Physiology and biochemistry of endothelial function in children with chronic renal failure

Premature atherosclerosis is a major cause of morbidity and mortality in chronic renal failure (CRF). Endothelial dysfunction is a key early event in atherogenesis .The aim of this study was to assess the effect of CRF on endothelial function using physiological and biochemical measures. To focus on the effect of CRF itself, 23 children (matched with 23 controls for age and vessel diameter) were selected because they were normotensive, had normal total cholesterol (TC (levels, and were not on vasoactive drugs. Their mean (range) age was 12.0 (7.8 to 17.0) years; GFR 17.5 (8.8 to 34.5) ml/min/1.73 m2. The physiology of endothelial function in the brachial artery was assessed using high resolution ultrasound by measuring its diameter at rest, during reactive hyperemia) endothelium dependent dilation) and after sublingual glyceryl trinitrate (GTN ; endothelium independent dilation). Nitric oxide (NO) metabolites and endogenous NO synthetase (eNOS) inhibitors were measured as an assessment of endothelial metabolism. Brachial artery dilation to flow [FMD, mean (SEM)%] was reduced in CRF to 4.9 (0.6) and controls 8.6 (0.6), P .0.0001 > In contrast, the response to GTN was similar in both groups: CRF 25.1 (1.6), controls 23.3 (1.2 ,(P = 0.31. There was no difference in TC, low density lipoprotein (LDL) or high density lipoprotein (HDL) between the patients and the controls. Triglycerides) TG) were higher in the patients but within the normal range. Antibodies against oxidized LDL (ox-LDL) were high in CRF. Endogenous NOS inhibitors were high in CRF, and intermediate NO metabolites were low. There was no

correlation between FMD of the brachial artery and lipid subfractions, or with NO metabolites or eNOS inhibitors. Endothelium dependent dilation of the brachial artery is impaired in children with CRF who do not have co-existing risk factors for atherosclerosis. This may represent early evidence of atherogenic vascular disease

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