ACUTE MYOCARDIAL INFARCTION AFTER BLUNT CHEST INJURY

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ABSTRACT

A 20-year-old male sustained an inferior myocardial infarction following blunt chest trauma, after a motor vehicle accident. Though coronary arteriograms 9 months later were normal, LV angiography revealed severe hypokinesia of the inferobasal and diaphragmatic segments. The literature is reviewed with respect to mechanisms of injury, autopsy and angiographic findings and clinical outcome in this condition.

Keywords : Blunt Chest Injury, Acute Myocardial Infarction, Normal Coronary Arteriograms

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INTRODUCTION

Coronary arterial lesions caused by penetrating chest injuries are common[1-3]; however, myocardial infarction caused by blunt chest trauma is rare[4,5]. Documentation of the latter with coronary arteriography and LV angiography is even more uncommon. To the best of our knowledge only 10 such cases have been reported in the literature[6-12]; 2 of these had normal coronary arteriograms. A further 8 cases have been documented at necropsy[13-20]. Postulated mechanisms of injury include coronary spasm and disruption of an atheromatous plaque.

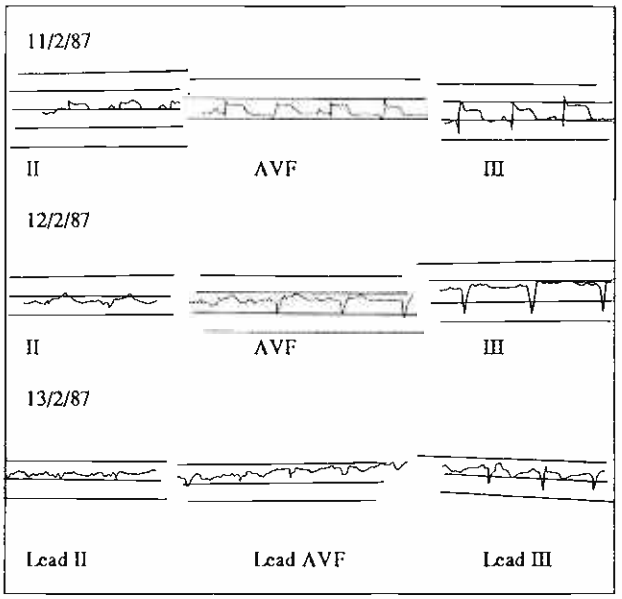
CASE PRESENTATION

NS, a 20-year-old Malay male, was seen in the Casualty Department of the Klang General Hospital, on 11th February 1987, after a motor vehicle accident. He was the co-driver of a car which collided head-on with another car. He was thrown forward and hit his chest hard against the dashboard. On admission to hospital he complained of inspiratory chest pains. Except for smoking 20 cigarettes per day for the past 1 year, there were no risk factors for coronary artery disease.

Clinical examination revealed an overweight young man with marked tenderness and bruising over the sternum and parasternal areas. Auscultation over the lungs and heart was normal. Patient was haemodynamically stable and the rest of the clinical examination was normal.

Chest X-rays did not reveal any fractures and the cardiac silhouette and lung fields were normal. Because of persistent chest pain, an electrocardiogram was done. This revealed an acute inferior myocardial infarction (Fig 1). Cardiac enzymes were elevated with a peak total creatine phosphokinase of 880 International Units/L(Normal: 60-110 International Units/L) and peak lactate dehydrogenase of 550 Units/L(Normal: 90-220Units/L). Serial ECG's over the next 3 days revealed classical changes of an evolving transmural inferior myocardial infarction. Sugar and lipid profiles were normal. NS made an uneventful recovery and was discharged 1 week after admission. A treadmill test done 6 months after his infarction did not reveal any features of residual myocardial ischaemia. He exercised for 12 minutes, attained a heart rate of 170 beats per minute with good blood pressure response (Bruce Protocol). Because of his youth, it was decided to go ahead with coronary arteriography. The right and left coronary arteries were normal (Fig. 2a & b). Left ventricular angiograms revealed severe hypokinesia of the inferobasal and diaphragmatic segments (site of the inferior infarction)(Fig 3a & b). At follow up 1 year after his accident, NS continues to be well and remains asymptomatic and active.

Fig 1 - Serial Electrocardiograms showing acute inferior myocardial infarction



DISCUSSION

Parmley and colleagues[4] in 1958, reviewed 546 postmortems of blunt chest injury; they did not find any case of coronary occlusion, but did observe 9 cases of complete rupture of a major coronary artery and 1 case involving an intimal tear.

