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Postoperative Nesiritide Use following High-Risk Mitral Valve Replacement

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ABSTRACT

Nesiritide is primarily used in the treatment of acutely decompensated chronic heart failure. It may also be useful in the postoperative management of patients with an exacerbation of heart failure after cardiac surgery. The management of a patient with cardiogenic shock after acute papillary muscle rupture is described. The patient exhibited signs of postoperative heart failure, and nesiritide therapy was instituted to lower filling pressures and achieve diuresis. This drug may be useful when patients with heart failure undergo cardiac surgery and continue to show evidence of heart failure in the postoperative period.

INTRODUCTION

Nesiritide is a recombinant form of human B-type natriuretic peptide (BNP) and has been approved for use in the United States in the management of acutely decompensated heart failure. To extend this application, some physicians have used nesiritide prior to operation to optimize the hemodynamic status of patients presenting in acute heart failure who need operative intervention. Some surgeons have recently begun to use nesiritide in the postoperative management of selected patients with heart failure. As more patients with heart failure are offered operations, evolving therapies, such as the perioperative use of nesiritide, simplify their management.

CASE REPORT

A 43-year-old man presented to his local hospital in cardiogenic shock with an inferior myocardial infarction and severe mitral regurgitation. Despite the use of epinephrine, norepinephrine, and an intraaortic balloon pump (IABP), the patient remained hypotensive with a systolic blood pressure of approximately 70 mm Hg. After approximately 8 hours, the systolic blood pressure stabilized between 80 and 90 mm Hg, and the patient was transferred to Hahnemann University Hospital. The results of serum chemistry tests were significant for elevated creatinine (3.0 mg/dL), aspartate aminotransferase (AST) (2403 U/L), and alanine aminotransferase (ALT) (1377 U/L), reflecting the severity of cardiogenic shock.

On arrival, the patient had a blood pressure of 86/74 mm Hg with a heart rate of 160 beats/min and was receiving norepinephrine (22 μg/min), epinephrine (7 μg/min), and IABP support at 1:1. He was hypoxic, with a saturation of 89% on 100% FIO_2, and had mixed respiratory and metabolic acidosis (pH 7.22). Echocardiography showed an ejection fraction of 35% with severe mitral regurgitation and a ruptured posteromedial papillary muscle. With evidence of impending multisystem organ failure, the patient underwent immediate placement of a left ventricular assist device (LVAD) (BVS 5000; Abiomed, Danvers, MA, USA) without the use of cardiopulmonary bypass to stabilize him prior to definitive repair.

Postoperatively the patient needed management with an open chest and exhibited worsening organ dysfunction. The chest was closed on the third postoperative day. By postoperative day 12, the creatinine, amylase, lipase, AST, and ALT values had stabilized at near-normal levels, and the patient underwent mitral valve replacement, bypass grafting to the posterior descending artery, and LVAD explantation. Operative findings included complete disruption of the posteromedial papillary muscle with a flail anterior leaflet. The patient was weaned from cardiopulmonary bypass without difficulty and was transferred to the intensive care unit receiving milrinone, norepinephrine, and aprotinin. The morning after the operation, the patient awoke with left arm and leg weakness.

In the early postoperative period, the patient exhibited a poor response to moderate doses of diuretics, associated with the slow rise in creatinine concentration, central venous pressure (CVP), and pulmonary artery diastolic (PAD) pressure (Figure 1). On the morning of the second postoperative day, nesiritide (Natrecor; Scios, Sunnyvale, CA, USA) was started without a bolus at a rate of 0.01 μg/kg per minute, and furosemide (20 mg intravenously) was given. The patient had an immediate increase in urine output (Figure 2) and a decline in CVP and PAD pressure. Nesiritide infusion was

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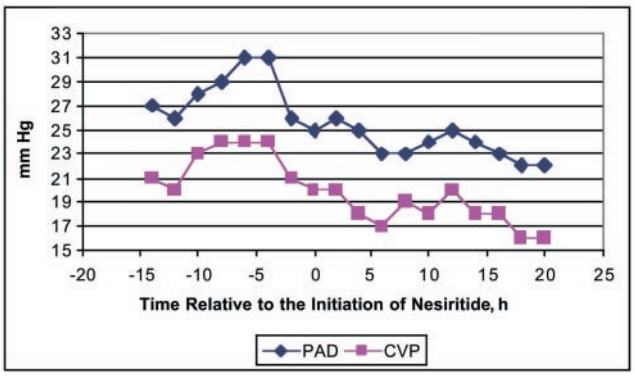

