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Letters

Hemorrhagic Myocardial Infarction

Mortality Compared With STEMI Patients Treated With Percutaneous Coronary Intervention

The natural course of myocardial infarction (MI) leads to ischemic necrosis (1). Experimental research indicates that hemorrhagic conversion of the myocardial necrotic area can increase the risk of serious complications of MI and may have a negative influence on its clinical course. After introduction of reperfusion therapy, primarily thrombolysis, hemorrhagic infarction has been described in autopsy studies (2,3). However, there are no data describing the hemorrhagic conversion of the MI in subsequent patients with MI treated with percutaneous coronary interventions (PCIs), with or without preceding thrombolysis, that combines clinical characteristics with autopsy findings.

TABLE 1 Baseline Characteristics and Autopsy Findings of Studied Groups

	Type of Myocardial Infarction (n = 129)		
	Hemorrhagic (n = 45)	Ischemic (n = 84)	p Value
Baseline characteristics			
Age, yrs	63.0 ± 11.3	65.0 ± 10.8	0.59
Female	26	27	0.99
Time of pain, h	6.25 ± 5.90	8.06 ± 6.90	0.44
Time door-to-balloon, min	33.7 ± 8.4	37.4 ± 9.1	0.51
Time from PCI to death, h	66.2 ± 7.4	62.6 ± 6.2	0.87
Diabetes mellitus type 2	24	37	0.17
Hypercholesterolemia	23	44	**0.01**
Myocardial infarction in medical history	20	39	0.11
Autopsy findings			
Left ventricular aneurysm	29	14	0.06
Left ventricular dilation	73	79	0.52
Left ventricular free wall rupture	31	12	**0.01**
Ventricular septal defect	0	1	0.99
Right ventricular infarction	0	7	0.09
Coronary arteries dissection	4	0	0.12
Aortic aneurysm	2	9	0.27
Old post-infarction scars	27	30	0.84
Hepatic steatosis	4	23	**0.01**
Red pulmonary infarction	7	11	0.53
Ischemic stroke	2	1	0.99
Hemorrhagic stroke	4	0	0.12
Focal infections	9	7	0.74

Values are mean ± SD or %. Values in **bold** indicate statistical significance.
PCI = percutaneous coronary intervention.

We enrolled patients with ST-segment elevation myocardial infarction (STEMI) who were hospitalized from 1998 to 2010, underwent PCI, died during the in-hospital period, and were subjected to autopsy. A conventional post-mortem examination was performed 12 to 24 h from the declaration of the patient's death by means of a 3-body-cavity autopsy. No less than 2 sections from opposite borders of the infarcted area were obtained. Each section was evaluated histopathologically. Hemorrhagic MI was assessed on the basis of gross and histopathological examination. Only confluent extravasation of erythrocytes with paucity of necrotic myocardial fibers was assumed as a hemorrhagic MI.

The pathomorphological evaluation was as follows:

1. *Ischemic myocardial infarction (IMI).* The heart muscle in the vicinity of infarction is macroscopically pale, with less compact tissue, and is surrounded by narrow, reddish and yellow borders. Histopathologically, the muscle fibers are defragmented with typical necrotic changes and have disappearing transverse striations, and neutrophils undergoing fat degradation appear on the border of the ischemia. In dubious cases, specific staining (fuchsinophilia or complement deposits) was applied.
2. *Hemorrhagic myocardial infarction (HMI).* Macroscopically, the muscle tissue was solid and dark red. In the histopathological examination, erythrocytes are present in the intercellular spaces. Fuchsinophilia and complement deposits were weaker and irregular.

In the analysis, 129 patients were included. During the autopsy examination, HMI and IMI were detected in 45 (34.9%) and 84 (65.1%) patients, respectively. Baseline characteristics are shown in Table 1. Patients with HMI received unsuccessful thrombolysis before the PCI more often (33% vs. 13%; p = 0.01). The mean number of occluded coronary vessels was lower in the HMI group (1.2 ± 0.9 vs. 1.7 ± 1.2; p = 0.035). The left

anterior descending artery was more often the infarct-related artery in patients with HMI (65% vs. 46%; p = 0.055).

