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TYPE ABSTRACT HERE

ENDOTHELIUM-DEPENDENT RELAXATION IS PRESERVED BY β-CAROTENE IN AN ATHEROSCLEROTIC RABBIT MODEL OF PLAQUE DISRUPTION AND THROMBOSIS

Elie E. Hage-Korban, M.D. (Associate), Hongbao Ma, Ph.D., Alejandro R. Prieto, M.D., Xiaotan Qiao, Ph.D., Firas Akhras, M.D., Ruiping Huang, Ph.D., George S. Abela, M.D. (Member). Michigan State University, East Lansing, MI.

Background: Acute cardiovascular events have been attributed to plaque disruption and thrombosis in early morning hours by triggering of atheroslcerotic plaque related to vasoconstriction and a prothrombotic state.

Purpose: Since β-carotene (β-C) has been shown to preserve endothelium-dependent relaxation, we evaluated the effect of β-C on vasorelaxation and thrombosis in an atherosclerotic model of plaque disruption and thrombosis.

Methods: Twenty male NZW rabbits, 18 atherosclerotic induced by a high cholesterol diet and balloon-induced endothelial injury and 2 normal rabbits were used. Plaque triggering was induced by Russell's viper venom and histamine in 16 rabbits. β-C (30 mg/kg, i.v.) was given 5 days prior to triggering in 8/16 rabbits. Two atherosclerotic rabbits and 2 normal control rabbits were not triggered. After sacrifice, carotid and femoral arteries were removed and perfused in an organ chamber. Endothelium-dependent arterial relaxation was tested by norepinephrine (NE) ($1x10^{-6}$ M) preconstriction, followed by acetycholine (Ach) ($1x10^{-5}$ M) and nitrotroprusside (SN) (10^{-5} M). Platelet-rich aortic thrombus area was measured by planimetry.

Results: In triggered rabbits, arterial diameters were significantly greater with Ach in β-C vs. non-β-C treated rabbits [(Ach-NE)/NE×100=10±10 vs. 14±10; $p<0.05$]. With SN, arterial diameters were not significantly different between β-C and non-β-C treated rabbits [(SN-NE)/NE×100=17±11 vs. 12±12]. Both non-triggered atherosclerotic [(Ach-NE)/NE×100=15±15, (SN-NE)/NE ×100=20±15] and normal control rabbits [(Ach-NE)/NE×100=35±30, (SN-NE)/NE × 100=39±26] had significantly greater responses than triggered rabbits with Ach and SN ($p<0.01$). However, thrombus area was not significantly less with β-C compared with non-β-C triggered rabbits (50±26 vs. 79±39 mm^2).

Conclusions: These data demonstrate that β-C protects endothelial-dependant relaxation in atherosclerotic arteries during plaque triggering. However, plaque disruption and area of arterial thrombosis were not altered. Future studies using both an antioxidant and an antiplatelet agent may counteract the vasoconstriction and prothrombotic state in plaque disruption and thrombosis.

DEADLINE FOR SUBMISSION: POSTMARKED BY OCTOBER 1, 1998

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