CASE REPORT

Anterior Penetrating Chest Injury with Left Ventricular Thrombus

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ABSTRACT

Urban cities are synonym with a high incidence of penetrating chest injuries either from accidents or interpersonal violence. The outcome of penetrating chest wound can vary from immediate death to a prolonged morbidity. We here report a case of 39-year-gentleman who presented to Emergency Department Hospital Raja Permaisuri Bainun, Ipoh, Perak after being stabbed to the chest. His anterior penetrating chest wound was located at the 5th intercostal space medial to the midclavicular line. The stab wound penetrated the myocardium, causing minimal myocardial rupture. He also suffered from left haemothorax and hemopericardium. The haemothorax was drained with insertion of 32 French chest tube. The patient

Kata kunci: embolisasi trombus, tikaman dada

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was admitted under the cardiothoracic team and discharged five days later without surgical intervention. He presented again to the Emergency Department with complains of shortness of breath and pleuritic pain. A left ventricular thrombus was detected via echocardiography. Unfortunately, he took his own discharge. Five days later he came again to Emergency Department with sporadic of loss of vision. The mural thrombus dislodged and embolized to the retinal artery causing amaurosis fugax. The patient was treated with aspirin 150mg and his symptoms subsequently resolved.

Keywords: embolization, penetrating chest wound, stab wound

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**CASE REPORT**

A 39-year man complained of pain, shortness of breath and bleeding over left anterior chest wall after being stabbed with a small knife. He was instantly rushed to the Emergency Department.

The victim was pallor and tachypnoaic with rate of 26 breaths per minute. Initial blood pressure was 80/40 mmHg, pulse rate of 110 beat per minute and saturation under room air was 94%. A 2 cm wound was visualized at the 5th intercostal space medial to the midclavicular line. Reduced chest expansion and dullness percussion was noticed on the left side of the chest. There was no muffled heart sound and jugular venous pressure was not elevated. Abdomen was soft and non tender.

Serial Extended Focused Assessment of Sonography in Trauma (EFAST) was performed. E-FAST revealed left side pleural effusion and accumulation of pericardial fluid measuring 1.3 cm anteriorly and 0.8 cm posteriorly. Electrocardiography showed sinus tachycardia with no poor R wave progression. Chest X-ray revealed left hemothorax with haziness over left lower zone of the lung. The heart was globular in shape. The mediastinum was normal in size. Blood investigations were normal.

He was resuscitated with intravenous crystalloid, colloid and two pints of whole blood. A 32 French chest tube was inserted to the left chest and drained only 250cc of blood. Vitals still showed class three hypovolemic shock, of a BP 80/40 mmHg and pulse rate of 110 beats/minute. Pulse oximetry was 99-100% on high flow mask. Since echocardiography revealed no pericardial tamponade, pericardiocentesis was not attempted. Repeated FAST was done and did not show any other intraabdominal bleeding. Source of the bleeding could not be determined; hence even in this unstable patient a CT (Computed Tomography) was performed.

Left lung contusion with haemothorax and minimal loculated pericardial effusion was evident on CT thorax. The patient was then referred to the cardiothoracic team. No surgical intervention was planned. He was then admitted to surgical high dependency ward for close observation and antibiotic treatment. His vitals stabilized
and he was discharged on the fifth day of admission.

The patient presented two days later to the Emergency Department with shortness of breath and pleuritic chest pain. He had no fever, cough nor any flulike symptoms. There were no complaints of calf pain or unilateral leg swelling, abdominal pain, vomiting, discharge or redness from the chest tube site. His blood pressure was of 130/80 mmHg, pulse of 80 beat per minute and oxygen saturation was 96% under room air. The air entry was reduced on the left side and was dull on percussion, correlated with the healing lung contusion. Left ventricular thrombus was visualized as a hypoechoic mass via the echocardiogram (Figure 1). The contractility was good and no regional wall motion abnormality was detected. The minimal pericardial fluid was still present. The patient was advised to be admitted be he refused and self-discharged at his own risk.

Five days later, he arrived again to the Emergency Department with sporadic loss of vision for the past two days. He described it as a curtain being closed in front of his right eye then subsequently left eye which lasted for 15 minutes. The symptoms were associated with pleuritic chest pain and shortness of breath on exertion. Vital signs were normal. Echocardiogram no longer detected the left ventricular thrombus, which assumed to have fragmented and embolized to the retinal artery. Plain CT brain showed no evidence of infarct. He was treated as amaurosis fugax secondary to thromoemboli and was started on Aspirin 150mg daily. Patients symptoms resolved and he was discharged.

**DISCUSSION**

The initial presentation of Stage 3 hypovolemic shock (hypotensive and tachycardia) did correlate with the blood lost due to hemothorax. The echocardiogram did not show any tamponade as the right ventricle was not collapsed in the diastolic phase.

![Figure 1: Bedside 2D Echocardiogram (4 chamber view) shows left ventricular thrombus at the apex](image.png)
Minimal pericardial effusion was detected. In a series done by Tayal et al. (2004) patients with penetrating anterior chest trauma, the Focused Assessment of Sonography in Trauma (FAST) examination was sensitive and specific in the determination of both traumatic pericardial effusion and intraperitoneal fluid indicative of injury, thus effectively guiding emergent surgical decision making (Tayal et al. 2004). Both ultrasound and CT scan explained the amount of blood loss that leads to this stage 3 shock. Only 250 ml of blood was drained from the chest tube, which was not indicative of a thoracotomy. After transfusion he stabilized, and classified as a responder, as there was no ongoing blood loss that need to be halted.

The second presentation to the ED with shortness of breath raised a few possibilities, which among them is the cardiac thrombus which resulted in pulmonary embolism. Bedside echocardiography in ED revealed a pedunculated mass in left ventricle where the patient was previously stabbed. This is likely to be a thrombus formation in result to the previous injury. Part of the thrombus must have fragmentized into embolus that lodged in the pulmonary vessels. Unfortunately, further workout of this patient at that time was not possible because he took his own discharge.

The patient presented again with symptoms of amarousis fugax, a transient loss of vision caused by lodgement of emboli on the retinal artery. Echocardiography at this time did not show any thrombus which has most likely dislodged. CT brain did not reveal any infarct.

Kertes et al. in 1983 reported a case of multiple peripheral emboli after cardiac trauma. It was a case of 25-year-old footballer that sustained non-penetrating (blunt) cardiac trauma after a blow to the chest six months before he had bilateral foot pain and numbness. He was then diagnosed to have left ventricular apical thrombus with multiple systemic emboli to both arterial supply of his feet and right coronary artery. He was treated surgically and discharged well (Kertes 1983).

Another case report by Neidlinger et al. in 2004 described a case of cardiac thromboemboli complicating a stab wound to the heart. It was a case of 27-year-old man with a single stab wound at left fifth intercostal space, where left anterior thoracotomy and primary repair of left ventricular apical laceration was done. Post operation day 4 complicated with thromboembolic phenomenon secondary to thrombus of the septal and posterior wall, which responded well with intravenous, subsequently oral anticoagulation and discharged well (Neidlinger et al. 2004).

Sharma et al. (2000) found that patients with an embolic secondary to left ventricular thrombus suffered a significantly higher mortality during follow-up when compared to those without an embolic event. In addition, the occurrence of an embolic event had an important bearing on survival, rather than the echocardiographic finding of LV thrombus. This implies that any anticoagulant therapy, if it is to impact on survival, probably should be given preemptively, before the appearance of a thrombus on echocardiography (Sharma et al. 2000). To the best of
our knowledge, no cases have been reported in which cardiac trauma specifically caused amaurosis fugax.

REFERENCES