Less Myocardial Harm in Endovascular AAA Repair

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SCOTTSDALE, ARIZ. — Patients who undergo endovascular repair of an abdominal aortic aneurysm are significantly less likely to experience myocardial damage than are patients undergoing open repair, Dr. James May said at an international congress on endovascular interventions sponsored by the Arizona Heart Foundation.

In a review of 149 consecutive patients undergoing elective abdominal aortic aneurysm (AAA) repair, Dr. May and colleagues found that 25% of 36 patients undergoing an open repair had elevated levels of troponin T following the procedure. By contrast, only 8% of 113 patients undergoing endovascular repair had elevated troponin T levels post procedure.

"This finding should be taken into account when deciding on the method of AAA repair, even when the patient is considered low risk for an open repair," said Dr. May, of the department of vascular surgery at the Royal Prince Alfred Hospital, Sydney, Australia.

He said although it has been assumed that endovascular repair of AAAs is generally less traumatic, this hypothesis had never been measured biochemically. Serum troponin T level is an accepted indicator of subclinical myocardial damage.

The study considered a positive finding of myocardial damage to be a 50% of greater increase in serum troponin T after the procedure, compared with before, without a significant increase in serum creatinine. Only one patient had clinically obvious myocardial ischemia after the procedure, and that patient was in the open repair group.

There were no significant differences in age between the two groups of patients, but the patients who underwent endovascular repair were significantly more likely to have had a previous myocardial infarction (41% vs. 21%). ■

Indication

NATRECOR® (nesiritide) is indicated for the intravenous treatment of patients with acutely decompensated congestive heart failure who have dyspnea at rest or with minimal activity. In this population, the use of NATRECOR® reduced pulmonary capillary wedge pressure and improved dyspnea.

The recommended dose of NATRECOR® is an intravenous bolus of 2 mcg/kg followed by a continuous infusion of 0.01 mcg/kg/min.

IMPORTANT SAFETY INFORMATION

HYPOTENSION

NATRECOR® may cause hypotension and should be administered only in settings where blood pressure can be monitored closely. If hypotension occurs during administration of NATRECOR®, the dose should be reduced or discontinued. At the **recommended dose** of NATRECOR®, the incidence of symptomatic hypotension (4%) was similar to that of IV nitroglycerin (5%). Asymptomatic hypotension occurred in 8% of patients treated with either drug. In some cases, hypotension that occurs with NATRECOR® may be prolonged. The mean duration of symptomatic hypotension was longer with NATRECOR® than IV nitroglycerin (2.2 versus 0.7 hours, respectively). NATRECOR® should not be used in patients with systolic blood pressure <90 mm Hg or as primary therapy in patients with cardiogenic shock. The rate of symptomatic hypotension may be increased in patients with a baseline blood pressure <100 mm Hg, and NATRECOR® should be used cautiously in these patients. In earlier trials, when NATRECOR® was initiated at doses higher than the 2 mcg/kg bolus followed by a 0.01 mcg/kg/min infusion, the frequency, intensity, and duration of hypotension was increased. The hypotensive episodes were also more often symptomatic and/or more likely to require medical intervention.

NATRECOR® is not recommended for patients for whom vasodilating agents are not appropriate and should be avoided in patients with low cardiac filling pressures.

RENAL

NATRECOR® may affect renal function in susceptible individuals. In patients with severe heart failure whose renal function may depend on the activity of the renin-angiotensin-aldosterone system, treatment with NATRECOR® may be associated with azotemia. In the VMAC trial, through day 30, the incidence of elevations in creatinine to >0.5 mg/dL above baseline was 28% and 21% in the NATRECOR® and nitroglycerin groups, respectively. When NATRECOR® was initiated at doses higher than 0.01 mcg/kg/min, there was an increased rate of elevated serum creatinine over baseline compared with standard therapies, although the rate of acute renal failure and need for dialysis was not increased.

MORTALITY

In seven NATRECOR® clinical trials, through 30 days, 5.3% in the NATRECOR® treatment group died as compared with 4.3% in the group treated with other standard medications. In four clinical trials, through 180 days, 21.7% in the NATRECOR® treatment group died as compared with 21.5% in the group treated with other medications. There is not enough information to know if there is an increased risk of death after treatment with NATRECOR®.

Please see brief summary of prescribing information for NATRECOR® on the following page.

References: 1. NATRECOR® Full Prescribing Information. **2.** Publication Committee for the VMAC Investigators (Vasodilation in the Management of Acute CHF). Intravenous nesiritide vs nitroglycerin for the treatment of decompensated congestive heart failure: a randomized controlled trial. *JAMA*. 2002;287:1531-1540. **3.** Nieminen MS, et al. Executive summary of the guidelines on the diagnosis and treatment of acute heart failure: the Task Force on Acute Heart Failure of the European Society of Cardiology. *Eur Heart J*. 2005;26:384-416. **4.** Data on file, Scios Inc. **5.** Cuffe MS, et al. Short-term intravenous milrinone for acute exacerbation of chronic heart failure. *JAMA*. 2002;287:1541-1547. **6.** Butler J, et al. Relationship between heart failure treatment and development of worsening renal function among hospitalized patients. *Am Heart J*. 2004;147:331-338. **7.** Emerman CL, et al. Impact of intravenous diuretics on the outcomes of patients hospitalized with acute decompensated heart failure: insights from the ADHERE registry. *J Card Fail*. 2004;10(suppl 4):S116-S117. Abstract 368. **8.** Silver MA, et al. Effect of nesiritide vs dobutamine on short term outcomes in the treatment of patients with ADHF. *J Am Coll Cardiol*. 2002;39:798-803. **9.** Philbin EF, et al. Association between diuretic use, clinical response, and death in acute heart failure. *Am J Cardiol*. 1997;80:519-522.