Increased cardiovascular risk associated with non-cardiac chest pain in patients with a prior psychiatric hospitalization: an opportunity and challenge for both the psychiatrist and the cardiologist

Bertram Pitt*

Division of Cardiology, University of Michigan School of Medicine, Ann Arbor, MI 48109-0366, USA

Online publish-ahead-of-print 1 December 2011

This editorial refers to 'Prior psychiatric hospitalization is associated with excess mortality in patients hospitalized with non-cardiac chest pain: a data linkage study based on the full Scottish population (1991–2006)'†, by M. Gillies et al., on page 760

The evaluation of patients with chest pain is a common problem for every cardiologist and one of the most frequent reasons for presentation to the emergency room. After diagnostic evaluation, only 15–25% of patients with chest pain are found to have an acute coronary artery syndrome (ACS).1,2 Improved strategies for stress testing, myocardial imaging, and the availability of sensitive biomarkers for myocardial damage such as high sensitivity troponin have reduced the risk of missing the diagnosis of an ACS. Once coronary artery disease, myocardial ischaemia, and/or injury are ruled out, both the patient and the physician often feel relieved. A specific diagnosis such as oesophageal reflux, peptic ulcer disease, herpes zoster, costochondritis, pulmonary embolism, or panic disorder often leads to specific therapy and pain relief. However, in many instances, despite intensive evaluation and reassurance as to the usually benign nature of non-cardiac chest pain (NCCP), the cause of the pain remains uncertain and/or the pain may reoccur with consequent patient frustration and distress. In these instances, the patient may be prescribed an antidepressant and/or enrolled in pain coping skills training (CST). For example, in a recent study, Keeffe et al.3 randomized patients with a diagnosis of NCCP to one of four treatments: (i) CST plus the antidepressant sertraline; (ii) CST plus placebo; (iii) sertraline alone; or (iv) placebo alone. CST and sertraline either alone or in combination were found to reduce pain intensity and pain unpleasantness significantly. The authors suggest that the combination of CST and sertraline may have the greatest promise in that when compared with placebo alone it reduced not only pain but also pain catastrophizing and anxiety. Thus it appears that there may be an important role for the psychiatrist in the care of patients with NCCP. However, there may also be an important role for the cardiologist in the care of patients with a psychiatric hospitalization.

Gillies et al.,4 on the basis of a population-based retrospective cohort study of > 150 000 first hospital discharges for NCCP in Scotland during the period between 1991 and 2006, have found that 3514 (4.4%) men and 3136 (3.9%) woman who had a first NCCP hospitalization had a prior psychiatric hospitalization during the 10 years preceding the incident hospitalization for NCCP. Patients with a diagnosis of NCCP and a prior psychiatric hospitalization, after adjusting for socio-economic deprivation and co-morbid diabetes mellitus and hypertension, had a significantly higher incidence of all-cause and cardiovascular disease-specific death at 1 year compared with those without a prior psychiatric hospitalization. The relative risk of total and cardiovascular-specific mortality associated with a prior psychiatric hospitalization was present in both men and woman, especially in the younger age groups. For example, the 1 year hazard ratio for all-cause death for men < 40 years of age the hazard ratio for all-cause mortality was 3.71, and 2.81 for cardiovascular mortality, whereas in woman < 40 years of age the hazard ratio for all-cause mortality was 2.94, and 3.71 for cardiovascular mortality compared with those without a prior psychiatric hospitalization. In part, the increased risk associated with NCCP in patients with a prior psychiatric hospitalization may have been due to the failure to detect coronary artery
disease and/or myocardial ischaemia in these patients whose symptoms are often difficult to interpret. The increased cardiovascular risk in patients with NCCP and a prior psychiatric hospitalization should therefore prompt a thorough review of the diagnostic parameters that led to a diagnosis of NCCP. In those individuals where high sensitivity troponin and stress testing are negative and a specific non-cardiac cause of the chest pain has not been identified, one might consider the use of N-terminal pro brain natriuretic peptide (NT-proBNP) which in a recent study had a sensitivity of 73% and a negative predictive ability of 90%, and/or the measurement of unbound free fatty acids (FFAuS) which had a sensitivity of 75%, a specificity of 72%, and a negative predictive ability of 92% while significantly adding to the net reclassification improvement analysis from high sensitivity troponin. In view of their finding of an increased cardiovascular risk associated with NCCP in patients with a prior psychiatric hospitalization, Gillies et al. suggest that the diagnosis of NCCP in patients with a prior psychiatric hospitalization is an opportunity to modify cardiac risk factors. Risk factor and lifestyle modification may, however, be particularly difficult in these patients, especially in younger individuals. Given the relatively short time period between the first hospitalization for NCCP and death (1 year) in those with a prior psychiatric hospitalization, risk factor and lifestyle modification may also be too late at the time of the NCCP hospitalization to alter the natural history of these patients. Thus, the initial episode of psychiatric hospitalization or possibly the diagnosis of psychiatric illness rather than the first episode of NCCP should be the time to consult a cardiologist, and the stimulus for intensive cardiac evaluation and risk factor control to prevent the development of coronary artery disease and its consequences.

