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Type D personality, but not Type A behavior pattern, is associated with coronary plaque vulnerability

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ABSTRACT

Personality traits are associated with major adverse coronary events (MACE) in patients with coronary artery disease (CAD). However, the link between personality traits and intravascular morphology in CAD patients is poorly understood. This study investigated the relationship between personality traits, specifically Type A behavior pattern and Type D personality, and plaque vulnerability. After adjustment for demographic and clinical factors, multivariable regression analysis showed no association between Type A and optical coherence tomography indices. However, Type D personality was independently associated with lipid plaque, thin cap fibroatheroma (TCFA), and fibrous cap thickness. More specifically, negative affectivity of Type D was related to lipid plaque, TCFA and fibrous cap thickness, and social inhibition was associated with plaque rupture. Our results show that Type D personality was associated with plaque vulnerability, independent of clinical factors. Measurement of negative affectivity and social inhibition will increase our understanding of the progressive phase of the plaque vulnerability, which can contribute to the early identification of high risk patients and reduce the incidence of MACE.

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Type A behavior pattern; Type D personality; plaque vulnerability; optical coherence tomography; major adverse coronary event

Introduction

Vulnerable plaque is associated with an increased incidence of major adverse coronary events (MACE) (Kubo et al., 2014). Previous studies revealed that traditional cardiac risk factors increased the likelihood of vulnerable plaques (Abtahian et al., 2014; Kato et al., 2012; Kimura et al., 2010). However, about 17% of patients with coronary artery disease (CAD) have MACE, despite having no traditional risk factors (Greenland et al., 2003). Hence, non-traditional risk factors may also promote the development of vulnerable plaques.

Personality traits contribute to CAD development and prognosis (Chida & Steptoe, 2009). Type A behavior pattern is characterized by a set of complex personality traits including

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ambitiousness, competitiveness, easily aroused hostility, and an exaggerated sense of time urgency (Friedman & Rosenman, 1959). Initial studies suggested a potential link between Type A and CAD; however, large clinical trials raised doubts about the influence of Type A on CAD (Kuper, Marmot, & Hemingway, 2002; Šmigelskas, Žemaitienė, Julkunen, & Kauhanen, 2015). In 1996, Denollet et al. (1996) introduced a new personality construct named Type D personality, which is defined by two dimensions: negative affectivity and social inhibition. Accumulating evidence shows that Type D is associated with an increased risk of adverse cardiac outcomes in CAD patients (Martens, Mols, Burg, & Denollet, 2010). However, two studies have not found any relationship between Type D personality and all-cause mortality in cardiac patients (Coyne et al., 2011; Grande et al., 2011). Thus, the importance of personality traits in CAD manifestations and development remains a debate. The objective of the present study was to examine the association between personality traits and the culprit plaque vulnerability evaluated by optical coherence tomography (OCT).

Methods

Study population and procedure

This study enrolled 198 consecutive patients with CAD who underwent OCT imaging between September 2011 and November 2014. Patients (aged 18–75 years) who each had at least one culprit coronary plaque were selected. Exclusion criteria included patients with poor OCT image quality, no clinical information, or no ability to participate in this study. Therefore, 152 subjects were included in this study.

Measures

The following constructs were measured.

1. *Type A behavior pattern questionnaire:* The questionnaire had 60 items, including 3 factors: time urgency, competitive hostility and lie components. Lie factors ≤ 7 proved questionnaire validity. Total score of time urgency and competitive hostility ≥ 28 was defined as Type A.
2. *Type D personality scale:* This scale consists of two seven-item subscales: negative affectivity and social inhibition. A score of 10 or more on both subscales denotes those with Type D personality.
3. *Demographics and clinical covariates:* Sociodemographic information included gender and age. Clinical variables were examined as follows: cardiac diagnoses, history of hypertension, diabetes mellitus, hyperlipidemia, smoking, and drinking. Laboratory measurements included total cholesterol, triglycerides, low density lipoprotein-cholesterol (LDL-C), high density lipoprotein-cholesterol (HDL-C), fasting blood glucose, and C-reactive protein (CRP).
4. *OCT data analysis:* A lipid-rich plaque was defined as the presence of lipids in ≥ 90° of the vessel wall circumference (Yabushita et al., 2002). Fibrous cap thickness in the lipid-rich plaque was measured in triplicate at the thinnest part and the average was calculated. The thin cap fibroatheroma (TCFA) was defined as a lipid-rich plaque, with the fibrous cap thickness ≤ 65 μm and necrotic lipid pools present in ≥ 90° (Prati et al., 2010). This index was used in our study to estimate

