

to the more potent anti-inflammatory and anti-oxidant activity of captopril which have been proved in the current study as it was stated in previous sections.

The probable mechanism for the protective role of captopril against colitis may be due to the induced reduction of intercellular adhesion molecules in both endothelial cells and leukocytes via angiotensin receptor 1 (AT 1) mediated mechanism. This observation was made by (6, 29). From another point of view it has been proved that angiotensin receptors 1 (AT1) are expressed by inflammatory cells such as macrophages and the stimulation of these receptors will cause increase in genes transcription with their products which are well known pro-inflammatory mediators such as transforming growth factor - β 1 (TGF- β 1) and tumor necrosis factor- α (TNF- α). So when the level of angiotensin I is reduced following administration of the angiotensin converting enzyme inhibitor (captopril), inflammation cascade will be reduced significantly (30,31,32)

CONCLUSIONS

Captopril has a potent anti-inflammatory and anti-oxidant effects that can be used successfully in treatment of experimentally acetic acid induced colitis in rats.

ACKNOWLEDGEMENTS

The author wishes to thank Dr. Eithar S Alkaragoli /College of dentistry /Al-Mustansiriya university for endless assistances during this research.

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