THE CCU CORNER

Blunt Cardiac Injury

BY GEORGE PHILIPPIDES, M.D., AND ERIC AWTRY, M.D. EDITED BY THOMAS J. RYAN, M.D.

The Patient

A 34-year-old man was the unrestrained driver in a head-on collision with a tree during which he sustained pelvic, femur, clavicular, and rib fractures, and also suffered a splenic rupture. He underwent emergent laparotomy, splenectomy, and reduction of the open femur fracture. Surgery was complicated by perioperative hypotension requiring transfusion and vasopressors. Postoperative ECG revealed a transient right bundle branch block and echocardiogram revealed hypokinesis of the right ventricle and the apex of the left ventricle. His creatine kinase was significantly elevated, although the MB fraction was normal, and the troponin was only mildly elevated.

He was diagnosed with a blunt cardiac injury, which was managed conservatively. Echocardiography several weeks later revealed resolution of left-ventricular and right-ventricular dysfunction.

The Problem

Blunt cardiac injuries (often inaccurately referred to as cardiac contusions) occur in 15%-30% of patients with thoracic trauma and may be associated with a variety of cardiac abnormalities, including conduction disturbances, myocardial hematoma, elevated cardiac biomarkers, ventricular septal defect, free wall rupture, and coronary artery injury. Often the cardiac injury goes unrecognized until complications develop.

Pathophysiology

The majority of blunt cardiac injury results from motor vehicle accidents; falls, crush injuries, sporting injuries, and assaults are less common causes. Cardiac injury may result from the direct impact on the heart by the sternum or compression of the heart between the sternum and the vertebrae. In addition, crush injuries of the abdomen and lower extremities may result in a sudden rise in venous return resulting in overdistension of the heart and subsequent rupture of the cardiac chambers or valves. The injured myocardium often contains patchy regions of hemorrhage that occur in a noncoronary distribution. The right ventricle is most often affected. When the left ventricle is involved, it is usually in the region of the interventricular septum or the apical anterior wall.

Clinical Findings

Patients with blunt cardiac trauma often have other significant thoracic injuries including sternal fractures, rib fractures, and pulmonary contusions. However, the absence of obvious chest wall trauma does not exclude blunt cardiac injury, especially in young patients in whom the chest cage is relatively flexible and can absorb significant force without resultant fracture. In one study, chest wall bruising was found in only 30% of patients with blunt cardiac injury, and although sternal fractures occur in 10%-70% of patients with blunt cardiac trauma, studies have failed to show a statistical link between the two injuries.

Patients may complain of chest pain; however, this may be difficult to distinguish from musculoskeletal injury. Similarly, the hypotension, hypoxemia, or shock associated with severe cardiac injury may be mistakenly attributed to blood loss and pulmonary injury in patients with significant thoracic trauma.

Diagnostic Modalities

There is no accepted standard for the diagnosis of blunt cardiac injury; however, the diagnosis should be suspected in any patient with significant chest trauma, especially if there are associated thoracic injuries. The ECG is often abnormal and may reveal sinus tachycardia, nonspecific ST or T wave abnormalities, conduction abnormalities (RBBB or various degrees of heart block), and premature atrial or ventricular contraction. More malignant arrhythmias are uncommon. Measurement of creatine phosphokinase–MB level is of uncertain utility in patients with significant muscular injury; however, cardiac troponin levels are highly sensitive markers of cardiac injury, are inversely related to cardiac function in the trauma patient, and may predict the risk of malignant arrhythmias.

While a normal ECG or biomarker level does not exclude cardiac injury, a recent study suggests that when both the ECG and troponin are normal at admission and 8 hours later, the incidence of significant blunt cardiac injury (i.e., cardiogenic shock, significant arrhythmias, or structural abnormalities) is exceedingly low. These patients may not require further cardiac evaluation or monitoring.

Echocardiography is currently the test of choice for identifying structural evidence of blunt myocardial injury (e.g., wall motion abnormalities, ventricular septal defect, valvular regurgitation, or pericardial effusion), and should be performed in any patient with thoracic trauma who has an abnormal ECG or hemodynamic instability. Transesophageal echocardiography should be considered in patients suspected of having traumatic cardiac injury when the transthoracic study is inadequate. Studies have shown the utility of radionucleotide imaging in the diagnosis of blunt cardiac injury; however, it may be impractical to obtain these studies in the trauma patient.

