Journal of Cardiology 66 (2015) 417–422

Contents lists available at ScienceDirect

Journal of Cardiology

journal homepage: www.elsevier.com/locate/jjcc

Original article

Effect of the Mehran risk score for the prediction of clinical outcomes after percutaneous coronary intervention

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ARTICLE INFO

Article history:
Received 31 October 2014
Received in revised form 13 December 2014
Accepted 24 December 2014
Available online 23 January 2015

Keywords:
Contrast-induced acute kidney injury
Mehran risk score
Clinical outcome

ABSTRACT

Background: The association of Mehran risk score (MRS) with long-term prognosis in patients treated with percutaneous coronary intervention (PCI) has not been fully reported. We investigated the association between MRS and clinical outcomes in patients who underwent PCI.

Methods: Study subjects comprised 2198 patients treated with PCI from the Ibaraki Cardiovascular Assessment Study multicenter registry, excluding patients receiving hemodialysis or who died within 7 days. We categorized them into 4 groups according to MRS (low-risk: ≤5; medium-risk: 6–10; high-risk: 11–16; and very high-risk: ≥16). Contrast-induced acute kidney injury (CI-AKI) was defined as an increase of 0.5 mg/dL or 25% in pre-PCI serum creatinine within 1-week post procedure. We evaluated CI-AKI and major adverse cardiac and cerebrovascular events (MACCE), and defined as all-cause death, myocardial infarction, congestive heart failure, or cerebrovascular disorder (stroke or transient ischemic attack).

Results: A total of 192 (8.7%) patients developed CI-AKI. At multivariate analysis, odds ratio for CI-AKI was 4.09 (95% CI: 1.72–9.17, $p = 0.002$) in the very high-risk group, 1.49 (95% CI: 0.89–2.42, $p = 0.120$) in the high-risk group, and 1.08 (95% CI: 0.74–1.54, $p = 0.693$) in the medium-risk group, as compared with the low-risk group. MACCE in the very high-risk group was more than 5-fold higher [hazard ratio (HR) 5.40, 95% CI: 2.96–9.28, $p < 0.001$] compared with the low-risk group and was also increased in the high-risk (HR 3.72, CI: 2.59–5.32, $p < 0.001$) and medium-risk groups (HR 1.97, CI: 1.45–2.69, $p < 0.001$). Kaplan–Meier analysis showed that increasing risk for MACCE was seen across the groups as MRS increased ($p < 0.001$).

Conclusion: MRS might provide potentially useful information for prediction of CI-AKI and clinical outcomes after PCI.

Introduction

Contrast-induced acute kidney injury (CI-AKI) is an important complication that can occur after percutaneous coronary intervention (PCI) [1–3]. It has been associated with both short- and long-term adverse outcomes, including the need for renal replacement therapy, major cardiac adverse events, and mortality [4]. Patients with chronic kidney disease (CKD), diabetes mellitus, congestive heart failure, older age, hypotension, and anemia are at particular risk of CI-AKI, and Mehran et al. devised a simple risk score for prediction of CI-AKI after PCI [5]. The Mehran risk score (MRS) is a scoring system based on comorbidities and procedural risk factors, including hypotension, intra-aortic balloon pumping, heart failure, age >75 years, anemia, diabetes mellitus, volume of contrast, and renal function [5] (Table 1). Predicted incidences of CI-AKI are reported to be 7.5%, 14%, 26.1%, and 57.3% for scores

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http://dx.doi.org/10.1016/j.jjcc.2014.12.016
0914-5087/

Table 1
Parameters in Mehran risk scores.

Risk factors	Integer score
Hypotension	5
IABP	5
CHF	5
Age > 75 years	4
Anemia	3
Diabetes	3
Contrast media volume	1 for each 100 cm^3
eGFR 40–60 mL/min/1.73 m^2	2
eGFR 20–40 mL/min/1.73 m^2	4
eGFR <20 mL/min/1.73 m^2	6

IABP, intra-aortic balloon pump; CHF, congestive heart failure; eGFR, estimated glomerular filtration rate.

of ≤5 (low-risk), 6–10 (medium-risk), 11–15 (high-risk), and ≥16 (very high-risk), respectively. Sgura et al. demonstrated that the MRS can also be applied in an ST-elevation myocardial infarction (STEMI) population and is able to predict CI-AKI development and to discriminate between low-, medium-, high-, and very high-risk patient subgroups for consequent poor clinical outcomes during both short- and long-term follow-ups [6]. Prediction of long-term clinical outcomes in patients with coronary artery disease (CAD) treated with elective and emergent PCI except for STEMI patients is also important as is the prediction of CI-AKI development and early risk stratification [7,8]. However, the association of the MRS with long-term prognosis in patients treated with PCI has not been fully reported. To address this issue, we investigated the association between MRS and clinical outcomes in patients who underwent PCI.

