

# Hemorrhagic myocardial infarction after percutaneous coronary intervention: Echographic *versus* autopsy findings

Becker S.N. Alzand, Casper Muhl, Emile C. Cheriex

Department of Cardiology, Maastricht University Medical Center, Maastricht, the Netherlands

## Abstract

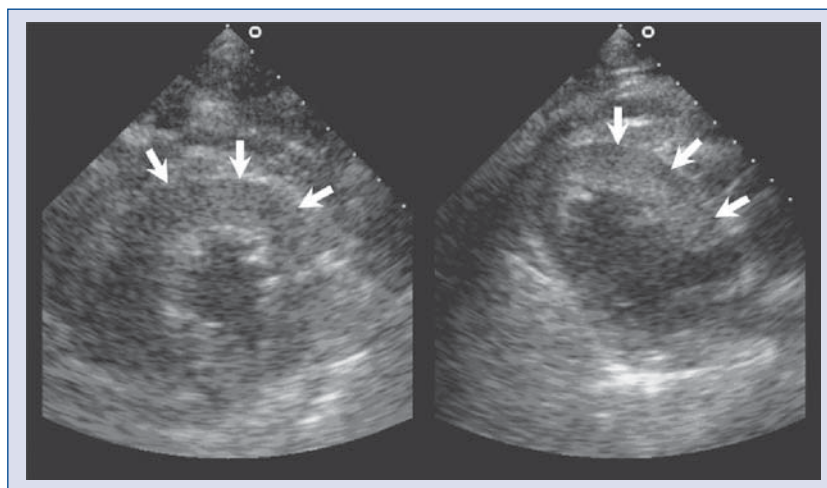
*We present a case of hemorrhagic myocardial infarction after early percutaneous coronary intervention which was suggested by cardiac echocardiography, and later confirmed by post-mortem examination. (Cardiol J 2011; 18, 4: 448–449)*

**Key words: intramyocardial dissection, intramyocardial hemorrhage**

## Introduction

A 74 year-old female presented with anterior wall myocardial infarction (MI). Percutaneous coronary intervention (PCI) of the proximal left anterior descending artery was performed two hours after onset of complaints, obtaining TIMI-II. Echocardi-

graphy revealed akinesia and speckled thickening of the antero-septal myocardium, leading us to suspect intramyocardial edema or bleeding (Fig. 1). Later she developed asystole and attempted resuscitation was in vain. Post-mortem autopsy revealed extensive transmural hemorrhage occupying the antero-septal myocardium extending to the lateral region (Fig. 2).

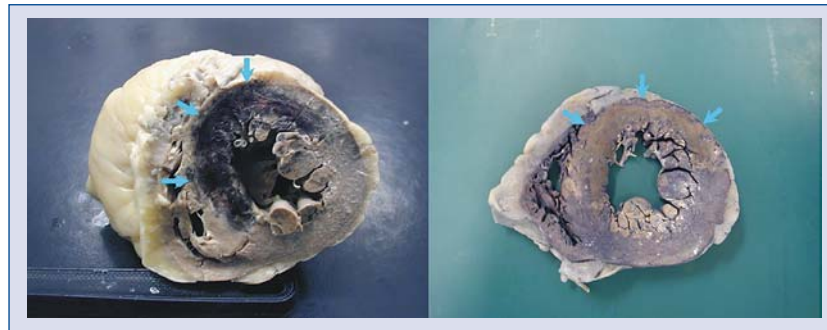


**Figure 1.** Transthoracic parasternal long and short axis echographic images demonstrating speckled thickening of the anterior-septal myocardium (arrows).

**Address for correspondence:** Becker S.N. Alzand, MD, Department of Cardiology, Maastricht University Medical Center, P. Debyelaan 25, PO Box 5800, 6229 HX Maastricht, the Netherlands, tel: +31 43 3876543, e-mail: alzand@hotmail.com

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**Figure 2.** Photograph of horizontal section of the heart; **A.** Gross hemorrhage occupies a large part of the region of distribution of the anterior descending coronary artery (arrows); **B.** Macroscopic NTB staining showing extensive transmural hemorrhagic infarction of the antero-septal myocardium with consequential thickening (arrows).

## Discussion

In contrast to non-reperfused infarctions, reperfusion may cause intramyocardial hemorrhage (IH) with massive extravasation of blood [1]. This has been described after prolonged bypass surgery, thrombolytic therapy and PCI performed late after onset of MI. It is not related to thrombolytic therapy, as was originally thought. The major determining factor is the time interval between coronary occlusion and reflow. IH after early reperfusion, as in our case, has previously been reported [2]. Hemorrhage is an important trigger for adverse left ventricle (LV) remodeling, myocardial stiffness, rupture and increased infarct size [3].

Progressive thickening after reperfusion and the failure to obtain TIMI-III flow are the most important diagnostic signs. Magnetic resonance imaging allows the detection of myocardial edema and hemorrhage. T2-images are sensitive to water-bound protons and hemoglobin. A hyperintense signal indicates edema, while a hypointense signal indicates hemorrhage [4]. Trials aimed to reduce reperfusion injury using nitric oxide donors, calcium channel blockers, or adenosine have not been encouraging. Drugs like  $\text{Na}^+/\text{Ca}^{2+}$  exchange inhibitors, protease inhibitors, and cyclic GMP mimetics are however promising [5].

In summary, we present a case of echocardiographic visualization of IH detected by increased LV mass and wall thickness after early PCI, which was later confirmed by autopsy.

## Acknowledgements

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