Uncontrolled hypertension and elevated NT-proBNP predict acute kidney injury and cardiac death in all-comer patients 1 year after acute coronary syndromes

A. Denegri[1], L. Raeber[2], S. Windecker[2], B. Gencer[3], F. Mach[3], N. Rodondi[4], D. Heg[5], D. Nanchen[6], R. Klingenberg[7], C.M. Matter[7], T.F. Luescher[8]

[1]Azienda Ospedaliero Universitaria, Modena, Italy; [2]Bern University Hospital, Cardiology, Bern, Switzerland; [3]Geneva University Hospitals, Cardiology, Geneva, Switzerland; [4]Bern University Hospital, Inselspital, Department of Family Medicine, Bern, Switzerland; [5]University of Bern, Institute of Social and Preventive Medicine, Bern, Switzerland; [6]University of Lausanne, Center for Primary Care and Public Health, Lausanne, Switzerland; [7]University Hospital Zurich, Cardiology, Zurich, Switzerland; [8]Harefield Hospital, Royal Brompton and Harefield NHS Foundation Trust, London, United Kingdom

Funding Acknowledgement: Type of funding source: None

Background: Hypertension is a recognized cardiovascular (CV) risk factor and, although many highly effective antihypertensive drugs have been developed, most patients fail to achieve recommended blood pressure target levels. This may increase major adverse CV events after acute coronary syndromes (ACS) such as acute kidney injury (AKI) and cardiac death (CD).

Purpose: We assessed the prognostic value of uncontrolled hypertension (UH) and elevated NT-proBNP among 2,168 all-comer patients admitted to 4 Swiss University Hospitals for acute coronary syndromes (ACS) enrolled in the prospective multicenter SPUM registry.

Methods: Patients with UH defined as a systolic blood pressure≥140 mmHg, and a NT-proBNP>900 ng/l were considered for the analysis. The composite primary endpoint was AKI and CD. Adjusted Cox proportional hazards regression models were implemented to determine risk prediction for UH and elevated NT-proBNP levels.

Results: Out of 2,168 ACS patients, 235 patients (10.8%) showed UH and NT-proBNP>900 ng/l (Fig. 1A). Compared to the general ACS population, those with UH and elevated NT-proBNP were more likely to be older (41.7% vs 20.0%, p<0.001), of female sex (36.2% vs 19.7%, p<0.001) and with a more complex history of CV disease, such as hypertension (77.0% vs 56.2%, p<0.001), diabetes (24.7% vs 17.5%, p=0.006), peripheral artery disease (9.4% vs 5.2%, p=0.011), cerebrovascular disease (6.8% vs 3.4%, p=0.013), chronic heart failure (3.4% vs 1.3%, p=0.025), dialysis (2.1% vs 0.3%, p=0.004) as well as prior CABG (9.4% vs 5.2%, p=0.010) and more often admitted as NSTEMIs (59.6% vs 40.9%, p<0.001). Although these patients were on a more aggressive antihypertensive therapy at admission (all p<0.05 for ACEi, ARB, Beta-blockers, calcium antagonists, nitrates and diuretics), there was a higher rate of death (OR 1.83, 95% CI 1.07–3.14, p=0.027), CD (OR 2.13, 95% CI 1.19–3.81, p=0.009), AKI (OR 2.83, 95% CI 1.41–5.67, p=0.002) and composite endpoint AKI+CD (OR 2.46, 95% CI 1.56–3.90, p<0.001) at one year. This combined risk persisted after adjustment for baseline differences, with a 71% (Adj. HR 1.71, 95% CI 1.44–1.84, p=0.003) increase for the composite endpoint (Fig. 1B).

Conclusions: Among a real-world cohort of ACS patients, coexistence of UH with elevated levels of NT-proBNP confers increased risk for AKI and CD up to one year after ACS. These observations might help clinicians to identify ACS patients at risk using simple clinical parameters and biomarkers and to target them for more intense preventive therapies.

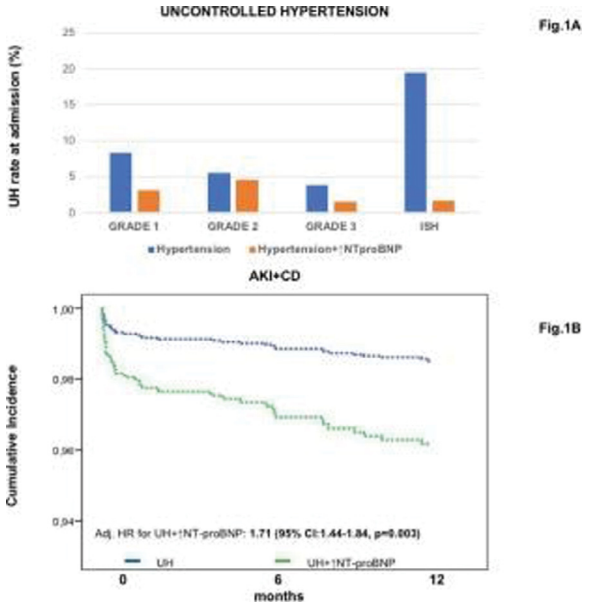

Figure 1. A: GRADE1 = 140–159 mmHg and/or 90–99 mmHg; GRADE2 = 160–179 mmHg and/or 100–109 mmHg; GRADE3 = ≥180 mmHg and/or ≥110 mmHg; ISH (isolate systolic hypertension) = ≥140 mmHg and <90 mmHg; NT-proBNP = N-terminal-pro B-type natriuretic peptide. B: UH = uncontrolled hypertension; AKI = acute kidney injury; CD = cardiac death.