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Results: After a median follow-up of 3 years, 11 cardiovascular and 16 non-cardiovascular deaths occurred. The study population consisted of 95 men (75%). Mean age was 70 years, mean office BP was 143/76 mmHg. The multivariate Cox regression analyses revealed that LVM index was independently associated with all-cause mortality (HR: 1.011, 95%CI 1.000–1.023, p = 0.049) after adjustment for age, sex, history of diabetes mellitus and hyperlipidemia, smoking, antihypertensive treatment and mean office blood pressure values.

Conclusions: Left ventricular mass index is an independent predictor of all-cause mortality in hypertensive patients with CKD.

RENIN, ERYTHROPOIETIN AND VITAMIN D RELEASE FROM HUMAN DONOR KIDNEYS DURING NORMOTHERMIC MACHINE PERFUSION: PREDICTORS OF POST-TRANSPLANTATION OUTCOME?

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Objective: Normothermic (37°C) machine perfusion (NMP) is a potential alternative to currently used hypothermic (4°C) machine perfusion (HMP) for donor kidney preservation before transplantation. NMP allows for metabolic activity and for functional assessment of donor kidneys. The kidneys are key producers of hormones and we investigated the release of prorenin/renin, erythropoietin (EPO), and vitamin D by kidneys on machine perfusion.

Design and method: Ten donor kidneys were subjected to HMP followed by 2 h of oxygenated NMP before transplantation. NMP perfusate was collected at three time points (0 h, 1 h, 2 h). Ten HMP perfusate samples were collected for the same measurements.

Results: Median release rates of prorenin (196 [i.q.r 28–266] ng/hour) and renin (228 [94–302] ng/hour) in the first hour of NMP were 83- and 37-fold higher than that in HMP perfusates respectively ($p = 0.0009$ and $p < 0.0001$). Median renin release rate showed a 3.2-fold downregulation during the second hour of NMP compared to the first hour. Median EPO release rate (14 [i.q.r 8–48] mIU/min) during the first hour of NMP was 2.9-fold higher than that in HMP perfusates ($p = 0.035$), while it remained stable in the second hour of NMP. Active vitamin D was undetectable in HMP perfusate samples, while there was a median vitamin D secretion of 56 (i.q.r 30–83) and 28 (8–50) pmol/hour in the first and second hour of NMP respectively ($p = 0.0001$ and $p = 0.003$). We then investigated whether there were correlations between the hormone releasing capacity and donor kidney status. Prorenin release rate in the second hour of NMP was lower as cold ischemia time (CIT) until NMP of donor kidneys increased ($r = -0.605$; $p = 0.049$). EPO release showed no correlation with donor age or CIT. Donor age and vitamin D release rate during two hours of NMP are highly correlated ($r = 0.652$; $p = 0.034$). Interestingly, donation after brain death (DBD) kidneys significantly released more vitamin D than donation after circulatory death (DCD) kidneys during the first and second hour of NMP ($p = 0.024$ and $p = 0.012$).

Conclusions: These data shows that NMP increases the hormone release capacity of transplant kidneys. Hormone release may represent a tool to assess kidney function during NMP.

HIGH FRUCTOSE DIET INCREASES RENAL CHREBP-BETA EXPRESSION AND LEADS TO INTRA-RENAL FAT ACCUMULATION IN A METABOLIC-LIKE SYNDROME RAT MODEL

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Objective: A high fructose diet induces metabolic syndrome (MeS) which includes ectopic lipid accumulation, such as fatty liver. MeS is also associated with chronic kidney disease (CKD). A novel isoform of ChREBP, ChREBPb, was recently reported to regulate liver fructose metabolism that leads to fatty liver development.
The aim of this study was to evaluate fructose metabolism in the kidney and whether this metabolism leads to intra-renal fat accumulation.

Design and method:
In-vivo: Sprague Dawley rats were fed either normal chow (Ctrl) or a high fructose diet (HFrD) for eight weeks. Blood pressure, fasting blood glucose and triglycerides were measured. The kidneys were harvested for ChREBPb and de novo lipogenesis (DNL) gene expression, triglyceride content and histopathology staining.
In-vitro: HK2 (human kidney) cells were treated with fructose for 48 h and gene expression for ChREBPb and DNL were determined.

