

Hypertension and Sudden Cardiac Death: Their Relationship in Post Infarction Cardiac Rupture

Abstract

The purpose of this study was to assess the relationship between hypertension (H) and sudden cardiac death (SCD), defined as a result of a cardiac event heralded by an abrupt loss of consciousness within one hour from the onset of acute symptoms in healthy individuals and in patients (pts) suffering from heart disease, in pts died from post infarction cardiac rupture (PCR). 168 autopsy cases (100%) with acute myocardial infarction were studied. 108 pts were males (64%) and 60 (36%) females with a mean age 64.3+/-18 years. 46 pts (27%) showed PCR. Clinically, the presence of H and symptoms of impending rupture was recorded. Pathological exam was conducted by analyzing gross and microscopic characteristics of the myocardium and coronary arteries of all pts. 2 pts (4.3%) showed sudden H as a symptom of impending rupture (respectively from 118/86 mmHg to 180/95 mmHg and 145/90 mmHg to 190/98 mmHg) and chest pain resistant to opiates. They deceased within 1 hour from increased H. The other 44 pts with PCR were hypertensive in 27 cases (61%) at the admission, while 17 (39%) displayed no H. Pts with rupture had a statistically significant increase of coronary alterations, cardiac hypertrophy, and up to 90% coronary stenosis (P<0.01). H was a symptom of impending rupture followed by SCD not frequently observed.

Keywords: Sudden cardiac death; Hypertension; Post infarction cardiac rupture

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Introduction

The relationship between hypertension (H) and sudden cardiac death (SCD), defined as a result of a cardiac event heralded by an abrupt loss of consciousness within one hour from the onset of acute symptoms in healthy individuals and patients suffering from heart disease, is far to be well established [1-3]. On the contrary, pre-existing hypertension has been identified to be a factor, which is often associated with post infarction cardiac rupture (PCR) [4-6]. Evidence indicates that cardiac rupture during an acute myocardial infarction occurs with a high rate ranging up 8% in the patients affected by the acute ischemic disease [3-4,7-10]. The purpose of this retrospective study was

to assess the relationship between H and SCD in pts died from post infarction cardiac rupture (PCR).

Material and Methods

168 autopsy cases with acute myocardial infarction were studied. 108 pts were males (64%) and 60 (36%) females with a mean age 64.3+/-18 years. 46 pts (27%) showed PCR. Clinically, the presence of H and symptoms of impending rupture was recorded. Pathological exam was conducted by analyzing gross and microscopic characteristics of the myocardium and coronary arteries of all patients using a method of investigation previously described [11]. The characteristics of study subjects are in Table 1.

Table 1: Study Population.

Number	168 (100%)
Males	108 (64%)
Females	60 (34%)
Mean age	64.3+/- 18 years
PCR	46 (27%)

Results

2 pts (4.3%) (Table 2) showed sudden H as a symptom of impending rupture (respectively from 118/86 mmHg to 180/95 mmHg and 145/90 mmHg to 190/98 mmHg) and chest pain resistant to opiates. They deceased within 1 hour from increased H. The other 44 pts with PCR were hypertensive in 27 cases (61%) at the admission, while 17 (39%) displayed no H. Pts with

rupture (Figures 1-4), and Table 3 had a statistically significant number of multiple coronary alterations, cardiac hypertrophy (mean heart weight 627+/-188 grams and left ventricle wall thickness 25+/-3 mm vs 400+/-75 grams for heart weight and 14+/-2 mm for left ventricle wall thickness in pts with no rupture), irregular areas of confluent fibrosis and coronary narrowing up to 90% with a high incidence of occlusive thrombi.

