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ABSTRACTS

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carbamazepine, valproic acid, phenytoin) and spectrophotometric endpoint method (lithium) in Siemens Dimension EXL200 model analyzer.

Results: Within a year, it was found that 1,330 lithium test results (60.1%) out of 2,213 were within the therapeutic range of 0.6 - 1.2 mmol / L, 89 test results were high levels (even 25 of them in toxic levels) and 794 results were below the therapeutic level. In the same period, 48 phenytoin levels out of 208 (23.1%) were within the therapeutic range (10 - 20 µg / mL); 2,311 (%68.9) valproic acid test results out of 3.355 were within the therapeutic range (50 – 100 µg/mL); 695 (%74.2) carbamazepine test results out of 937 were within the therapeutic range (4 – 12 µg/mL). In total, the percentage of drug monitoring tests that were within the mean therapeutic ranges was 65.3% and, approximately 35% was out of the therapeutic range.

Conclusion: The causes of this inconvenience may be preanalytical factors, errors in analysis or patient-caused factors. By sharing this information with clinicians, more appropriate and effective therapeutic drug levels could be obtained. Besides, by consulting with clinicians, we could find the causes of this inappropriate levels which were outside of the therapeutic range and contribute to reaching the target values as much as possible.

Keywords : Keywords: Phenytoin, valproic acid, carbamazepine, lithium, therapeutic drug analysis

Therapeutic Drug Monitoring and Toxicology

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Carnosine Protects Acetaminophen-Induced Liver İnjury Via Activation Of The Nuclear Erythroid-Related Factor-2 /Heme Oxygenase-1

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Aim: Acetaminophen (APAP) is an antipyretic and analgesic drug. APAP overdose causes severe hepatic injury. Its hepatotoxic potential result from the generation of a toxic metabolite N-acetyl-p-benzoquinone imine (NAPQI). Excessive NAPQI depletes hepatic glutathione (GSH) and then binds covalently to cellular proteins. Depletion of GSH and excessive production of NAPQI creates oxidative stress, which leads to hepatic necrosis. Nuclear erythroid-related factor 2 (Nrf2) is a transcription factor that regulates cellular defences by inducing the expession of various detoxification and antioxidant genes, such as heme oxygenase (HO-1). Nrf2 deficient mice exhibit increased sensitivity to APAP. Therefore, Nrf2 activation may serve as a shield for the prevention of APAP hepatotoxicity by combating oxidative stress. Carnosine (β-alanyl-L-histidine; CAR) is a dipeptide having anti-inflammatory and anti-oxidant properties. CAR pretreatment was found to decrease lipid peroxidation, inflammation and improve antioxidant system in APAP-treated rats. This study was aimed to investigate the role of CAR posttreatment in APAP-induced acute liver injury by activating Nrf2/HO-1 system. The efficiency of CAR was also compared with N-acetylcysteine (NAC) which is widely used in the treatment of APAP hepatotoxicity.

Materials and Methods: Sprague-Dawley rats were injected with APAP (500 mg/kg) intraperitoneally. One hour after APAP, CAR (250 mg/kg) or NAC (300 mg/kg) were administered to rats, intraperitoneally. Liver samples were collected 8 and 24 hours after APAP. Hepatic malondialdehyde (MDA) levels, Nrf2 and HO-1 mRNA and protein expressions were determined.

Results: APAP increased serum transaminases and hepatic MDA levels, CAR and NAC treatment decreased these elevated levels at 24 h after APAP injection. CAR and NAC caused activation of Nrf2 and HO-1 mRNA and protein expressions after APAP treatment.

Conclusion: Our results indicate that activation of Nrf2 /HO-1 system may play a role in improvement of liver injury due to NAC and CAR treatments in APAP-treated rats.

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Keywords : Carnosine, acetaminophen, liver injury, oxidative stress

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Elevated Symmetric Dimethylarginine Levels In Manganese-Exposed Welders

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Aim: SDMA, as an indirect inhibitor of NOS, has been demonstrated to act as a impotent molecule for neuronal NOS.Homoarginine (HArg) blocks the endogeneous NO synthesis via competing with arginine (1).The aim of this study was to determine the relation between serum symmetric dimethyl arginine levels and manganese exposure.

Methods: Serum SDMA was analyzed with the Shimadzu LC-20AD system coupled with Applied Biosystems MDS SCIEX (USA) API 3200 mass spectrometry (2). 100 microliters (µL) of internal Standard (d7-ADMA) in methanol were added to 200 µL of serum and centrifuged at 13.000 rpm for 10 minutes to remove the precipitated proteins. The supernatant was collected and dried under a nitrogen gas flow at 60°C. The derivatization step was performed dissolving the dried extract in 200 µL of a freshly prepared butanol solution containing 5% (v/v) acetyl chloride and kept at 60°C for 20 minutes. The solvent was removed by evaporation under nitrogen flow at 60°C. The derivatized samples were dissolved in 100 µL of water–methanol (90:10, v/v) containing 0.1% (v/v) formic acid and 40 µL was injected into the ultra pressure liquid chromatography (UPLC) analytical column.

Results: Serum symmetric dimethylarginine (SDMA) levels (0.33 ±0.07 µmol/L vs 0.22±0.04 µmol/L, $p<0.001$) were found to be statistically higher in welders compared to controls.

Conclusions: This result suggest that some of the oxidative stress-producing molecules may suppress the activity of the dimethylarginine dimethylaminohydrolase (DDAH) enzyme that metabolizes SDMA. Serum symmetric dimethylarginine levels might be in a relation with cardiovascular effects of manganese exposure.

Keywords : Symmetric dimethylarginine, Manganese toxicity, Cardiovascular risk, Welder