Spinal Cord Stimulation for Treatment-Refractory Angina Pectoris

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Introduction

Refractory angina pectoris (RAP) is a chronic condition that manifests as angina caused by coronary insufficiency in the presence of coronary artery disease (CAD) that cannot be controlled by a combination of medical therapy, angioplasty, and coronary bypass surgery. There is considerable number of patients with RAP worldwide.

Spinal Cord Stimulation (SCS) is a well-established therapy that stimulates the spinal cord with a low voltage current, which is very effective for nociceptive and neuropathic pain syndromes. SCS has been shown to be efficacious for the treatment of chronic pain-associated RAP by decreasing sympathetic tone and augmenting myocardial blood flow to protect the myocardial cells. This intervention is especially useful when conservative therapeutic measures fail. However, a formal guideline for its use as routine therapy for RAP does not exist.

Case Presentation

45-year-old-female with a past medical history of non-obstructive CAD presenting with severe, recurrent chest pain. Anginal episodes with no clear trigger, not exercise-induced or clearly associated with any exposures. Patient was admitted for worsening angina and NSTEMI in setting of coronary vasospasm (of unclear etiology), now s/p nitroglycerin drip and stellate ganglion block and catheter placement with minimal relief.

Prior Therapies Include:
Procedures: stellate ganglion block and catheter insertion with bupivacaine infusion

Additional Therapies: s/p nitroglycerin infusion and high-dose steroids; antianginal regimen (verapamil, amlodipine, and isosorbide mononitrate), magnesium supplement, amiloride (for magnesium absorption)

Chronic Pain Management Consulted for:
Evaluation and consideration of SCS trial

Evaluation

Patient endorses pain in left chest. Often occurs in the morning between 0600-0800. Rates pain 6-10/10 on a numeric rating scale. Prior therapies have minimal effect.

Interventions

Stage 1

- **SCS Trial**
  - Intraoperative sedation and antibiotics given
  - Lumbar 1-2 (L1-2) interspace identified with fluoroscopy
  - A 14-gauge Tuohy epidural needle was advanced with a paramedian approach into the epidural space
  - A 16-contact lead was advanced under fluoroscopic guidance until the 0-position lead located at bottom of Thoracic-1 (T1). Second lead was placed from the L1-2 interspace on the left until the 0-position was at bottom of T1
  - The needles were removed and leads were secured to the skin with a fixation device. Proper placement of the leads confirmed with fluoroscopy before anchoring device
  - Device placement confirmed



Stage 2

- **Post-operative Evaluation & Treatment**
  - Tolerated the procedure well. No apparent complications
  - Patient prescribed antibiotics upon return to hospital floor
    - Bactrim DS one tab BID x 10 days
  - SCS stimulation settings
    - Amplitude: 2.5mA, pulse width 220μs, Rate 90Hz

Stage 3

- **Post-interventional Follow Up:**
  - **Patient with significant chest pain relief**
  - Stellate ganglion catheter weaned off
  - SCS ran around 20-30% of perception threshold with boluses
  - One episode of subjective warmth sensation in chest & face, and bilateral conjunctival injection on day of stress test
  - Device turned off by patient on POD#4
  - Trial leads removed POD #6; no SCS implant
  - **No additional angina episodes prior to discharge**
- **Continuation of medication regimen:**
  - Anti-anginal and magnesium therapy per Cardiology team

Figure 1. Stages of Therapeutic Course

Discussion

When using SCS for RAP, leads are placed in the higher thoracic epidural space (C7–T1), providing stimulation at the level of myocardial neurons. The leads are connected to an implantable pulse generator in the abdomen/buttock. Proposed mechanisms of action: direct inhibition of pain, decreased sympathetic tone and changes in myocardial blood flow with a reduction in myocardial oxygen demand, and increased coronary microcirculatory blood flow.

The patient experienced stimulation-dependent relief of anginal symptoms during the SCS trial, including a reduction in pain intensity and frequency. This relief lasted through the termination of the stellate ganglion catheter, nitroglycerine infusion, and during a stress test. This relief lasted despite the device being turned off on POD #4 and removed on POD #6. No additional anginal episodes were reported through discharge on POD #10. She was continued on anti-anginal medication management.

This case raises the possibility that short-term neuromodulation may produce sustained physiologic or autonomic effects in refractory angina, even beyond active stimulation.

Conclusions

SCS remains an effective intervention for patients with chronic RAP refractory to medical therapy. This intervention may also lead to a reduction of anginal episodes, decreased use of nitrates, and a significant improvement in quality of life for patients. Additional research is needed to provide recommendations on SCS intensity and stimulation duration for this condition.

References

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