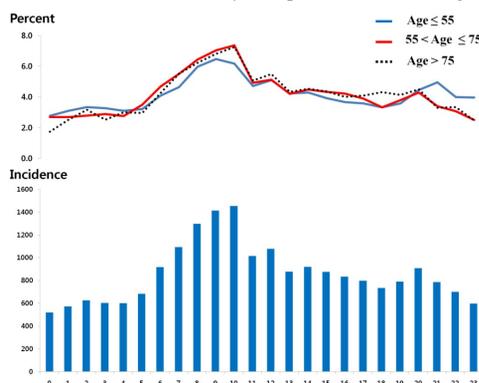


## Relationship between age distribution and circadian pattern in Myocardial Infarction

1Division of Cardiology, Department of Internal Medicine, Kyung Hee University Hospital, Kyung Hee University, Seoul,

\*Jung Rock Moon<sup>1</sup>, Jong Shin Woo<sup>1</sup>, Jae Min Kim<sup>1</sup>, Hyung Oh Kim<sup>1</sup>, Hyemooon Chung<sup>1</sup>, Jung Myung Lee<sup>1</sup>, Soo Joong Kim<sup>1</sup>, Myung Ho Jeong<sup>2</sup>, Weon Kim<sup>1</sup>

**Background:** Many epidemiologic studies have been reported the morning peak incidences of acute myocardial infarction (AMI). However, short and long-term clinical outcomes and relationship between age distribution and circadian pattern have not been fully investigated in patients with AMI. **Methods:** From Korea Acute Myocardial Infarction Registry (KAMIR) database, we analyzed 20684 eligible patients (11513 STEMI, 9172 NSTEMI; age = 64±13 years) who had primary percutaneous coronary intervention (PCI) and early invasive PCI. The clinical impact of circadian variation was evaluated among four 6-hour intervals (12:00 midnight-6:00 AM, 6:00 AM-12:00 noon, 12:00 noon-6:00 PM, 6:00 PM-12:00 midnight). Major adverse cardiac events (MACEs) including death, nonfatal MI, repeated revascularization at 12-month were evaluated. **Results:** There was a marked circadian variation with increased incidences of AMI during the second quarter of a day (6:00 AM to 12:00 noon) regardless of age distribution and other factors. Patients with second quarter of a day showed significantly low portion of Killip class higher than II, smoking history and lower levels of peak CK-MB and troponin-I. In-hospital mortality, MACEs were not significantly different for 12-month follow-up. Instead, old age, hypertension, diabetes, dyslipidemia and Killip class higher than II were independent factors for 12-month MACEs. **Conclusions:** Age distribution do not related with onset time of AMI, but old age and additional comorbidities are likely to explain the deteriorating outcomes in patients with primary PCI.



## In-Stent Restenosis-prone Lesion Characteristics

Cardiology, Konyang University Hospital, Daejeon, Korea

\*Duck-Jun Seo, Yong-Kyun Kim, Ki-Hong Kim, Taek-Geun Kwon, In-Geol Song, Dong-Ju Yang, Wan-Ho Kim, Yo-Han Park, Hwan-Hyi Cho, Young-Hoon Seo, Hyun-Woong Park, Jang-Ho Bae

**Objective:** In-stent restenosis (ISR) remains still an important issue even in drug-eluting stents era. We hypothesized that higher inflammatory reaction, which is known as an important atherosclerotic process, at the culprit lesion may have higher restenosis. We sought to evaluate the baseline coronary plaque composition in patients with ISR. **Methods:** Study population consisted of 241 patients with coronary artery disease, who underwent percutaneous coronary intervention with virtual histology- intravascular ultrasound (VH-IVUS) and 9 months follow up coronary angiography. We compared coronary plaque composition between patients with ISR and those without ISR. **Results:** Patients with ISR (n=27, 11.2%) were likely to be older (66.2±9.5 years vs. 58.7±11.7 years, p=0.002), increased high sensitivity C-reactive protein (hs-CRP, 1.60±3.59 mg/dl vs. 0.31±0.76 mg/dl, p<0.001) than those without ISR (n=214, 88.8%). Baseline angiographic, procedural findings and gray scale IVUS findings showed no significant differences between 2 groups. VH-IVUS examination showed necrotic core volume (22.1±19.9 mm<sup>3</sup> vs. 14.2±12.7 mm<sup>3</sup>, p=0.045) and percent necrotic core volume (14.3±8.7% vs. 19.5±9.1%, p=0.005) were higher in those without ISR than those with ISR, whereas percent fibrofatty volume was higher (16.6±9.7% vs. 12.4±8.4%, p=0.018) in those with ISR than those without ISR. The independent predictors for ISR were hs-CRP (Odd ratio=3.334, 95% confidence interval (CI); 1.158-9.596, p=0.026), and age (Odd ratio=3.557, 95% CI; 1.242-10.192, p=0.018). **Conclusions:** This study suggests that ISR was not associated with baseline coronary plaque composition but old age and an increased inflammatory marker such as hs-CRP. **Keywords:** Intravascular Ultrasound, In-stent Restenosis, Inflammation and restenosis after percutaneous coronary intervention