

105. Impaired Peripheral Skin Microvascular Reactivity in Non-Obstructive Coronary Artery Disease Patients

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Body

Background: Coronary endothelial dysfunction is believed to be associated with non-obstructive coronary artery disease (NOCAD), however there was no concrete evidence on the involvement of peripheral endothelial dysfunction in NOCAD patients. Therefore, this study aimed to non-invasively evaluate the peripheral skin microvascular reactivity in NOCAD patients as compared to obstructive coronary artery disease (OCAD) and non-symptomatic subjects.

Methods: Stable angina patients who scheduled for coronary angiography were recruited and categorized in OCAD (stenosis $\geq 50\%$) or NOCAD (stenosis $< 50\%$) groups. The forearm skin microvascular reactivity was measured using the laser Doppler blood perfusion monitor and the process of post-occlusive skin reactive hyperaemia (PORH) by a single-blinded operator. The real-time blood perfusion was monitored and parameters specifically maximum change in perfusion after occlusion compared to baseline (PORHmax), time to reach peak perfusion after occlusion release (Tp) and PORH percent change, PORH% (PORHmax – mean baseline perfusion/mean baseline perfusion) were calculated. A non-symptomatic controls group was also recruited and underwent PORH procedure.

Results: Forty-two and forty patients were categorized as OCAD and NOCAD respectively, whereas thirty-nine subjects were categorized into the control group. There were significant differences in PORHmax ($P = 0.003$) and PORH% ($P = 0.002$) between the all groups. However, Tp values were not significantly different between the groups. Post-hoc analysis showed significant reductions in PORHmax between the OCAD and control groups ($P = 0.025$), while significant reduction of PORH% can be seen between the OCAD and control groups ($P = 0.002$), as well as NOCAD and control groups ($P = 0.016$). No significant difference was seen between the OCAD and NOCAD groups.

Conclusion: The impairment in peripheral skin microvascular reactivity in NOCAD patients was similar to OCAD patients, but was worse compared to non-symptomatic control subjects. Peripheral microvascular abnormalities is clearly manifested in NOCAD patients and may contribute to the symptoms experienced by this group of patients.

Table 1: Baseline characteristics and skin microvascular reactivity of study groups

Parameters	Control (n = 39)	NOCAD (n = 40)	OCAD (n = 42)	P value ^a
Age (years)	49.36 (8.00)	51.73 (8.80)	53.48 (7.90)	0.083
Body mass index, BMI (kg/m ²)	28.52 (4.39)	29.16 (4.98)	29.14 (4.12)	0.777
Systolic blood pressure (mmHg)	125.91 (12.53)	124.34 (18.46)	125.72 (15.71)	0.902
Diastolic blood pressure (mmHg)	82.40 (8.70) ^{b,e}	75.71 (10.33)	77.29 (7.89)	0.004
Gender, male/female, (n/n)	21/18	17/23	31/11	0.015
Family history, n (%)	4 (10.3)	17 (43.6)	20 (47.6)	0.001
Diabetes, n (%)	7 (17.9)	11 (28.2)	21 (50.0)	0.007
Hypertension, n (%)	11 (28.2)	26 (66.7)	29 (69.0)	< 0.001
Hyperlipidemia, n (%)	27 (69.2)	35 (89.7)	35 (83.3)	0.062
Smoking, n (%)	7 (17.9)	6 (15.4)	11 (26.2)	0.443
PORHmax (perfusion unit)	136.68 (39.76) ^b	121.31 (60.70)	109.03 (36.36)	0.033
PORH time to peak, T _p (s)	7.74 (3.41)	9.30 (3.45)	8.85 (4.76)	0.196
PORH% (%)	497.54 (215.53) ^{c,d}	384.13 (170.77)	358.93 (145.31)	0.002

Data presented as Mean (SD). ^aOne-way ANOVA. Post-hoc analysis with Tukey's HSD: ^bControl vs OCAD, $P < 0.05$. ^cControl vs NOCAD, $P < 0.05$. ^dControl vs OCAD, $P < 0.01$. ^eControl vs NOCAD, $P < 0.01$.

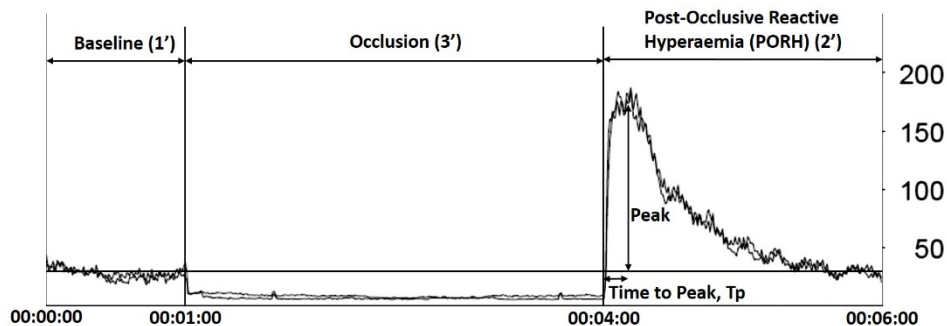


Figure 1: A sample of skin blood perfusion during post-occlusive reactive hyperaemia procedure

Clinical Implications: understand the fundamental knowledge on the roles of peripheral microvascular endothelial dysfunction in the occurrence of NOCAD, thus will offer a significant step in the prevention and effective treatment of NOCAD, reducing hospital admission, improving quality of life & increasing productivity in a significant number of people.