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Editorial

The Effect of n-3 PUFA on the Development of Abdominal Aortic Aneurysm

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Abdominal aortic aneurysm (AAA) is characterized by a gradual dilation of abdominal aorta, which is typically asymptomatic until rupture. There is currently no drug for preventing AAA development. Several studies have recently reported the effect of n-3 polyunsaturated fatty acid (PUFA), particularly eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA), on AAA development, which is formed in AAA animal models. EPA treatment reportedly attenuates $CaCl_2$-induced AAA formation, elastic lamina destruction, and aortic calcification[1]. Yoshihara *et al*. reported that EPA and DHA suppress AAA development induced by angiotensin Ⅱ infusion in apolipoprotein E-deficient mice[2]. Using the same experimental mouse model, Russell *et al*. reported that dietary supplementations of n-3 PUFA attenuate matrix metalloproteinase 9 immunoreactivity[3]. They also reported a trend of a delay in AAA-related death in mice that were fed n-3 PUFA diet[4]. Using a newly developed AAA animal model[5], Kugo *et al*. reported that EPA-rich fish oil prevented AAA development[6] induced by hypoperfusion of the vascular wall. These studies suggested that n-3 PUFA has preventive effects on AAA development in different AAA animal models. The mechanism of action of n-3 PUFA is generally considered to be associated with antiinflammatory and antioxidant activities[7]. Recent studies show that abnormal appearance of adipocyte in vascular wall may induce AAA rupture[8, 9]. The risk for AAA rupture decreased following fish oil administration, with the decreased number and size of ectopic adipocytes in the vascular wall[8]. The hypolipidemic effect of n-3 PUFA might be involved in the preventive effect on AAA development or rupture.

In contrast to the increasing evidences from animal studies, the effect of n-3 PUFA on human AAA remains unknown. In the current issue of *Journal of Atherosclerosis and Thrombosis*, Aikawa *et al*. analyzed 67 patients who were admitted for elective surgical repair of AAA. They estimated the correlation of serum EPA, DHA, and EPA/arachidonic acid (AA) ratio with the size and growth rate of AAA[10]. Their study indicated that low serum EPA levels (low EPA/AA ratio) were significantly associated with the size and growth rate of AAA diameter ($r=-0.43$ and $r=-0.33$, respectively). Interestingly, serum DHA levels did not correlate with AAA formation in their study group. It is of interest to clarify the difference between the effects of each n-3 PUFA on AAA development.

Their cross-sectional study did not show the causal association between n-3 PUFA and AAA development. However, this first study in a clinical setting provided an important finding to estimate the effect of n-3 PUFA on human AAA development. Further investigation should be performed to clarify the association between n-3 PUFA and human AAA development.

Conflicts of Interest

None.

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