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EDITORIAL

Contrast agent-induced nephrotoxicity: role of oxidative stress and apoptosis through the mitochondrial pathway

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Iodinated contrast media may lead to a sudden deterioration in renal function (Laville and Juillard 2010). Approximately 10% of all hospital-acquired cases of renal failure are caused by contrast agents. However, relatively little is known about the responsible mechanisms in vivo. Therefore, the editors are happy that Carlo Briguori, Cristina Quintavalle, Francesa De Micco and Gerolama Condorelli from Naples and Milan in Italy have contributed a review of the molecular mechanisms of contrast agent-induced nephrotoxicity (Briguori et al. 2010, this issue). Several human and animal studies have given strong evidence that oxidative stress plays an important role in contrast agent-induced acute kidney injury. Furthermore, as caspase 9 and 3 are activated but caspase 8 and 10 are not, apoptosis is therefore induced via the mitochondrial pathway. Since both, reactive oxygen species (Bolt and Hengstler 2010; Nishimura et al. 2010; Cederbaum et al. 2009; Wang et al. 2009a, b; Schumann et al. 2009; Schug et al. 2008; Glahn et al. 2008) and apoptosis (Han and Park 2010; Ogata et al. 2010; Wang et al. 2009a, b; Plöttner et al. 2009; Borza et al. 2008; Lehmann et al. 2010) represent cutting-edge topics of our journal, the current review may be of high interest to our readers.

Moreover, the authors have reviewed 17 clinical studies on prophylaxis of contrast agent-induced nephrotoxicity. As a result, they conclude that oral treatment with the potent antioxidant N-acetylcysteine appears to be effective in preventing renal failure.

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