Conclusions: Complement 3, not other components of complement system, was activated and associated with vascular lesion in arterionephrosclerosis.

No conflict of interest

POS-405

SINGLE KIDNEY WITH RENAL ARTERY STENOSIS CAUSING PICKERING SYNDROME

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Introduction: Pickering syndrome, defined as recurrent 'flash' pulmonary oedema in patients with renal artery stenosis (RAS), is associated with a high mortality. Our case highlights the importance of careful patient selection for percutaneous renal artery stenting (PRAS) to ensure the benefits of revascularisation.

Methods: A 51-year-old female, with a single functioning kidney secondary to reflux nephropathy, known hypertension, ischaemic heart disease, and a 10 pack-year smoking history, presented to the emergency department with a two week history of dyspnoea, 'flash' pulmonary oedema, a progressive pericardial effusion, acute kidney injury and severe hypertension despite treatment with frusamide and four anti-hypertensive agents. Her blood pressure (BP) remained poorly controlled with systolic readings between 170 and 190 mmHg and diastolic levels between 90 and 110 mmHg. She subsequently developed acute respiratory failure requiring ventilator support. Computed tomographic (CT) angiography revealed severe ostial left RAS and near-occlusive distal aortic atheromatous disease (Figure 1).

Results: Following extensive multidisciplinary discussion, a catheter-based selective renal angiogram and stenting were performed, with access from the brachial artery. This resulted in marked improvement in renal perfusion, BP control and diuresis (3-6 litres per day for a week). She was extubated within 48 hours and her kidney function normalised.

Conclusions: Percutaneous stenting for the treatment of atherosclerotic RAS continues to be a controversial topic because of variable results from several randomised clinical trials (including ASTRAL, DRASTIC, STAR and CORAL). The primary patency of an optimally sized and access from the brachial artery. This resulted in marked improvement in renal perfusion, BP control and diuresis (5-6 litres per day for a week). She was extubated within 48 hours and her kidney function normalised.

Conclusions: Percutaneous stenting for the treatment of atherosclerotic RAS continues to be a controversial topic because of variable results from several randomised clinical trials (including ASTRAL, DRASTIC, STAR and CORAL). The primary patency of an optimally sized and access from the brachial artery. This resulted in marked improvement in renal perfusion, BP control and diuresis (5-6 litres per day for a week). She was extubated within 48 hours and her kidney function normalised.

No conflict of interest

POSTER SESSION: PHYSIOLOGY OF TUBULAR FUNCTION AND TUBULOINTERSTITIAL KIDNEY DISEASE

POS15
15/04/2021
Poster Area
05:00 – 06:00

POS-407

BARIATRIC SURGERY REPAIRS RENAL TUBULAR URIC ACID TRANSPORT IN MS PATIENTS THROUGH CIRCULATING EXOSOMES

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Introduction: Metabolic surgery is increasingly becoming recognized as a more effective treatment for patients with metabolic syndrome as compared to lifestyle modification and medical management alone. Kidney plays a crucial role in maintaining the homeostasis of uric acid. The aim of the study was to imply that Whether circulating exosomes are involved in the change of renal uric acid transporter after weight loss surgery in patients with metabolic syndrome.

Methods: We analyzed the basic data and blood and urine biochemical indicators of 30 normal people and 30 patients with metabolic syndrome preoperative and postoperative. The plasma exosomes of this cohort was extracted, and the human renal tubular epithelial cells were co-cultured with EVs. We determined the expressions of uric acid transporters URAT1, GLUT9 and ABCG2 on renal tubular epithelial cells by q-PCR and Western Blot.

No conflict of interest
Results: Compared with normal cohort, patients with metabolic syn-
drome have higher blood pressure, higher BMI, higher blood sugar, higher blood lipids and uric acid levels (P<0.001), but these biochemical indexes can return to near normal levels after bariatric surgery. The BMI of patients with metabolic syndrome was positively correlated with blood uric acid (R²=0.14, P<0.001); then there was no significant correlation between BMI and 24-hour uric acid. The human renal tubular epithelial cells were co-cultured with EVs. The expression of URAT1, GLUT9 and ABCG2 on the cells was detected by q-PCR and Western Blot. Compared with the renal tubular epithelial cells stimulated by circulating EV after operation, the expression of URAT1 and GLUT9 on renal tubular epithelial cells stimulated by circulating EV from the preoperative increased (P<0.05), and the expression of ABCG2 decreased (P<0.05).

Conclusions: Our results confirm that patients undergoing bariatric surgery changes in the composition of circulating extracellular vesicle, which can regulate renal tubular uric acid transporter, thereby affect renal uric acid metabolism.

No conflict of interest

POS-408

ETIOLOGIES AND CLINICAL FEATURES OF RENAL TUBULAR ACIDOSIS IN INFANTS AND CHILDREN

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Introduction: In children, renal tubular acidosis (RTA) is due to either an inherited or acquired defect that affects the kidney’s ability to absorb filtered bicarbonate, or excrete ammonia or titratable acid. The most common pediatric forms of RTA are distal (type 1) and proximal (type 2). The two other types of RTA are mixed (type 3) and hyperaldosteronism (type 4).

Methods: The aim of this study is to review the etiologies and clinical manifestations of the different forms of RTA in 37 infants and children from our center. This is a retrospective study from January 2005 to June 2020.

