Elevated Troponin a Red Flag in Heart Failure

High levels of the protein were associated with worse outcomes in acute decompensated heart failure.

BY SHERRY BOSCHERT
San Francisco Bureau

SAN FRANCISCO — Patients seen in the emergency department for acute decompensated heart failure fared much worse if they had elevated serum troponin, W. Frank Peacock IV, M.D., said in a poster presentation at the annual meeting of the American College of Emergency Physicians.

The results should have a profound impact on controversy about the clinical implications of elevating troponin in patients with heart failure, several speakers said in a session discussing cutting-edge research in emergency medicine at the meeting.

The analysis of data on 67,924 patients in the Acute Decompensated Heart Failure National Registry (ADHERE) showed that 6% had elevated troponin levels, and the rest were considered troponin-negative. Patients with elevated serum troponin were more likely than troponin-negative patients to develop systolic heart failure (61% vs. 51%) or undergo coronary artery bypass grafting (4% vs. 1%), intraaortic balloon counterpulsation (3% vs. less than 1%), mechanical ventilation (11% vs. 4%), or cardioversion (3% vs. 2%), said Dr. Peacock of the Cleveland Clinic and his associates.

Patients with acute decompensated heart failure and elevated serum troponin also had longer hospitalizations (median 5.1 vs. 4.1 days) and longer ICU stays (a median of 2.9 vs. 2.3 days) and were more likely to die in the hospital (8% vs. 3%), compared with troponin-negative patients.

The study defined elevated serum troponin as a level of at least 1 ng/mL for troponin I or at least 0.1 ng/mL for troponin T. Patients with levels below those cutoffs were considered troponin-negative.

"This [study] is important, because cardiologists everywhere—particularly our heart failure cardiologists—tend to pooh-pooh troponin leaks," according to Judd E. Hollander, M.D., professor of emergency medicine at the University of Pennsylvania, Philadelphia.

Elevated troponin in heart failure does not necessarily indicate underlying coronary disease, he said. "It's not something that cardiologists can fix in the cath lab—and that's what cardiologists look for. What this doesn't tell us is whether there's something we can fix in the hospital to decrease that mortality" associated with elevated troponin, he added.

Charles V. Pollack Jr., M.D., agreed: Cardiologists "tend to talk about benign troponin leaks. We've got to be careful about that." Particularly in older patients, elevated troponin has been a marker for sick patients in studies of sepsis, shock, chest pain, or congestive heart failure. "It's a worrisome marker and should be treated as such," said Dr. Pollack, chair of emergency medicine at the University of Pennsylvania.

'People are realizing [that elevated troponin] is a good marker of high-risk patients independent of whether the arteries have blockages or not.'

Troponin is a structural protein, and elevated levels are produced by cell death, noted Brian J. O'Neil, M.D., of Wayne State University, Detroit. "These are not 'leaks,' " he said.

In a separate interview, cardiologist Christopher P. Cannon, M.D., agreed that some of his colleagues have been misled by the common use of elevated troponin levels as a marker for acute coronary syndrome. When catheterizations found no arterial blockage in some patients with elevated troponin, the marker gained a reputation for false positives.

"We've learned that there are other things that cause elevations in troponin. We're all learning how to use this in these other patient groups. People are realizing it's a good marker of high-risk patients independent of whether the arteries have blockages or not," said Dr. Cannon of Brigham and Women's Hospital, Boston.

Previous studies have shown that troponin is a biomarker for myocardial injury. In earlier studies of patients hospitalized for heart failure, troponin elevations have been associated with lower ejection fractions, worse functional status, repeat hospitalizations for heart failure, and death. Studies on the clinical implications of troponin in heart failure are few, however, and have been plagued by methodological problems.

Although speakers at the emergency medicine meeting lauded the current study for the number and breadth of patients in the database, Jerome R. Hoffman, M.D., pointed out one major limitation: possible incorporation bias. Higher rates of procedures and longer hospitalizations may be due to physicians' reactions.

