High Thoracic Epidural Block as a Cardioprotective Adjunct for Severe Left Main Coronary Disease

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Citation

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Abstract

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INDUCTION

We were presented with the clinical dilemma of controlling angina and allowing the patient to benefit from the effects of HTEA in the face of an 80% left main lesion and a recent non-Q-wave infarct.

Running Head: Thoracic Epidural in Coronary Ischemia

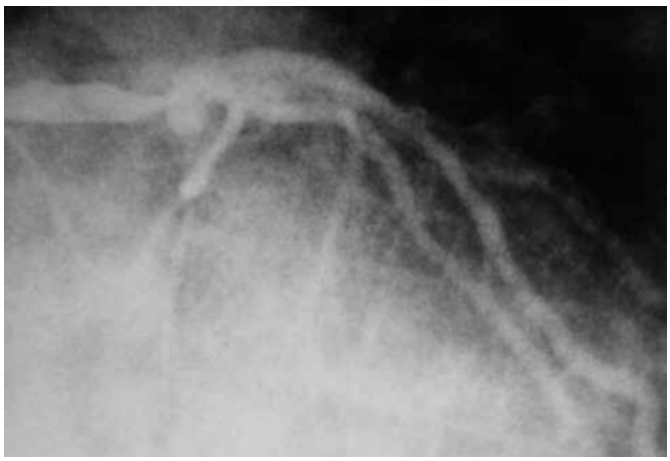
CASE REPORT

Our patient is a 58 year-old man admitted for crescendo angina and congestive heart failure (CHF). His past medical history was significant for hypertension, transient ischemic attacks, alcohol abuse, smoking, COPD and HTN induced chronic renal insufficiency (baseline Cr=5.3). Admission echocardiography revealed mild global hypokinesis with inferior and posterior akinesis, an ejection fraction of 40-50% and moderate mitral regurgitation. He ruled-in for an anterolateral myocardial infarction with a positive CK-MB fraction and anterolateral ECG ST-depression without Q-waves. He was transferred to our medical center and I.V. heparin, metoprolol, furosemide, and nitroglycerin were optimized.

Cardiac catheterization, using minimal contrast, revealed an 80% distal left main coronary stenosis (Fig. 1A) and a 90% proximal right coronary stenosis (Fig. 1B). Although excellent target vessels for CABG, the following morning surgery was canceled when the patient developed productive sputum. He was placed on cefotaxime and bronchodilators but within the next 24 hours developed hemoptysis. A rising creatinine, most likely contrast induced, requiring treatment with hemodialysis complicated the clinical picture . Echocardiography now demonstrated a worsening of LV systolic function to an estimated FAC of 30% with severe global hypokinesis. Unfortunately he then developed severe substernal chest pain, CHF, hypotension, acute anterolateral ECG changes requiring intubation and inotropic support. CXR showed severe pulmonary edema (Fig. 2) and CABG was not felt to be a viable option because of significant multi-organ involvement.

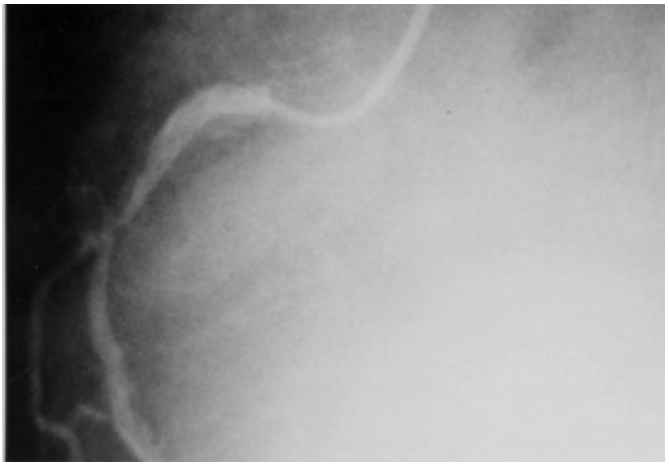
Figure 1

Fig. 1: Coronary arteriography



1A: Shallow RAO magnified projection of the proximal left coronary system. The catheter-engaged left main coronary artery shows an 80% distal lesion prior to the bifurcation of the left anterior descending and circumflex coronary arteries. Although there were diffuse luminal irregularities throughout the coronary vasculature, there were no other high grade (> 40%) lesions in the left system.

