Cardiogenic shock in acute myocardial infarction: clinical outcome and predictors
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Preface and outline of the thesis
Cardiogenic shock is the most extreme clinical manifestation of acute myocardial infarction. It is associated with the loss of more than 40% of the contractile left ventricular myocardium and extensive myocardial destruction. The clinical presentation is driven by acutely decreased myocardial contractility and pump failure developing early and late in the course of acute myocardial infarction. Acute decreased cardiac output is accompanied by increased left ventricular loading pressure, hypotension and forward failure. The result is a further reduction of myocardial perfusion and a deteriorating vicious circle. The inability to build up adequate stroke volume and maintain sufficient end organ perfusion leads to the Low Output Syndrome. This syndrome is characterized by reduced systolic blood pressure (<90 mmHg) in combination with evidence of end organ hypoperfusion such as cold extremities, molten skin, decreased urine output (< 30 ml/hour) and acidosis. This clinical syndrome is associated with a high mortality rate.

Early identification and subsequent revascularization, has reduced the incidence of cardiogenic shock late in the course of acute myocardial infarction. Nevertheless, the incidence of acute myocardial infarction patients that present with cardiogenic shock on admission has remained stable and accounts for approximately 7% of all acute myocardial infarction patients. Despite, early revascularization this ‘vicious circle of death’ is still the leading cause of in hospital mortality for patients admitted with acute myocardial infarction, with short term fatality rates around 44%.

**THIS THESIS**

In order to identify potentially modifiable predictors that may improve prognosis of these severely ill patients, several studies were undertaken and are presented in this thesis.

Our academic hospital is a large tertiary referral hospital for primary percutaneous intervention (pPCI) with one of the largest single center cohorts of patients presenting with cardiogenic shock described in current literature. For comparison, the well known multicenter SHOCK trial was performed in the nineties of the last century and represents a cohort of similar magnitude of around 300 patients that was gathered from 30 centers during a period of 5 years. For an important part of this the-
sis we performed several analyses in our single centre cohort comprising 292 patients that were treated over the course of 8 years.

OUTLINE

PART I portrays the predictive value of several biochemical parameters taken just before primary PCI.

CHAPTER 1 describes the prognostic value of hemoglobin. We investigated the relation between admission hemoglobin (Hb) concentration and one-year mortality in patients, with ST-segment elevation acute myocardial infarction and cardiogenic shock on admission, who were treated with primary percutaneous coronary intervention. Furthermore, in CHAPTER 2 we investigated the impact of admission glucose concentration and one year mortality in our cohort of patients referred for primary PCI and presenting with cardiogenic shock. CHAPTER 3 presents the strong association between the prognostic value of creatinine clearance on admission and one year mortality. The previous three parameters are closely related in daily clinical practice. We therefore investigated the independent value of these three parameters in a separate study, presented in CHAPTER 4.

PART II Comprises angiographic parameters in patients with acute myocardial infarction complicated by cardiogenic shock.

CHAPTER 5 is an extensive review on the topic of cardiogenic shock in patients with acute myocardial infarction. Insight in pathophysiology of circulatory failure with all compensatory mechanisms to preserve end organ perfusion is provided. Treatment modalities according to the guidelines besides our single centre experiences are described. Early recognition and prehospital (in ambulance) stratification in patients prone to develop cardiogenic shock are potential targets that may lead to improved clinical outcome. Future directions towards a new era of percutaneous available left ventricular (LV) support devices are outlined. As mentioned before, cardiogenic shock occurs in patient with a large area of myocardium at risk. As such, patients with acute myocardial infarction due to left main related culprit lesions and subsequent PCI usually present with cardiogenic shock. However, the absolute number of
these patients is small and reported clinical outcome varies greatly. We therefore performed a systematic review and meta-analysis of all available cohorts in the current literature on primary PCI for unprotected left main disease in acute myocardial infarction patients. **CHAPTER 6** describes this systematic review. We calculated an average mortality for patients treated for left main coronary artery disease with or without cardiogenic shock on presentation. These mortality rates can be used as a benchmark for further studies. In **CHAPTER 7** we present our own single centre experience on the clinical outcomes after percutaneous or surgical revascularization of unprotected left main coronary artery related myocardial infarction. Determinants for either types of revascularization are depicted. Some of these findings may set the stage for future strategy in patients while glancing at the next chapter. The following **CHAPTER 8** describes the influence of multivessel disease with and without a chronic total occlusion in cardiogenic shock on short and long term prognosis.

**PART III** depicts several hemodynamic factors in cardiogenic shock.

In **CHAPTER 9** and **10** we present our research on the influence of mitral regurgitation and right ventricular dysfunction as an independent predictor of one-year mortality in ST-elevation myocardial infarction patients presenting with cardiogenic shock on admission. Finally, **CHAPTER 11** describes a systematic review and meta-analysis of intra-aortic balloon pump therapy in patients with acute myocardial infarction.
REFERENCES