Materials and methods

Study design

The Ibaraki Cardiovascular Assessment Study (ICAS) registry was designed as a retrospective multicenter observational study of CAD in Ibaraki Prefecture, Japan. All consecutive CAD patients who underwent PCI at 12 cooperating centers were enrolled in this registry [9]. Study subjects are summarized in Fig. 1. A total of 2657 patients were enrolled from April 2007 to August 2010. We excluded the patients on dialysis (n = 83), those who died within 7 days of their PCI (n = 34), and those with no information regarding clinical outcome or serial measurement of creatinine (n = 342). The study population thus comprised 2198 patients who were divided

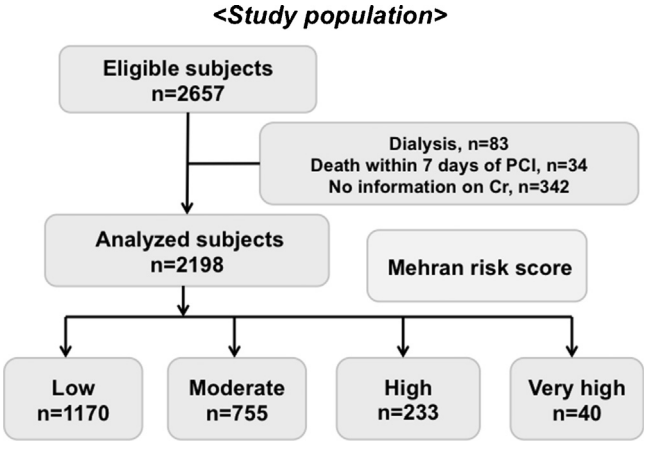

Fig. 1. Study flow chart. PCI, percutaneous coronary intervention; Cr, creatinine.

into 4 groups according to MRS: low-risk, ≤5 (n = 1170); medium-risk, 6–10 (n = 755); high-risk, 11–15 (n = 233), and very high-risk, ≥16 (n = 40). Patients with baseline renal impairment [estimated glomerular filtration rate (eGFR) <60 mL/min/1.73 m^2] would receive approximately 12 h of pre-hydration with 0.9% normal saline at a rate of 1 mL/kg body weight per hour in the elective setting. Saline hydration would continue during the PCI procedure until 12 h post-PCI. In the urgent setting, hydration would be started immediately if renal impairment had been identified. The use of prophylaxis strategies, including N-acetylcysteine and sodium bicarbonate, was made at the discretion of treating physicians. This study was approved by the institutional review boards or ethics committees of all participating institutions.

Clinical data collection

Patient demographic information, cardiovascular risk factors, laboratory findings, angiographic findings, and PCI procedure characteristics were recorded according to medical charts on initial enrollment in the registry. The serum concentration of creatinine was measured serially, before the exposure to contrast and at 24, 48, and 72 h after PCI. When the creatinine concentration was not available for day 2 or 3, we used the value obtained within 1 week after contrast exposure. An eGFR was calculated by using the Modification of Diet in Renal Disease study equation modified for the Japanese population [10]. Contrast volume used during each PCI was recorded. The MRS was calculated on the basis of information collected before the procedure.

Study endpoints

All follow-up data and clinical events were surveyed once a year during the study period. Data for patients who were lost to follow up were censored at the time of the last contact. The primary endpoint for the present analysis was the incidence of major adverse cardiac and cerebrovascular events (MACCE), which were defined as all-cause death, myocardial infarction, congestive heart failure, or cerebrovascular disorder (stroke or transient ischemic attack). Secondary endpoints included the development of CI-AKI defined as an increase in serum creatinine of more than 25% or 0.5 mg/dL from the baseline within 1 week after contrast exposure [7]. The serum creatinine concentration was not available on day 2 or 3 for 25% of the patients, and we used the value obtained within 1 week after contrast exposure. Additional secondary endpoints included the individual endpoint of requiring dialysis or all-cause death, myocardial infarction, congestive heart failure, or cerebrovascular disorder. All deaths were confirmed by medical charts or by contacting the referring physician and/or patient's family, and all events were registered by the attending physicians.

Coronary angiography and PCI procedure

PCI was performed according to standard techniques. All patients received treatment with aspirin (100 mg/day) and clopidogrel (75 mg/day following a 300-mg loading dose). A glycoprotein IIb/IIIa receptor inhibitor is not yet available in Japan. Operators selected interventional devices and performed PCI through either the radial, brachial, or femoral artery using 5–7 French catheters. A low-osmolality, non-ionic contrast agent was used (iopamidol 350 mg I/mL; Schering AG, Berlin, Germany). Before starting the procedure, 8000 units of heparin were given intravenously, and the activated clotting time was maintained at >300 s. Once the guide wire passed through the culprit lesion, balloon dilatation or stent placement was performed. Treatment was regarded as successful when the luminal diameter of the target lesion increased by at least by 50%, residual stenosis was less