Right Atrial and Ventricular Thrombi after Blunt Chest Trauma

Miin-Yaw Shyu, MD, Andrew Ying-Siu Lee, MD, PhD, Bor-Jen Lee, MD, Yen Chang, MD, Hao-Jei Wei, MD, Michael Chich-Kuang Chang, MD, Wen-Fung Chang, MD, and Tien-Jen Chen, MD

J Trauma. 2002;52:765–766.

Blunt chest trauma may provoke many different kinds of cardiac injuries ranging from myocardial contusion to cardiac tamponade, aortic tears and cardiac rupture, myocardial aneurysm, valvular insufficiency, septal defects, and coronary artery injury.[1] Intracardiac thrombus is an unusual complication of blunt cardiac injury. There was no incidence of thrombi in two prospective echocardiographic studies involving 213 patients who suffered blunt cardiac injury.[2,3] Others have reported transient left ventricular thrombi following blunt chest trauma.[4] We describe a young man with blunt chest trauma caused by the steering wheel in a car crash 3 years ago, who progressively developed congestive heart failure and subsequently large thrombi formation in both the right atrium and the ventricle together with pulmonary embolism. To our knowledge, this is the first reported case of echocardiographic demonstration of right atrial and ventricular thrombi occurring concomitantly after blunt chest trauma.

CASE REPORT

A 42-year-old man with no history of heart disease was admitted to our hospital with massive hemoptysis and dyspnea. He had a history of car crash with chest blunt trauma by the steering wheel 3 years ago. Although initially well after discharge, he experienced dyspnea on minimal exertion, paroxysmal nocturnal dyspnea, orthopnea, cough, hemoptysis, and pedal edema 1 year following the crash. He was treated symptomatically by local medical clinics until, on the date of admission, he had massive hemoptysis, dyspnea, and cyanosis of the lips.

On examination, the patient was dyspneic and cyanosed. His blood pressure was 120/60 mm Hg, pulse 108/min, respiration 26/min, and temperature 36°C. His jugular veins were markedly engorged. He had coarse breathing sounds with basal moist rales. The heart sounds were indistinct, with pansystolic murmur over the left sternal region, which increased in intensity on inspiration. The abdomen was soft and nontender. The liver and spleen were not felt. The patient had bilateral pedal edema. The remainder of the physical examination was normal.

Complete blood count and blood chemistry profile were normal. The electrocardiogram showed normal sinus rhythm with right axis deviation, left atrial enlargement, and ST-segment and T-wave changes. The chest radiograph showed cardiomegaly with pulmonary congestion and oligemia of the right lung, suggesting pulmonary embolism. Lung perfusion scan revealed no perfusion of the right lung, indicating high probability of pulmonary embolism. Chest CT showed cardiomegaly, pericardial and pleural effusion, complete occlusion of the right pulmonary artery, and filling defects in the right atrium. Abdominal sonogram was normal. Alpha-fetoprotein, carcinoembryonic antigen (CEA), and proteins C and S were within normal limit.

Echocardiography was performed (Fig. 1). There was right atrial and ventricular enlargement, septal and right ventricular hypertrophy, moderate pericardial effusion, and moderately severe tricuspid insufficiency, with severe generalized hypokinesis of the right but preserved left ventricular systolic functions. One large thrombus (4 × 3 cm) was detected in the right atrium, and multiple thrombi in the right ventricle were observed. The thrombi were smooth, homogenous, and well demarcated without a discrete attachment or evidence of tissue invasion and infiltration.

A diagnosis of intracardiac thrombi in the right atrium and ventricle, together with tricuspid insufficiency and pulmonary embolism, was made. Surgical thrombectomy, pulmonary artery embolectomy and endarterectomy, tricuspid repair, and patch repair of the right atrium were immediately performed, which subsequently confirmed the above diagnoses. The patient recovered and was discharged, maintained with Coumadin at our outpatient department. Follow-up echocardiography 5 months later showed no evidence of intracardiac thrombi, with minimal tricuspid insufficiency, and preserved right and normal left ventricular systolic functions.

DISCUSSION

Blunt cardiac injury is defined as cellular damage due to nonpenetrating chest trauma, resulting in muscle necrosis and

Submitted for publication March 12, 2001.
Accepted for publication August 14, 2001.

From the Division of Cardiology, Jen Ai Hospital, Tali (M.-Y.S., A.Y.-S.L., M.C.-K.C., W.-F.C., T.-J.C.), and Divisions of Cardiology (B.-J.L.) and Cardiovascular Surgery (Y.C., H.-J.W.), Taichung Veterans General Hospital, Taichung, Taiwan, Republic of China.
Address for reprints: Andrew Ying-Siu Lee, MD, PhD, Division of Cardiology, Jen Ai Hospital, Tali, Taichung, Taiwan, Republic of China.

