One-Time Pulmonary Edema Prognosis

1. Does a single episode of hypertensive crisis complicated by pulmonary edema imply a diagnosis of chronic congestive heart failure (CHF) with its attendant poor prognosis and increased mortality?

Question submitted by: Dr. Keith Graham, Campbell River, British Columbia

Grouped together, patients with acute heart failure represent the most common discharge diagnosis in patients > 65-years-old and have high mortality and hospital readmission rates.[1] However, the clinical syndrome of acute heart failure comprises a heterogeneous group of disorders, including pulmonary edema, hypertensive crisis, worsening exacerbated CHF and cardiogenic shock and spans a wide prognostic spectrum. The long-term prognosis of patients with acute heart failure syndromes depends upon their left ventricular function, the presence and severity of coronary artery disease and comorbid factors, such as atrial fibrillation and Type 2 diabetes mellitus.[2] Patients who develop acute pulmonary edema in the context of a hypertensive crisis need to undergo further diagnostic assessment (after they have been rescued from the jaws of death), to better determine their long-term prognosis.

References
1 De Luca L, Fonarow GC, Adams KF Jr, et al: Acute Heart Failure Syndromes: Clinical Scenarios And Pathophysiologic Targets For Therapy. Heart Fail Rev 2007; 12(2):97-104.
2 Flaherty JD, Bax JJ, De Luca L, et al: Acute Heart Failure Syndromes In Patients With Coronary Artery Disease Early Assessment and Treatment. J Am Coll Cardiol 2009; 53(3):254-63.

Answered by:
Dr. Theodore Fenske

Testing for Atypical Chest Pain

2. A 75-year-old mildly overweight smoker who plays hockey, develops atypical chest pain but has a normal stress test. Do I push the investigating? What are my criteria for pursuing it further?

Question submitted by: Dr. Mitch Shulman, Cote Saint Luc, Quebec

I presume the patient is a male and has no other CV risk factors. If he achieved 10 METS or more on the Exercise Treadmill Test, without noteworthy symptoms or ECG changes, I would not investigate further.

If he were diabetic, hyperlipidemic, hypertensive or had a strong family history, stress imaging via sestamibi or echocardiography would be warranted.

Answered by:
Dr. Thomas Wilson

Elevated Triglycerides in Association with Increased CV Risk

3. Elevated triglycerides are associated with an increased CV risk. Have there been studies that show lowering triglycerides will reduce CV mortality?

Question submitted by: Anonymous

If we base our practice on evidence-based medicine, then the quick answer to your question is that no evidence exists that treating isolated high triglyceride levels, in the absence of other risk factors, prevents coronary events. The association of elevated triglycerides with coronary events weakens when the studies are controlled for factors such as diabetes, HDL levels and LDL-C levels, BMI and other known risk factors.

As you know, the most impressive risk reductions demonstrated come from patients with the triad of low HDL, high LDL and high triglycerides. All studies showing improved outcomes have simultaneously increased HDL while lowering triglycerides. It appears that all professional societies recommend that elevated triglycerides should prompt a rigorous identification of risk factors for CV morbidity and mortality high dietary fat, excess alcohol, drugs such as steroids, β-blockers and high estrogen OCs and medical conditions such as hypothyroidism, renal failure, liver disease and lupus and that LDL lowering remains the primary target for therapy based on the risk profile.

Answered by:
Dr. Wayne Warnica

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Inflammation Causing Atherosclerotic Heart Disease

4. What kind of inflammatory process causes atherosclerotic heart disease, besides lipids and abdominal obesity? Are there other pathologies involved?

Question submitted by: Anonymous

Inflammation is a key etiologic component of acute coronary syndromes and coronary atherosclerosis. The causes of inflammation include lifestyle factors (*e.g.,* smoking, physical inactivity, obesity), systemic inflammatory diseases (*e.g.*, rheumatoid arthritis, influenza) and metabolic abnormalities (*e.g.,* diabetes, dyslipidemia).

The mechanisms whereby these cause plaque deposition or plaque rupture are multifactorial and ultimately result in endothelial dysfunction and disruption.

Of the many inflammatory markers studied, C-reactive protein (CRP) has gained prominence since the publication of the Justification for the Use of Statins in Primary Prevention (JUPITER) Trial, in which CRP was used to identify a high-risk group of primary prevention patients, who were shown to benefit from statin therapy. The clinical trials of antibiotics to treat coronary atherosclerosis have not demonstrated benefit.

Multiple biomarkers and imaging modalities are currently being studied in order to achieve the "holy grail" of preventive cardiology—the identification of inflamed vulnerable plaques before they rupture.

Answered by:
Dr. Brett Heilbron

> Inflammation is a key etiologic component of acute coronary syndromes and coronary atherosclerosis.