**INTRAMYOCARDIAL HEMORRHAGE AFTER ACUTE MYOCARDIAL INFARCTION; PATHOGENESIS AND CLINICAL IMPLICATIONS**

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Intramyocardial hemorrhage after acute myocardial infarction was frequently observed in post-mortem studies in the era of thrombolysis treatment. Scientific interest waned after the introduction of primary percutaneous coronary intervention which was believed not to be associated with intramyocardial hemorrhage. However, using dedicated cardiac magnetic resonance, intramyocardial hemorrhage can now be visualized in patients and recently published large series of patients showed that intramyocardial hemorrhage actually occurs in up to 50% of patients with successfully PCI-treated acute myocardial infarction. Intramyocardial hemorrhage is of prognostic importance and is associated with a larger final infarct size, adverse left ventricular remodelling and a higher incidence of major adverse cardiac events. It is believed that the hemorrhage is linked to adverse remodelling via the propagation of an inflammatory response. Especially iron particles, as breakdown products of haemoglobin play a role in the sustained inflammatory response. The sequel of events leading to intramyocardial hemorrage is not fully understood but the extravasation of erythrocytes occurs at a very early stage and is accompanied by massive destruction of the microcirculation upon reperfusion. These new insights have opened new avenues for additional treatment strategies in patients with acute myocardial infarction. Pharmaceutical protection of the endothelium, basal membrane and pericytes, or adjusting the way reperfusion is established, may prevent microvascular damage, attenuate intramyocardial hemorrhage development and prevent adverse remodelling.