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Diabetic neuropathic pain development in type 2 diabetic mouse model and the prophylactic and therapeutic effects of coenzyme Q10

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Highlights

- Type 2 diabetes-induced neuropathic pain was investigated in NZO/HILtJ mice.
- Diabetic pain was indicated by mechanical, thermal and chemical-induced hyperalgesia.
- Treatment of CoQ10 decreased pain-related hypersensitivity.
- Lipid peroxidation increased, CoQ10 content in neurons decreased in diabetic mice.
- Treatment of CoQ10 decreased lipid peroxidation and complemented CoQ10 in neurons.

Abstract

The early onset of type 2 diabetes mellitus (DM), driven by increasing obesity, is associated with peripheral neuropathy. Here, we characterize diabetic neuropathic pain in New Zealand obese diabetic mice (NZO/HILtJ) as a polygenic model of obesity with type 2 diabetes and investigate the role of coenzyme Q10 (CoQ10) in the prevention and treatment of diabetic neuropathic pain. Since the overexpression of mitogen-activated protein kinase (MAPK), nuclear factor-κB proteins (NF-κB), toll-like receptor 4 (TLR4) and downstream cytokines (such as CCL2, CXCL10) are considered important factors contributing to the development of neuropathic pain, the expression of these factors and the inhibitory effects of CoQ10 were evaluated.

NZO/HILtJ mice spontaneously developed type 2 DM and increased body mass with diabetic neuropathic pain. CoQ10 treatment decreased pain hypersensitivity and long-term supplementation prevented the development of diabetic neuropathic pain but did not attenuate diabetes. Spinal cord, blood serum, liver tissue, and dorsal root ganglia (DRG) from diabetic mice demonstrated increased lipid peroxidation, which was decreased by CoQ10 treatment. The percentage of positive neurons of p65 (the activated marker of NF-κB) and MAPK in DRG were significantly higher in DM mice compared to controls. However, CoQ10 treatment significantly decreased p65 and MAPK positive neurons in the DRG of DM mice. RT-PCR demonstrated that elevated levels of mRNA of CCL2, CXCL10 or TLR4 in the spinal cord of DM mice decreased significantly when DM mice were treated with CoQ10.


Conclusion

This model may be useful in understanding the mechanisms of neuropathic pain in type 2 DM induced neuropathic pain and may facilitate preclinical testing of therapies. CoQ10 may decrease oxidative stress in the central and peripheral nervous system by acting as an anti-oxidant and free-radical scavenger.

These results suggest that CoQ10 might be a reasonable preventative strategy for long-term use and using CoQ10 treatment may be a safe and effective long-term approach in the treatment of diabetic neuropathy.

Keywords

Type 2 diabetes; Neuropathic pain; Coenzyme Q10; Oxidative stress; Anti-oxidant

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