Rosacea and Oxidative Stress: Mini Review

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Abstract

Rosacea is a chronic inflammatory skin disorder. The average incidence is around 10%. The prevalence of the disease is higher in females and ages between 20-50 years. Oxidative stress is an disequilibrium among oxidants and antioxidants. According to many of studies, reactive oxygen species along with decreased antioxidant levels are responsible for rosacea pathogenesis.

Keywords: Rosacea; Oxidative stress; Pathogenesis; Inflammatory dermatosis; Telangiectasia

Abbreviations: OS: Oxidative Stress; ROS: Reactive Oxygen Species; TLR2: Toll Like Receptor 2

Introduction

Rosacea is a long-standing is an inflammatory dermatosis characterized by flare-ups and remissions. Patients apply to the dermatologists with complaints such as telangiectasia, edema, erythema, acneiform eruptions [1,2]. Individuals aged 20-50 years are more commonly affected and the disease is more prevalent in women than in men. The average incidence is around 10% [1,3]. In the previous studies, rosacea is reported to be related to systemic diseases such as hyperlipidemia, hypertension, insulin resistance, metabolic and cardiovascular diseases [4-9]. Its etiology is exactly unknown. Factors involved in the inflammatory processes are also responsible for the pathogenetic factors in the rosacea. Moreover, oxidative stress affects both the onset and the progression of rocasea [1-3].

Oxidative Stress (OS) is an disequilibrium among oxidants and antioxidants. It causes a impairment of redox signaling and controlor molecular abnormality. Excess levels of Reactive Oxygen Species (ROS) may give irreversible damage to various molecules such as protein, DNA and lipids. Increased IL-1 and TNF-a levels cause damage in keratinocytes, fibroblasts and endothelial cells resulting in the recruitment of leucocytes from periheral blood to the tissues [2]. In the recent studies, it has been found that production of ROS from neutrophils are related to inflammation in rosacea [1-5]. According to these studies, elevated ROS substances and ROS together with decreased antioxidant levels are responsible for rosacea pathogenesis [1-3,5,6]. Now-a-days, it has been discovered that levels of cathelicidin, trypsin-like serine protease kallikrein 5 (KLK5) and Toll Like Receptor 2 (TLR2) are high in rosacea patients [7,8].

Rosacea is thought to be a cutaneous vascular disorder but inflammation also plays an important role in the pathogenesis of rosacea. This inflammation is related to vascular and oxidative tissue changes and sunlight damage [2,4]. Oxidative modulation of proteins and lipids, deactivation of natural defences by ROS, altered lipid balance and the ROS-related stimulation of inflammatory mediators compose the inflammation of rosacea [2,4]. Recent studies have shown that rosacea is associated with an increase in ROS production and OS markers and a decrease in antioxidant levels [1-3,6,7]. ROS levels were evaluated in skin biopsy samples from rosacea and healthy individuals, and higher ROS activity in rosacea skin lesions than healthy controls was observed [5]. A defective antioxidant system in rosacea patients reported by serum total antioxidative potential levels [1], serum paraoxonase and arylesterase activities [2], superoxide dismutase activities [3] and antioxidant potential levels [10]. In the study, rosacea patients were divided into three groups based on clinical severity: stage I, II, and III [3]. They found no significant differences between Malondialdehyde (MDA) levels and Superoxide Dismutase (SOD) activities of the control and stage I and stage II groups classified as mild forms of rosacea. On the otherhand, in the severe involvement group (stage III), the SOD activity was lower than in the control groupwith decreased MDA levels. The authors hypothesised that in the mild involvement phase of rosacea patients, SOD activity was stimulated to protect the skin against ROS so that the MDA levels were maintained. In contrast, inmure severe disease form, due to a decrease in the capacity of the antioxidant defence system, the MDA levels were increased.Baz and colleagues [6] examinedplasma
MDA, and antioxidant potential levels in rosacea patients and control subjects. They discovered higher MDA levels and lower antioxidant potential levels in rosacea when compared to the control group, regardless of the severity of the disease. A positive correlation was detected between MDA and antioxidant potential levels in rosacea patients compared with the healthy subjects. We also found increased disulfide levels, and disulfide/native thiol ratios in rosacea patients compared to healthy subjects, suggesting increased oxidative stress in rosacea, which was similar to the results of previous studies [1-3].

Since the subtypes of rosacea are clinically heterogeneous, rosacea studies were diversely conducted based on the findings in clinical manifestations, histology, and factors exacerbating the skin disorder. From the diverse findings, the complex network in the pathophysiology of all rosacea subtypes is still unclear and expected to be from multiple factors: innate immunity, vascular changes, reactive oxygen species, ultra violet radiation, and microbes [1-3]. Regarding the role of oxidative stress in different types of rosacea patients, Takci Z et al. [2] reported decreased paraoxonase and arylesterase activities and increased serum lipid hydroperoxide levels suggesting elevated oxidative stress in patients with rosacea compared with controls, however, there were no significant differences in these parameters among ERT, PPT and phymatous subtype of the rosacea patients.

Conclusion

The pathophysiology of rosacea is multifactorial and not completely understood. It is reported that inflammation is a main process in rosacea. This inflammation is associated with many factors including UV damage, vascular changes, and oxidative tissue damage. In order to determine role of OS on this disease or to understand the association between oxidative stress, inflammation and rosacea, further studies including large number of patients are needed.

References