Letter to the Editor — Editöre Mektup

Another therapeutic target for atherosclerosis

Dear Editor,

We congratulate Dr. Kılıçkap on an intriguing review emphasizing the blurred relations between traditional infection/inflammation and atherosclerosis.[1] As already mentioned by the author, according to Bradford Hill's criteria on the causal link between events, the link in this case, between infection and atherosclerosis, remains obscure. Dr. Kılıçkap concluded that although convincing evidence about this link is lacking, the infection hypothesis still merits more evaluation.

I believe a new microbiological mechanistic link is presently being forged. Gut microbiota might have a role in development of atherosclerosis. Two high quality papers published recently have served to reignite discussion. Tang WHW et al. showed that a supposed proatherosclerotic metabolite, namely trimethylamine-N-oxide (TMAO), an intestinal microbiota-dependent metabolite of choline, has an indisputable association with cardiovascular events.[2] They claimed that high plasma levels of TMAO provoke increased incidences of major cardiovascular endpoints.

A report also appeared in Nature Medicine.[3] Koeth RA et al. named TMAO as an independent predictor of clinical cardiovascular risk.[3] Intestinal microbiota metabolism of choline and phosphatidylcholine produces trimethylamine, which is further metabolized into a strong proatherogenic substrate, namely TMAO. Koeth's paper showed that red meat contains high quantities of these kind of substances, and that production of high quantities of TMAO by gut bacteria accelerates atherosclerosis in mice.

Antibiotic-assisted suppression of bacterial activity producing TMAO might be a therapeutic accomplishment against atherosclerosis in the future. Even newer studies show that bacterial activity in the gut may affect the mind via some actions of byproducts of the bacteria.[4]

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