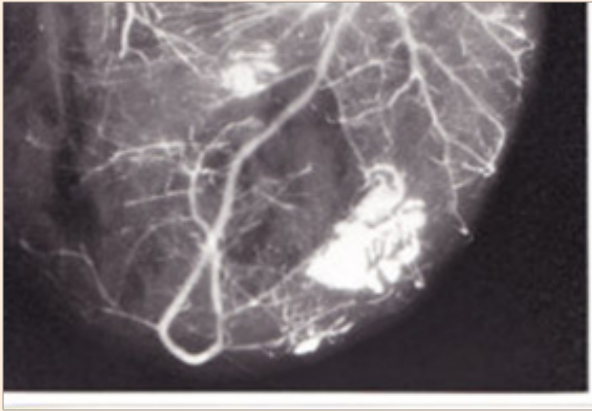


Figure 1: Postmortem angiography of heart with post infarction rupture. It can be seen an irregular filling of the coronary tree and a large avascular area at the site of the infarction.

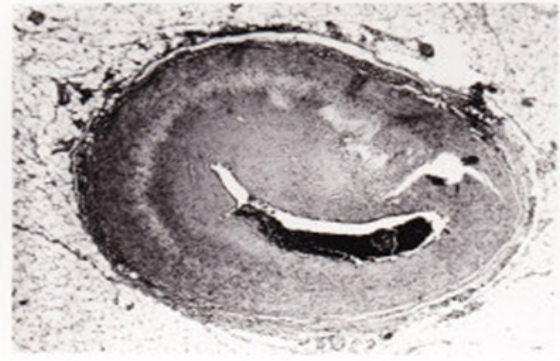


Figure 4: Severe stenosis of the left coronary descending artery in PCR.

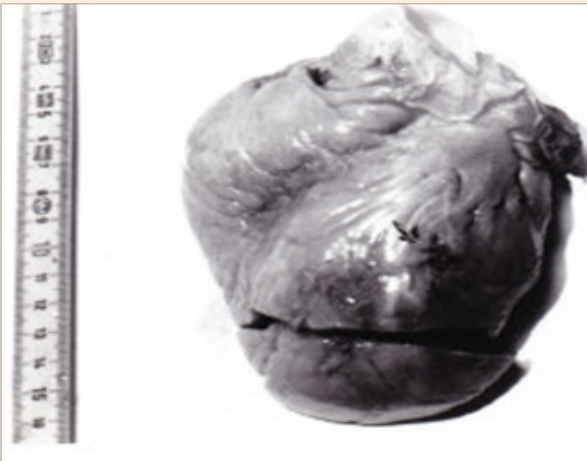


Figure 2: Irregular rupture line of the left ventricle anterior wall and marked cardiac hypertrophy of a patient acutely deceased from PCR.

Table 2: BP increase in the 2 subjects before PCR.

Subjects (n°2; 4.3%)	BP Baseline	BP Before Rupture
1st	118/86mmHg	145/90 mmHg
2nd	180/95 mmHg	190/98 mmHg

Table 3: Parameters in PCR related to baseline H.

BP	Number	Heart Weigh (grams)	LV Wall Thick-ness (mm)
H	27 (61%)	627+/-188	25+/-3
NoH	17 (39%)	400+/-75	14+/-2

Conclusion

H was a symptom of impending rupture followed by SCD not frequently observed in contrast to the fact that pre-existing H appeared to be a variable often associated with the development of myocardial rupture.

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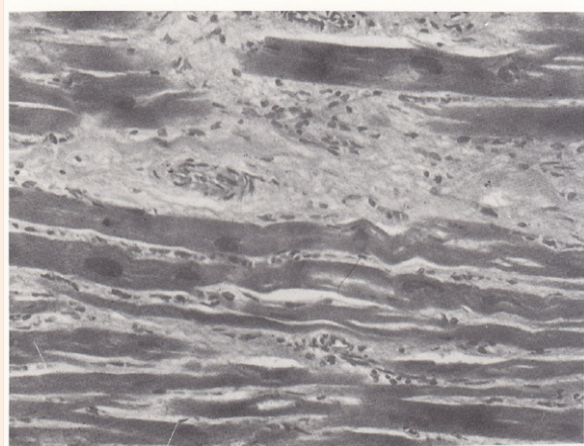


Figure 3: Microscopic changes in PCR. It can be seen confluent areas of fibrosis with a hemorrhagic occlusion of intramural coronary artery and myocardial fiber fragmentation.

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