

GW23-e1936

ROLE OF ALDOSTERONE IN MYOCARDIAL INJURY DURING HEPATIC ISCHAEMIA/REPERFUSION

doi:10.1136/heartjnl-2012-302920b.36

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Objectives Hepatic ischaemia reperfusion (HIR) injury is common in major liver surgery including liver transplantation and hepatectomy. Furthermore, hepatic ischaemia reperfusion is associated with excess aldosterone production and greatly increases risk of postoperative cardiac complications. The objective of this study was to investigate the role of aldosterone in myocardial injury following HIR.

Methods Using a 20 min total warm ischaemia model in rats, heart tissues and blood were sampled at 2–72 h after reperfusion for detection of myocardial damage, myocardial and plasma aldosterone, lipid peroxidation and inflammatory factors. Myocardial mitochondrial functions were also compared.

Results HIR resulted in myocardial injury early during the reperfusion period ($p < 0.05$). These effects were correlated with enhanced levels of myocardial and plasma aldosterone. The levels of aldehyde dehydrogenase (ALDH₂) and calcineurin (CaN) mRNA expression in hearts were decreased by HIR. Administration of spironolactone (20 mg/kg/day, P.O.) significantly attenuated the severity of heart injury and inhibited malondialdehyde (MDA) and tumour necrosis factor (TNF) α , which were elevated by HIR. Moreover, spironolactone significantly improved heart mitochondria function and up-regulated ALDH₂ expression accompany with the down-regulation of CaN mRNA.

Conclusions In conclusion, aldosterone contributes significantly to myocardial injury in HIR, and may be a potential therapeutic target for cardiac injury in clinical situations involving remote ischaemia/reperfusion.