Effects of Ghrelin on Testicular Ischemia/Reperfusion-Induced Injury

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Abstract- Ischemia-reperfusion injury is a possible cause of testicular damage and infertility after testicular torsion and detorsion. The purpose of this study was to evaluate the effect of ghrelin on testicular Ischemia-reperfusion damage. A total of 30 adult male rats were selected for the study and divided randomly into 3 groups, each containing 10 rats. Animals in the testicular torsion and ghrelin treated groups were subjected to unilateral 720°counterclockwise testicular torsion for 1 hour, and then reperfusion was allowed after detorsion for 7 and 30 days. The ghrelin-treated group and the other two groups received intraperitoneally 40 nmol of ghrelin and physiological saline 15min before detorsion, respectively. The animals were sacrificed at the end of reperfusion times, and their testes were taken for later histopathological examination. The seminiferous tubules diameter, germinal epithelium height, as well as volume densities in testicular torsion / detorsion plus saline group, were significantly lesser versus control group, which clearly indicates an ischemia-reperfusion injury. Ghrelin treatment resulted in a partial increment in examined histological parameters on day 30 after reperfusion. Current results showed that ghrelin ameliorates the testicular ischemia-reperfusion damage.

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Introduction

Testicular torsion is one of the emergency conditions which require immediate surgery to reperfuse the affected testis, however, attempt to reperfuse of ischemic tissue may cause further damage to the testis. Many studies reported a loss of germ cells and disruption of the seminiferous epithelium after ischemia-reperfusion (IR) injury of the testis (1,2). A possible cause of IR damage is the reactive oxygen species (ROS) produced during its process (3). This ROS interact with lipids, proteins and nucleic acids leading to the loss of membrane integrity, structural or functional changes in proteins and genetic mutations, respectively (4). In a tissue like the testis, with its high rates of metabolism and cell replication, excessive production of ROS can especially be damaging, which makes the antioxidant capacity of the tissue very important. Prevention of reperfusion injury using a combination of enzymes and drugs has been studied along with the assessment of histopathological changes after testicular torsion/detorsion (2,3,5). They were intended to inhibit oxidative stress. For instance zinc aspartate, curcumin and dexamethasone reduce IR injury and increase the activity of antioxidant enzyme systems (6-8).

Ghrelin is a 28 amino acids orexigenic hormone produced principally in the stomach and has been identified as the endogenous ligand for the growth hormone secretagogue receptor (GHSR) (9). Ghrelin and its receptors were detected in testicular tissues indicating that the peptide may play a role in testicular regulation (10). The possible involvement of ghrelin in the protection of numerous tissues against IR injury has been shown (11). Antioxidant properties of ghrelin have been demonstrated in the rat testis (12).

Recently, we showed that administration of ghrelin increases antioxidant enzyme activities and reduces the lipid peroxidation in the testicular tissue exposed to IR (12).

In spite of remarkable antioxidative properties of ghrelin, few studies have been conducted on IR injury using ghrelin. Therefore, the present study was designed to investigate the histopathological effect of ghrelin on the testes of Wister rats following an IR injury. The

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