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Prediction of cardiovascular events by atheromatous plaques detected by non-obstructive general angioscopy: two-year results of EAST-NOGA Registry

T. Kakuta[1], S. Komatsu[2], K. Kojima[3], H. Fujii[4], S. Kimura[4], K. Dai[5], H. Kawakami[6], H. Matsuoka[7], Y. Higuchi[8], H. Abe[9], T. Inoue[9], Y. Okumura[3], M. Asakura[10], A. Hirayama[8], K. Kodama[2]

[1]Tsuchiura Kyodo Hospital, Tsuchiura, Japan; [2]Osaka Gyoumeikan Hospital, Osaka, Japan; [3]Nihon University School of Medicine, Tokyo, Japan; [4]Yokohama Minami Kyosai Hospital, Yokohama, Japan; [5]Hiroshima City Hospital, Hiroshima, Japan; [6]Ehime Prefectural Imabari Hospital, Imabari, Japan; [7]Ehime Prefectural Central Hospital, Matsuyama, Japan; [8]Osaka Police Hospital, Osaka, Japan; [9]Dokkyo Medical University, Mibu, Japan; [10]Hyogo College of Medicine, Nishinomiya, Japan

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Background: Non-obstructive general angioscopy (NOGA) has revealed the intimal damages or atheromatous plaques as well as its spontaneous rupture of the aorta. Recent study revealed that plaque debris or different size of cholesterol crystals were detected in the blood above the spontaneous ruptured aortic plaque observed by NOGA and these plaque materials might cause the peripheral organ damages as the embolic source. These various morphological changes may cause the acute aortic events or atheroembolic events on the peripheral organs, such as brain, kidney, peripheral artery and so on.

Purpose: EAST-NOGA (Evaluation of AtheroSclerotic and rupture events by Non-Obstructive General Angioscopy) is a multi-center prospective observational study to assess the relationship between the findings of NOGA and future cardiovascular events.

Methods: Five hundred and seventy-seven patients with atherosclerotic cardiovascular disease who underwent NOGA study. The major cerebrocardiovascular events including cardiovascular death, non-fatal myocardial infarction, non-fatal cerebral infarction, and acute aortic syndrome were accumulated during the 2-year follow-up after NOGA study.

Results: The median number of aortic atheromatous plaques was 6 [IQR: 3–12]. A total of 514 patients were followed up (89.1%). The mean follow-up duration was 757±120 days. Major adverse cardiovascular events developed in 23 (4.5%) during 2 years follow-up. Patients with MACE and cerebral infarction, had significantly greater number of aortic atheromatous plaques (11 [5–19] vs. 6 [3–11], $p<0.001$, 12 [4–20] vs. 6 [3–12], p=0.014, respectively). In a univariate analysis, the number of aortic atheromatous plaques and ruptured plaque were significant predictors of MACE (HR: 1.09 95% confidence interval 1.05–1.14, $p<0.001$) and (HR: 1.12, 95% confidence interval 1.02–1.23, p=0.02). In a multivariate logistic analysis, the number of aortic atheromatous plaques is one of the independent predictors of MACE (HR 1.05, 95% confidence interval 1.00–1.10, p=0.032).

Conclusion: The number of atheromatous plaques identified by NOGA has a significant relation to the onset of cerebral infarction, which suggest the atheromatous plaque were vulnerable and ruptured spontaneously, then cause the aortogenic cerebral infarction. The NOGA study would be useful for predicting the futured atheroembolic events.