
Rupture of the Right Coronary Artery Due to Nonpenetrating Chest Trauma*

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We describe a case of traumatic myocardial infarction due to rupture of the right coronary artery. Isolated rupture of a coronary artery is rare and often recognized only at autopsy. Absence of cardiac tamponade due to laceration of the pericardium was partly responsible for the relatively long survival time.

Injury to the heart and the great vessels after nonpenetrating trauma to the chest is a frequent occurrence and an inevitable penalty of modern traffic. Myocardial damage has been reported in 16 to 76 percent of the cases after blunt chest trauma.1 Myocardial contusion is mostly of minor importance and does not bear consequences.2 Among the rapidly fatal lesions, myocardial rupture and laceration of the great vessels are by far the most frequent.3

With the improvement in first aid and prompt evacuation, more patients with severe cardiac injuries now reach the hospital emergency room sooner and benefit from prompt recognition of the nature of their injuries and subsequent adequate treatment. This eventually proves life-saving.4

The following patient is presented because the exceptional concurrence of injuries was demonstrated at autopsy. As the patient survived for a few hours after the accident, interesting conclusions could be formulated as to diagnosis and treatment of such lesions.

CASE REPORT

A 62-year-old man suffering from mild diabetes, but without previous history whatsoever of ischemic heart disease, was involved in a car accident and sustained blunt trauma to his chest. On admission to the emergency room, half an hour after the crash, he was in severe shock. On inspection there were only a few skin lacerations. Blood pressure was 70/50 mm Hg, pulse rate was regular at 50 beats per minute. He was conscious, had tachypnea and complained of dyspnea and severe thoracic pain.

An electrocardiogram showed an acute inferior myocardial infarction with complete atrioventricular block (Fig 1). On chest roentgenogram, four rib fractures at least were visible, as well as closure of the left costodiaphragmatic sinus. The heart was slightly enlarged.

Significant laboratory findings included a hematocrit of 46 percent and moderately increased levels of SGOT and CPK (73 U and 158 U respectively).

![Figure 1. Electrocardiogram on admission showing acute myocardial infarction with complete atrioventricular block.](http://journal.publications.chestnet.org/pdfs supplements/chest20951/00_062017)
Resuscitative measures were instituted immediately and cardiac pacing was insured by introducing a catheter transvenously into the right ventricle. The blood pressure rose to 100/80 mm Hg, but despite temporary improvement the patient's condition deteriorated three hours later. Blood pressure had decreased to 80/40 mm Hg. Central venous pressure was increased to 16 cm H2O. The patient became obtunded and persistent labored respiration necessitated use of assisted ventilation. On a control roentgenogram of the chest, the heart appeared slightly more enlarged than before. Hematocrit was nearly unchanged (43 percent). Blood gas measurement revealed a severe metabolic acidosis and profound hypoxemia. Despite the absence of a pulsus paradoxus, muffled heart sounds and marked distension of the neck veins, pericardiocentesis was performed, in order to exclude cardiac tamponade. On two attempts, no blood could be aspirated. Blood pressure became unrecordable and the patient died nine hours after the original trauma in irreversible shock. No cardiac massage was performed.

Postmortem examination revealed the presence of 500 ml bloody fluid in the left hemithorax and 250 ml in the right. There were 13 rib fractures. A bone splinter had perforated the left parietal pleura. There was a longitudinal laceration of the pericardium, with total herniation of the heart. The right coronary artery showed a practically complete rupture 0.5 cm from its orifice, covered by a small fresh thrombus (Fig 2). There was also a small transverse tear of the intima of the left coronary artery.

Both coronary arteries were slightly atheromatous but patent. In the aorta a nonperforating horizontal tear of 2.5 cm was found, distal to the origin of the left subclavian artery, at the insertion of the ligamentum arteriosum. There was a large hematoma in the adventitia, at this site.

Routine histology (hematoxylin, erythrosine safran and phosphotungstic hematoxylin) revealed only a few foci of recent ischemic necrosis of the muscle fibers of the posterior septum, under the form of kariolysis and myofibrillar alterations, with discrete leukocytic infiltration of the interstitium. Quantitative enzymatic studies, which will be reported elsewhere, showed the existence of a recent extensive inferior wall infarction of degree III, according to the WHO classification of the histochemical ischemic alterations of the myocardium.

