Editorial

Hypertroponinemia, Structural Cardiac Disease, and Stroke Mortality

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See related article, p 1226.

High cardiac troponin T (cTn) levels reflect myocardial damage in heart failure patients and predict poor prognosis in various cardiomyopathies and stroke. Cerebrovascular and cardiovascular diseases are major causes of death and disability worldwide. Ischemic stroke is a frequent complication in cardiac diseases, and cardiac complications commonly cause early clinical worsening and death after stroke. Acute myocardial infarction is a common complication of acute ischemic stroke (AIS); ≈4% of AIS patients will die from myocardial infarction in the 3 months after stroke, and 19% will suffer a cardiac event.[1,2] AIS is an established cause of nonacute myocardial infarction hypertroponinemia, previously reported as 20% to 60%.[3–5]

Can these risk factors, such as cTn elevation and cardiac changes, influence mortality after stroke?

To this regard, Wrigley et al[6] analyzed data of biracial population of 1.3 million is largely representative of the United States in terms of median age, proportion of black subjects, and economic status. The analysis was done in adult patients (aged 20 years and older) with AIS who presented to an emergency department.

The authors investigated the positivity of serum cTn levels and echocardiogram testing within a large biracial AIS population (1.3 million), and the association with poststroke mortality. Hypertroponinemia was defined as elevation in cTn above the standard 99th percentile. Of 1999 AIS cases, 85.3% had a cTn drawn, and 79.5% had echocardiograms. Hypertroponinemia occurred in 20.7% of these patients, and 10.1% had echocardiogram findings of clinical interest (eg, intracardiac thrombus 0.9%, low ejection fraction ≤35%–7.8%, valvular vegetation 0.6%, and akinetic wall segment 3.6%). When the concomitant myocardial infarction (3,5%) was excluded from hypertroponinemia was also associated with increased mortality at 1 (35%) and 3 years (60%).[6]

The results from this elegant and sophisticated statistical model evidence that the increase of cTn in patients with structural heart disease and who suffered a stroke has the highest risk of mortality. However, the results cannot be interpreted as if they reflected the real picture throughout the world.

In fact, the increased cTn concentrations in acute coronary syndrome identify patients with ongoing cardiomyocyte necrosis who are at increased risk for adverse outcome in patients with various cardiovascular, that is, conditions heart failure, stroke, myocarditis, Takotsubo cardiomyopathy, aortic dissection, supraventricular arrhythmias, valve disease, pulmonary arterial hypertension, and in the perioperative setting.[7]

Madsen et al[8] reported a study with 487 patients with suspected stable typical angina pectoris (AP) underwent invasive or computed tomography coronary angiography (significant stenosis ≥50%). Typical AP and high-sensitive troponin I (hs-TnI) elevation were associated with increased risk of having significant coronary artery disease (typical AP, odds ratio, 3.46; hs-TnI, odds ratio, 1.50) and experiencing future cardiovascular events (typical AP, hazard ratio, 2.64; hs-TnI, hazard ratio, 1.26). Patients in the lowest hs-TnI tertile, without typical AP (n=107), had a 1.9% absolute risk of significant coronary artery disease and a 3.7% absolute risk of long-term cardiovascular events.[8]

In another study, the authors evaluate the prognostic performance of high-sensitivity cardiac troponin T in a low-risk outpatient population presenting for nonsecondary and secondary prevention. In 693 patients with a median follow-up of 796 days, the authors observed 16 deaths, 32 patients with end point 2, and 83 patients with end point 3; Hs-cTnT provides excellent risk stratification on all-cause mortality, acute myocardial infarction, and stroke.[9]

Pokharel et al[10] evaluated associations between metabolic syndrome and groups with similar number of metabolic syndrome components and incident heart failure hospitalization, coronary heart disease, stroke, and death using high-sensitivity cardiac troponin T categories after adjusting for risk factors/markers in 8204 with mean age of 63 years (56% women, 19% Blacks). Hs-cTnT is useful for identifying metabolic syndrome patients with increased hazards for coronary heart disease, death, and particularly heart failure.[10]

Elevated troponin levels are commonly found in patients with acute stroke, and ≈60% of stroke patients experienced an accompanying coronary artery disease, and in patients with acute stroke and elevated troponin levels.[11]

Electrocardiography abnormalities such as QTc prolongation, left ventricular hypertrophy Q wave, and ST elevation as cardiac variables associated with troponin elevation, and higher National Institutes of Health Stroke Scale score and insular cortical lesions as neurological variables associated with troponin elevation. Incidence of troponin elevation and QTc prolongation was increased further in combination with cardiac and neurological factors. Certain cardiac and neurological conditions in AIS may contribute to troponin elevation.[12]

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(***Stroke*. 2017;48:1134-1135.**
DOI: 10.1161/STROKEAHA.117.017061.)

***Stroke* is available at http://stroke.ahajournals.org**
DOI: 10.1161/STROKEAHA.117.017061

Among the limitations of the study, we can mention the noninclusion in the data of the diagnosis of the metabolic syndrome, the waist circumference, the measurement of the triglycerides, and the medications in use that can influence the risk of stroke and the mortality.

Another limitation was related to the stroke culprit artery by cerebral angiography, as well as the neutrophil count and the neutrophil ratio for lymphocytes that could influence mortality.

In conclusion, this study provides an associated with hypertroponinemia and AIS and with structural heart disease predicts a higher mortality.

The encouraging results of this analysis should be used to formally implement early mortality assessment and prevention standards in patients presenting with stroke, increased cTn, and structural heart disease.

Disclosures

None.

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KEY WORDS: Editorials ■ acute coronary syndrome ■ heart failure ■ myocardial infarction ■ stroke ■ troponin

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Stroke. 2017;48:1134-1135; originally published online April 5, 2017;
doi: 10.1161/STROKEAHA.117.017061
Stroke is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231

Print ISSN: 0039-2499. Online ISSN: 1524-4628