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Letter to the Editor:

Effects of Omega-3 Fatty Acids on Erectile Dysfunction in a Rat Model of Atherosclerosis-induced Chronic Pelvic Ischemia

Dear Editor,

I have read the article "Effects of Omega-3 Fatty Acids on Erectile Dysfunction in a Rat Model of Atherosclerosis-induced Chronic Pelvic Ischemia" written by Shim et al. (1), which was an interesting study investigating the protective effect of of omega-3 fatty acids on erectile dysfunction in a rat model of atherosclerosis-induced chronic pelvic ıschemia. In this study, they showed that omega-3 fatty acids improved intracavernosal pressure and had a beneficial role against pathophysiological consequences such as fibrosis or hypoxic damage on chronic pelvic ischemia rat model, which represents a structural erectile dysfunction model. Yet, this article is open to critism from several aspects.

Ischemic and hypercholesterolemic conditions resulted in erectile dysfunction as stated in this experiment. In ischaemic conditions reactive oxygene species, nuclear factor kappa b and inducible nitric oxide synthase pathway is involved (2,3). It would be better if this pathway had been studied. Transforming growth factor β (TGF-β) expression in cavernosal tissues had been evaluated, but TGF-β indirectly informed about collagen expression and fibrosis. Together with TGF-β1, it would be better if collagen, especially type-1, and fibrosis were observed (4). Most of the tissue responses during ischaemic conditions occur through the induction of the transcription factor hypoxia-inducible factor-1 (HIF-1) which regulates many processes needed for tissue repair during ischemia in the damaged tissue. Vascular endothelial growth factor (VEGF) is one of the transcriptionally regulated soluble growth factor by HIF-1 (5). It would be better if the effect of omega-3 fatty acids on VEGF expression were studied in penile tissues. By using corpus cavernosum tissues to evaluate the contractile responses to phenylephrine and relaxation responses to carbachol is a good method in organ bath, which enables to investigate isometric tension changes in response to various bioactive agents and electrical field stimulation in in vitro models. This method gives us functional information about penile function and nitric oxide (NO)/cyclic guanosine monophosphate pathway (5,6). Intracavernosal presssure measurement following cavernosal nerve stimulation without study of organ bath is another flaw of this experiment. It is well known that omega-3 fatty acids are strong antioxidant and used as a ROS scavenging agents in literature (7). In the current study, antioxidant efficacy of omega-3 fatty acids had not been evaluated. It reported that atherosclerosis-associated endothelial dysfunction reduced nitric oxide bioavailability and caused nitric oxide synthase (NOS)-uncoupling (8,9). These are main results of increased ROS and caused erectile dysfunction. Certain antioxidants prevent NOS-uncoupling and reverse nitric oxide bioavailability (10). It would be better when the preventive effect of omega-3 fatty acids was studied against reduced NO bioavailability and NOS-uncoupling.

Finally, preventive effects of omega-3 fatty acids in the ischemic penile tissues could be evaluated in line with my recommendations and used as a supplementary agent in aged patients with suspected pelvic ischemia.

DISCLOSURE

The author has no potential conflicts of interest to disclose.

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