plaque vulnerability. Macrophage infiltration was identified by signal intensity of the plaque with heterogeneous backward shadows (MacNeill et al., 2004). A plaque rupture was characterized as a discontinuity of the fibrous cap and a clear cavity formation in the plaque (Jang et al., 2005). A thrombus was defined as an irregular mass (diameter ≥ 250 mm) attached to the arterial lumen (Kitabata et al., 2010).

Procedure

Patients were asked to fill out questionnaires the next day after OCT when they were in a stable condition. Clinical data were acquired from our health recording system. This study was approved by the Ethics Committee of the Second Affiliated Hospital of Harbin Medical University. Informed consent was signed by all participants and the acquired data were kept confidential.

Statistical analysis

For analysis of patient characteristics, categorical data were presented as a percentage and compared using the chi-square test. Continuous measurements were presented as mean ± standard deviation (SD) for normal distribution variables and median and interquartile range (IQR) for skewed variables. The continuous measurements in the vulnerable and non-vulnerable plaque groups were compared using the Student's *t* test for normally distributed variables or Mann–Whitney test for skewed variables. All covariates were selected a priori based on the literature in the multivariate regression model. A probability value of $p < .05$ was considered statistically significant.

Results

Baseline characteristics

We first evaluated the possible differences in the baseline characteristics between the vulnerable and non-vulnerable plaque groups. There were no significant differences in these characteristics between the two groups except for acute coronary syndrome (ACS), hyperlipidemia and smoking (Table 1), indicating that these three factors play an important role in the development of vulnerable plaques.

Contributing factors to coronary plaque vulnerability

We next asked if there was any association between personality traits and plaque vulnerability using the multivariate regression model with the categorized personality construct. Type A behavior pattern had no significant effect on plaque vulnerability, but Type D personality was the independent contributor to lipid plaque, TCFA, rupture, and fibrous cap thickness after adjustment for other relevant covariates (Table 2).

We next examined whether different components of Type A behavior pattern and Type D personality – time urgency, competitive hostility, negative affectivity, and social inhibition (*z* scores) – had different correlations with plaque vulnerability. The multivariate analysis confirmed that negative affectivity was related to lipid plaque, TCFA and fibrous

Table 1. Demographic and baseline clinical characteristics.

Variable	Total sample (n = 152)	Vulnerable plaque (n = 83)	Non-vulnerable plaque (n = 69)	Test values	p-Value
Age, *M* (SD), yr	57.9 (10.51)	57.5 (10.63)	58.3 (10.45)	t = .43	.67
Male, *n* (%)	114 (75.0)	64 (77.1)	50 (72.5)	x^2 = .43	.32
BMI, *M* (SD), kg/m^2	24.4 (2.79)	24.7 (3.03)	234.0 (2.59)	t = −1.72	.09
ACS, *n* (%)	123 (81.0)	73 (88.0)	50 (72.5)	x^2 = 5.85	.013
Hyperlipidemia, *n* (%)	66 (44.0)	44 (54.3)	22 (31.9)	x^2 = 7.61	.005
Diabetes, *n* (%)	43 (28.9)	25 (31.3)	18 (26.1)	x^2 = .48	.31
Hypertension, *n* (%)	82 (54.3)	49 (59.8)	33 (47.8)	x^2 = 2.15	.10
Smoking history, *n* (%)	74 (49.3)	49 (60.5)	25 (36.2)	x^2 = 8.78	.002
Drinking history, *n* (%)	80 (52.6)	48 (57.8)	32 (46.4)	x^2 = 1.20	.11
Laboratory findings					
Total cholesterol, *M* (SD), mmol/L	4.6 (1.21)	4.7 (1.28)	4.4 (1.18)	t = −1.19	.24
Triglycerides, *M* (IQR), mmol/L	1.7 (1.35)	1.9 (1.39)	1.5 (1.28)	z = −1.65	.10
LDL-C, *M* (IQR), mmol/L	2.1 (.94)	2.1 (.90)	2.0 (1.01)	z = −.40	.69
HDL-C, *M* (IQR), mmol/L	1.3 (.48)	1.2 (.64)	1.3 (.42)	z = −.47	.64
Blood glucose, *M* (IQR), mmol/L	5.7 (1.32)	5.7 (1.42)	5.7 (1.21)	z = −.10	.92
C-reactive protein, *M* (IQR), mmol/L	1.6 (2.01)	1.7 (2.11)	1.4 (1.84)	z = −.31	.76