The causes of blunt chest injury resulting in myocardial infarction, have varied from major injuries due to blast trauma[4] and motor vehicle accidents as in our case, to relatively minor trauma as being struck on the chest by a football[8], a softball[16] and a punch on the chest[17]. Mechanisms of injury, have been

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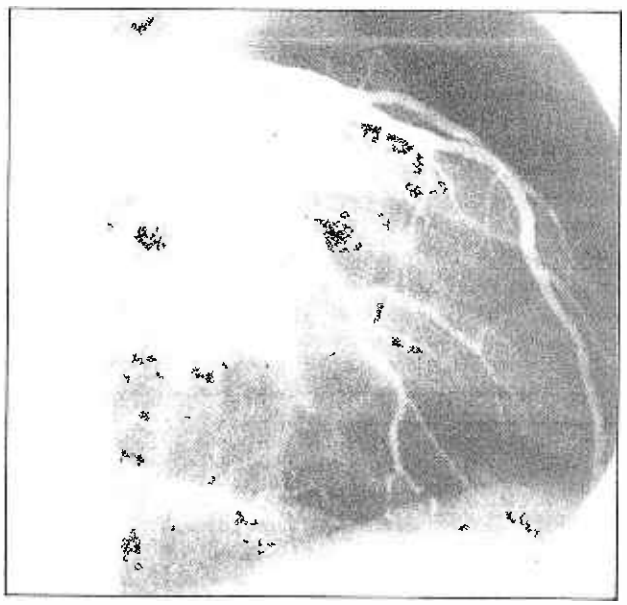
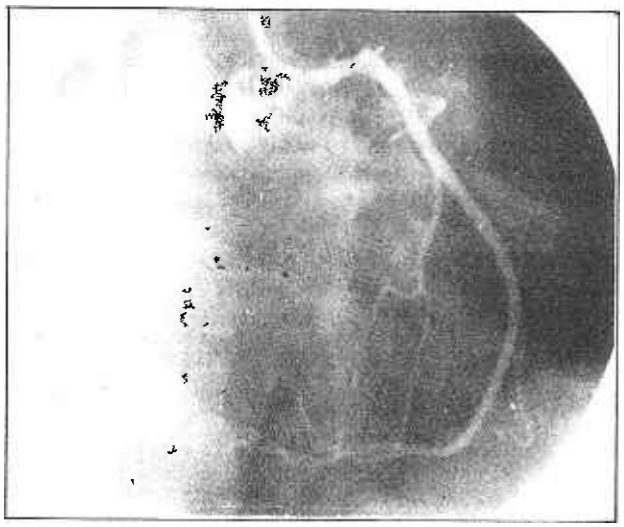
attributed to sudden acceleration/deceleration, causing the heart to be forcefully thrusted against the sternum, compression of the heart between the sternum and vertebra[12] and sudden increase in intrathoracic pressure.

