

Abstract

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Indications of the mechanisms involved in improved sperm parameters by zinc therapy.

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OBJECTIVE: To determine possible indications of the mechanisms involved in improved sperm parameters by zinc therapy in asthenozoospermic men.

SUBJECTS AND METHODS: Forty-five men with asthenozoospermia ($\geq 40\%$ immotile sperm) were randomized into four therapy groups: zinc only: $n = 11$; zinc + vitamin E: $n = 12$ and zinc + vitamins E + C: $n = 14$ for 3 months, and non-therapy control group: $n = 8$. Semen analysis was done according to WHO guidelines. Malonaldehyde, tumour necrosis factor- α (TNF- α), total antioxidant capacity, superoxide dismutase (SOD) and glutathione peroxidase were determined in the semen and serum. Antisperm antibodies IgG, IgM and IgA were evaluated by immunobeads. Sperm chromatin integrity was determined by acid denaturation by acridine orange and sperm apoptosis by light and electron microscopy. The effect of zinc on in vitro induced sperm oxidative stress by NADH was evaluated.

RESULTS: Asthenozoospermia was significantly associated with oxidative stress with higher seminal malonaldehyde (8.8 vs. 1.8 mmol/l, $p < 0.001$) and TNF- α (60 vs. 12 pg/l, $p < 0.001$), and low total antioxidant capacity (1.8 vs. 8.4, $p < 0.01$), SOD (0.8 vs. 3.1, $p < 0.01$) and glutathione peroxidase (1.6 vs. 4.2, $p < 0.05$), compared to normozoospermia. Zinc therapy alone, in combination with vitamin E or with vitamin E + C were associated with comparably improved sperm parameters with less oxidative stress, sperm apoptosis and sperm DNA fragmentation index (DFI). On the whole, there was no difference in the outcome measures between zinc only and zinc with vitamin E and combination of vitamins E + C. In the in vitro experiment zinc supplementation resulted in significantly lower DFI (14-29%, $p < 0.05$) compared to zinc deficiency.

CONCLUSION: Zinc therapy reduces asthenozoospermia through several mechanisms such as prevention of oxidative stress, apoptosis and sperm DNA fragmentation.

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