On admission, the mean hemoglobin concentration and mean total cholesterol concentration were higher in the group of patients with HMI (9.2 ± 1.5 mmol/l vs. 8.4 ± 1.4 mmol/l; p = 0.018; and 5.7 ± 1.8 mmol/l vs. 4.9 ± 1.5 mmol/l; p = 0.041, respectively). Size of the infarct, as assessed indirectly by maximal creatine kinase-MB concentration, was similar in both groups (194 ± 180 IU/l and 176 ± 198 IU/l; p = 0.75 for HMI and IMI, respectively).

Ventricular fibrillation in the intensive care unit occurred less often (25% vs. 47%; p = 0.03) in the HMI group. The rate of cardiogenic shock was numerically lower with HMI (61% vs. 78%; p = 0.10). A comparison of the autopsy parameters is shown in **Table 1**.

In the multivariate analysis, anterior wall MI and thrombolytic treatment before PCI were associated with greater risk of hemorrhagic conversion of the infarct area in analyzed patients (odds ratio [OR]: 3.45; 95% confidence interval [CI]: 1.30 to 9.14; p = 0.01; and OR: 3.21; 95% CI: 0.99 to 10.39; p = 0.04, respectively). Risk of hemorrhagic conversion of necrotic area was lower in patients with cardiogenic shock (OR: 0.36 95% CI: 0.14 to 0.96; p = 0.03.

In our study, one-third of the patients with HMI received unsuccessful thrombolysis before PCI, but most were treated with PCI only. Until now, HMI found in autopsy was observed in patients treated with thrombolysis (2,3). Our results support the role of thrombolysis in the occurrence of intramyocardial hemorrhage, but also suggest that reperfusion by itself may lead to this phenomenon. Although there are doubts whether hemorrhagic conversion of necrosis area deteriorates the course of MI, there is evidence that the healing process may be slowed down in this zone (4). Our results also may suggest that hemorrhagic conversion of infarct area may influence the incidence of free wall rupture (FWR).

The main limitations of the study are that the presented analysis has a retrospective nature and the material was collected in 1 cardiology center. Moreover, the study evaluates only those patients who have died, and as such, the prevalence, clinical importance, and association with fibrinolysis and infarct location of hemorrhagic conversion may differ amongst those who survive PCI after STEMI. Furthermore, as one-fifth of patients received unsuccessful thrombolysis before PCI, results of this analysis may not apply to patients with STEMI treated with primary PCI.

In conclusion, hemorrhagic conversion of the infarct area occurs in approximately one-third of patients who die after STEMI treated with PCI. Free wall rupture occurs more often in patients with HMI. Independent predictors of HMI were anterior wall MI and thrombolytic treatment before PCI.

***Michał Hawranek, MD, PhD**
Michał Wróbel, MD, PhD
Jerzy Nożyński, MD, PhD
Damian Pres, MD, PhD
Marek Gierlotka, MD, PhD
Przemysław Trzeciak, MD, PhD
Krzysztof Dyrbuś, MD, PhD
Jacek Piegza, MD, PhD
Andrzej Lekston, MD, PhD
Mariusz Gąsior, MD, PhD

*IIIrd Department of Cardiology
School of Medicine with the Division of Dentistry in Zabrze
Medical University of Silesia
Silesian Centre for Heart Disease
ul. Skłodowkiej-Curie 9
41-800 Zabrze
Poland
E-mail: mhawranek@poczta.fm

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Pedicled Vein Grafts in Coronary Surgery Exhibit Reduced Intimal Hyperplasia at 6 Months

Despite strong evidence for superior patency and improved clinical endpoints using arterial grafts in coronary artery bypass graft surgery, the saphenous vein (SV) is still the most widely used graft in addition to left internal mammary artery (LIMA) (1). Consequently, improving SV patency continues to be a major goal. Souza et al. (2) introduced a “no-touch”