Novel risk factor for fatal arrhythmia in Brugada syndrome

R. Nishinarita[1], S. Niwano[1], N. Ishizue[1], T. Satoh[1], G. Matsuura[1], Y. Arakawa[1], S. Kobayashi[1], Y. Shirakawa[1], A. Horiguchi[1], H. Nakamura[1], J. Oikawa[1], J. Kishihara[1], H. Fukaya[1], H. Niwano[2], J. Ako[1]

[1]Kitasato University School of Medicine, Department of Cardiovascular Medicine, Sagamihara, Japan; [2]Tamagawa University, Department of Education, Machida, Japan

Funding Acknowledgement: Type of funding source: None

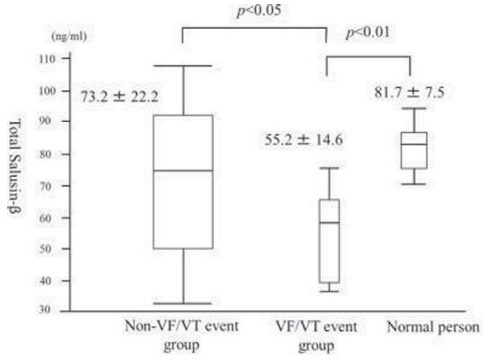
Background: Brugada syndrome (BrS) is a genetic disease associated with increased risk of ventricular fibrillation (VF)/ventricular tachycardia (VT). The VF/VT in BrS occurs more frequently during a night time or at a rest with parasympathetic nerve activation. Some risk factors of VF/VT occurrence in BrS have been elucidated, however, it remains controversial about risk stratification. Salusin-β is an endogenous bioactive peptide that systemically exerts rapid and profound hypotensive and bradycardic activities and parasympathomimetic hemodynamic actions in vivo. Previous reports suggested that salusin-β is suppressed following physiological parasympathetic stimulation and appears to constitute a negative feedback relationship with the parasympathetic nervous system.

Purpose: We hypothesized that salusin-β is associated with the occurrence of VF/VT in BrS.

Methods: The study population consisted of 26 BrS patients with newly implantation of implantable cardioverter defibrillator (ICD) during 2003–2008. In all patients, salusin-β was measured in supine position after 20 minute rest. The date of salusin-β sampling was set as the registration point for this study. The VF/VT was defined as any episode of fatal ventricular tachyarrhythmia or any appropriate shock. In accordance with the presence or absence of VF/VT events within 5 years, all patients were divided into VF/VT group (n=6) and non-VF/VT group (n=20).Various clinical parameters were compared between the two groups. For analysis of autonomic nervous function, heart rate variability (HRV) and pupil function were evaluated.

Results: The mean age was 54±17 years old. There is no differences between the two groups in clinical parameters. In analysis of HRV, the high-frequency component (0.15–0.40 Hz; HF), low frequency component (0.04–0.15 Hz; LF) and the LF/HF ratio were analyzed over 24 h. LF/HF ratio was significantly lower over 24h in VF/VT groups in comparison with non-VF/VT groups [day-time; 1.8 (1.2–5.6) vs. 5.2 (3.4–8.8), p=0.048, night-time; 1.2 (1.1–1.3) vs. 3.9 (2.5–8.7), p=0.003]. Furthermore, in analysis of pupil function, right/left miosis ratio was higher in VT/VF groups in comparison with non-VT/VF groups [right pupil; 0.39 (0.37–0.59) vs. 0.34 (0.28–0.38), p=0.035, left pupil; 0.43 (0.36–0.50) vs. 0.33 (0.28–0.40), p=0.049]. In plasma total salusin-β levels, the VF/VT groups exhibited significantly lower than non-VF/VT groups (55.2±14.6 vs. 73.2±22.2, p=0.039, Figure).

Conclusions: Salusin-β was associated with the occurrence of VF/VT in Brugada syndrome. Salusin-β might be useful to identify high-risk patients for the occurrence of VT/VF events in Brugada syndrome.



Difference of plasma total salusin-β levels among the three groups (non-VF/VT group, VF/VT group, normal person)