## CASE REPORT

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# Post-Traumatic Left Ventricular False Aneurysm

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**ABSTRACT:** Most false aneurysms of the heart represent contained ventricular free wall ruptures after myocardial infarction. Post-traumatic aneurysms also may follow penetrating or non-penetrating trauma to the chest. Regardless of the origin of the false aneurysm there is a propensity for aneurysm rupture.

We report a patient who developed a false aneurysm of her left ventricle that developed post-motor vehicle accident. Her orthopedic problems were the clinical problems identified and after a hospital admission of 10 days she was discharged home. Four weeks later she died suddenly from anterior left ventricle false aneurysm rupture and tamponade. Patients with significant chest wall trauma should be assessed for cardiac pathology prior to discharge. Presentation may be delayed and be overshadowed by more evident pathology.

Trauma-related aneurysms may cause sudden death, and this may occur some later time after the trauma. Attributing the cause of death to the trauma, which may be remote, is important for the forensic investigator to remember.

**KEYWORDS:** forensic science, false aneurysm, pseudoaneurysm, left ventricle, trauma

### Case Report

A previously healthy 17-year-old female sustained multiple injuries after a high-speed motor vehicle accident. The car, in which she was a passenger, hit a tree head on and she was thrown 70 ft from the vehicle, as she wore no restraint.

In emergency, chest radiography showed a small left pneumothorax and there was an open proximal fracture with neck displacement of her right proximal humerus, a left zygoma fracture with orbital blowout fracture and a thoracic spinal subluxation injury with compression/lateral flexion injury at T10-11. She had chest tube insertion. She underwent open reduction and debridement of her humerus. Her thoracic spine injury was treated by open reduction, debridement and internal fixation with Harrington rod and fusion of T9-12. Further investigation revealed C-spine was unremarkable, but CT of her head demonstrated the aforementioned orbital and facial fractures.

She did well in hospital and was discharged home after 10 days of hospitalization and plans were made for follow up in ambulatory care and Plastic Surgery clinic for her facial injuries.

One month after discharge, six weeks after her accident, she had a witnessed abrupt onset of dyspnea and arrested. In the emergency room she was hypotensive with cyanosis and distended neck veins. Despite resuscitation attempts, including pericardiocentesis, she could not be revived. Since the death was unexpected and occurred a few weeks after major trauma, the death was investigated by a coroner. A complete autopsy was performed.

At autopsy there were sequelae of the prior surgeries with scars on her central back, right lower back and shoulder. A spinal rod was noted. No pneumothoraces were seen. There were no pulmonary contusions. The sternal skin had numerous punctures from pericardiocentesis and there were peripheral arterial and venous punctures and chest paddle marks.

Her cause of death related to cardiac tamponade. The pericardium contained 450 mL of blood. The heart had severe fibrinous pericarditis and a large abnormal aneurysm was noted on the anterolateral aspect of the left ventricle just under the left atrial appendage. This was a 4.8 by 4.0 cm false aneurysm with a thin wall and a 1-cm defect that represented the area of rupture. Bisecting the aneurysm showed it to take origin in the anterobasal left ventricle and possess a narrow neck and contain a small amount of organizing thrombus (Fig. 1). The contralateral inferior wall of the left ventricle showed an organizing epicardial contusion with fat necrosis and organizing hemorrhage and myocardial necrosis.

Microscopic examination of the aneurysm showed the wall to consist of only fibrous tissue and thrombus. No myocardium was present except at the edge of the aneurysm; it was clearly seen that the myocardium abruptly ended and the false aneurysm began. The coronary arteries were carefully dissected and examined. No coronary artery pathology was found.

The patient's cause of death was cardiac tamponade related to rupture of a post-traumatic left ventricle false aneurysm. Her manner of death was accidental.

### Discussion

Ventricular aneurysms may be true aneurysms or false aneurysms. True aneurysms represent transmural myocardial infarcts that have remodeled and thinned. Their wall consists of all the myocardial elements including epicardium, myocardium, and endocardium. They contain a prominent component of collagen and fibrous tissue and thus do not tend to rupture. In contrast false aneurysms represent free wall ruptures that are contained by adherent pericardium (1).

