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What is the evidence for EUS-guided celiac plexus block/neurolysis?

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The management of pain associated with pancreatic cancer or chronic pancreatitis is often challenging. Nociceptive afferent nerve fibers from the pancreas as well as other abdominal and pelvic viscera are relayed through the celiac plexus. This dense network of multiple ganglia and nerve fibers, located anterolateral to the aorta in the vicinity of the celiac trunk, is an attractive target for interventions when pancreatic pain is refractory to medical therapy. Celiac plexus neurolysis (CPN) is defined as the ablation of celiac plexus neurons by the injection of chemical cytolytic agents such as alcohol or phenol. Celiac plexus blockade (CPB) is defined as the injection of a local anesthetic with the aim of inhibiting neurotransmission without the ablation of neurons. CPN has been performed by a variety of radiological guided techniques for several decades. An alternative approach has been to perform CPN at the time of surgery. EUS guidance for performing CPN is relatively new, being first reported just over a decade ago.¹

Alcohol and phenol are the two chemical agents that have been widely used for performing CPN. Alcohol has become the favored agent in the United States because of a perception that it leads to more complete ablation and avoids the potential mutagenicity of phenol. Despite the numerous series and the large number of reported cases in the radiology literature there is a dearth of investigations on the histopathological effects of CPN. An experimental study that injected various amounts and concentrations of phenol and alcohol directly into various cat sympathetic ganglia actually found 50% phenol resulted in more neuronal loss than absolute alcohol.² Alcohol injection resulted in only moderate neuronal degeneration within sympathetic ganglia and was associated with residual fibrosis. Postmortem examinations of two patients who had undergone CPN with alcohol found the persistence of neurons within fibrotic fascicles with only moderate neuronal degeneration.³ Thus, the available histopathological evidence would suggest that CPN with chemical injections will not result in complete long-term resolution of pain and will at best provide mild to moderate pain relief when used clinically.

Radiologic CPN can be performed under Xray, fluoroscopy, sonography, CT, or MR guidance using posterior or anterior approaches. The highest level of evidence for effectiveness of CPN comes from 5 published randomized controlled trials in pancreatic cancer patients.⁴⁻⁸ Even these studies were of limited quality because the methods of allocation, allocation concealment and double blinding were not defined. The study by Lillemoe et al⁸ performed CPN at surgery regardless of whether patients had pain whereas the remaining 4⁴⁻⁷ used radiologic guidance for CPN in selected patients with pancreatic cancer pain. A 10 point visual analog scale was

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the primary outcome. A meta-analysis by Yan and Bryer calculated that CPN decreased pain by a mean visual analog score of 0.6 (95% CI, 0.37 – 0.82) at 8 weeks in cases compared to controls.⁹ Mean daily opioid use was also lowered by 80.45 mg morphine equivalents at 8 weeks following CPN in pancreatic cancer cases. Side effects of CPN were transient local pain, decreased constipation, and a trend towards hypotension. Although the Lillemoe study reported an improved survival in patients receiving CPN when all five of the studies were pooled there was no survival advantage for CPN.⁹ The weight of the evidence suggests that CPN results in a mild to moderate sustained reduction of pain in pancreatic cancer. CPN results in decreased opioid use but does not eliminate the need for opioids.

Radiologic CPN is largely being supplanted by EUS guided CPN because the location of the celiac plexus can be easily accessed using a transgastric anterior approach at EUS. However, the published evidence for EUS CPN is largely limited to observational uncontrolled series. Gunaratnam et al. measured visual analog scores and opioid usage in 58 patients with pancreatic cancer following EUS CPN.¹⁰ Pain scores were lower in 78% of patients and pain relief lasted for over 24 weeks. However, the decrease in pain scores was less than 2 points on the visual analog scale in nearly half of the patients. Furthermore, the efficacy of CPN was lower if patients did not receive adjuvant therapy and opioid usage was not eliminated. There is one published randomized trial by Gress et al. comparing EUS vs. CT guided celiac blockade (local anesthetic plus steroid injection) in patients with chronic pancreatitis pain.¹¹ This study compared 10 patients treated with EUS with 8 patients treated with CT. The pain relief with either technique was not persistent and the sample size was too small for reliable conclusions. The same group reported on the efficacy of this CPB technique in a larger observational series¹² but the pain relief was not sustained and the series did not have a comparative control group. Newer EUS techniques that directly target celiac ganglia have been reported but not studied in controlled trials.¹³ Since the celiac plexus consists of more than two ganglia it is not evident that this approach will prove to be more effective than existing approaches.

Although the published evidence of EUS CPN is of poor quality it is safe to suggest that EUS guided CPN is equivalent to radiologic CPN. As such, it is likely that EUS CPN will result in mild to moderate pain relief in patients with pancreatic pain. Sustained pain reduction can be achieved but EUS guided alcohol injection in the celiac plexus should be considered an adjunct to opioid use and adjuvant therapy in patients with pancreatic cancer. There is little convincing physiologic or pathologic evidence to support the use of steroid injection in chronic pancreatitis pain.

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