between the groups ($p = .109$), however, disease activity index (SLEDAI score) was significantly higher in the active group ($p < .001$). Patients with active LN demonstrated significantly higher urinary MCP-1 and TWEAK concentrations than patients in the quiescent group [data described as median (P25; P75): 1440 (683; 2729) vs 256 (175; 477) pg/mL; $p < .001$ and 209 (117; 312) vs 74 (11; 173) pg/mL; p ⟨001]. When the patients' MCP-1 and TWEAK results were corrected for their urinary creatinine concentrations, the active group demonstrated values that were again significantly higher than the quiescent group: cMCP-1 [1093 (577; 2014) vs 286 (138; 774) pg/mgCr; $p < .001$] and cTWEAK [159 (89; 296) vs 63 (26; 160) pg/mg Cr; $p = .02$]. Furthermore, the results for MCP-1 and TWEAK, and cMCP-1 and cTWEAK were correlated in the active group (R-squared = 0.8, $p < .001$ and 0.9, $p < .001$, respectively).

Conclusions

As per previous studies in other countries, our data suggests that the urinary biomarkers MCP-1 and TWEAK reflect renal disease activity in our South African cohort of LN patients and could be useful for screening, diagnosis and disease activity monitoring.

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T468

Etiology of chronic kidney disease influences on values of lipids, lipoproteins, apolipoproteins and lipid indices

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Background-aim

To investigate the association of lipid profile parameters and etiology of chronic kidney disease (CKD).

Methods

The study enrolled patients ($n = 73$) recruited from the Nephrological Counselling Centre of the University Clinical Centre, Sarajevo whose estimated glomerular filtration rate (eGFR) was <60 ml/min/1.73 m^2. Estimated GFR was calculated by The Modification of Diet in Renal Disease (MDRD) equation. Chronic kidney disease patients were distributed into three groups according to etiology of disease. Concentration of lipids, lipoproteins and apolipoproteins were measured, and indices such as atherogenic index of plasma (AIP), Castelli risk index I (CRI-I), II (CRI-II), atherogenic coefficient (AC), ApoB/AI, lipid tetrad indeks (LTI) and lipid pentad index (LPI) were calculated.

Results

Apo AI and lipoprotein (a) levels were not significantly different among groups. In vascular etiology of CKD, significantly higher values of TCh, TG, VLDLc, LDLc, apoB, CRI-I, CRI-II, AIP, AC, ApoB/AI, LTI and LPI were observed in comparison to inflammatory etiology of the disease. Significantly lower HDLc and higher TG, VLDLc, CRI-I, CRI-II, AIP, AC, ApoB/AI LPI were found in vascular compared to morphological CKD type.

Conclusions

Besides eGFR, CKD etiology should be taken into account when lipid profile parameters and lipid indices are analyzed in CKD patients.

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T469

The role of copeptin and corticotropin-releasing factor in children with monosymptomatic and nonmonosymptomatic nocturnal enuresis

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Background-aim

Nocturnal enuresis is defined as bed wetting in children older than 5 years of age that occurs during sleep. Nocturnal enuresis is a complicated disease that includes multiple pathogenetic factors. Nocturnal enuresis is divided into two subgroups: monosymptomatic and non-monosymptomatic. The aim of this research was to evaluate the possible role of copeptin and corticotropin-releasing factor (CRF) levels in monosymptomatic and non-monosymptomatic nocturnal enuresis cases.

Methods

One hundred nineteen children with nocturnal enuresis and forty healthy children have enrolled in the study. Of the nocturnal enuresis group, forty-nine were monosymptomatic and 70 were non-monosymptomatic. Copeptin and CRF were measured via a competitive inhibition method with ELISA.

Results

The study group was composed of 119 children with nocturnal enuresis; forty-nine were monosymptomatic and 70 were non-monosymptomatic. Only nine patients had received desmopressin in the past, and all of them were nonresponders. The mean copeptin and CRF levels were significantly lower in children with both monosymptomatic and non-monosymptomatic nocturnal enuresis. The mean CRF level was significantly higher in nine patients who had received desmopressin and had not responded in comparison with the other nocturnal enuresis patients.

Conclusions

We demonstrated that the central nervous system and neurotransmitters such as copeptin and CRF play a role in children with both monosymptomatic and non-monosymptomatic nocturnal enuresis. We also found increased CRF levels in children who were nonresponsive to desmopressin acetate. Our study suggested that high plasma CRF levels before treatment may be a predictive marker for resistance to desmopressin acetate.

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