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Protective effects of vitamins on Diclofenac induced hepatotoxicity in adult male Wistar albino rats

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Introduction: Drug induced liver injury (DILI) possesses a major clinical problem and has become leading cause of acute liver failure and transplantation. Overstressed liver compromises its detoxification role which may expose it to a variety of diseases and disorders. Diclofenac sodium is a phenyl acetic acid derivative, a widely used NSAID for treatment of inflammatory conditions like osteoarthritis, rheumatoid arthritis, polymyositis, dermatomyositis, dental pain, spondyloarthritis, acute migraine, gout attacks and pain management in gall and renal stones. Though the exact mechanism by with Diclofenac injuries liver is not understood, some studies explain the toxicity by affecting cytochrome P 450 leading to production of active metabolites. This study was done to show the changes in the liver following Diclofenac treatment and to study the heapto-protective effects of vitamin A and C in Diclofenac treated rats.

Methodology: Rats were divided into four groups each 6 rats. Group-1: (n=6) control rats, Group-2: (n=6) rats treated with Diclofenac at dose of 75 mg/kg IP for seven days, Group-3: (n=6) rats treated with vitamin A at dose of 10 mcg/kg orally followed by Diclofenac at 75 mg/kg IP 2 hours later for seven days for seven days, Group 4: (n=6) rats treated with vitamin C at dose of 200 mg/kg orally followed by Diclofenac at 75 mg/kg IP 2 hours later for seven days.

Findings: Following Diclofenac treatment there the liver function test was elevated in Diclofenac treated group which was significantly reduced by the vitamin C compared to vitamin A. The liver acinus showed centriacinar necrosis of hepatocytes after seven days of Diclofenac treatment, which was prevented by administration of vitamin A and C. The hepatocyte necrosis was well prevented by administering vitamin C. So the hepato-protective effects of vitamin C were better compared to vitamin A following treatment with NSAID. So it may be necessary to administer vitamin C in patients treated with Diclofenac.

Recent Publications

1. Karthikeyan G, Sankaran P K, Kumaresan M, Zareena begum, Yuvaraj M (2017) localization of gap junction and neuropeptide as a determinant of neuropathic pain. *Indian Journal of Clinical Anatomy and Physiology*; 4(2): 70-71.
2. Sankaran P K, Jeevapriya t, Vinay Jadhav (2016) Expression of calcitonin gene related peptide (CGRP) in small neurons of trigeminal ganglion and its implications in migraine. *J Pharm Bio Sci*; 7(2): (b)52-55.

References

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2. Maria Francis Yuvaraj, Ponuswamy Kasirajan Sankaran, Gunapriya Raghunath, Zareena Begum, Kumaresan (2017) Thanatophoric Dysplasia; a Rare Case Report on a Congenital Anomaly. *Int J Pediatr*; 5: N.1(37).

Biography

Sankaran P K is currently working as an Associate Professor in Department of Anatomy, Saveetha Medical College in India. He has been working in developing pain models related to trigeminal neuralgia and its treatment module in animals.

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