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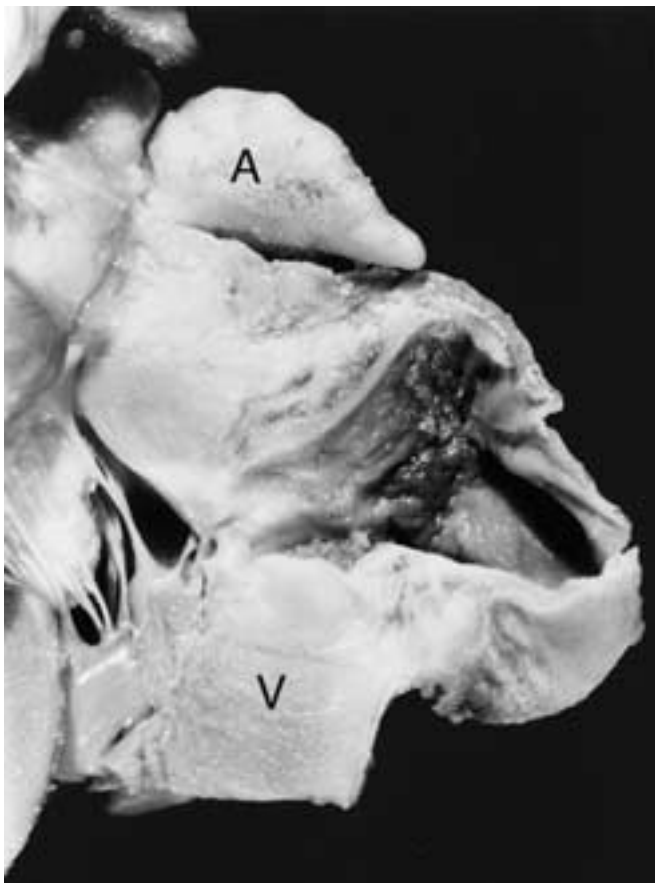


FIG. 1—*Cut surface of lateral left ventricle (*V*) demonstrating the ruptured false aneurysm. The aneurysm is below the left atrial appendage (*A*) and shows a narrow neck and a fibrous wall. Thrombus is noted in the lumen.*

Most false aneurysms represent ruptured myocardial infarcts, but other causes include post-traumatic causes, both penetrating and non-penetrating trauma, complication of infectious endocarditis, syphilis, rheumatic fever, and post-operative complications (2). They develop after adherent reactive pericardium walls off a myocardial rupture. Since the only components in the aneurysm wall are fibrous tissue and thrombus these aneurysms have a tendency for rupture (3,4).

Penetrating trauma usually results in false aneurysm formation where as non-penetrating trauma may result in either false or true aneurysm formation (5).

The majority of false aneurysms are the result of contained ruptured myocardial infarcts with a gradual wall disruption. This is similar to the usual fashion of conventional free wall rupture that usually occurs in the first week after infarct (1,4,6).

Post-traumatic false aneurysms are postulated to develop from several mechanisms including: (a) traumatic compression of the myocardial chambers due to chest deformation, (b) deceleration injury with differential mobility of the ventricles in comparison to the heart base, which is attached via the venous insertions, and (c) direct penetration of the chambers from rib fractures (7). These develop due to a tear in the myocardium or with a contusion, as a gradual rupture (6,8). Others have postulated that traumatic aneurysms, either true or false, develop due to coronary artery injury (9–11).

Clinically it is not uncommon for these aneurysms, regardless of their pathoetiology, to be clinically silent (2,12). If they are symptomatic they may present as dyspnea, chest pain and palpitations. Clinically an important sign is an unusual prominence on chest X-ray on the cardiac silhouette (12–14). By clinical examination a new murmur should be sought for. A to and fro quality may be a feature of the murmur noted (7,12). Signs and symptoms of congestive heart failure may be present.

The most important complication of these false aneurysms is rupture with subsequent tamponade and death of the patient (4,12). Emboli from the thrombus within the aneurysm may also occur (5,9,14). Others may cause congestive heart failure, chest pain, syncope, and arrhythmia (2).

Diagnosis of post-traumatic false aneurysms depends primarily upon clinical suspicion. Since these important pathological lesions may be silent, they must be actively sought for by clinical examination in cases of significant chest trauma. If clinically suspected, confirmation may be performed by angiography, echocardiography (ECHO), computerized tomographic (CT) or magnetic resonance imaging (MRI) scans (3,12).

False aneurysms of the left ventricle may also co-exist with similar aneurysms of the atrium or the aorta (7,8). Concomitant repair should be planned after adequate pre-operative evaluation. Coronary artery injury may also be noted (10).

Treatment of ventricular false aneurysms is by surgical excision and repair of the defect. Some have proposed a more conservative approach but this is generally not accepted (15,16).

The presentation of these aneurysms may be delayed and present anytime from three months to several years after the injury (5,6,14). The clinically silent myocardial injury may be overshadowed by more evident bone, great vessel and abdominal or extremity trauma.

In the current case, there was no new murmur recognized prior to the hospital discharge. The aneurysm developed in the month after the initial trauma. If her chest had been ascultated during this time, it is almost certain a murmur would have been present that could have led to further investigation and subsequent excision or repair. Unfortunately this aneurysm was silent until it ruptured. The patient died in emergency and thus detection or intervention was not possible.

Patients with significant chest trauma from non-penetrating or penetrating injury should be followed up and re-examined for new murmurs or abnormal cardiac silhouettes in order to detect these important pathological aneurysms prior to rupture (14). Follow up is important even if the initial assessment for injury is negative in these patients with significant traumatic injury.

These aneurysms may be a cause of sudden death weeks after the trauma has occurred. The forensic investigators, including pathologist, coroner, medical examiner or other professional, must correctly attribute the cause of death to be due to the trauma that initiated the aneurysm formation.

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