

Seasonal Flu Can Trigger MI and CV Death

BY MITCHEL L. ZOLER

Consistent evidence from more than 40 studies shows that seasonal influenza can trigger acute myocardial infarction and cardiovascular death.

The review also found “some evidence that influenza vaccines are effective at reducing the risk of cardiac events in those patients with established cardiovascular

disease,” said Dr. Charlotte Warren-Gash of the Centre for Infectious Disease Epidemiology at the Royal Free Hospital in London and her associates (*Lancet Infect. Dis.* 2009;9:601-9).

A large body of published work suggests that a range of acute and chronic bacterial and viral infections might be associated with an increased risk of acute MI. Influenza, however, stands out because of its substantial clinical and

public health effects, the authors wrote.

Influenza might acutely stimulate inflammation or coagulation. The virus may also affect endothelial function or stimulate coronary artery remodeling.

Their research identified 42 studies published between 1932 and 2008 that examined the outcomes of MI or death from cardiovascular disease in patients infected with influenza or an acute respiratory infection or in people who

received influenza immunization. They included 12 case-control studies that looked for associations between acute MI and either presumed influenza infection or nonspecific respiratory infection. Four other case-control studies examined links between flu immunization and acute MI or cardiovascular disease death.

The review also included two intervention studies that tested whether flu immunization prevented MI or cardiovascular death. The two intervention studies were relatively small, involving a total of just under 1,000 patients. The meta-analysis showed a statistically significant 61% reduction in cardiovascular deaths with influenza immunization, compared with no immunization, but the analysis also showed no significant effect of immunization on the MI rate.

The authors said that they had no conflicts of interest. ■



3rd in a series of 4

Addressing The Burden Of Cardiovascular Disease

Cardiovascular (CV) disease claims the life of 1 American every 37 seconds.¹ Because of this devastating burden, The National Heart, Lung, and Blood Institute (NHLBI) is currently developing comprehensive guidelines to help primary care physicians reduce the risk of CV disease in their patients (http://www.nhlbi.nih.gov/guidelines/cvd_adult/background.htm).

As you already know, patients suffering from hypertension, high cholesterol, diabetes, and obesity are at a higher risk for developing CV disease.¹ In addition to recommending lifestyle modifications, you may also be prescribing, as appropriate, lipid lowering agents, antiplatelet medications, and antihypertensive therapies in your practice to help reduce CV risk in your patients.² **What if** this isn't enough?

Minimizing The Impact Of Hypertension

As stated in the Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (JNC 7), the relationship between blood pressure and the likelihood of a CV event is continuous, consistent, and independent of other risk factors. As blood pressure increases, so does the risk of experiencing a CV event.² Again, you can reduce this risk in patients with hypertension by encouraging lifestyle modification to achieve BP goal.² When lifestyle modification is not enough, however, an antihypertensive therapy should be added to the treatment regimen.² There are a number of factors to consider when prescribing an antihypertensive therapy:

- **What if my patient does not tolerate initial therapy?**
 - Switch to another class of antihypertensive agent that is proven to reduce CV events²
- **What if BP control is not maintained in high-risk patients?**
 - Failure to maintain tight BP control over the long term in high-risk patients will not sustain the CV benefits gained by BP reduction,³ so consider an agent with long-term efficacy
- **What if you could go beyond BP control?**
 - Some antihypertensive therapies have clinical data demonstrating CV risk reduction, allowing you to optimize CV management by choosing an antihypertensive that goes beyond BP control

Before choosing an antihypertensive therapy, revisit current formulary access information to help you select the best antihypertensive agent available to your patient.

References:

1. American Heart Association. Heart Disease and Stroke Statistics – 2009 Update. Dallas, Texas: American Heart Association; 2009. ©2009, American Heart Association.
2. Chobanian AV, Bakris GL, Black HR, et al. Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure. *Hypertension.* 2003;42:1206-1252.
3. Holman RR, Paul SK, Bethel MA, et al. Long-term follow-up after tight control of blood pressure in type 2 diabetes. *N Engl J Med.* 2008;359:1565-1576.



RSV Appears to Cause Heart Damage Directly

SAN FRANCISCO — Respiratory syncytial virus itself, and not the bronchiolitis associated with the infection, appears to be the cause of the heart damage often seen in young children with the virus, according to a prospective study involving 74 children.

All 74 children were less than 12 months of age and were admitted to the hospital for bronchiolitis. Dr. Susanna Esposito explained in a poster at the Interscience Conference on Antimicrobial Agents and Chemotherapy. Aside from their bronchiolitis, all 74 were healthy.

The investigators from the University of Milan collected specimens with nasopharyngeal swabs to detect respiratory syncytial virus (RSV) types A and B. As it turned out, 35 patients (47%) tested positive for RSV infection, and the remaining 39 (53%) did not.

Patients with RSV had significantly more cardiac arrhythmias and a significantly greater degree of abnormal heart rate variability than those without RSV. For example, approximately 25% of the patients with RSV had cardiac arrhythmias, compared with about 5% of those without RSV. Approximately 60% of the patients with RSV exhibited abnormal heart rate variability, compared with approximately 40% of those without RSV.

The heart involvement appeared to be related to an RSV viral load of 100,000 copies per milliliter or more, and not to drug use or the disease's severity.

“This last finding suggests that RSV can be the direct cause of the heart damage and that arrhythmias can be found also in children with very mild RSV bronchiolitis in whom pulmonary hypertension and lung damage are nonexistent or marginal,” wrote the investigators, who reported that they had no conflicts of interest.

—Robert Finn