

doi: 10.13241/j.cnki.pmb.2023.03.020

CRP、PTH、Alb 及 Hb 水平变化与尿毒症周围神经病变程度的相关性 *

鲍楠¹ 朱燕亭¹ 张蓬杰¹ 吴冰¹ 李娜^{2Δ}

(1 陕西省人民医院肾内科 陕西 西安 710068; 2 西安交通大学第一附属医院检验科 陕西 西安 710061)

摘要 目的:探讨 C 反应蛋白(CRP)、甲状旁腺素(PTH)、血清白蛋白(Alb)及血红蛋白(Hb)水平变化与尿毒症周围神经病变程度的相关性。**方法:**选取我院 2019 年 4 月到 2022 年 4 月收治的 340 例需要进行维持性血液透析治疗的尿毒症患者作为研究对象,依照是否存在周围型神经病变的情况分为非神经病变组(n=132)和神经病变组(n=208)。对比两组患者的临床相关资料,CRP、PTH、Alb 及 Hb 水平变化,其他血清指标变化,并分析 CRP、PTH、Alb 及 Hb 水平变化与尿毒症周围神经病变程度的相关性。**结果:**两组患者高血压患病人数对比无差异($P>0.05$),两组患者的糖尿病患病人数、血液透析频率以及血液透析时间对比差异显著($P<0.05$);神经病变组患者的 CRP、PTH 水平明显高于非神经病变组($P<0.05$),神经病变组患者的 Alb、Hb 水平明显低于非神经病变组($P<0.05$);两组患者血浆胆固醇(CHO)、红细胞(RBC)、三酰甘油(TG)、血肌酐(SCr)水平对比无显著差异($P>0.05$),神经病变组患者血清前白蛋白(PA)、二氧化碳结合力(CO_2CP)水平低于非神经病变组,神经病变组患者血清 BUN、FBG 水平高于非神经病变组($P<0.05$);Spearman 相关分析结果显示:CRP、PTH 与尿毒症周围神经病变程度呈正相关($P<0.05$),Alb、Hb 与尿毒症周围神经病变程度呈负相关($P<0.05$)。**结论:**CRP、PTH、Alb 及 Hb 水平变化与尿毒症周围神经病变程度具有明显关系,可以用于判断尿毒症患者的周围神经病变程度。而且尿毒症周围神经病变患者多数原发疾病为糖尿病,血液透析频率较低,维持血液透析时间较长。

关键词:尿毒症;周围神经病变;C 反应蛋白;甲状旁腺素;血清白蛋白;血红蛋白

中图分类号:R692;R459.5 **文献标识码:**A **文章编号:**1673-6273(2023)03-500-05

Correlation between The Changes of CRP, PTH, ALB and Hb Levels and The Severity of Uremic Peripheral Neuropathy*

BAO Nan¹, ZHU Yan-ting¹, ZHANG Peng-jie¹, WU Bing¹, LI Na^{2Δ}

(1 Department of Nephrology, Shaanxi Provincial People's Hospital, Xi'an, Shaanxi, 710068, China;

2 Department of clinical lab, The First Affiliated Hospital of Xi'an Jiaotong University, Xi'an, Shaanxi, 710061, China)

ABSTRACT Objective: To investigate the correlation between the changes of C-reactive protein (CRP), parathyroid hormone (PTH), serum albumin (ALB) and hemoglobin (HB) and the degree of uremic peripheral neuropathy. **Methods:** From April 2019 to April 2022, 340 patients with uremia who need maintenance hemodialysis in our hospital were selected as the research objects, and they were divided into neuropathy group (n=132) and non neuropathy group (n=208) according to whether there was peripheral neuropathy. The clinical data, the changes of CRP, PTH, ALB and Hb levels and other serum indexes of the two groups were compared, and the correlation between the changes of CRP, PTH, ALB and Hb levels and the degree of uremic peripheral neuropathy was analyzed. **Results:** There was no difference in the number of patients with hypertension between the two groups ($P>0.05$), but there were differences in the number of patients with diabetes, hemodialysis frequency and hemodialysis time between the two groups ($P<0.05$); the levels of CRP and PTH in the neuropathy group were higher than those in the non neuropathy group ($P<0.05$), and the levels of ALB and Hb in the neuropathy group were lower than those in the non neuropathy group. There was no significant difference in plasma cholesterol (CHO), red blood cells (RBC), triacylglycerol (TG), and blood creatinine (SCr) levels between the two groups ($P>0.05$). The serum prealbumin (PA) and carbon dioxide binding force (CO_2CP) levels in the neuropathy group were lower than those in the non-neuropathy group, and the serum BUN and FBG levels were higher than those in the non-neuropathy group ($P<0.05$). The results of correlation analysis showed that CRP and PTH were positively correlated with the degree of uremic peripheral neuropathy ($P<0.05$), while ALB and Hb were negatively correlated with the degree of uremic peripheral neuropathy ($P<0.05$). **Conclusion:** The levels of CRP, PTH, ALB and Hb are significantly related to the degree of uremic peripheral neuropathy, which can be used to judge the degree of uremic peripheral neuropathy. Moreover, most of the primary diseases of uremic peripheral neuropathy patients are diabetes, the frequency of hemodialysis is low, and the maintenance of hemodialysis time is long.

