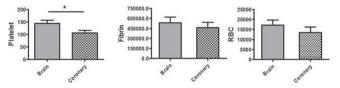
in patients with ST-segment elevated myocardial infarction and from 12 middle cerebral arteries in patients with cardioembolic infarction. The thrombi sectioned at a thickness of 5-mm were treated with an anti-PECAM-1 (#3528, Cell Signaling) and glycophorin A (sc-20628, Santa Cruz Biotechnology). The staining for fibrin was performed using PTAH stating kit (ab150683, Abcam). The density of all signals was analyzed in 400 x magnification using NIS-Elements BR 3.2 (Nikon, Japan).

Results: Intracerebral thrombi had higher proportion of platelets than intracoronary thrombi (P=0.021) (Figure). However, the composition of intracerebral and intracoronary thrombi did not differ significantly for fibrin (P=0.525) and red blood cell (P=0.443) (Figure).



Conclusion: Intracerebral thrombi from patients with cardioembolic infarction showed high density of platelet. This suggests that antiplatelet agents may prevent a thrombus growth in acute phase after stroke.

OUTCOME OF ACUTE CORONARY SYNDROMES

P811

Impact of non-infarct-related artery occlusion on short-term mortality in STEMI patients: insight from Tokyo CCU network database

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Background: A potential benefit of CTO recanalization on long-term survival could be related to protection from future coronary events. Actually, concurrent occlusion in non-infarct-related artery (non-IRA) has been reported in previous studies to have prognostic impact on short-term and long-term mortality in STEMI patients. However, data are still limiting.

Methods and results: This study was performed by using data from Tokyo CCU Network registered cohort in 2010–2015. Tokyo CCU Network is operated through over 70 hospitals with the help of ambulance units through the control room of the Tokyo Fire Department. For evaluating clinical impact of concurrent occlusion in non-IRA, a total of 11809 STEMI patients without history of CABG and OMI were analyzed. Among those, 8.0% of all patients had non-IRA occlusion. Patients with non-IRA occlusion were more frequently to be in shock status and needed circulation assist device including IABP and PCPS more often than patients without non-IRA occlusion. Kaplan-meier estimates revealed that 30-day mortality of patients with non-IRA occlusion was 16.8%, which was significantly higher than that of those without non-IRA occlusion. (30-day mortality: 7.3%).

Predictors for all-cause death

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Variables	OR (95% CI)	P value
Age	1.024 (1.014-1.034)	< 0.001
Male		
BMI		
Smoking	0.683 (0.548-0.852)	0.001
Hypertension		
Dyslipidemia	0.637 (0.510-0.797)	< 0.001
Diabetes		
CKD	2.721 (2.143-3.455)	< 0.001
Hemodialysis		
Prior PCI		
Prior stroke		
Hemoglobin		
Noninfarct-related artery stenosis		
Noninfarct-related artery occlusion	2.417 (1.901-3.073)	< 0.001

Stepwise multivariate Cox proportional hazard model for all-cause death

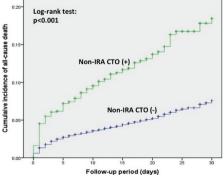


Figure 1. Kaplan-Meier curve for all-cause death

Multivariate cox regression analysis showed that non-IRA occlusion was an independent predictor of 30-day mortality. (HR:2.417 (1.901–3.073), p<0.001)

Conclusion: Non-IRA occlusion may contribute to worse short-term outcome in STEMI patients.

P812

Clinical outcomes of ST-elevation myocardial infarction secondary to stent thrombosis treated by percutaneous coronary intervention

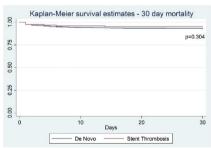
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Background: Stent thrombosis (ST) is one of the rare but recognized serious complications behind coronary stenting with substantial associated morbidity and mortality. Data regarding the long-term outcomes of ST-elevation myocardial infarction (STEMI) secondary to ST compared to those with de novo culprit lesions are sparse.

Purpose: The objective of this study was to assess the outcomes of patients presenting with STEMI and treated by percutaneous coronary intervention (PCI), comparing ST versus de novo culprit coronary lesions from a single multi-centre Australian registry.

Methods: We studied consecutive patients who underwent PCI for STEMI from 2005 to 2013, enrolled prospectively in the Melbourne Interventional Group (MIG) registry. Patients were retrospectively divided into two groups according to the cause of STEMI; the ST group comprised patients with STEMI due to stent thrombosis. The de novo group comprised the remainder of the STEMI cohort. The primary endpoint was long-term all-cause National Death Index-linked mortality which was obtained by linkage to the Australian National Death Index (NDI).

Results: Compared to the de novo group (n=3,835), the ST group (n=128) had higher rates of cardiovascular disease risk factors including diabetes, hypertension and dyslipidemia (all p<0.05). In addition, established cardiovascular diseases were more prevalent in the ST group such as previous myocardial infarction, previous coronary artery bypass graft surgery, heart failure and peripheral vascular disease (all p<0.01). The ST group had shorter symptom-to-door time compared to the de novo group (140.1min ± 122.9 vs. 197.8min ± 170.4, p=0.032) and although there was no significant difference in the door-to-balloon time between the two groups, the ST group had a shorter symptom-to-balloon time (219.37min \pm 146.92 vs. 269.01min \pm 220.58, p=0.051). Within the ST group, very late stent thrombosis was the most common form of ST, followed by late and early stent thrombosis (64.2%, 19% and 16.8% consecutively). There was no significant difference in the primary outcome of long-term NDI-linked mortality between the ST group and the de novo group (7% vs. 11.9%, p=0.16). Similarly, there were no differences in mortality, major adverse cardiac outcomes (MACE) during in-hospital, at 30-days and 12-months follow-up between the two groups. In-hospital complications of recurrent stent thrombosis, bleeding and blood transfusion requirement were significantly higher in the ST group (all p<0.01). On multivariable analysis, ST was not independently associated with outcomes for 30-day mortality (OR 0.62, 95% CI 0.07-1.09, P=0.068) and 30-day MACE (OR 0.88, 95% CI 0.41-1.8, p=0.75),



Kaplan-Meier 30-day survival estimates

Conclusion: The long-term prognosis of patients with ST presenting with STEMI and treated by PCI is equivalent in that of patients with STEMI due to de novo lesions. However, the ST group presented to hospital sooner and sustained higher rates of in-hospital complications.