

Systemic immune-inflammation index predicts major adverse cardiovascular events in patients with ST-elevation myocardial infarction

A. Denegri¹, S. Obeid², L. Raeber³, S. Windecker³, B. Gencer⁴, F. Mach⁴, N. Rodondi⁵, D. Heg⁶, D. Nanchen⁷, C.M. Matter⁸, R. Klingenberg⁹, T.F. Luescher¹⁰

¹Azienda Ospedaliero Universitaria, Modena, Italy; ²Cantonal Hospital Aarau, Division of Cardiology, Aarau, Switzerland; ³Bern University Hospital, Cardiology, Bern, Switzerland; ⁴Geneva University Hospitals, Cardiology, Geneva, Switzerland; ⁵University Hospital, Department of Family Medicine, Bern, Switzerland; ⁶Institute of Social and Preventive Medicine, University of Bern, Bern, Switzerland; ⁷Centre for Primary Care and Public Health (Unisante), Lausanne, Switzerland; ⁸University Hospital Zurich, Cardiology, Zurich, Switzerland; ⁹Kerckhoff Heart and Thorax Center, Department of Cardiology, Bad Nauheim, Germany; ¹⁰Royal Brompton and Harefield Hospital, London, United Kingdom

Funding Acknowledgement: Type of funding sources: None.

Background: ST-elevation myocardial infarction (STEMI) represents the life-threatening manifestation of atherosclerosis, a chronic inflammatory disease of arterial wall, and is associated with high rate of morbidity and mortality. Thus, inflammatory biomarkers may be useful in identifying high inflammatory burden patients who may benefit from tailored high-intensity secondary prevention therapy.

Purpose: We therefore assessed the relationship between the systemic immune-inflammation index (SII) and CV outcomes among 1144 all-comers patients admitted to four Swiss University Hospital for STEMI and enrolled in the prospective multicenter SPUM registry cohort I (NCT 01000701).

Methods: SII was calculated as platelet counts x neutrophil counts / lymphocyte counts. Patients were subdivided into three groups according to SII tertiles. The composite primary endpoint was major adverse cardiac and cerebrovascular events (MACCE: stroke, myocardial infarction, CV death). Adjusted Cox proportional hazards regression models were implemented to determine the risk associated with SII and outcomes.

Results: Out of 1144 STEMI patients, 912 patients (79.7%) had avail-

able for SII. Patients within the highest tertile were slightly more frequently male (23.0 vs 22.0%, $p=0.05$), with higher plasma values of neutrophils (11.4 ± 2.4 vs 6.5 ± 3.7 G/l, $p<0.001$), platelets (275.3 ± 97.5 vs 202.5 ± 51.6 G/l, $p<0.001$) and lower levels of lymphocytes (1.0 ± 0.6 vs 2.1 ± 1.1 G/l, $p<0.001$) and LVEF ($46.4\pm 11.5\%$ vs $50.4\pm 10.3\%$, $p<0.001$) (Fig. 1A). At 1 year, these patients presented the highest rate of all-cause mortality (7.2% vs 2.6%, $p=0.02$) and MACCE (8.2% vs 3.3%, $p=0.03$). This enhanced risk persisted for all-cause mortality and MACCE, after adjustment for age, sex, ace-inhibitors and statin therapy (Adj. HR 2.85, 95% CI 1.30–6.70, $p=0.03$ and Adj. HR 2.63, 95% CI 1.25–5.55, $p=0.03$, respectively, Fig. 1B).

Conclusions: Among a real-world cohort of STEMI-patients, SII highlights the highest inflammatory risk phenotype, being associated with significant increased rates of MACCE and all-cause of death. These observations might help clinicians to furtherly identify patients who may derive the greatest benefit from tailored more intense secondary prevention therapies including inflammatory modulation.

	Low Tertile	Mid Tertile	High Tertile	p value
Age (years)	62.9±12.5	62.5±12.2	62.6±12.6	0.932
Female	23.0	15.8	22.0	0.055
CAD	23.7	22.5	22.0	0.894
PAD	3.0	3.0	2.3	0.554
Hypertension	54.9	48.7	52.9	0.267
Smoking	56.2	48.3	53.0	0.108
Neutro (G/l 10 ⁹)	6.5±3.7	8.9±2.6	11.4±2.4	<0.001
Lympho (G/l 10 ⁹)	2.1±1.1	1.5±0.6	1.0±0.6	<0.001
Platelets (G/l 10 ⁹)	202.5±51.6	230.4±57.5	275.3±97.5	<0.001
Glucose (mmol/l)	7.1±2.5	7.5±2.8	8.1±4.1	0.002
LVEF(%)	50.4±10.3	49.2±11.0	46.4±11.6	<0.001

Fig.1A

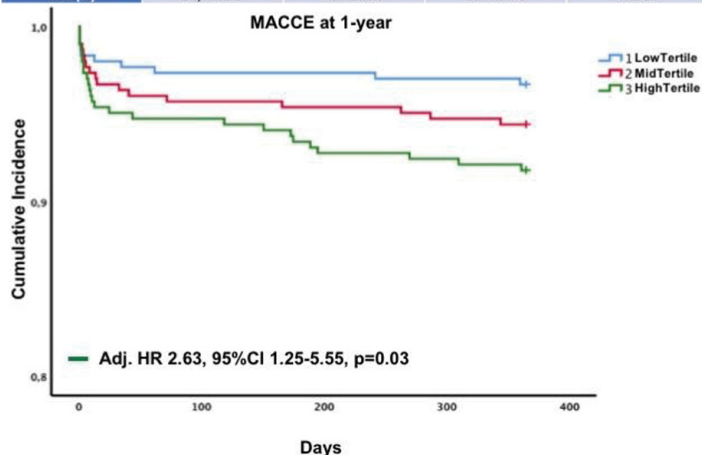


Fig.1B

Figure 1