Results: The median age was five years. Twenty of our patients were girls, and 19 were from first-degree consanguineous parents, while four of them had similar cases in siblings. The youngest patient had 22-year-old, and the oldest had nine-year-old. Twenty-two of these children had stunting at diagnosis. The other clinical features at diagnosis were: dehydration (75%), polyuria-polydipsia syndrome (90%), and rickets (53%). Deafness was associated with RTA in five cases, meanwhile, nephrocalcinosis was more common with 62.5% of cases. All the children had metabolic acidosis with hypokalemia, hyperchloremia, and normal plasma anion gap. As for the type, 73% were identified as distal RTA (type 1), and 27% as proximal RTA (type 2). No case of mixed RTA (type 3) or hyperaldosteronism (type 4 RTA) was identified in this series. As for type 2 RTA, six cases were primitive (two of which were a Toni-Debré-Fanconi two cases of type 11 glycogenosis (Fanconi-Bickel syndrome), one case of tyrosinemia, one case of congenital glucose-galactose malabsorption. Management consisted of symptomatic measures: urine alkalinization, potassium supplementation, and vitamin D in case of rickets. Overall, evolution was good with height adjustment in 53% of the children. Complications were dominated by urinary tract infections seen in 72% of the patients. Unfortunately, we lost one child (Fanconi-Bickel syndrome) following an acute liver failure.

Conclusions: Renal tubular acidosis must be suspected in every child with polyuria-polydipsia syndrome. Secondary etiologies should be investigated from the perspective of starting a curative treatment. Genetic testing can help genetic counseling for parents with an affected child.

No conflict of interest

POS-409

THE ROLE AND SEX DIFFERENCES OF THE MITOCHONDRIAL PROTEIN SIRT3 IN THE DEVELOPMENT OF ANGIOTENSIN II-INDUCED HYPERTENSION

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Introduction: The proximal tubule of the kidney is one of the most energy-demanding tissues due to its unique capacity of reabsorbing 70 % of the filtrates. Sirtuin 3 (SIRT3) is a NAD-dependent deace-
tylase in the mitochondrial matrix and plays an important role in regulating cellular metabolism and oxidative stress in hypertensive diseases. The present study tested whether SIRT3 in the proximal tubules of the kidney is involved in angiotensin II (Ang II)-induced hypertension.

Methods: To test our hypothesis, we generated mutant mice with proximal tubule-specific knockout of SIRT3 (PT-SIRT3−/−) and treated male and female PT-SIRT3−/− mice with or without infusion of Ang II (15 ng/min, i.p.) plus 2% NaCl to slowly induce Ang II-dependent hypertension. The blood pressure response was evaluated by the tail-cuff method, whereas 24 h urinary excretions of Na+, K+, and Cl− were determined using metabolic cages. Whether there are significant sex differences in the pressor and natriuretic responses to Ang II and 2% NaCl were studied in male and female PT-SIRT3−/− mice.

Results: Both male and female PT-SIRT3−/− mice developed Ang II-induced hypertension in a time-dependent and similar manner within 2 weeks of Ang II infusion (P<0.01). There was no difference in the development of Ang II-induced hypertension with or without a 2% NaCl diet (n.s.), as systolic blood pressure (SBP) increased similarly to 135 ± 6 mmHg in both groups. These were compared with their respective basal blood pressure at 95 ± 3 mmHg in male and female PT-SIRT3−/− mice (P<0.01). Likewise, there was no significant difference in basal SBP between male (98 ± 3 mmHg) and female PT-SIRT3−/− mice (93 ± 3 mmHg, n.s.); nor was there a significant difference in the pressor response to Ang II and 2% NaCl diet between male (136 ± 6 mmHg) and female PT-SIRT3−/− mice (135 ± 7 mmHg, n.s.). Heart rate decreased similarly in response to Ang II and 2% NaCl in both male and female PT-SIRT3−/− mice during Ang II-induced hypertension (n.s.). In male and female PT-SIRT3−/− mice, 24 h water intake was significantly decreased when compared with basal conditions (P<0.05). However, a significant sex difference was found where female PT-SIRT3−/− mice had a lower urine output (ml/24 h) when compared with males, in basal and Ang II-induced hypertensive groups (P<0.05). 24 h urinary Na+ and Cl− excretions, but not K+ excretion, were significantly increased in response to Ang II-induced hypertension, compared with basal conditions in male and female PT-SIRT3−/− mice (P<0.01). No significant sex differences were detected in these responses.

Conclusions: Our results demonstrated that deletion of SIRT3 selectively in the proximal tubules of the kidney exacerbated Ang II-induced hypertension and increases natriuretic responses in both male and female PT-SIRT3−/− mice. However, no significant sex differences were established in the key pressor and natriuretic responses during Ang II-induced hypertension in PT-SIRT3−/− mice.

No conflict of interest

POSTER SESSION: ESSENTIAL HYPERTENSION AND RENOVASCULAR KIDNEY DISEASE

POS16
15/04/2021
Poster Area
05:00 – 06:00

POS-410

PREVALENCE OF RENAL MANIFESTATION AND HYPERTENSION AMONG CHILDREN WITH SICKLE CELL DISEASE; A SINGLE-CENTER RETROSPECTIVE STUDY

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Introduction: Sickle cell disease (SCD) is an autosomal recessive inherited blood disorder. A broad spectrum of renal abnormalities is associated with sickle cell disease. The renal impact of sickle cell disease among children remains an under-investigated area, especially the