* 基金项目:陕西省自然科学基金研究计划项目(2021JQ-905)

作者简介:鲍楠(1983-),女,大学本科,主治医师,研究方向:肾内相关疾病,E-mail:baonan830106@163.com

Δ 通讯作者:李娜(1982-),女,硕士研究生,副主任技师,研究方向:临床检验诊断及妇幼健康研究,E-mail:baonan830106@163.com

(收稿日期:2022-05-05 接受日期:2022-05-29)

Key words: Uremia; Peripheral neuropathy; C-reactive veneer; Parathyroid hormone; Serum albumin; Hemoglobin

Chinese Library Classification(CLC): R692; R459.5 **Document code:** A

Article ID: 1673-6273(2023)03-500-05

前言

尿毒症周围神经病变是维持性血液透析患者常见的一种临床症状,目前其发病机制尚无明确定论^[1]。该病多发于慢性肾衰竭期,是一种以肢体远端对称性感觉障碍为主的多发性神经病变,以感觉障碍为主的一种多发性周围神经病变,多为下肢发病^[2]。一般慢性肾衰竭6个月之后的患者会出现周围神经病变情况,若不进行及时治疗,患者会随着发病时间的延长导致病情加重。典型尿毒症周围神经病变的患者多发生在进展期的尿毒症患者之中,他们内生肌酐清除率多数大于12毫升/分钟,对感觉与运动神经产生严重影响,当患者出现周围性神经病变则需要应用肾脏替代疗法进行治疗^[3,4]。临床上对于周围性神经病变的患者多以临床症状进行诊断,患者多以对称性感觉障碍为主,患者在早期会感觉肢体远端出现刺痛感、灼痛感等,还可出现感觉丧失、麻木感和痛觉过敏现象^[5,6]。虽然以症状进行诊断的准确率较高,但为了丰富尿毒症周围神经病变的诊断标准,应用血液指标对该疾病进行诊断成为了研究的热点话题^[7,8],因此本文选取我院2019年4月到2022年4月收治的340例需要进行维持性血液透析治疗的尿毒症患者作为研究对象,探讨CRP、PTH、Alb及Hb水平变化与尿毒症周围神经病变程度的相关性,具体报告如下。

1 资料与方法

1.1 一般资料

选取我院2019年4月到2022年4月收治的340例需要进行维持性血液透析治疗的尿毒症患者作为研究对象,依照是否存在周围型神经病变的情况分为非神经病变组($n=132$)和神经病变组($n=208$)。

纳入标准:所有患者对本研究知情并签署同意书;所有患者符合尿毒症诊断标准^[9];所有患者均长期性维持血液透析治疗;神经病变组患者符合周围性神经病变的诊断标准。

排除标准:合并多器官功能障碍综合征;合并呼吸衰竭;合并血液透析严重并发症;合并恶性肿瘤;合并精神障碍者。

其中神经病变组患者中男性117例,女性91例,年龄为23-71岁,平均(45.43 ± 2.53)岁;非神经病变组患者中男性67例,女性65例;年龄为25-75岁,平均(45.26 ± 2.46)岁。两组患者一般资料对比无显著差异($P>0.05$),可以进行对比分析。

1.2 方法

对两组患者的血液灌流治疗、血液率过指标、血液透析时间、血压情况、糖尿病情况以及高血压情况进行调查分析。

对所有患者在清晨空腹采取静脉血2毫升,防止室温下进行血清分离,应用免疫比浊法对血清中C反应蛋白(C-reactive protein, CRP)、甲状旁腺素(Parathyrin, PTH)、血清白蛋白(Albumin, Alb)、血红蛋白(Hemoglobin, Hb)、前白蛋白(Prealbumin, PA)、血浆胆固醇(Cholesterol, CHO)、红细胞(Red blood cell, RBC)、三酰甘油(Triacylglycerol, TG)、尿素氮(Urea nitro-

gen, BUN)、血肌酐(Serum creatinine, SCr)、二氧化碳结合力(CO_2CP)、血浆纤维蛋白原(Fibrinogen, FBG)水平情况进行测定。

1.3 统计学方法

本研究数据采取统计学软件SPSS 23.0进行数据分析,计数资料以($n\%$)表示,进行 χ^2 检验;计量资料以($\bar{x} \pm s$)表示,组间比较采用t检验;采用Spearman相关分析方法分析CRP、PTH、Alb及Hb水平变化与尿毒症周围神经病变程度的相关性;以 $P<0.05$ 为差异有统计学意义。