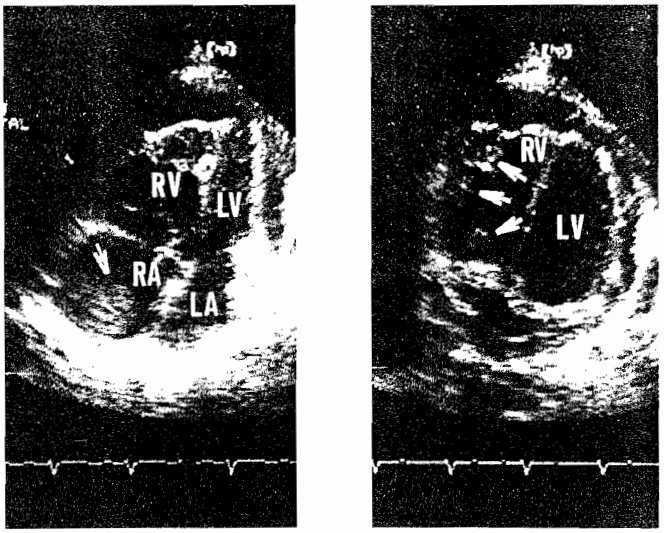

Fig. 1. *Apical two-dimensional echocardiograms showing intracardiac thrombi at right atrium* (left, arrow) *and ventricle* (right, arrows). *RA = right atrium; RV = right ventricle; LA = left atrium; LV = left ventricle.*

hemorrhagic infiltrates that may impair ventricular function.[5] The right heart is more often affected than the left because of its proximity to the sternum. The incidence and etiology of cardiac injury after blunt chest trauma vary widely. Mural thrombosis is an unusual complication of blunt cardiac injury. Timberlake and McSwain reported thromboembolism as a late complication of blunt cardiac trauma in 9 of a total of 14 patients.[6] To our knowledge, the occurrence of thrombi in both the right atrium and the ventricle concomitantly due to blunt cardiac injury as demonstrated in our case has never been reported.

Traumatic tricuspid insufficiently from blunt chest trauma is relatively uncommon.[7] It may be secondary to a severe elevation of the right ventricular intracavitary pressure from compression of the full heart in diastole. Chordal rupture, rupture of the papillary muscles, and leaflet tears may cause the subsequent tricuspid insufficiency. Pulmonary embolism is also a rare complication of blunt cardiac injury. Previous studies have reported an incidence of pulmonary embolism in chest trauma ranging from 0.2 to 1.29%.[8] The pathophysiological mechanisms remain to be elucidated, although a number of risk factors such as age, nature and severity of the injury, and prolonged immobilization have been suggested.[8]

In our patient, we speculate that his intracardiac thrombi formation may have resulted from direct myocardial injury after the blunt cardiac injury, which may cause traumatic tricuspid insufficiency and impaired right ventricular function. The abnormal wall motion may lead to aberrant blood flow dynamics within the heart and consequent thrombi formation in the right atrium and ventricle. His subsequent pulmonary embolism was most likely secondary to systemic embolization from the heart.

As blunt thoracic trauma has been associated with unusual complications such as valvular insufficiency, thrombus, embolism, and constrictive pericarditis, we suggest that systemic and serial investigation of all cases of chest trauma by transthoracic or, preferably, transesophageal echocardiography should be performed so as to avoid delayed or missed cardiovascular diagnoses or complications.

REFERENCES

1. Pretre R, Chilcott M. Blunt trauma to the heart and great vessels. *N Engl J Med.* 1997;336:626–632.
2. Fabian TC, Mangiante EC, Patterson CR, et al. Myocardial contusion in blunt trauma: clinical characteristics, means of diagnosis and implications for patient management. *J Trauma.* 1990; 28:50–57.
3. Hiatt JR, Yeatman LA, Child JS. The value of echocardiography in blunt chest trauma. *J Trauma.* 1988;28:914–922.
4. Hung JL, Wang CH, Cherng WJ. Left ventricular thrombus after seat-belt-related chest trauma. *J Trauma.* 1999;47:599–601.
5. Potkin RT, Werner JA, Trobaugh GB, et al. Evaluation of noninvasive tests of cardiac damage in suspected cardiac contusion. *Circulation.* 1982;66:627–631.
6. Timberlake GA, McSwain NE. Thromboembolism as a consequence of myocardial contusion: a new capricious syndrome. *J Trauma.* 1988;28:535–540.
7. Perlroth MG, Hazan E, Lecompte Y, Gougre G. Chronic tricuspid regurgitation and bifascicular block due to blunt chest trauma. *Am J Med Sci.* 1986;291:119–125.
8. Tuttle-Newhall JE, Rutledge R, Hultman S, Fakhry SM. Statewide population-based, time-series analysis of the frequency and outcome of pulmonary embolus in 318,554 trauma patients. *J Trauma.* 1997; 42:90–99.