Fig 2 - Coronary Artery Angiograms showing Normal Coronary Arteries

(a) Left Coronary Artery



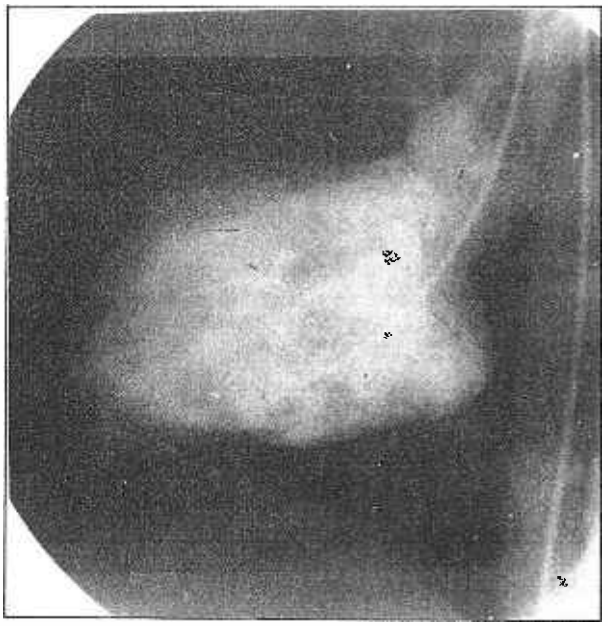
(b) Right Coronary Artery



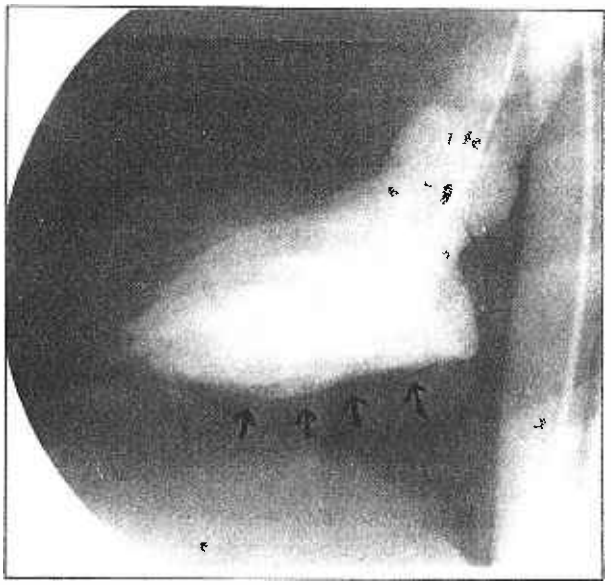
In the past coronary artery thrombosis due to blunt chest injury was thought to occur only if there was underlying coronary artery disease[4]. Rupture of an atherosclerotic plaque or embolism of atheromatus debris was the postulated mechanism. Of the 18 cases of blunt chest injury studied with selective coronary arteriography and/or autopsy, referred to in this article, only 8 had evidence of atherosclerotic disease - one of these 18, at autopsy had insignificant atheromatus disease but a large organizing thrombus in the left circumflex artery. In 8 other patients[5,9,11] severe obstruction of a single coronary artery with no evidence of pre-existing atherosclerotic disease was noted. Besides our case, 2 other reports[8,9] exist of patients who have sustained similar injury, documented to have myocardial infarcts and subsequently shown to have normal coronaries. As in our patient, the interval between injury and coronary arteriography, was several months. In the first of 2 cases reported by Olivia[12] serial coronary arteriograms revealed lysis of an intracoronary thrombus and improved coronary flow 3 months after the injury. These cases suggest that blunt chest trauma may damage normal coronary arteries leading to obstruction to flow either by an intimal flap or thrombus and thereby lead to myocardial infarction. The prolonged interval between injury and coronary arteriography most likely lead to dissolution of thrombus and recanalization in our case and that of deFeytor[8] and Hartone[9]; this is also the postulate of the latter author. In these cases, severe regional wall motion abnormalities confirmed the past occurrence of myocardial infarction though coronary angiograms were normal. Coronary artery spasm is the other possibility. Site of infarction in both our patient and that of Hartone[9] was isolated to the diaphragmatic portion of the LV wall(inferior infarct); myocardial contusion, thus, is not the explanation for the injury as this portion of the LV wall would not be in the direct line of injury.

Fig 3 - Left Ventriculogram showing posterobasal and diaphragmatic hypokinesia

(a) Diastole



(b) Systole



The left anterior descending artery was most commonly affected[11-18] - Table I. This is surprising as one would have expected the more anteriorly placed right coronary artery to be involved more frequently. The interval between

Table I - Myocardial infarction following chest trauma documented with coronary arteriography/autopsy – Coronary artery involvement

Total Number of Cases	18
Left aneterior Descending Artery	12
Right Coronary Artery	5
Circumflex Artery	1
Presence of Atherosclerotic Coronary Artery Disease	8

injury and development of symptoms of myocardial ischemia varied between immediately after injury as in our case to 9 days[13]; the coronary artery injury probably initiated a progressive thrombotic process which gradually reduced coronary flow to a critical point, to explain for the delay in development of symptoms.

Ten patients died - Table II. Causes of death included cardiac failure in the majority[8,13,15,18,21], sudden death from pulmonary embolism[9] and ventricular arrhythmias[2,13]. In cases of heart failure the interval between injury and death varied from 2 weeks[13] to 2 hours[15]. Given the cause of the myocardial infarction, thrombolytic therapy would be contraindicated. Coronary angioplasty would probably be dangerous when applied to a contused vessel wall. However in the patient who presents early and is noted to be haemodynamically compromised, early coronary bypass surgery may alter the outcome[1-3,10].

Table II - Myocardial infarction following chest trauma documented with coronary arteriography/autopsy - Complications

DEATH	Congestive heart failure	7
	Ventricular arrhythmia	2
	Pulmonary embolism	1
	Total	10
COMPLETE HEART BLOCK		2

This patient illustrates well the fact that traumatic coronary artery disease should be considered in all patients with closed chest trauma. If not for the vigilance of the casualty officer, the extensive bruising on this patient's chest could have easily caused his symptoms to be misinterpreted and the diagnosis missed. Early recognition would mandate close monitoring and facilitate the institution of appropriate emergency treatment.

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