Wakeboarding-related water impact trauma as a cause of fatal cardiac rupture

Jang Wen Su, FRCSCTh,^a Chong Hee Lim, FAMS,^{a,b} Ju Le Tan, FAMS,^c Yeow Leng Chua, FAMS,^a and Paul Peng Sun Chui, MRCPATH,^d Singapore

Wakeboarding is a very popular water sport, with high potential for injury.^{1,2} We report the case of a young male patient who had a severe cardiac contusion with tamponade from hemopericardium after a fall during wakeboarding. The subsequent progression to myocardial necrosis resulted in fatal left ventricular free wall rupture. We believe this to be the first reported case of wakeboarding-related severe, fatal cardiac injury in the literature.

Clinical Summary

We present the case of a 25-year-old man who fell from a 1-m height during wakeboarding. The automated trailer was traveling at a speed of 25 km/h when he fell prone into the water.

He presented 3 hours later with persistent dull central chest pain, increasing dyspnea, and orthopnea. He was hemodynamically unstable, with a heart rate of 140 beats/min and a blood pressure of 76/40 mm Hg. Clinically, he was in cardiac tamponade, as evidenced by increased jugular venous pressure and muffled heart sounds. Pulsus paradoxus was also detected on the arterial waveform. However, there was no sign of external chest injury.

A chest radiograph revealed a globular cardiac shadow. An electrocardiograph demonstrated sinus tachycardia of 141 beats/min with ST-segment elevation on the inferolateral leads. The creatine kinase-MB level was 73.5 μ g/L, the creatine kinase level was 1502 U/L, and the troponin-T level was 2.27 μ g/L. Arterial blood gas analysis showed metabolic acidosis. Based on urgent transthoracic echocardiography (TTE), the cardiac contractility was found to be markedly impaired, with akinetic lateral and inferior segments. The anterior segment was hypokinetic. Left ventricular ejection fraction (LVEF) was 15% to 20%. There was a circumferential pericardial effusion of 2.5 cm in thickness. Tamponade was featured echocardiographically by right atrial and ventricular diastolic collapse and a plethoric inferior vena cava. Urgent computed tomography of the thorax was performed, which further confirmed hemopericardium with no other injury (Figure 1).

The thickness of the myocardium was within normal limits, with no extravasation of contrast.

Subsequently, the patient underwent emergency exploratory left anterior thoracotomy and pericardial drainage. Left anterior thoracotomy was chosen over the subxiphoid approach to allow assessment of the right ventricle and the lateral aspect of the left ventricle. In addition, computed tomographic scanning excluded active extravasation of contrast and hence rendered conversion to full sternotomy unlikely. Intraoperatively, 350 mL of blood with blood clot was evacuated from the pericardial cavity. Patchy epicardial bruises were found on the anterolateral surface of the right ventricle. After drainage, there was no reaccumulation of blood within the pericardial space. Hence full sternotomy was not performed.

After pericardial drainage, his hemodynamic status improved remarkably, with resolution of tachycardia and stabilization of blood pressure. He was monitored in the cardiothoracic surgery intensive care unit for 36 hours. TTE was repeated on 2 occasions, which showed improvement of cardiac contractility with an LVEF of 40%. However, the inferior and posterior segments remained akinetic. There was no recollection of pericardial fluid. In view of persistent akinetic inferoposterior segments, coronary artery computed tomography was carried out, which did not show any coronary artery pathology. During the course of hospitalization, serial cardiac enzyme levels were on a sharp downward trend, and no cardiac arrhythmia was detected on telemetry.

On the fifth postoperative day, he was found to be in asystole. Postmortem examination was performed, which revealed extensive necrosis of the posterior left ventricular wall with free wall rupture (Figure 2).

Discussion

Blunt thoracic trauma is a common injury worldwide and is one of the leading causes of accident-related death.³ Blunt cardiac trauma

From the Departments of Cardiothoracic Surgery^a and Cardiovascular Medicine,^c National Heart Centre; Singapore General Hospital^b; and the Centre for Forensic Medicine,^d Singapore General Hospital, Singapore.

Received for publication March 15, 2007; accepted for publication April 19, 2007.

Address for reprints: Jang Wen Su, FRCSCTh, Department of Cardiothoracic Surgery, National Heart Centre, Mistri Wing, 17 Third Hospital Ave, Singapore 168752 (E-mail: bottle1001@yahoo.com).

J Thorac Cardiovasc Surg 2007;134:506-7

0022-5223/\$32.00

Copyright © 2007 by The American Association for Thoracic Surgery

doi:10.1016/j.jtcvs.2007.04.015

Figure 1. Computed tomographic scan demonstrating hemopericardium with preserved myocardial thickness.

Figure 2. Postmortem image showing necrotic posterior left ventricular rupture.

commonly manifests as myocardial contusion. These contusions can cause cardiac enzyme leaks, electrocardiographic abnormalities, and cardiac contractile dysfunction, making it difficult to differentiate a myocardial contusion from a peritraumatic myocardial infarction. In our patient, cardiac enzyme levels and serial ECG and echocardiographic results all indicated myocardial contusion in this setting of blunt thoracic injury.

Our patient experienced an enormous amount of compressive force on the heart between the sternum and vertebral body. Because of the much higher intracardiac pressure of the left heart, the degree of injury was much more severe than for the right ventricle. The resultant contusion led to myocardial necrosis and subsequent fatal rupture. Throughout the course of management, we experi-

enced difficulty in the attempt to diagnose the development of myocardial necrosis and prevent rupture. In retrospect, we acknowledge that magnetic resonance imaging of the heart might possibly provide more accurate information on the state of the myocardium. However, in a setting in which the patient's hemodynamic parameters and cardiac enzyme levels are sharply normalizing and serial TTE shows improved LVEF, magnetic resonance imaging is not clearly indicated.

Had an early diagnosis of impending rupture been made, because of the extent of injury, the options of treatment were still very limited. Possible options include total artificial heart implantation and cardiac transplantation, which are not readily available in most centers. Successful treatment options reported in the literature include infarct exclusion, as well as epicardial patching with glutaraldehyde-preserved equine pericardium and various types of biologic glue.^{4,5}

In conclusion, wakeboarding is a water sport that is gaining increasing popularity. The nature and severity of injuries sustained are generally under-recognized. This case illustrates the need for a high index of suspicion to diagnose one of the fatal injuries sustained from wakeboarding-related water trauma. We have highlighted the challenges in managing this extreme form of myocardial injury.

References

1. Hostetler SG, Hosteler TL, Snith GA, Xiang H. Characteristics of water skiing and wakeboarding-related injuries treated in emergency departments in the United States, 2001-2003. *Am J Sports Med.* 2005;33:1065-70.
2. Carson WG Jr. Wakeboarding injuries. *Am J Sports Med.* 2004;32:16-73.
3. Fitzgerald M, Spencer J, Johnson F, Marasco S, Atkin C, Kossmann T. Definitive management of acute cardiac tamponade secondary to blunt trauma. *Emerg Med Australas.* 2005;17:494-9.
4. Padro JM, Mesa JM, Silvestre J, Larrea JL, Caralps JM, Cerron F, et al. Subacute cardiac rupture: repair with a sutureless technique. *Ann Thorac Surg.* 1993;55:20-4.
5. Prêtre R, Benedikt P, Turina MI. Experience with postinfarction left ventricular free wall rupture. *Ann Thorac Surg.* 2000;69:1342-5.