Figure 1. Central venous pressure (CVP) and pulmonary artery diastolic (PAD) pressure relative to the initiation of nesiritide infusion (0.01 µg/kg per minute) over time. The nesiritide infusion was initiated at time zero.

continued for 72 hours with maintenance of brisk diuresis during this period without administration of additional diuretics. The remainder of the hospitalization was unremarkable. The neurological deficit resolved by the time the patient was ready for immediate rehabilitation. The patient continued to do well in follow-up.

DISCUSSION

Patients who present for surgery in acute cardiogenic shock have a high mortality rate. Despite a technically successful operation, many continue to experience signs of heart failure, and fluid overload can become a problem as third-space fluids are mobilized in the face of diminished cardiac function. This problem may be compounded by renal insufficiency. Nesiritide may help in the management of these patients.

Nesiritide is a recombinant form of BNP that has many properties that are beneficial in the management of heart failure. This agent can rapidly decrease PAD pressure and CVP, improve diuresis, and lead to symptomatic improvement in these patients [Colucci 2000]. Nesiritide is typically used in the management of acute exacerbations of chronic heart failure, shortens the time to symptomatic relief, decreases the incidence of hospital readmission for heart failure in these patients, and improves 6-month mortality [Silver 2002]. Patients with acute or chronic heart failure presenting for cardiac surgery may also benefit from treatment with this agent [Moazami 2003]. These patients have diminished cardiac function that may be temporarily worse in the postoperative period. Fluid mobilization often leads to intravascular volume overload accompanied by increased filling pressure, worsening of peripheral edema, and the onset of hypoxia and shortness of breath. Although it is predictable, the onset is not always avoidable in the presence of significant cardiac dysfunction.

Nesiritide should produce a decrease in filling pressure soon after administration. Although nesiritide, like naturally occurring BNP, is not a strong diuretic, it may promote diuresis in some patients. Our patient did not respond to diuretic doses that had previously produced good diuresis. However, after nesiritide administration, the patient had a sustained increase in urine output in response to 20 mg of furosemide and had a fall in CVP and PAD pressure. Thus in the setting of significant cardiac dysfunction and fluid overload, nesiritide infusion decreased filling pressure and improved urine output, as expected.

Nesiritide is often given as a bolus followed by continuous infusion in the setting of decompensated heart failure [Hobbs 2001]. The incidence of symptomatic hypotension is low with this agent. Patients should have adequate filling pressure and not be hypotensive at the initiation of drug therapy. Bolus therapy may not be necessary in postoperative patients because the onset of fluid overload usually is predictable, and fluid shifts often begin within 24 to 48 hours after operation. When increases in CVP and PAD pressure are not responsive to standard therapies, nesiritide infusion can be initiated before the patient becomes severely compromised. This measure potentially avoids the need for a bolus.

In the postoperative period the behavior of patients with either acute or chronic heart failure appears to be similar to that of patients presenting with decompensated heart failure. Although nesiritide is beneficial in patients with heart failure and theoretically can be helpful in the postoperative management of these patients, further study is required before a final determination can be made.

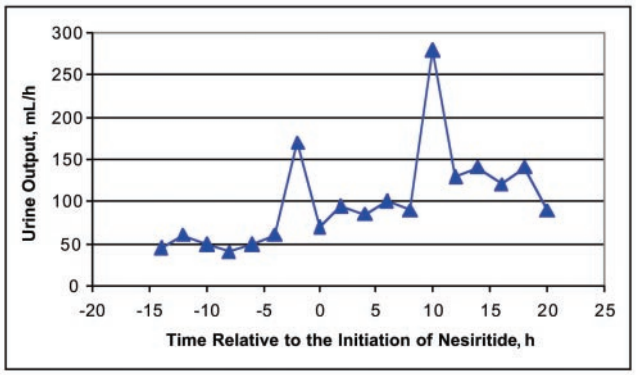

Figure 2. Urine output relative to the initiation of nesiritide infusion (0.01 µg/kg per minute) over time. The nesiritide infusion was initiated at time zero.

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