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 omega-3 fatty acids on erectile dysfunction in a rat model of atherosclerosis-induced chronic pelvic ischemia. 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 criticism from several aspects.

Ischemic and hypercholesterolemic conditions resulted in erectile dysfunction as stated in this experiment. In ischemic conditions reactive oxygen 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 ischemic 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 vitro models. This method gives us functional information about penile function and nitric oxide (NO)/cyclic guanosine monophosphate pathway (5,6). Intracavernosal pressure 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.

ORCID

Emin Özbek https://orcid.org/0000-0002-8699-2383

REFERENCES

1. Shim JS, Kim DH, Bae JH, Moon DG. Effects of omega-3 fatty acids on erectile dysfunction in a rat model of atherosclerosis-induced chronic pelvic ischemia. J Korean Med Sci 2016; 31: 585-9.
2. Csomka C, Sárközy M, Pipicz M, Dux I, Csont T. Modulation of hypercholesterolemia-induced oxidative/nitrative stress in the heart. Oxid Med Cell Longev 2016; 2016: 3863726.
3. Xia Z, Li H, Irwin MG. Myocardial ischaemia reperfusion injury: the challenge of translating ischaemic and anaesthetic protection from animal models to humans, Br J Anaesth 2016; 117 Suppl 2: ii44-62.
4. Çevik Ö, Çadırcı S, Şener TE, Tinay I, Akbal C, Tavakçu HH, Çetinel S, Kran D, Şener G. Quercetin treatment against ischemia/reperfusion injury in rat corpus cavernosum tissue: a role on apoptosis and oxidative stress. Free Radic Res 2013; 47: 683-91.
5. Lokmic Z, Musyoka J, Hewitson TD, Darby IA. Hyposia and hypoxia signaling in tissue repair and fibrosis. Int Rev Cell Mol Biol 2012; 296: 139-85.
6. Toksoz S, Erdem SR, Peskircioğlu CL, Keskin U. The effect of long-term oral tadalafil treatment on corpus cavernosum function in an experimental spinal cord transection rat model. Spinal Cord 2013; 51: 663-7.
7. Bas O, Songur A, Sahin O, Mollaoglu H, Ozen OA, Yaman M, Eser O, Fi-
6. dan H, Yagmurca M. The protective effect of fish n-3 fatty acids on cerebral ischemia in rat hippocampus. Neurochem Int 2007; 50:548-54.
8. Schächinger V, Zeiher AM. Atherosclerosis-associated endothelial dysfunction. Z Kardiol 2000; 89 Suppl 9: IX/70-4.
9. Charles S, Raj V, Arokiaraj J, Mala K. Caveolin1/protein arginine methyltransferase1/sirtuin1 axis as a potential target against endothelial dysfunction. Pharmacol Res 2017; 119: 1-11.
10. Xia N, Förstermann U, Li H. Resveratrol and endothelial nitric oxide. Molecules 2014; 19: 16102-21.

Emin Özbek, MD
Department of Urology, Health Sciences University, Istanbul Research & Education Hospital, Sisli, Fatih- Istanbul 34384, Turkey
E-mail: ozbekemin@hotmail.com
Received: 9 February 2017 / Accepted: 24 October 2017