function as antigen presenting cells, and could explain the observed relation between viral and allogeneic immunity.

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HERPES VIRUSES ORAL SHEDDING IN CHRONIC RENAL PATIENTS

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Introduction: Chronic renal failure (CRF) patients present inability of the kidney to maintain normal product of protein metabolism, blood pressure and hematocrit, and also the control of sodium, water, potassium and acid-base balance. Cellular and humoral immune response and can be depressed so the existence of disorders in oral health may represent foccus of infection. According the literature patients under hemodialysis have a 40-50-fold increased risk of mortality from sepsis compared with the general population. The monitoring of opportunistic viruses is an extreme importance, since they are related to comorbidities in these individuals. The objective of this study was to analyze the presence of human herpesviruses (HSV1, HSV2, VZV, EBV, CMV, HHV6, HHV7 and HHV8) in saliva of CRF patients.

Methods: 105 individuals divided into 3 groups according to the stage of renal disease. Group 1 (n=13) control group; Group 2 (n=53) patients with chronic renal disease and group 3 (n=33) end-stage renal disease (ESRD) patients in hemodialysis. For saliva collection was used the technique of salivary flow non-stimulated. Samples were evaluated by multiplex PCR for herpesviruses.

Results: The herpesviruses CMV, HSV-2, and HHV-8 were not found in any of the saliva samples. The distribution of herpesviruses in the study patients is shown in Table 1. The comparison of the prevalence of HSV-1 between group 3 and 2 showed significant difference ($p = 0.0307$). EBV, VZV, HHV-6 and HHV-7 did not show any difference.

Table 1. Distribution of herpesviruses in the 3 groups (number of patients and percentage)

GROUP	N	NEGATIVE	HSV-1	EBV	VZV	HHV-6	HHV-7
1	13	3 (23.07	1 (7.69)	7 (53.84)	0	1 (7.69)	7 (53.84)
2	53	18 (33.96)	4 (7.54)	25 (47.16)	3 (5.66)	4 (7.54	26 (49.05)
3	33	9 (27.27)	8 (24.24)	18 (54.54)	4 (12.12)	1 (3.03)	17 (51.51)

The most prevalent co-infection was EBV/HHV-7 detected in 40% of all patients. The distribution of the co-infection of the 3 groups are shown in Table 2. The statistical analysis did not show difference between the groups, but we detected the association of the HSV-1/EBV/HHV-7 just in the group 3.

Table 2 - Herpesviruses co-infection in saliva samples (number of patients and percentage).

GROUP	N	HSV-1/ EBV	EBV/ HHV-7	EBV/HHV-6/ HHV-7	EBV/VZV/HHV-7	HSV-1/EBV/HHV-7
1	10	1 (10.00)	3 (30.00)	1 (10.00)	0	0
2	35	1 (2.85)	15 (42.85)	15 (42.85)	2 (5.71)	0
3	24	2 (8.33)	5 (20.83)	5 (20.83)	2 (8.33)	5 (20.83)

Conclusions: With the results obtained we can conclude that serological screening for herpesvirus in chronic renal failure patients and hemodialysis , which are transplant candidates is essential to determine its been immune. The prevalence of co-infection in hemodialysis patients as HSV -1 / EBV / HHV -7 can cause complications in these patients.

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BETA-HYDROXYBUTYRATE INHIBITS NLRP3-MEDIATED INFLAMMATION AND DELAYS PROGRESSIVE RENAL FAILURE DURING PRIMARY HYPEROXALURIA RELATED KIDNEY STONE DISEASE

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Introduction: Primary hyperoxaluria is a condition characterized by overproduction of oxalate. When available in excess, oxalate combines with calcium to form crystals of calcium oxalate leading to kidney stone disease. NLRP3 inflammasome-mediated renal inflammation was identified as a prime pathomechanism of acute oxalate nephropathy (Mulay et. al., JCI 2013) as well as kidney stone disease (Knauf et. al., KI 2013). In addition, a recent report demonstrated that -hydroxybutyrate (BHB) inhibited NLRP3-mediated inflammation (Youm YH et. al., Nat Med 2015). Therefore, we speculated that BHB would ameliorate calcium oxalate (CaOx) crystals-induced NLRP3-mediated inflammation in acute oxalate nephropathy as well as primary hyperoxaluria related kidney stone disease.

Methods: C57BL/6 mice at the age of 6-to 8-week old were purchased from Charles River, which received a single intraperitoneal injection of 100 mg/kg sodium oxalate for the acute calcium oxalate nephropathy model and kidneys were harvested after 24 hours. For the chronic model, mice were fed with a low calcium plus Na-Oxalate diet. In both models, mice were fed with a ketogenic diet (KD) containing 1,3 butanodiol ketone diesters for 5-7 days before induce kidney damage. Urine and blood samples were collected at different time intervals (before and at 3, 7 and 14 days after damage) and kidneys were used for RNA isolation and histological analysis (IHC). The kidney function was quantified by the glomerular filtration rate (GFR), plasma creatinine and BUN levels at indicated times. Markers of kidney damage (Kim-1), inflammation (IL-6, MCP-1, RANTES, TNF-) and fibrotic markers (SMA, Col1A1, FSP-1, Fib-1) were analyzed by RT-PCR and IHC. The BHB levels were assayed in serum samples with a colorimetric assay kit (Cayman Chemicals. MI, USA).

Results: We observed that BHB inhibited CaOx crystals-induced NLRP3-mediated IL-1ß release from bone marrow derived dendritic cells in a dose dependent manner in vitro. Moreover, feeding of ketogenic diet (KD) to C57Bl6 mice elevated their plasma BHB levels and protected them from acute oxalate nephropathy as seen by decreased plasma BUN and creatinine levels despite similar renal crystal deposition. KD also reduced tubular injury (PAS, Kim-1), renal neutrophil infiltration and renal inflammation (IL-6, TNF, RANTES and CXCL2) during acute oxalate nephropathy. Similarly, KD did not change the renal CaOx crystal deposition and plug formation during kidney stone disease, however still protected mice from progressive renal failure as seen by improvement in glomerular filtration rate (GFR) and decreased plasma BUN and creatinine levels. Elevated plasma BHB levels after KD administration