"When somebody tells you a patient has a high troponin level, you might keep them in the hospital or ICU a little longer. It may be a self-fulfilling prophecy" and not necessarily an appropriate step, said Dr. Hoffman of the University of California, Los Angeles.

Cardiologist Sorin J. Brener, M.D., called the study "important and well executed" but agreed with Dr. Hoffman's criticism. A multivariate logistic regression analysis controlling for the differences between patients in the two troponin groups would be necessary to isolate the independent effect of elevated troponin on outcomes, he said in a separate interview. "Elevated troponin levels are indeed a marker of adverse prognosis and cannot be ignored. Unfortunately, more often than not there is no specific intervention tailored to this finding in patients with decompensated heart failure that one would not apply in patients without elevated troponin," said Dr. Brener, director of the angiography core laboratory at the Cleveland Clinic. ■

Adverse Outcomes Tied to Elevated Troponin in Heart Failure

Adverse event	Troponin-positive group	Troponin-negative group
In-hospital mortality	8%	3%
CABG	4%	1%
Intraaortic balloon counterpulsation	3%	1%
Cardiac catheterization	24%	10%
Mechanical ventilation	11%	4%
Cardioversion	3%	2%
Time in ICU/CCU	2.9 days	2.3 days
Length of hospitalization	5.1 days	4.1 days

Note: Based on ADHERE data on 4,240 troponin-positive and 63,684 troponin-negative patients with decompensated heart failure.
Source: Dr. Peacock

Heart Failure More Common in Rheumatoid Arthritis Patients

BY TIMOTHY F. KIRN
Sacramento Bureau

SAN ANTONIO — Rheumatoid arthritis patients develop heart failure more frequently than the general population, and this increase does not appear to be explained by traditional risk factors, Cynthia Crowson said at the annual meeting of the American College of Rheumatology.

There have been many studies of heart disease in rheumatoid arthritis, but no one has previously looked at heart failure in particular, said Ms. Crowson, a statistician at the Mayo Clinic, Rochester, Minn.

The study followed 575 rheumatoid arthritis patients and 583 control subjects from the time they were 50-60 years of age (mean age 57 years) for 11-15 years, to see how many developed heart failure and what role was played by known cardiovascular risk factors.

Over the course of the study, 165 of the rheumatoid arthritis (RA) patients developed heart failure, as did 115 of the control subjects.

A statistical analysis of the subjects with heart failure—one that took into account each individual's risk factors—indicated that risk factors such as sedentary lifestyle and smoking played less of role in the heart failure of the RA patients than did that of the controls. Instead, the pathogenesis of RA itself may be to blame for the rates of heart failure, Ms. Crowson suggested.

Overall, the analysis indicated that 83% of the heart failure in the control subjects could be attributed to known cardiovascular risk factors and ischemic heart disease. By comparison, 45% of the heart failure in the rheumatoid arthritis patients could be attributed to such factors.

In the control subjects, 64% of the risk of heart failure was attributable to hypertension, but only 18% of the risk was associated with hypertension in the rheumatoid arthritis patients.

A history of ischemic heart disease (myocardial infarction, silent myocardial infarction, angina, or a revascularization procedure) was present in 26% of the control subjects, but only 17% of the risk in the RA patients.

Smoking accounted for 14% of the attributable risk in the control subjects, but only 3% in RA patients.

Body mass index tended to be fairly similar in the two groups; 23% of the RA patients had a BMI greater than 30, compared with 24% of the controls.

Smoking or a history of smoking was more common in the RA patients, but not dramatically so (55% versus 45%). ■

DATA WATCH

Top 10 Causes of Death, 2002

Diseases of the heart	696,947
Malignant neoplasms	557,271
Cerebrovascular diseases	162,672
Chronic lower respiratory diseases	124,816
Unintentional injuries	106,742
Diabetes mellitus	73,249
Influenza and pneumonia	65,681
Alzheimer's disease	58,866
Nephritis, nephrotic syndrome, and nephrosis	40,974
Septicemia	33,865

Source: Centers for Disease Control and Prevention

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