J Atheroscler Thromb, 2017; 24: 910-911.
http://doi.org/10.5551/jat.ED075

Editorial

Histopathology of Asymptomatic Iliac Atherosclerosis: From Autopsy to Practice

Kensaku Nishihira and Satoshi Yasuda

Department of Cardiovascular Medicine, National Cerebral and Cardiovascular Center, Suita, Japan

See article vol. 24: 921-927

Lower extremity atherosclerotic peripheral arterial disease (PAD) developed in more than 200 million adults worldwide, and it is associated with morbidity and mortality because of the systemic nature of atherosclerosis[1, 2]. The prevalence of PAD is age-related, and more than 30% increase in deaths and disability have been reported from 2005 to 2015[1-4]. Additionally, many clinical studies have shown the significant overlap of atherosclerosis involving multiple arterial territories in patients with coronary artery disease, cerebrovascular disease, or PAD. More than 50% of patients with PAD have coronary artery disease and/or cerebrovascular disease[1, 3, 4]. The presence of PAD is, therefore, associated with worse clinical outcomes. It should also be noted that asymptomatic PAD is several times more common than symptomatic PAD[4]. Noninvasive measures of ankle-brachial index, brachial-ankle pulse wave velocity, and ultrasound could detect systemic atherosclerotic burden and predict clinical outcomes in patients with symptomatic and asymptomatic PAD[1, 4-6]. However, little is known about the histopathology of PAD in the asymptomatic period and about its relationship with systemic atherosclerosis.

In the present study, Nakamura *et al* pathologically investigated atherosclerotic plaques of the common iliac, common carotid, coronary, and renal arteries using 121 autopsy cases without symptoms of PAD, and they evaluated the relationship between atherosclerotic severity of the iliac artery and that of other arteries and cardiovascular disease (CVD) events[7]. They found that advanced atherosclerotic plaques were observed in 72% of the common iliac arteries in asymptomatic subjects. Intraplaque hemorrhage and plaque disruption, features of vulnerable atherosclerotic plaque, were also detected in 42% and 24% subjects, respectively. These advanced lesions in the iliac arteries were associated with age, sex, hypertension, diabetes mellitus, and smoking habit. Additionally, they also showed that the presence of asymptomatic iliac plaques was closely associated with any CVD (odds ratio [OR], 95% confidence interval [CI]; 6.2, 2.2−22.3), myocardial infarction (6.4, 1.2−18.6), stroke (8.7, 1.7−16.4), and renal failure or hemodialysis (5.8, 1.1−10.8). Importantly, although subjects having symptomatic PAD are excluded from the present study, the prevalence of advanced iliac plaques seems to be relatively high compared with the results of previous studies that conducted evaluations by noninvasive measures[4, 6].

Some study limitations and perspective should be stated. This study might have a selection bias as the number of subjects is relatively small. It would have been interesting if the authors had evaluated the relationship between asymptomatic iliac plaques and aortic aneurysm and had performed analyses of inflammatory cells and mediators in the systemic atherosclerotic plaques as inflammation plays a major role in the pathogenesis of atherosclerosis and plaque instability[8].

In conclusion, the present study suggests that detection of iliac atherosclerotic plaques in its subclinical stage may help in predicting systemic atherosclerotic disease.

Disclosures

None.

Address for correspondence: Kensaku Nishihira, Department of Cardiovascular Medicine, National Cerebral and Cardiovascular Center, 5-7-1 Fujishiro-dai, Suita, Osaka 565-8565, Japan
E-mail: nishihira@ncvc.go.jp
Received: April 4, 2017
Accepted for publication: April 6, 2017

References

1) Hamburg NM, Creager MA: Pathophysiology of intermittent claudication in peripheral artery disease. Circ J,

Copyright©2017 Japan Atherosclerosis Society
This article is distributed under the terms of the latest version of CC BY-NC-SA defined by the Creative Commons Attribution License.

2017; 81: 281-289
2) Steg PG, Bhatt DL, Wilson PW, D'Agostino R Sr, Ohman EM, Röther J, Liau CS, Hirsch AT, Mas JL, Ikeda Y, Pencina MJ, Goto S; REACH Registry Investigators: One-year cardiovascular event rates in outpatients with atherothrombosis. JAMA. 2007; 297: 1197-1206
3) Bhatt DL, Steg PG, Ohman EM, Hirsch AT, Ikeda Y, Mas JL, Goto S, Liau CS, Richard AJ, Röther J, Wilson PW; REACH Registry Investigators: International prevalence, recognition, and treatment of cardiovascular risk factors in outpatients with atherothrombosis. JAMA. 2006; 295: 180-189
4) Criqui MH, Aboyans V; Epidemiology of peripheral artery disease. Circ Res. 2015; 116: 1509-1526
5) Joo HJ, Cho SA, Cho JY, Lee S, Park JH, Hwang SH, Hong SJ, Yu CW, Lim DS; Brachial-ankle pulse wave velocity is associated with composite carotid and coronary atherosclerosis in a middle-aged asymptomatic population. J Atheroscler Thromb. 2016; 23: 1033-1046
6) Fernandez-Friera L, Penalvo JL, Fernandez-Ortiz A, Ibanez B, Lopez-Melgar B, Laclaustra M, Oliva B, Mocoroa A, Mendiguren J, Martinez de Vega V, Garcia L, Molina J, Sanchez-Gonzalez J, Guzman G, Alonso-Farto JC, Guallar E, Civeira F, Sillesen H, Pocock S, Ordovas JM, Sanz G, Jimenez-Borreguero LJ, and Fuster V; Prevalence, vascular distribution, and multiterritorial extent of subclinical atherosclerosis in a middle-aged cohort: The PESA (Progression of Early Subclinical Atherosclerosis) Study. Circulation. 2015; 131: 2104-2113
7) Nakamura E, Sato Y, Iwakiri T, Yamashita A, Moriguchi-Goto S, Maekawa K, Gi T, Asada Y; Asymptomatic plaques of lower peripheral arteries and their association with cardiovascular disease: an autopsy study. J Atheroscler Thromb. 2017; 24: 921-927
8) Mizuno Y, Jacob RF, Mason RP; Inflammation and the development of atherosclerosis. J Atheroscler Thromb. 2011; 18: 351-358