). Pain scores showed significantly greater improvement in the epidural group than in the PCA group ($p = 0.034$). No complications related to the epidural procedure were observed in this study.²⁰

Similarly, a study by Bulyez et al. (2017) conducted a prospective multicenter randomized controlled trial (the EPIPAN trial) aimed at investigating the effects of epidural analgesia on organ failure, mortality, and other clinical outcomes in critically ill patients with acute pancreatitis. This study included a total of 148 patients with acute pancreatitis who were assigned to either a thoracic epidural analgesia intervention group or a control group. The study reported a 43% increase in pancreatic perfusion in the intervention group compared with only 7% in the control group ($p = 0.0025$). The authors also concluded that thoracic epidural analgesia improved respiratory function, reflected by a higher number of ventilator-free days by day 30 in patients with acute pancreatitis. Furthermore, no epidural-related procedural complications were reported in this study.

Jabouden et al. (2017) conducted a study involving 1,003 patients with acute pancreatitis who received thoracic epidural analgesia compared with those who did not. The investigators analyzed the 30-day mortality risk over a three-year period among patients with acute pancreatitis who underwent thoracic epidural analgesia. The study concluded that the 30-day mortality rate was significantly lower in the intervention group than in patients who did not receive thoracic epidural analgesia (2% vs. 17%; $p = 0.01$).¹⁸

Segmental sympathetic blockade from thoracic epidural analgesia depends on dose, anatomical site, and the extent of neural spread. Potential risks and complications associated with thoracic epidural analgesia in patients with acute pancreatitis have been reported. Two major complications that may occur are hypotension and infectious neuraxial complications. The incidence of hypotension related to epidural use in acute pancreatitis is 37.5%. This hypotension typically occurs in patients who have already developed SIRS. However, it can be managed with fluid therapy and the administration of vasopressors such as norepinephrine. Recent studies indicate that the incidence of infectious neuraxial complications associated with epidural blockade in critically ill patients is very low, approximately 0.8%.²¹ Other possible complications include failed catheter placement (1.1%), dural puncture (0.7%), postoperative radicular pain (0.2%), and peripheral nerve injury (0.2%).⁴

3. Conclusion

Thoracic epidural analgesia appears to offer multiple clinical benefits in the management of acute pancreatitis, including enhanced pancreatic perfusion, improved pain control, and reduced reliance on systemic opioids. Its safety profile is generally acceptable when coupled with appropriate hemodynamic monitoring. Further clinical studies are warranted to refine indications and protocols for its optimal application.

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