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RESPONSIBLE EDITORS

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NKA in a human renal proximal tubular epithelial cell line (HK-2) and its comparison with previous results in HRTEC and in studies *in vivo* with ovariectomized (oVx) Wistar rats. HK-2 were treated with 17 β E (10 nM, 24 h) with or without an agonist (G-1) or an antagonist (G-15) of GPER-1. Cell proliferation was measured by bromodeoxyuridine (BrdU) uptake. The expression of NKA was assayed by western blot. In HK-2, 17 β E stimulated the BrdU uptake (26%) compared with control cells ($p < 0.05$). The treatment of HK-2 with G-15 (100 nM) inhibited 17 β E effect on cell proliferation. The treatment with G-1 (1000 nM) inhibited the BrdU uptake as observed in HRTEC ($p < 0.05$). The treatment of HK-2 with 17 β E and with G-1 (10 nM, 24 h) decreased NKA expression compared with control cells ($p < 0.05$), demonstrating that estradiol exerts these effects through GPER-1. These results agree with previous studies in HRTEC, where an increase of D1DR (dopamine receptor) expression was associated with the decrease of NKA. These results also match with previous studies in female adult Wistar rats, in which the oVx produced an increase of NKA expression in renal medulla while there was a decrease of D1DR, both in cortex and medulla. Likewise, hormonal replacement on oVx animals with 17 β E diminished the expression of NKA in renal medulla. In conclusion, our present results show that HK-2 cell line can be a valid *in vitro* model for better understanding of molecular and cellular renal mechanisms regulated by female sex hormones like estrogen.

186. (444) A MULTIVARIATE RELATIONSHIP BETWEEN LABORATORY DATA DURING THE EVOLUTION OF TYPICAL HEMOLYTIC UREMIC SYNDROME CHILDREN POPULATION

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Hemolytic uremic syndrome (HUS) is a systemic disease characterized by variable degrees of acute nephropathy, thrombocytopenia and microangiopathic hemolytic anemia. Laboratory and clinical parameters contribute very closely to progression of HUS. To better understand HUS evolution, the association between a set of laboratory data and a set of clinical parameters of a HUS population is investigated in this study.

We conducted a retrospective study of patients (n = 20) attended with diagnosis of typical HUS in the Pediatric Service of the Hospital Posadas from January 2012 to July 2020. 70% were women, with a mean age of 2.19 year. All laboratory data including those from the emergency department (admission), hospitalization, up to the first post-discharge check-up by external clinics were standardized in innovative report formats.

We perform the graphical representation of the evolution over time of several of the important clinical parameters (creatinine, hemato-crit, hemoglobin, among others). We find the creatinine curve relevant with well-defined moments in its evolution: rise, plateau and decline. We emphasize that 50% of the patients present a similar descent slope (- 0.353 +/- 0.022 mg/dL/day) regardless of the maximum value reached by creatinine. Also, analytic platform KNIME was used to evaluate the multivariate relationship between laboratory data and the evolution plasma creatinine values. We observed a strong correlation between the plasma values of creatinine-urea (positive, $r = 0,818$), platelets-uric acid (negative, $r = 0,610$) and direct bilirubin-uric acid (positive $r = 0,735$).

The study should be complemented with the comparison of qualitative variables, as well as with new parameters such as albuminuria, podocyturia, etc.), in order to generate a model of prediction of patient evolution during the acute period of HUS the disease and after it.

187. (468) NATRIURETIC PEPTIDES AND RENAL INFLAMMATION IN TWO MODELS OF HYPERTENSION: DEOXYCORTICOSTERONE ACETATE-SALT AND RENOVASCULAR 1 KIDNEY-1 CLIP

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Objectives: The aim was to demonstrate the hypothesis that in DOCA-salt (DS) and renovascular 1K-1C (RV) hypertensive models, the development of hypertension and the changes in natriuretic peptides (NP) secretion could be related to simultaneous over expression of NP receptors and inflammatory and fibrotic biomarkers in kidney.

Materials: Male Sprague-Dawley rats were randomly divided in three groups: Control (C), DS (30 mg/kg-Salt 1% W/W and RV. After 6 and 12 weeks, systolic blood pressure (SBP) was measured by the tail cuff method; plasma ANP and BNP levels were determined by commercial radioimmunoassay; mRNA expression of receptors NPR-A and NPR-C were measured in kidney by RT-PCR; and immuno-expression of inflammatory biomarkers (IL-6, TNF- α and NF- κ B) and fibrotic biomarker TGF- β , by immunohistochemistry. Statistical analysis was performed by two-way ANOVA followed by a Tukey-Kramer test.

Results: SBP increased in DS and RV at 6 and 12 weeks (mmHg: C6:118 \pm 2; DS6:194 \pm 2*; RV6:184 \pm 2*; C12:121 \pm 1; DS12:193 \pm 4*; RV12:203 \pm 4*). Plasma ANP increased in DS and RV (pg/mL: C6:170 \pm 68; DS6:689 \pm 143*; RV6:387 \pm 90*; C12:87 \pm 39; DS12:609 \pm 38*; RV12:294 \pm 57*), while plasma BNP rose only in RV (pg/mL: C6:39 \pm 6; RV6:99 \pm 8*; C12:42 \pm 7; RV12:94 \pm 10*). mRNA levels of NPR-A (AU, C6:0.096 \pm 0.011; DS6:0.232 \pm 0.015*; C12:0.092 \pm 0.020; DS12:0.328 \pm 0.039*) and NPR-C (AU, C6:0.184 \pm 0.020; DS6:0.721 \pm 0.131*; C12:0.131 \pm 0.026; DS12:0.302 \pm 0.039*) increased only in DS. All biomarkers increased in DS at 6 and 12 weeks (*), while TGF- β and TNF- α enhanced in RV only at 12 weeks (* $p < 0.05$ vs C).

Conclusion: Results suggest that ANP is a better marker for volume overload, while BNP level was increased only in the RV model. mRNA expression of both NP receptors increased only in DS and correlated with plasma ANP levels. Furthermore, inflammatory biomarkers expression increased faster in kidney DS than in RV, suggesting that volume overload induces earlier inflammation than pressure overload.

188. (490) ERYTHROPOIETIN IN URINE AS A NOVEL EARLY BIOMARKER OF CISPLATIN-INDUCED NEPHROTOXICITY IN RATS.

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Erythropoietin (EPO) is a glycoprotein hormone produced primarily in the kidney in response to hypoxic conditions. There is little information about the role of endogenous EPO in urine. In this regard, we were pioneering in detecting EPO in rat urine. Cisplatin is a chemotherapeutic agent broadly used. Its primary dose-limiting side effect is nephrotoxicity. The aim of this work was to analyse EPO urinary excretion (EPOu) in rats treated with Cisplatin and compare it with traditional and novel markers of renal injury. Male Wistar rats were treated with different single doses of Cisplatin (2, 5 and 10 mg/kg b.w., i.p.; Cis2, Cis5 and Cis10, n=4, respectively). A Control group of rats (C, n=4) was processed. After 48 h of Cisplatin administration plasma and urine samples were collected. Urea and creatinine plasma levels (U_p and Cr_p) and total proteins levels in urine (Pr_u) were determined spectrophotometrically. EPOu and Neutrophil gelatinase-associated lipocalin (NGALu) were eval-