Results: The HFrD rats exhibited higher blood pressure (152 vs. 138 mmHg), glucose (146 vs. 127 mg/dl) and triglyceride (280 vs. 143 mg/dl) levels. Kidneys weight normalized to body weight of the HFrD rats were significant higher than the Ctrl (7.32 vs. 5.87). The difference can be explained by the higher triglyceride content in the HFrD kidneys (16.4 vs. 12.4 mg). Oil red staining revealed higher lipid droplet formation in the HFrD kidneys, which was also supported by higher adipophilin mRNA expression. The expression level of ChREBPb and its downstream genes, scd and fasn, were elevated in the HFrD kidney.
Treating HK2 cells with 40 mM fructose increased ChREBPb expression levels. Its downstream genes, fasn and acc, also showed an increasing trend. In three out of five experiments, adipophilin was increased as well.

Conclusions: In this study, we demonstrated that fructose consumption leads to intra-renal lipid accumulation and to the formation of a fatty kidney. This suggests a potential mechanism that can partially explain CKD development in fructose induced MeS.

ACUTE RENAL FAILURE PROGRESSING TO ESRD IN A PATIENT WITH UNTREATED HIGH GRADE HYPERTENSION – THE MALIGNANT NATURE OF "BENIGN" NEPHROSCLEROSIS

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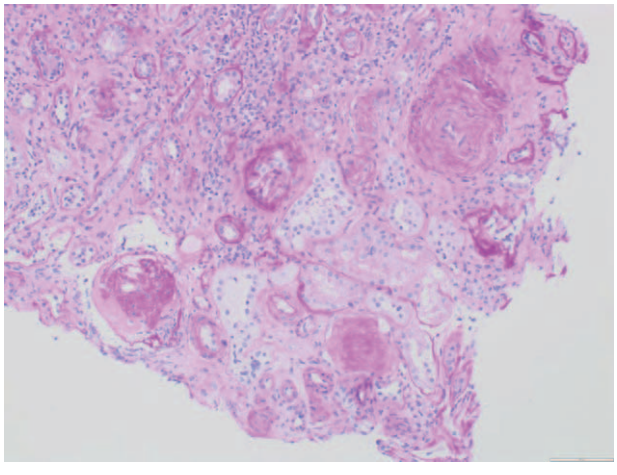
Objective: A 43 year old male with history of hypertension, non-adherence to medications, heavy smoking (since age 8) and alcohol abuse was admitted with epigastric pain, heartburn and upper GI bleeding (hematochezia) for which he had been recently taking PPI. On arrival his BP was 182/100 mmHg, HR-89, decreased breath sounds + wheezes.



Design and method: Hemoglobin was 7.4 mg/dL, Creatinine 11.2 mg/dl (recent baseline 1.1 mg/dl) Urea- 200 mg/dl.
He soon after needed HD which he continues a thrice weekly basis.
Four months prior to this admission he was hospitalized with hemoptysis, BP up to 261/136mmHg, proteinuria (1.5 gram/gram) and microhematuria, mild creatinine elevation to 1.3 mg/dL and hypokalemia of 2.6 mg/dL. Fundus examination revealed high grade retinopathy with cotton wool spots and flame hemorrhages but no papilledema. Supine Renin was 41.37microunits/ml, aldosterone was 30ng/dl.

Results: Since his renal function has not improved, and in parallel with HD treatments he undergone a kidney biopsy which revealed advanced benign nephrosclerosis (figure 1) with some FSGS, a single crescent and marked interstitial inflammation with eosinophils (figure 2)

Conclusions: Untreated high-grade hypertension and severe "benign" nephrosclerosis represent a microvascular ischemic nephropathy (similar to critical bilateral RAS) in which a relatively minor insult can deteriorate the patient into ESRD. While being non-adherent to different anti hypertensive medications, patient did take omeprazole which triggered acute interstitial nephritis on the background of advanced ischemic nephropathy, leading to permanent loss of renal function.