Revascularization in Post-STEMI Cardiogenic Shock

By George Philippides, M.D., and Eric H. Awtry, M.D.

The Patient

A previously healthy, active 76-year-old man presents to his local hospital 3 hours into an extensive acute anterior STElevation MI. He is treated with intravenous fibrinolytic therapy and is admitted to the CCU 90 minutes later, “hemodynamically stable” and pain free. Over the ensuing 6 hours he becomes progressively hypotensive and oliguric. An urgent echocardiogram and right heart catheterization confirm the diagnosis of severe left-ventricular failure and cardiogenic shock. Before he can be transferred to a percutaneous coronary intervention-capable facility, he arrests and dies.

The Problem

Cardiogenic shock occurs in approximately 7%-8% of all MIs, is the most common cause of death for patients hospitalized with acute MI, and is historically associated with a 70%-80% mortality rate.

The Data

Nonrandomized studies have suggested that when reperfusion with PCI, CABG, or thrombolysis with hemodynamic support with an intra-aortic balloon pump (IABP) have been improving survival in such patients. In most of these reports it is clear that restoration of coronary blood flow correlates with in-hospital survival, regardless of how patency is achieved. Thrombolysis alone has shown modest benefit, compared with PCI. This may reflect the relatively low rate of clot lysis and rate of TIMI 3 flow achieved in hypertensive patients treated solely with fibrinolytic agents.

The SHOCK (Should We Emergently Revascularize Occluded Coronaries for Cardiogenic Shock) trial was the first multicenter, prospective study to support this “aggressive approach.” SHOCK randomized 302 patients with predominant LV failure/shock within 36 hours of MI to an early revascularization (ER) strategy (PCI or coronary artery bypass graft as soon as possible) vs. initial medical stabilization (IMS) consisting of thrombolysis unless contraindicated and delayed revascularization, if needed. IABP was strongly recommended and ultimately used in 86% of both treatment arms.

While the 30 day mortality rate for ER and IMS did not differ significantly, the mortality rate at 6 and 12 months was significantly lower in the ER group (N Engl J Med. 1999;341:625-34).

The researchers determined cardiac outcomes in STEMI patients with cardiogenic shock. The primary end points within 10 days of presentation for clinical variables, they found that cystatin C levels in quintile 5 were 0.82 for quintile 1; 0.83-0.91 for quintile 2; 0.92-1.00 for quintile 3; 1.01-1.14 for quintile 4; and 1.15 or more for quintile 5.

The Future

The risk of developing and dying from cardiogenic shock post MI increases with age. Considering that most episodes of cardiogenic shock become clinically apparent after admission to the CCU, and that fewer than 20% of U.S. hospitals have revascularization facilities, it is crucial that future CCU physicians are well trained in recognizing, treating, and triaging these critically ill patients.

Boston Medical Center is a member of the Emergency Medical Services Point of Entry Program in Boston, a coalition of hospitals working to develop a regional system of evidence-based care for STEMI patients in the greater Boston area. We believe that the continued development and widespread adoption of these regional systems, which aim to increase the number of STEMI patients with timely access to PCI facilities, represent the greatest chance for improving outcomes in STEMI patients with cardiogenic shock.

Elevated Cystatin C Is Harbinger of Adverse Events in ACS

By Doug Brunk

San Diego Bureau

SAN DIEGO — Elevated baseline cystatin C levels in patients who present with acute coronary syndrome are strongly linked with adverse cardiovascular outcomes, results from a large study showed.

“Cystatin C has been shown to be a strong and independent predictor of cardiovascular events and overall mortality in elderly subjects, but its prognostic performance in patients with acute coronary syndrome is less well studied,” reported Dr. Stacy E. Melanson on behalf of coauthor Dr. Steven D. Wiviott and researchers from the Thrombolysis In Myocardial Infarction (TIMI) Group at Brigham and Women’s Hospital, Boston.

In a presenter at the annual meeting of the American Association for Clinical Chemistry, the researchers analyzed levels of cystatin C in blood samples from 1,714 patients that were collected within 10 days of presentation with ACS. The primary end points were death, MI, and heart failure.

The researchers determined cardiovascular outcomes for each quintile of cystatin C. Cut points for cystatin C, in mg/L, were less than 0.82 for quintile 1; 0.83-0.91 for quintile 2; 0.92-1.00 for quintile 3; 1.01-1.14 for quintile 4; and 1.15 or more for quintile 5. Patients who had elevated cystatin C levels were more likely to have hypertension, diabetes, and a history of MI. They were also more likely to be older than 75 years. Additionally, the median age of patients in quintile 5 was 68 years, while the median ages of patients in quintiles 1, 2, 3, and 4, were 52, 54, 57, and 61 years.

Between cystatin C quintiles 1 and 5, the risk of death rose from 0.7% to 8.4%; the risk of MI rose from 5.4% to 10.6%; the risk of heart failure rose from 1.0% to 8.3%; and the risk of a composite of death and heart failure rose from 1.7% to 11.6%.

After the researchers adjusted for clinical variables, they found that cystatin C levels in quintile 5 independently predicted recurrent cardiovascular events, compared with the levels in quintile 1. Specifically, the hazard ratios between quintile 5 and quintile 1 were 2.5 for death, 1.6 for MI, 4.2 for heart failure, and 1.1 for a composite of death and heart failure.

When other markers of hemodynamic stress were added to the model, including C-reactive protein and B-type natriuretic peptide, cystatin C remained a significant predictor of recurrent cardiovascular events.

Dr. Melanson is associate medical director of clinical chemistry at Brigham and Women’s Hospital.