Original article

Augmented oxidative stress in infertile women with persistent chlamydial infection

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ABSTRACT

There is established association between oxidative stress, infections of genital tract and fertility. Genital tract infections may provoke increased production of free radicals and generate oxidative stress that can be involved in pathophysiology of a number of reproductive diseases and complications during pregnancy. The aim of this study was to determine connection between oxidative stress and infertility associated with persistent chlamydial infection. Serum samples of infertile women with tubal factor infertility (TFI), women with multiple spontaneous abortions (MSA) and fertile women was screened for C. trachomatis MOMP specific IgG and IgA antibodies and chHSP60 specific IgG antibodies using ELISA. The levels of superoxide anion radical, nitric oxide and reduced glutathione were determined spectrophotometrically. Serum levels of testosterone, luteinizing hormone and follicle stimulating hormone were determined by enzyme-linked fluorescent immunoassay method. Our results showed that persistent infection was more prevalent in TFI than in MSA group, whereas seropositivity was higher in MSA than in TFI group of patients. We also found that superoxide anion was significantly lower, while LH was markedly higher in TFI and MSA group of patients. However, when our results were analyzed according to the serological status of chlamydial infection, we found that parameters of oxidative stress, superoxide anion and index of oxidative stress, defined as relative ratio between superoxide anion and nitrites sum and glutathione ([O₂⁻ + NO₃⁻]/[GSH]) were significantly elevated in infertile patients with persistent chlamydial infection compared to seropositive and seronegative patients. Our findings point to the possible impact of Chlamydia trachomatis infection on prooxidative-antioxidative balance that can influence fertility potential in women with persistent chlamydial infection.

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1. Introduction

Oxidative stress (OS) is a condition characterized by an imbalance between prooxidants and antioxidants. This relationship can be disrupted by increased levels of reactive nitrogen species (RNS) and/or reactive oxygen species (ROS), or by reduction of antioxidant defense mechanisms [1]. A certain amount of ROS is required for maintenance of normal cellular functions. On the other hand, overproduction of ROS can overpower antioxidant defense mechanisms, thereby creating an environment that is unsuitable for normal physiological reactions. In women, this imbalance has been involved in the pathophysiology of a number of reproductive diseases including tubal factor infertility, polycystic ovary syndrome, endometriosis and unexplained infertility. In addition, this condition is associated with pregnancy complications such as miscarriage, recurrent pregnancy losses, preeclampsia, as well as intrauterine growth restriction [2]. Nitric oxide (NO) is a signal molecule with vasodilatory properties involved in several physiological and pathological processes [2]. Although the vasodilatory effect of NO may be therapeutic, excessive production

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can influence the structure and function of proteins, thereby causing changes in the catalytic activities of enzymes, changes in the organization of the cytoskeleton, and disturbances in cell signal transduction. Moreover, NO is a key factor that leads to the endothelial dysfunction associated with infertility states in both men and women [3].

Among others, C. trachomatis genital tract infection is widely associated with the failure of human reproduction. Spread of chlamydial infection to the upper part of the female genital tract can cause pelvic inflammatory disease with serious scarring in the fallopian tubes leading to tubal factor infertility [4]. Inflammatory responses to genital chlamydial infection lead to activation of polymorphonuclear leukocytes and macrophages, resulting in an increased production of ROS and oxidative stress. In that way, OS may possibly participate in multiple pathological changes that affect reproductive function of both men and women. Pathological mechanisms include lipid peroxidation, oxidative DNA damage, modulation of gene expression and inhibition of protein synthesis [5]. In several cell lines, chlamydial infection has proved to be the cause of the release of ROS and the products of lipid peroxidation [6]. Peroxidation of surrounding cells may induce cell lysis and consequently can facilitate spreading of chlamydial elementary bodies. This may partially explain the inflammation and cell damage occurring during chlamydial infections [7]. Moreover, infection of epithelial cells of fallopian tubes can cause oxidative damage to DNA, which may result in elevated levels of 8-OHdG (8-hydroxy-2-deoxyguanosine), as observed in women with chlamydial infection and tubal infertility [8]. A biomarker of endogenous oxidative DNA damage, 8-OHdG is also associated with a lower rate of fertilization and low quality oocytes [9].

It has been reported, in both human and animal studies, that female sex hormones might influence host susceptibility, innate and adaptive immune response and outcome of chlamydial infection. [10–13]. Other findings suggest that oxidative stress genes are, at least in part, under the control of sex hormones which may exert either antioxidative [14–16] or prooxidative effects [17,18]. Finally, hormonal imbalance is considered as a common cause of female infertility [19–21].

The complex interplay between oxidative stress response, sex hormones and persistent chlamydial infection in the etiology of female infertility is poorly understood. Understanding the mechanisms of their mutual relationships may provide useful for treatment and/or prognosis of infertility patients. Therefore, serum levels of oxidative stress biomarkers and female reproductive hormones were estimated in Chlamydia positive and negative women with tubal factor infertility, multiple spontaneous abortions and in fertile women with the aim to determine their possible association with persistent chlamydial infection and tubal factor infertility.
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