Notes: *M* (SD) = mean (stand deviation); *M* (IQR) = median (interquartile range); TCFA = thin cap fibroatheroma; BMI = body mass index; ACS = acute coronary syndrome; LDL-C = low density lipoprotein-cholesterol; HDL-C = high density lipoprotein-cholesterol.

Table 2. Multivariate regression models for coronary plaque vulnerability with personality traits categorized.

	Lipid plaque (n = 152)			TCFA (n = 152)			Rupture (n = 152)			Fibrous Cap Thickness (n = 152)		
Variable	OR	95% CI	p	OR	95% CI	p	OR	95% CI	p	β	SE	p
Type A	.25	.05–1.13	.07	1.04	.50–2.17	.91	.54	.24–1.25	.15	.003	.007	.68
Type D	4.56	1.84–6.63	.01	3.15	1.48–6.71	.003	2.52	1.18–5.39	.02	−.031	.008	.00
Covariates												
Age	1.05	.98–1.13	.19	1.00	.96–1.04	.99	1.01	.97–1.05	.76	.000	.000	.25
Female	.70	.15–3.26	.65	.78	.31–1.92	.59	.34	.13–1.00	.05	.002	.008	.86
Smoking	3.73	1.14–5.60	.03	2.56	1.21–5.41	.01	2.33	1.07–5.07	.03	−.023	.007	.003
HDL-C	.26	.04–1.86	.18	.38	.08–1.72	.21	.30	.06–1.59	.16	−.002	.006	.77
LDL-C	.62	.29–1.32	.21	2.37	1.02–5.78	.03	2.86	.67–5.15	.15	−.002	.003	.51
Diabetes	2.24	.41–3.32	.35	1.17	.51–2.70	.72	.52	.21–1.32	.17	.000	.008	.95
Log C-reactive protein	.35	.07–1.78	.21	.69	.25–1.79	.42	.59	.21–1.68	.33	.012	.009	.18
Statins	.60	.08–4.53	.60	.96	.31–2.95	.95	1.54	.45–5.29	.50	−.011	.011	.33

Notes: TCFA = thin cap fibroatheroma; HDL-C = high density lipoprotein-cholesterol; LDL-C = low density lipoprotein-cholesterol; Log C-reactive protein = logarithm 10 of C-reactive protein; OR = odds ratio; CI = confidence interval; β = beta; SE = standard error.

cap thickness, and that social inhibition was associated with plaque rupture (Table 3). When the interaction (z scores) in the dimension of personality was included in the model, time urgency/competitive hostility and negative affectivity/social inhibition showed no significance (Supplementary Table). Taken together, Type D personality, particularly negative affectivity and social inhibition, are closely related to development and rupture of lipid plaque, respectively.

Discussion

In our study, we researched the relationship between personality and plaque vulnerability from the microstructure view. The findings indicate that Type A does not correlate

Table 3. Multivariate regression models for coronary plaque vulnerability with TH × CH and NA × SI statistical interaction.