**Discussion**

Myocardial infarction after nonpenetrating trauma is not uncommon. It is either due to contusion or to laceration of the heart. Thrombosis of a major coronary artery as a result of nonpenetrating trauma, even in cases of pre-existing coronary heart disease is very rare, or even denied. Coronary artery injury in the form of laceration or rupture is likewise infrequent. In a series of more than 500 autopsies of nonpenetrating trauma to the heart, Parmley and colleagues reported only ten cases. Eight patients, with associated rupture of the heart and the aorta, died immediately and the two others survived for two and six hours respectively. Both sustained rupture of the descending branch of the left coronary artery. In neither was myocardial infarction demonstrated. Cardiac tamponade rather than myocardial infarction was the major cause of death. Rea et al pointed out that in the case of penetrating injuries of the coronary artery, the mortality rate rises when the arterial lesions are more proximal. This also apparently applies to nonpenetrating injuries. In our case, only minor contusion of the endocardium could be observed with the naked eye. Apart from the rupture of the right coronary artery, the myocardial wall and the heart valves were healthy.

The relatively long survival (nine hours) in this patient is probably due to the absence of concomitant lesions of the heart wall and valves, but undoubtedly also to the laceration of the pericardium which prevented cardiac tamponade.

The early diagnosis and treatment of heart injuries following nonpenetrating chest injuries have been discussed recently.** Clinical signs of either cardiac tamponade or hypovolemic shock and hemothorax are considered to be reliable indicators of severe cardiac injuries and they should prompt immediate exploratory surgery. From this case report, it appears that the absence of these signs in the presence of definite myocardial infarction does not necessarily point to a milder lesion as, for example, simple cardiac contusion. Rupture of a coronary artery does not inevitably lead to severe hemorrhage as confirmed by the absence of pure blood in the thoracic cavity.

We therefore believe that coronary arteriography should be performed as soon as possible if the clinical situation deteriorates in definite myocardial infarction after blunt chest trauma, even in the absence of cardiac tamponade or major hemorrhage. Using this technique, coronary artery lacerations can be identified. A complete angiographic investigation is also justified in that the degree of severity of the visceral lesions and their possible association with tears in the wall of the great vessels may be underestimated.

Surgical treatment of coronary artery lacerations now seems to be in the realm of possibility, but it remains fraught with difficulties. Successful ligation has been reported for distal lesions. However, main stem lesions should be repaired or saphenous vein bypass grafting should be performed. This obviously requires the use of emergency cardiopulmonary bypass and the total heparinization undoubtedly increases the risk of severe bleeding in a recently traumatized patient.

Finally, this case reminds us opportunely that the right coronary artery, at its point of origin, is situated more anteriorly than the left and is therefore more vulnerable.

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Late Esophagopleural Fistula after Pneumonectomy for Bronchial Carcinoma

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We report one case of esophagopleural fistula occurring after right pneumonectomy. Pathogenesis and management of this complication are briefly discussed. Drainage of the empyema and direct closure of the fistula, with coverage by a pedicle flap of the intercostal muscle are the best methods of treatment.

The appearance of an esophagopleural fistula after pneumonectomy is a rare complication. In an extensive review of the literature, 1-4 35 cases were reported. Most of these cases developed following pneumonectomy for tuberculosis or suppurative pulmonary disease. Only six cases were found in which fistulae developed as early or late complication after pneumonectomy for carcinoma of the lung. Because of the rarity of this complication we are reporting one case.

CASE REPORT

This 65-year-old man underwent right pneumonectomy for bronchial carcinoma on Dec 10, 1968. The pathology report showed undifferentiated carcinoma, requiring extensive resection of infiltrated glands. The patient made an uneventful recovery and on the 21st day after operation he was sent home. Because of the type of carcinoma and the glandular involvement, the prognosis was poor.

Six months later the patient was readmitted with a high temperature and chest pain. A diagnosis of empyema necessitatis was made. Drainage was accomplished through a basal tube. There was no bronchial fistula. After the drainage of the empyema, the condition of the patient improved and thoracoplasty for closure of the empyema cavity was suggested, but the patient refused.

He was again discharged and was quite well until June, 1971. By that time he noticed that food was leaking through the drainage tube. The patient was readmitted to the clinic in a very poor general condition. Radiologically, a diagnosis of esophagopleural fistula in the middle of the esophagus was made (Fig 1). Our first impression was that the fistula was due to neoplastic infiltration of the esophagus, although the radiologic findings of esophageal distention were not indicative of metastasis. Esophagography at a depth of 30 cm revealed a small fistula in the right lateral wall of the esophagus without neoplastic infiltration. Biopsy findings were negative. Bronchoscopy showed no recurrence in the bronchial stump. The patient was fed lying on his left side, averting the necessity for gastrostomy, and after six weeks his general condition improved. The empyema cavity shrank, but the esophageal leak persisted. In August, 1971, a thoracotomy was carried out through the same incision. A small empyema cavity was found, which was drained. A hole of 2 cm in length was found at the lateral wall of the esophagus near the bronchial stump. Microscopically, there was no neoplastic disease. After mobilizing the esophagus, the edges of the fistula were excised and the opening was closed in two layers. The suture was reinforced and covered with a flap of...