CT Angiography Speeds Triage for Chest Pain

BY DAMIAN McNAMARA
Miami Bureau

MIAMI BEACH — Coronary CT angiography might improve detection of significant coronary risk in an emergency department and help physicians decide which chest pain patients can be discharged, according to a presentation at a symposium on emergency radiology sponsored by Baptist Health South Florida.

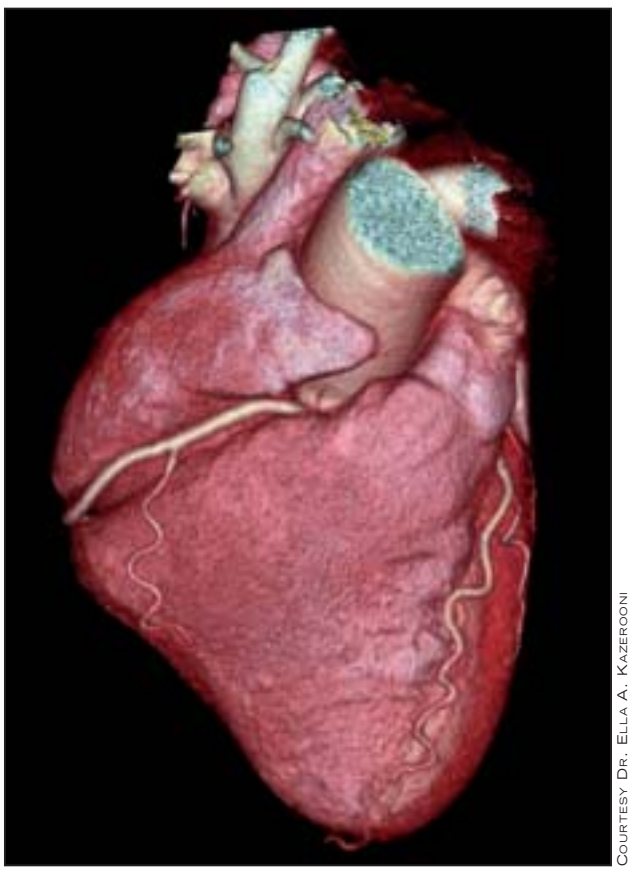
"In low-risk patients, we need a test with a high negative predictive value. Essentially we are screening for [those patients] we can send home. Many people think coronary CTA is that test," Dr. Ella A. Kazerooni said.

Nonspecific chest pain is the second most common reason that people seek emergency department care, but only about 10%-15% of those patients have an acute coronary syndrome. "On the other hand, 2%-5% with acute coronary syndromes are mistakenly sent home. Often they do not meet risk criteria. And later they are admitted with a severe MI, or they die at home," said Dr. Kazerooni, professor and director of the division of cardiothoracic radiology at the University of Michigan, Ann Arbor.

An estimated 5 million to 8 million Americans present to an emergency department with nonspecific chest pain each year, she said. The approximate cost for taking care of these patients is $10 billion.

Identification of which low-risk patients will progress to an acute coronary event has proved difficult, Dr. Kazerooni said. Other researchers performed a meta-analysis to determine potential risk factors (JAMA 1998;280:1256-63), but most likelihood ratios were not robust enough "to say 'send them home,' " said Dr. Kazerooni.

A total of 240 patients (12%) had a confirmed cardiac etiology among those presenting with chest pain during a 1-month study at the University of Michigan emergency department chest pain center. This finding supports what is in the literature, Dr. Kazerooni said. Annually, the mean length of stay is 21 hours, and the total room cost alone for this group of patients is close to $4 million.



COURTESY DR. ELLA A. KAZEROONI

The high negative predictive value of normal coronary CT angiograms like this one, in a 52-year-old woman presenting with chest pain, can expedite diagnosis.

"If you can do something to expedite triage, you can diagnose them earlier, at a lower cost, and use those rooms for other patients," Dr. Kazerooni said.

What is the role of coronary CT angiography in the emergency department? Immediate coronary CT angiography can safely triage low-risk acute chest pain patients home if they have negative ECG/enzymes for ischemia/infarction, Dr. Kazerooni said. "Also, it can provide a significant reduction in length of stay and cost of care."

In another study, 31 patients who presented to an emergency department with at least 30 minutes of chest pain had coronary CT angiography performed (Circ. J. 2005;69:1047-51). These patients "were already going to the cath lab, so it was not a low-risk population," Dr. Kazerooni said. A total of 93% of the patients were men, and 71% had acute coronary syndrome. "Remember I told you in general 12%-15% of patients [have acute coronary syndrome] in other studies, so this was biased." This was essentially a feasibility study that demonstrated coronary CT angiography can be performed in an emergency department, she added.

Some of the best data on coronary CT angiography in the emergency department came from another study that found 75% of 69 patients had no significant CT findings, Dr. Kazerooni said (Am. J. Roentgenol. 2005;185:553-40). About half the participants were men. Outside of the study, 45 patients would not have had a chest CT. A total of 19% patients had significant CT findings that explained their chest pain. There were two false negative CT findings because the images suffered from motion artifact, she said.

The 16-slice ECG-gated multidetector CT had a negative predictive value of 96% in this study. "They concluded this was a pilot study demonstrating feasibility, and that the greatest potential is for exclusion of significant coronary disease," Dr. Kazerooni said.

