The protective role of heme oxygenase-1 induction on testicular tissues after testicular torsion and detorsion

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Abstract

Purpose

Testicular torsion-detorsion has been identified as an ischemia-reperfusion type of injury. We elucidated the protective role of heme oxygenase-1 super induction on testicular torsion-detorsion injury.

Materials and Methods

Adult male Sprague-Dawley rats were randomly allocated to undergo testicular torsion-detorsion, immediately followed by injection of normal saline, the heme oxygenase-1 inducer hemin or hemin plus the heme oxygenase-1 inhibitor tin protoporphyrin. Another set of rats that underwent sham operation, immediately followed by injection of normal saline, hemin or hemin plus tin protoporphyrin, served as controls. Testes were harvested 4 and 24 hours after detorsion, respectively, in the experimental groups or at comparable time points in the control groups.

Results

Histological evaluation confirmed that torsion-detorsion caused significant testicular tissue injury. Torsion-detorsion also caused significant increases in the testicular levels of nitric oxide, malondialdehyde, myeloperoxidase activity and heme oxygenase-1. The heme oxygenase-1 inducer hemin significantly enhanced the heme oxygenase-1 expression induced by torsion-detorsion and in turn attenuated testicular injury, and increases in nitric oxide, malondialdehyde and myeloperoxidase activity. In addition, the protective effects of hemin were significantly offset by the heme oxygenase-1 inhibitor tin protoporphyrin.

Conclusions

Super induction of heme oxygenase-1 protects testes from torsion-detorsion injury.

Key Words: testis; heme oxygenase (decyclizing); spermatic cord torsion; hemin; rats; Sprague-Dawley