HIGH RISK OF SEMINAL OXIDATIVE STRESS IN SMOKER INFERTILE MEN

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Introduction and Objectives:
The impact of cigarette smoking on male fertility is highly controversial. The objective of this study was to investigate semen quality, levels of seminal oxidative stress (OS) and sperm DNA damage in a population of infertile men with a history of cigarette smoking.

Methods:
Information on smoking habits (number of cigarettes per day and number of years of smoking) was obtained from 48 infertile men at the time of clinical examination. Patients were classified into 4 groups: group 1 (smokers with normal genital examination; n = 8), group 2 (non-smokers with normal genital examination; n = 25), group 3 (smokers with abnormal genital examination; n = 7), and group 4 (non-smokers with abnormal genital examination; n = 8). A group of normal non-smoker donors (n = 13) was included as a control. Semen samples were examined according to the World Health Organization (WHO) guidelines (1999). Seminal leukocyte concentrations were determined by a myeloperoxidase-staining test. Levels of reactive oxygen species (ROS) were determined in washed sperm suspensions by a chemiluminescence assay and results expressed as X 10^6 counted photons per minute (cpm)/20 X 10^6 sperm/mL. Total antioxidant capacity (TAC) was measured in seminal plasma by an enhanced chemiluminescence assay and results expressed as Trolox equivalents. A composite value of ROS-TAC score was calculated as an index for OS. Seminal OS increases as ROS-TAC score decreases. Sperm nuclear DNA damage was assessed by sperm chromatin structure assay (SCSA). Results were expressed as %COMPα which represents the percentage of cells outside the main population of sperm, which have abnormal chromatin structure.

Results:
Smoking resulted in 48% increase in seminal leukocyte concentrations (P < 0.0001), 107% increase in ROS levels (P = 0.0003) and 11% decrease in ROS-TAC score (P = 0.002).

Conclusions:
Standard sperm parameters (sperm concentration, motility & morphology) or sperm DNA damage were not significantly different in smoker infertile men compared to non-smokers. However, cigarette smoking was strongly correlated with increased seminal leukocyte concentrations and OS. Smoking metabolites may cause sub-clinical inflammation of male reproductive tract and recruitment of leukocytes. Activation of leukocytes results in oxidative burst and OS. Our results indicate the importance of counseling these patients on the potential adverse effects of smoking on their fertility.

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