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Scientific Contributions

Low-Dose Estrogen Supplementation Improves Vascular Function in Hypogonadal Men

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Abstract

It is widely accepted that in women, estrogens provide protection against the development of cardiovascular disease. However, the cardiovascular role of estrogens in men remains uncertain, despite preliminary evidence that endogenous estrogens produced by aromatization of androgenic precursors are of physiological importance. Hypogonadal men have very low levels of circulating estrogen. We studied the responsiveness of forearm resistance arteries to vasoconstrictor and vasodilator agents in 12 men (mean±SEM age, 68.7±2.6 years) rendered hypogonadal as a result of treatment for prostatic cancer, before and after 8 weeks of estrogen supplementation (estradiol valerate 1 mg daily; n=7) or placebo (n=5). Forearm blood flow was measured by venous occlusion plethysmography, and vasoactive agents were infused through a brachial artery cannula in doses that did not affect blood pressure or heart rate. Estrogen supplementation was well tolerated, with no adverse effects. After estrogen treatment, mean estradiol levels increased from <30 to 308±65 pmol/L, and both systolic and diastolic blood pressures were reduced. HDL cholesterol levels increased significantly, and vasoconstrictor responses to the NO synthase inhibitor N^G-monomethyl-L-arginine (1, 2, 4 μmol/min) were enhanced. Vasoconstrictor responses to angiotensin II (8, 16, 32 ng/min) were markedly attenuated by estrogen treatment, as were vasoconstrictor responses to norepinephrine (25, 50, 100 ng/min). Estrogen did not alter the vasodilator responses to acetylcholine (9.25, 18.5, 37 μg/min) or to the endothelium-independent agent sodium nitroprusside (1.6 μg/min). Responses to all vasoactive agents were unchanged after administration of placebo. We conclude that low-dose estrogen supplementation in hypogonadal men is well tolerated, lowers blood pressure, and may affect vascular reactivity in a manner that is potentially beneficial, through several mechanisms, including enhancement of basal NO release and attenuation of vasoconstrictor responses to angiotensin II and norepinephrine. These findings suggest the need to consider a possible clinical role for estrogenic compounds in cardiovascular risk reduction in some groups of men.

Key Words:
estrogen
men