**CT Angiography Speeds Triage for Chest Pain**

**BY DAMIAN McNMARA**

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.

Non-specific 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 3 million to 8 million Americans present to an emergency department with nonspecific chest pain each year, she said. The approximate cost for treatment 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,’" 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,’" Dr. Kazerooni said.

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.

"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 be performed in an emergency department with 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;114:2221-60). 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 MI occurs 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 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.

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 days post MI, Dr. Gibson noted. The two groups differed at baseline in several key ways. NSAID users were older, 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. It’s continued. It’s known that many over-the-counter NSAIDs interfere with access of aspirin’s binding site to cyclooxygenase-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, cyclooxygenase-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: 5.8% 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.

**Incidence of Death or MI in EXTRACT-TIMI 25**

<table>
<thead>
<tr>
<th>NSAID users (n = 572)</th>
<th>NSAID nonusers (n = 19,907)</th>
</tr>
</thead>
<tbody>
<tr>
<td>15.9%</td>
<td>10.8%</td>
</tr>
</tbody>
</table>

Source: Dr. Gibson

**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.**