Exp Clin Endocrinol Diabetes. 2001;109(6):330-6.

Efficacy of benfotiamine versus thiamine on function and glycation products of peripheral nerves in diabetic rats.

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Abstract

In rats with streptozotocin (STZ) induced diabetes the effect of (watersoluble) thiamine nitrate and of (lipidsoluble) benfotiamine on peripheral nerve function (motor nerve conduction velocity) as well as on the formation of advanced glycation end-products in peripheral nerve tissue was studied. In one group of animals drug administration was started immediately after diabetes induction (prevention study) and in another group two months after diabetes induction (treatment study). Motor nerve conduction velocity (NCV) dropped by 10.5% in diabetic animals, carboxymethyl-lysine (CML) rose to a 3.5fold concentration, deoxyglucosone (3DG)-type AGE formation was increased 5.1fold compared with controls. After three months preventive administration of both vitamin B(1) preparations NCV had increased substantially compared with results in diabetic controls. It was nearly normal after six months with benfotiamine, while the administration of thiamine nitrate resulted in no further amelioration. NCV was nearly normalized after six months of benfotiamine application but not with thiamine. Furthermore, benfotiamine induced a major inhibition of neural imidazole-type AGE formation and completely prevented diabetes induced glycoxidation products (CML). Treatment with thiamine did not significantly affect AGE or cmL levels. Unlike treatment with water-soluble thiamine nitrate timely administration of liposoluble prodrug benfotiamine was effective in the prevention of functional damage and of AGE and cmL formation in nerves of diabetic rats.

PMID: 11571671 [PubMed - indexed for MEDLINE]