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Monitoring of Renal Function in Patients with Chronic Headache Treated with NSAID

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Abstract

Headache is one of the most common symptoms encountered in the general population as well as in medical practice worldwide. Migraine is the most frequent cause of headache and a common disabling neurological disorder with a serious socio-economical burden. Despite the introduction more than a decade ago of a new class of migraine-specific drugs with superior efficacy, the triptans, NSAIDs remain the most commonly used therapies for the migraine attack. Some are available over the counter and likely to be abused. Nonsteroidal anti-inflammatory drugs (NSAIDs) are capable of inducing a variety of renal function abnormalities. The adverse effects of nonsteroidal anti-inflammatory drugs (NSAIDs) are mediated via inhibition of prostaglandin synthesis by non-specific blocking cycloxygenase, leading to vasoconstriction and reversible mild renal impairment in hypoperfusion. When unopposed, this may lead to acute kidney injury (AKI). Although this presents as AKI, chronic use of NSAIDs may result in chronic kidney disease (CKD).The standard metrics to follow the progression of AKI, like serum creatinine and blood urea levels, are inconvenient and depend on kidney injury. That's why we must use specific markers for early detection. In the present review we will follow the levels of specific urinary biomarkers which we can use as signals for early detection of nephrotoxicity. There has not been perspective study for nephrotoxicity of NSAID used in long term by a patient with chronic pain.

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Keywords: AKI, biomarker, nonsteroidal anti-inflammatory drugs, migraine.

Nephrotoxicity of NSAIDs and MTX

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Abstract

Rheumatoid arthritis (RA) is a chronic disease requiring potencial nephrotoxic therapy with nonsteroidal antinflamatory drugs (NSAIDs) and disease modifying antireumatic drugs (DMARDs). The aim of this study is to identify early renal changes by means of specific biomarkers,as the most the sensitive parameter. 100 patients (80-RA^{sero+},20-RA^{sero-}) with chronic rheumatic pain were treated with NSAIDs and 8 to16 weeks by metotrexat (MTX) in comparison with the control group. The follow up was 3 time intervals. Besides conventional markers of renal function (serum/urine creatinin determined by Jaffe methods, enzymaticassay for urea serum and GFR calculated by Cockcroft Gaunt formulas) we used imunoturbodimetric assay for urine α1 Microgloglobulin (α1M) and microalbuminuria, to monitor glomerular and tubular functioning. Any history of kidney disease was exclusion criteria to enter the study. Following 16 weeks treatment with combined therapy with NSAIDs and MTX, no changes were found in the serum creatinine and serum urea, compared with the specific biomarker α1 Microgloglobulin (α1M) and microalbuminuriain all patients (RA $^{sero+}$ and RA $^{sero-}$) with 99% interval of confidence(CI), and probability of $p<0.01$ compared with the control group of healthy patients. We found changes on the glomerular and tubular level, despite the normal values of all the assayed conventional markers for renal function, and we confirmed the sensitivity of the used. We can't confirm by nephrotoxicity, but if we follow the elevation of the level of the specific biomarkers, we can use them as early signals for nephrotoxicity.

Keywords: Biomarkers, nephrotoxicity, nonsteroidal antiinflamatory drugs, rheumatoid arthritis.