Management

Patients with an abnormal ECG, elevated biomarkers, or abnormal echocardiogram should be monitored on telemetry for at least 48 hours. Arrhythmias should be treated similarly to those occurring in other settings; β-blockers are the agents of choice for the treatment of supraventricular tachycardia or ventricular ectopy. There are no data to guide the management of trauma-induced ventricular dysfunction; in general, we have treated these patients similarly to those with other forms of cardiomyopathy. β-blockers and ACE inhibitors should be given in hemodynamically stable patients. In patients with severe left-ventricular dysfunction and cardiogenic shock, intravenous inotropic agents and/or intra-aortic balloon counterpulsation is occasionally required.

The main differential diagnosis of traumatic cardiac injury is an acute myocardial infarction. We have seen several patients who have suffered a myocardial infarction while driving, resulting in syncope, collision, and thoracic trauma. The diagnosis may be clarified by the patient's history or by the presence of regional ST abnormalities on ECG and a focal wall motion abnormality on echocardiography that correlates with a coronary distribution. Such patients should receive standard treatment for acute coronary syndrome (antiplatelet therapy, consideration of cardiac catheterization), provided their injuries allow such therapy.

Dr. Awtry *is assistant professor of medicine and director of education at Boston Medical Center.* Dr. Philippides *is assistant professor of medicine and director of the Coronary Care Unit at BMC. To respond to this column or suggest topics for consideration, write to our editorial offices or e-mail us at cardnews@elsevier.com.*

Noninvasive Ventilation Bests Oxygen for Acute Symptoms

BY MARY ANN MOON
Contributing Writer

Both continuous positive airway pressure (CPAP) and noninvasive intermittent positive-pressure ventilation (NIPPV) yield earlier resolution of dyspnea, respiratory distress, and metabolic abnormalities than does standard oxygen therapy in patients hospitalized with acute cardiogenic pulmonary edema, according to a report.

However, those treatments did not affect short-term mortality, the primary objective of the Cardiogenic Pulmonary Oedema (3CPO) trial.

Nonetheless, because of the clinical improvements, "We recommend that CPAP or NIPPV be considered as adjunctive therapy in patients with acute cardiogenic pulmonary edema who have severe respiratory distress or whose condition does not improve with pharmacologic therapy," said Dr. Alasdair Gray of the Royal Infirmary of Edinburgh and his associates.

CPAP maintains the same positive airway pressure support throughout the respiratory cycle; NIPPV raises airway pressure more during inspiration than expiration. Both methods, which deliver oxygen through a face mask, have shown benefit in case series or small trials that have been conducted primarily at single centers. "Therefore, it is uncertain whether these results are either generalizable or robust," Dr. Gray and his colleagues noted.

3CPO was a large, randomized, controlled trial in 26 emergency departments throughout the United Kingdom that compared the two techniques against standard oxygen therapy and against each other. A total of 367 patients were randomly assigned to standard oxygen therapy, 346 to CPAP, and 356 to NIPPV.

The mean patient age was 78 years, and subjects had marked tachycardia, tachypnea, hypertension, acidosis, and hypercapnia.

Both CPAP and NIPPV yielded greater reductions in dyspnea, heart rate, acidosis, and hypercapnia than did standard oxygen therapy, and the two methods performed similarly, the investigators said (N. Engl. J. Med. 2008;359:142-51).

Standard oxygen therapy was associated with a greater rate of failure due to respiratory distress, while both CPAP and NIPPV were associated with higher rates of noncompletion due to patient discomfort.

There were similar rates of tracheal intubation, admission to the critical care unit, and myocardial infarction among the three groups.

Unfortunately, there were no significant differences among the three groups in the primary end point of 7-day mortality, which was 9.5% with CPAP and NIPPV and 9.8% with standard oxygen therapy. The rates of 30-day mortality also were not significantly different (16.4% with CPAP and NIPPV vs 15.2% with oxygen).

Thus, early improvements in symptoms and surrogate measures of disease severity did not translate into improved short-term or long-term mortality for the two new techniques.

Previous trials have indicated that the physiologic improvements seen with noninvasive ventilation caused a reduced use of tracheal intubation, but this benefit was not observed in this study, Dr. Gray and his associates said. ■