4

INDOXYL SULFATE SERUM LEVEL IN CHRONIC RENAL FAILURE PATIENTS DETECTED USING FLUORESCENCE-HPLC

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Indoxyl sulfate (IS) is a protein-bound typical uremic toxin that accumulates in patients with impaired kidney function. The laboratory methods used to quantify serum concentrations of IS require improvement. We report an optimal, cost-effective alternative to the methods currently used.The methods is as follows: serum samples were extracted and deproteinized using acetonitrile and then further purified using dichloromethane. The samples were then analyzed using high-performance liquid chromatography (HPLC) with a fluorescence detector at a flow rate of 1.0 mL/min. The isocratic mobile phase consisted of acetonitrile – 0.2% (V/V) trifluoroacetic acid in phosphate buffered saline, pH 2.5 (8:92, V/V). Detector settings were λex 280 nm/λem 390 nm. Under the indicated conditions, IS was well separated. The retention time was approximately 6.59 ± 0.11 min. The results of the precision and reproducibility tests were < 3%, and the recovery rate was greater than 95%,(98.44 ± 3.21%).

In conclusion, HPLC with a fluorescence detector is a simple, sensitive, and reliable method for quantifying IS concentration in the serum of patients with chronic renal failure.

http://dx.doi.org/10.1016/j.krcp.2012.04.363

40

THE CARDIOVASCULAR EFFECTS OF CINACALCET IN HEMODIALYSIS PATIENTS WITH SECONDARY HYPERPARATHYROIDISM

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Vascular changes characterized by calcification of either intima or media result in arterial stiffness and cardiac hypertrophy, especially in hemodialyais patients with secondary hyperparathyroidism. The purpose of this study is to evaluate the effects of cinacalcet on arterial compliance and cardiac hypertrophy. We studied 14 patients with ESRD who had high levels of intact PTH (iPTH, > 300 pg/mL) and of corrected serum calcium (cCa, > 9.0 mg/dL) with cinacalcet over 20-week period prospectively. After 20 weeks treatment, we performed flow-mediated dialation (FMD), cadio-ankle vascular index (CAVI) and echocardiographic analyses. Twenty weeks cinaclacet treatment significantly decreased blood levels of iPTH (628.2 ± 250.8 vs. 251.7 ± 237.4 pg/ml, $p < 0.01$), calcium (9.7 ± 0.7 vs.8.7 ± 0.6 mg/dl, $P < 0.01$), phosphorus (6.8 ± 1.3 vs. 5.0 ± 1.4 mg/dl, $P < 0.01$), calcium x phosphorus product (64.8 ± 15.4 vs. 43.5 ± 14.9, $P < 0.01$) and 25(OH) vitamin D (9.9 ± 3.4 vs. 8.2 ± 2.7 ng/mL, $P < 0.05$). There were no significant changes in LV mass, the ejection fraction and fractional shortening. In contrast, cinacalcet significantly improved FMD (8.6 ± 2.9 vs. 14.3 ± 2.8%, $P < 0.01$) and enhanced CAVI (8.8 ± 2.3 vs. 7.6 ± 2.4, $P < 0.05$), respectively. In conclusion, cinacalcet treatment in hemodialysis patients with secondary hyperparathyroidism ameliorates endothelial dysfunction and arterial compliance.

http://dx.doi.org/10.1016/j.krcp.2012.04.364

41

PLASMA BRAIN-DERIVED NEUROTROPHIC FACTOR LEVELS OF PATIENTS UNDERGOING HEMODIALYSIS AND PERITONEAL DIALYSIS

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Recent studies have shown that brain-derived neurotrophic factor (BDNF) is associated with neurotoxic, inflammatory and nutritional status. The purpose of this study is to investigate the changes in plasma BDNF concentration that were induced by chronic uremic condition. Using an enzyme-linked immunosorbent assay (ELISA), we measured peripheral BDNF levels in pre-hemodialysis (pre-HD), peritoneal dialysis (PD) patients and in a control group. The plasma BDNF concentrations of pre-HD, PD patients were significantly higher compared with those of controls (856.3 ± 879.1, 1167.9 ± 647.5 versus 403.1 ± 288.9 pg/ml, respectively). The plasma BDNF in dialysis patients was positively correlated with high-sensitivity C reactive protein (hs-CRP) and with serum albumin negatively.

Plasma BDNF	hs-CRP		Albumin	
	r	p	r	p
Pre-HD	0.321	0.025	-0.298	0.021
PD	0.414	0.070	-0.527	0.008
Pre-HD + PD	0.446	0.001	-0.453	0.001

In conclusion, BDNF might plays some role in the neurotoxicity of uremia and associated with inflammation and malnutrition in dialysis patients.

http://dx.doi.org/10.1016/j.krcp.2012.04.365

42

SURVIVAL DURING RENAL REPLACEMENT THERAPY OF PATIENTS PREVIOUSLY TREATED WITH A VERY LOW-PROTEIN DIET SUPPLEMENTED WITH KETOACIDS : THE ITALIAN EXPERIENCE.

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In the course of chronic renal failure, low protein-diets allow better control of metabolic disorders and may delay the start of renal replacement therapy (RRT). However, concerns exist that a very low protein diet supplemented with ketoacids (sVLPD) worsens survival after starting RRT. To evaluate whether a prolonged sVLPD regimen may affect all-cause mortality during the following RRT period, we studied time to all-cause death during RRT in patients previously followed in renal clinics either treated with sVLPD (sVLPD group, $n = 184$, age 67 ± 18 yrs) or not (CKD group, $n = 334$, age 66 ± 14 yrs). A Control group including 9,092 patients (age 66 ± 14 yrs) was selected from the Italian dialysis & transplantation registry (RIDT).

In sVLPD, CKD and Control groups, the prevalence of an history of cardiovascular disease was 41, 31, 25% and of diabetes was 18%, 31%, 17%, respectively; the median follow-up time in RRT (36, 32, 36 months) did not differ among groups.Cumulative survival was similar in sVLPD and CKD groups ($p = 0.638$), but significantly higher than in the Control group (Long-rank test, 20,62; $p < 0.0001$). These results persisted in the Cox model adjusted for age, gender, diabetes, history of cardiovascular disease; as compared with controls, the HRs [95% CI] for death were 0.57 [0.45 – 0.74] ($p = 0.0001$) in sVLPD and 0.65 [0.53 – 0.80] ($p = 0.001$) in CKD group. Due to age interaction with survival in sVLPD ($p = 0.024$), HRs for death reduced to 0.34 [0.16 – 0.73] and 0.58 [0.37 – 0.92] in sVLPD and CKD pts < 70 yrs.

In conclusion, prescription of sVLPD during the conservative phase of chronic renal failure does not worsen, or even improves, survival after starting RRT. This survival advantage is more evident in patients younger than 70 years.

http://dx.doi.org/10.1016/j.krcp.2012.04.366