Although the exact cause of the psychiatric hospitalization preceding the diagnosis of NCCP in the study by Gillies et al. is uncertain, as is the specific cause of the increase in total and cardiovascular mortality, it is known that patients with a diagnosis of anxiety or depression have an increased cardiovascular risk. Patients with a psychiatric illness may be at increased cardiovascular risk due to an inappropriate lifestyle such as smoking, alcohol abuse, poor diet, and/or a sedentary lifestyle. There is, however, evidence that patients, for example with anxiety disorder and those with depression, have an increased cardiovascular risk independent of their lifestyle. Although the mechanistic links between psychiatric illness and cardiovascular mortality are incompletely understood, patients with anxiety disorder and depression have been shown to have increased inflammatory cytokine expression, decreased vascular nitric oxide (NO) availability, decreased circulating endothelial progenitor cells, endothelial dysfunction, sympathetic nervous system activation, decreased parasympathetic activity, neurohumoral activation, and increased platelet activation. These mechanisms are common to patients with both vascular and psychiatric disease. It can be postulated that these mechanisms in conjunction with lifestyle triggers, such as loss of a loved one, loss of a job, or a natural disaster, could predispose to sudden cardiac death. Further understanding of the mechanistic links between psychiatric illness and the development of cardiovascular risk, and further insight into the co-morbid conditions that predispose to cardiovascular disease that are common to both, such as obstructive sleep apnoea, as well as the triggers precipitating sudden cardiac death will be necessary to develop strategies to detect subclinical coronary vascular disease and to design appropriate therapeutic strategies to reduce the increased cardiovascular and total mortality associated with psychiatric hospitalization and subsequent NCCP. One might, for example, consider the use of an angiotensin-converting enzyme inhibitor or angiotensin receptor blocker and/or a statin, which have been shown to reduce inflammatory cytokine activation, improve endothelial function, restore autonomic nervous system balance, and reduce platelet activation. The value of these agents under these conditions is uncertain, as is the specific cause of the increase in total and cardiovascular mortality, it is known that patients with a diagnosis of anxiety or depression have an increased cardiovascular risk.
circumstances will, however, require prospective evaluation in adequately powered randomized studies. New strategies to prevent the development of coronary artery disease in patients with a psychiatric illness such as anxiety or depression may be of special importance in view of the lack of evidence that psychiatric medications such as antidepressant medications decrease cardiovascular risk.6

Thus, while the exact mechanisms linking a prior psychiatric hospitalization and a first hospitalization for NCCP to increased cardiovascular and total mortality remain uncertain, we are indebted to Gilles et al.4 for pointing out the increased cardiovascular risk and the need for cardiovascular evaluation of these patients. The increasing evidence that both vascular disease and psychiatric illnesses such as anxiety and depression share common mechanisms suggests challenges and opportunities for both the psychiatrist and the cardiologist to improve risk detection and to prevent cardiovascular and total mortality in patients with psychiatric illnesses both with and without NCCP. One might consider a strategy such as outlined in Figure 1 for patients hospitalized with a psychiatric illness such as depression if we are to prevent the development of cardiovascular disease before the patient presents with chest pain and an increased risk of death. This will, however, require a further understanding of the links between psychiatric illness and cardiovascular disease as well as prospective evaluation.

Conflict of interest: none declared.

References


CARDIOVASCULAR FLASHLIGHT

Implantable cardioverter defibrillator avoids shock during electrocution

Ardan M. Saguner and Firat Duru

A 30-year-old electrician with an implantable cardioverter defibrillator (ICD) who had previous history of idiopathic ventricular fibrillation presented to emergency room after incurring electrocution injury while accidentally grasping a 230 Volt 50 Hz alternating current power line at work. He was in contact with the power line for approximately 3 s though he remained conscious. At presentation, the patient was clinically in stable condition. Physical examination revealed two grade IIa entrance burns of the right palm (Panel A, bold arrows) and one grade I exit burn of the left palm (Panel A, thin arrow), which served as the ground. The burns were surgically treated by cautious debridement. Subsequently, ICD interrogation revealed appropriate ‘noise reversion’ (Panel B) due to detection of continuous high-frequency sensing in the ventricular channel for 3.3 s avoiding shock delivery. No ventricular tachyarrhythmia was induced during electrocution. However, the heart rate was more rapid after the event, possibly due to sinus tachycardia. All ICD measurements were within the normal range. Twelve-lead surface ECG and the cardiac biomarkers remained normal after the event. The patient was discharged on the same day.