

DEFINITION OF HYPOXIC-ISCHEMIC INJURY OF KIDNEY IN IUGR NEWBORNS

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Background and aim: Intrauterine growth restriction (IUGR) is a serious complication of pregnancy leading to an increased risk of perinatal hypoxia, intrauterine and neonatal death.

Circulatory adaptive responses to perinatal hypoxia may lead to renal injury as consequence of decreased perfusion of the kidney. Hypoxic stress usually results a tubulo-interstitial damage in newborns. One of the sensitive biomarker of proximal tubular impairments is a kidney injure molecule-1(KIM-1).

KIM-1 is a transmembrane protein that is not detectable in normal kidney tissue and expressed at very high levels in urine after ischemic and toxic injury.

The aim of this study was to estimate renal function in newborns with IUGR by determination the urinary level of KIM-1.

Methods: Urinary samples of 33 infants with IUGR collected at first 3 days of life to determine urinary KIM-1 concentration. Human KIM Rapid Test was used for this determination.

Results: KIM-1 was detected in urine samples of 20/33 IUGR newborns. Range of urinary level of KIM-1 was 0.5-4 ng/ml. High concentration of this marker was revealed in newborn with pulmonary hypertension and hypoxic encephalopathy. Range of seric creatinine levels was 38-42mkmol/l.

Conclusions: In population of newborns with IUGR fetoneonatal hypoxia can cause kidney impairment which is often not clinically overt. Correlation between urinary level of KIM-1 and severity of perinatal hypoxia indicate prerenal injury of kidney in newborns with IUGR.

Further investigations will be conducted in order to define the correlation between severity of IUGR and levels of KIM-1 and other biomarkers of kidney injury.