A prospective, blinded study of 103 patients, 60% of whom were men, found CT angiography had a negative predictive value of 100% (Circulation 2006:114:2251-60).

Despite its impressive negative predictive value, the safety of screening nonspecific chest pain patients in the emergency department with coronary CT angiography still needs to be established, Dr. Kazerooni said. ■

NSAID Use at Time of MI Increases Risk of Death by 29%

BY BRUCE JANCIN
Denver Bureau

CHICAGO — Being on a nonsteroidal anti-inflammatory drug when an ST-elevation MI strikes is associated with markedly worse 30-day outcomes in fibrinolytic-treated patients, Dr. C. Michael Gibson said at the annual scientific sessions of the American Heart Association.

This new observation, coupled with the recent report that patients placed on an NSAID after MI had worse outcomes, indicates the peri-infarct period "should be a nonsteroidal-free time," according to Dr. Gibson, director of the Thrombolysis in Myocardial Infarction (TIMI) data coordinating center at Brigham and Women's Hospital, Boston.

"I think if someone [with an MI] is on an NSAID, you probably need to be very vigilant in your antiplatelet therapy," he added.

The increased risk of developing an MI while on nonaspirin NSAIDs has received enormous publicity, with some cyclo-oxygenase-2–selective NSAIDs being withdrawn from the market for that reason. Dr. Gibson and his TIMI coinvestigators asked a different question: What's the impact of being on an NSAID when an MI occurs?

For answers, they conducted a retrospective secondary analysis of the prospective Enoxaparin and Thrombosis Reperfusion for Acute Myocardial Infarction Treatment, TIMI 25 (EXTRACT-TIMI 25) study, in which more than 20,000 patients undergoing thrombolysis for ST-elevation MI were randomized to enoxaparin or unfractionated heparin. Within 7 days prior, 572 had taken an NSAID, whereas 19,907 had not.

'If someone [with an MI] is on an NSAID, you probably need to be very vigilant in your antiplatelet therapy.'

DR. GIBSON

The incidence of recurrent MI within 30 days was 6.5% in the NSAID group, compared with 4.1% in patients who had not been on an NSAID. The rate of death or MI was 15.9% in NSAID users and 10.8% in nonusers. Incorporating indicators of pump failure into the outcome, the rate of death, recurrent MI, severe heart failure, or shock was 18.2% in NSAID users and 12.6% in nonusers. Most of these end points occurred within the first 7-10 days post MI, Dr. Gibson noted.

The two groups differed at baseline in several key ways. NSAID users were older, were more likely to have hypertension, had slightly worse renal function, and had a 20% prevalence of diabetes, compared with 15% in NSAID nonusers. Thus, NSAID users were a higher cardiovascular-risk cohort, and hence more likely to be on aspirin and other cardiac drugs.

After researchers adjusted for these and other potential confounders in a multivariate logistic regression analysis, NSAID use at the time of MI was associated with a 44% greater relative risk of recurrent MI and a 29% increased risk of death, MI, severe heart failure, or shock; both relative risks were statistically significant.

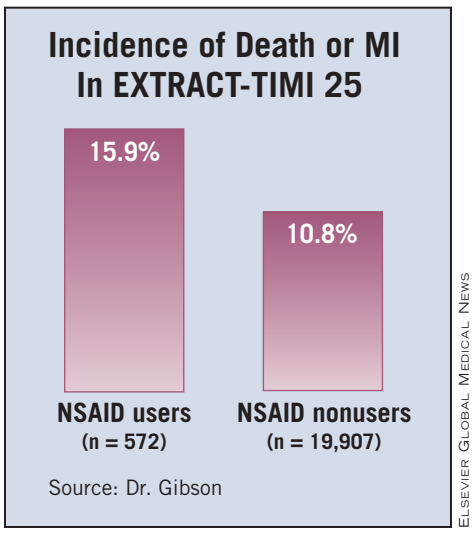
Dr. Gibson stressed that as a retrospective analysis of a study in which patients weren't randomized to NSAID use, these data must be considered hypothesis generating. There is no information as to which specific NSAIDs patients were on or what doses were used. It's possible that the worse outcomes in NSAID users were due to unidentified confounders.

Nevertheless, several biologically plausible potential mechanisms exist for the observed association between NSAID use at the time of a major MI and worse outcomes, he continued. It's known that many over-the-counter NSAIDs interfere with access of aspirin's binding site to cyclo-oxygenase-1, which might lessen aspirin's cardioprotective effect.

Moreover, NSAID inhibition of prostaglandin E2 may lead to hypertension and increased afterload, which could account for the observed high rates of heart failure and shock. NSAID inhibition of prostaglandin E1 can cause hyperkalemia, increasing risk of sudden arrhythmic death. And as is now well known, cyclo-oxygenase-2 inhibition may increase the risk of thrombosis.

Audience members expressed surprise at the trend for more TIMI-grade major and minor bleeding in NSAID nonusers: 3.8% compared with 3.5% in NSAID users. Dr. Gibson agreed this finding was "very odd."

"If these drugs are prothrombotic, that might explain it in part—but that's pure speculation," he said. ■



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