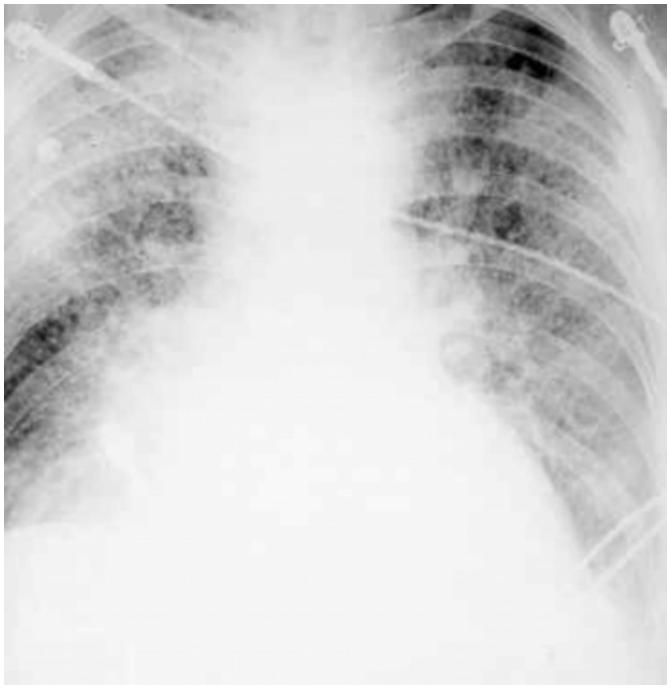
Figure 2



1B: LAO projection of the right coronary artery showing a 90% proximal lesion just prior to the origin of a right ventricular branch.

Figure 3

Fig. 2: Chest x-ray with anterior-posterior portable technique. There is prominent diffuse vascular congestion compatible with severe pulmonary edema



After three days of maximal medical management and aggressive dialysis, he was extubated. It was felt that the patient's high Cleveland Clinic score made his risk unacceptable for surgical intervention. The option was to allow the patient to recover and return for surgery in six weeks. The patient continued to have a pattern of ischemic angina and was forced to remain very inactive despite medical management. Our Anesthesia service was consulted since we had substantial experience with high thoracic epidural analgesia (HTEA) for relief of angina ([1,2]). After informed consent, the patient was taken to the OR and a DuPen catheter (Bard) placed under MAC anesthesia. An externalized catheter was appropriate due to the short length of time it would be indwelling and the ease of removal. His catheter was placed at a T1-2 position and confirmed by radiographic means. He was placed on a solution of 0.25% bupivacaine with 3 m fentanyl/cc at a bolus dose of 3.5 cc with a 180 minute lockout time. He self-dosed on a tid basis and prior to hemodialysis as well as with the first symptom of angina (along with sublingual nitroglycerin). The patient was closely monitored to assure no blood pressure change with each dose since hypotension would cause devastating changes in the coronary perfusion pressures. Other medications included amlodipine, isosorbide-dinitrate and ASA (b -blockers were stopped because of COPD).

There was great concern about the patient going home with a left main lesion for a six week period but the patient strongly desired discharge from the hospital. He did exceptionally well, not having any emergent evaluations during catheter therapy or reported hemodynamic changes with bolus dosing. He did not have more than one to two mild anginal episodes per day. After six weeks CXR showed only emphysema and ECG had returned to baseline. Echocardiography revealed an improved ejection fraction of 50% with resolution of all regional wall motion abnormalities and no evidence of mitral regurgitation. He underwent a successful 3-vessel CABG. The catheter was used as an adjunct to isoflurane/sufentanil/midazolam anesthesia and for post-op pain control. Intra-op/post-op the patient was maintained on bupivacaine 0.25% with fentanyl 5 m /cc infusion at 3 cc/hour. The catheter was discontinued on the third post-op day with discharge home on day 7 still requiring hemodialysis but otherwise stable. He has done well long-term.

DISCUSSION

Cardiogenic shock in patients with severe left main coronary artery disease (³ 75% stenosis) after myocardial infarction has a grave prognosis. In-hospital mortality without invasive therapy is as high as 94% ([3]). Medical stabilization is often ineffective, intra-aortic balloon pumps only temporizing and revascularization strategies such as emergent CABG or angioplasty (with stenting) extremely risky ([4,5]). Invasive revascularization in the presence of acute myocardial infarction or cardiogenic shock has mortality reported up to 38.5% ([6]). Accordingly, clinicians must often focus on

stabilization to allow a subsequent invasive procedure.

There are several animal studies supporting (HTEA) may have cardioprotective properties ([7,8,9,10,11]). For example, Shibata et al. demonstrated reduction in both mortality and detrimental cardiac hemodynamics in profoundly hypotensive dogs that had dosed epidurals in place prior to insult when compared to controls (8). Other studies have mapped increased endocardial blood flow using tagged microspheres as well as shown decreases in myocardial oxygen consumption (9-11). This experimental data strongly suggests the mechanism is improved blood flow within the myocardium via sympathetic blockade. Blomberg, Toft and Liem ([12,13,14,15]) have reported favorable outcomes with HTEA in chronic and acute coronary artery disease and in CABG surgery. Our case expands these observations to include benefit from HTEA in high grade coronary disease as adjunctive therapy prior to revascularization. We had done similar therapy in a hospitalized patient (2) but not to a patient with a high grade lesion discharged to home. Of note is the patient's outcome in a complicated situation. His ECHO had significantly improved at the time of surgery and did not demonstrate new or worsening coronary disease. Whether HTEA had any impact in the improvement process or whether normal resolution of stunned myocardium is the only process is difficult to ascertain. These findings support further investigation of HTEA in advanced coronary artery syndromes.

IMPLICATIONS

A 58 year-old man presents with chest pain and severe coronary disease. Coronary bypass surgery must be delayed because of cardiogenic shock with pulmonary and renal complications. In addition to aggressive medical management, high thoracic epidural analgesia is used as an adjunct to treat ischemia and allow sufficient recovery to undergo successful surgery.

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