	Lipid plaque (n = 152)			TCFA (n = 152)			Rupture (n = 152)			Fibrous cap thickness (n = 152)		
Variable	OR	95% CI	p	OR	95% CI	p	OR	95% CI	p	β	SE	p
TH (z scores)	.50	.17–1.47	.21	.83	.45–1.52	.55	1.38	.72–2.63	.33	−.001	.006	.85
CH (z scores)	2.00	.54–7.44	.30	1.06	.56–1.98	.86	.65	.34–1.26	.21	.005	.006	.39
TH × CH (z scores)	1.14	.46–2.79	.78	.80	.55–1.15	.23	.84	.58–1.23	.38	.005	.003	.12
NA (z scores)	3.93	1.64–9.43	.002	1.77	1.05–2.99	.03	1.66	.99–2.79	.06	−.011	.005	.02
SI (z scores)	1.81	.52–6.37	.36	1.19	.76–1.87	.45	1.73	1.09–2.96	.05	−.007	.004	.13
NA × SI (z scores)	.49	.20–1.21	.12	1.38	.91–2.10	.13	.95	.62–1.47	.83	.003	.003	.30
Covariates												
Age	1.04	.96–1.12	.35	1.00	.99–1.04	.91	1.00	.95–1.05	.99	.001	.001	.44
Female	1.39	.21–9.05	.73	.90	.36–2.24	.82	.36	.13–1.05	.06	.001	.009	.94
Smoking	2.87	1.05–4.21	.04	2.65	1.22–5.78	.01	1.67	.72–3.86	.23	−.023	.008	.003
HDL-C	.07	.01–.98	.05	.29	.06–1.37	.12	.16	.03–.98	.05	.017	.015	.26
LDL-C	1.13	.47–2.67	.79	3.03	1.51–7.19	.02	2.13	.78–4.00	.10	−.006	.004	.10
Diabetes	1.30	.22–7.76	.78	1.01	.41–4.51	.59	.50	.19–1.13	.16	−.002	.008	.71
Log C-reactive protein	.13	.01–1.36	.09	.67	.24–1.85	.44	.30	.10–1.03	.06	.016	.010	.11
Statins	1.75	.14–2.29	.39	1.38	.42–2.46	.99	1.08	.54–2.05	.29	−.019	.012	.12

Notes: TCFA = thin cap fibroatheroma; TH = time hurry; CH = competition hostility; NA = negative affectivity; SI = social inhibition; HDL-C = high density lipoprotein-cholesterol; LDL-C = low density lipoprotein-cholesterol; Log C-reactive protein = logarithm 10 of C-reactive protein; OR = odds ratio; CI = confidence interval; β = beta; SE = standard error.

with vulnerable plaque, which was in line with other studies investigating the association between personality and subclinical atherosclerosis (Abtahian et al., 2014). The present results highlight the need to clarify and refine the relationship between Type A behavior pattern and CAD.

We also demonstrated that vulnerable plaque was significantly more frequent in Type D patients compared to Type A patients. Furthermore, multivariate analysis found that Type D personality was a powerful predictor of plaque vulnerability (lipid plaque, TCFA, plaque rupture, and fibrous cap thickness), even after adjustment for other risk factors. Analysis of the main effects of Type D revealed that negative affectivity was an independent influential factor of lipid plaque, TCFA and fibrous cap thickness, and that social inhibition was an influential factor of plaque rupture. Vulnerable plaque development is a dynamic process, and lipid plaques are prone to increase in the presence of TCFA. TCFA is known as the precursor of plaque rupture, which accounts for the majority of MACE (Sakakura et al., 2013). Hence, our findings demonstrate that two dimensions affected the development of plaque vulnerability in different stages, and we speculate that the increased plaque vulnerability may partly explain the higher prevalence of MACE in Type D personality patients.

Limitations

First, this study was conducted with a relatively small sample size and our study population consisted entirely of patients with culprit lesions. Thus, our study results do not represent all CAD patients and all diseased coronary arteries. Second, although OCT can depict the lipid component and fibrous cap clearly, it was unable to visualize the entire plaque and quantify all OCT indices because of limited penetration depth.

Conclusions

We found that Type A behavior pattern does not affect plaque vulnerability. However, there is a relationship between Type D personality and vulnerable plaque characteristics. We also demonstrate that negative affectivity and social inhibition affect the progression of plaque vulnerability in different stages. Therefore, we believe that measurement of negative affectivity and social inhibition will provide important information about the progressive phase of plaque vulnerability, which can contribute to early identification of high risk patients and reduce the incidence of MACE.

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Disclosure statement

None of the authors has any conflict of interest.

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