



*The Medical **Bulletin***

Drug Corner

Increase in plasma ADMA (asymmetric dimethyl arginine) induced by PPI may potentially explain the association of PPI's with cardiovascular events in patients with Acute coronary syndrome. PPI's inhibit an enzyme DDAH (Dimethylarginine dimethylamino hydrolase, present in every cell) which degrades ADMA, the endogenous inhibitor of NO Synthetase. Thus chronic use of PPI may increase plasma ADMA level, inhibiting the generation of vascular NO, resulting in increased risk of MACE, because NO inhibit platelet aggregation and adherence to the veseel wall.

A PPI induced reduction in NO levels increases the risk of coronary thrombosis. In addition, chronic PPI use impairs lysosomal enzyme activity resulting in cellular proteostatis(protein aggregation)....induces oxidative stress...impairs NO synthetase pathway. Chronic PPI use causes hypomagnesemia, hypocalcemia and vitamin B12 deficiency.

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