2 结果

2.1 两组患者的相关信息对比

两组患者高血压患病人数对比无显著差异($P>0.05$),两组患者的糖尿病患病人数、血液透析频率以及血液透析时间对比差异显著($P<0.05$),如表1所示。

2.2 两组患者CRP、PTH、Alb及Hb水平变化对比分析

神经病变组患者的CRP、PTH水平明显高于非神经病变组($P<0.05$),神经病变组患者的Alb、Hb水平明显低于非神经病变组($P<0.05$),如表2所示。

2.3 两组患者其他血清指标水平对比分析

两组患者CHO、RBC、TG、SCr水平对比无显著差异($P>0.05$),神经病变组患者血清PA、 CO_2CP 水平低于非神经病变组,神经病变组患者血清BUN、FBG水平高于非神经病变组($P<0.05$),如表3所示。

2.4 CRP、PTH、Alb及Hb水平变化与尿毒症周围神经病变程度的相关性

Spearman相关分析结果显示:CRP、PTH与尿毒症周围神经病变程度呈正相关($P<0.05$),Alb、Hb与尿毒症周围神经病变程度呈负相关($P<0.05$),如表4所示。

3 讨论

尿毒症性周围神经病变是当前尿毒症患者常见的一个并发症,发病率约为50%^[10,11]。但当前对于尿毒症周围神经病变的发病机制并不明确,患者临床上多以血管运动症状障碍、腱反射减退、肌肉萎缩、肌肉无力以及感觉丧失等为主要症状^[12,13]。临床上对尿毒症周围神经病变的治疗方法主要为肾脏移植、血液透析以及药物治疗等^[14]。近年来有研究发现^[15,16],尿毒症周围神经病变的严重程度与氨基酸和白蛋白水平具有明显相关性,为了揭示尿毒症周围神经病变的发生与发展特征,并丰富该疾病的诊断标准,本文主要针对CRP、PTH、Alb及Hb与尿毒症周围神经病变程度的相关性展开研究,为临床诊断奠定理论基础。

本研究结果显示:两组患者高血压患病人数对比无显著差异,糖尿病患病人数、血液透析频率以及血液透析时间对比差异显著,与Hayden MR等^[17]研究结果类似。这主要是因为,尿毒症周围神经病变患者的尿毒症原发疾病多为糖尿病,且患者

表 1 两组患者相关信息比较(n)

Table 1 Comparison of relevant patient information between the two patient groups (n)

Indexs	Non neuropathy group(n=132)	Neuropathy group(n=208)
Hypertension		
Yes	94	125
No	38	83
Diabetes		
Yes	77	170*
No	55	38*
Hemodialysis frequency		
No regular perfusion and hemofiltration	17	179*
Regular perfusion and hemofiltration	115	29*
Less than twice a week	43	123*
Twice a week or more	89	85*
Hemodialysis time		
<2 years	60	34*
>2 years	72	174

Note: compared with the non neuropathy group, * $P<0.05$, the same below.

表 2 两组患者 CRP、PTH、Alb 及 Hb 水平变化对比分析($\bar{x} \pm s$)Table 2 Comparative analysis of CRP, PTH, ALB and Hb levels between the two groups ($\bar{x} \pm s$)

Groups	n	CRP	PTH	Alb	Hb
Non neuropathy group	132	4.42± 0.71	84.31± 2.43	36.31± 3.42	101.24± 9.24
Neuropathy group	208	14.31± 0.53*	334.32± 35.21*	31.22± 3.21*	86.32± 5.35*

表 3 两组患者其他血清指标水平对比分析($\bar{x} \pm s$)Table 3 Comparative Analysis of other serum index levels between the two groups ($\bar{x} \pm s$)

Indexs	Non neuropathy group(n=132)	Neuropathy group(n=208)
PA(mg/L)	263.23± 12.43	214.24± 10.46*
CHO(mmol/L)	4.71± 1.31	4.68± 1.25
RBC(× 10 ¹² /L)	3.64± 0.69	3.72± 0.58
TG(mmol/L)	1.52± 0.83	1.51± 0.74
BUN(mmol/L)	18.64± 3.16	27.53± 2.13*
SCr(μmol/L)	974.23± 108.43	982.34± 113.43
CO ₂ CP(mmol/L)	22.35± 3.14	13.45± 3.23*
FBG(mmol/L)	4.87± 0.67	13.46± 2.51*

表 4 CRP、PTH、Alb 及 Hb 水平变化与尿毒症周围神经病变程度的相关性

Table 4 Correlation between the changes of CRP, PTH, ALB and Hb levels and the degree of uremic peripheral neuropathy

Indexs	Degree of peripheral neuropathy	
	r	P
CRP	0.586	0.013
PTH	0.579	0.018
Alb	-0.374	0.009
Hb	-0.484	0.023

血液透析频率低也会导致疾病的发生,随着患者疾病进程的发展也会导致尿毒症周围神经病变程度加重^[18]。此外,尿毒症性周围神经病变可能是由于微量元素及电解质等低分子尿毒素的毒素在体内聚集所导致,例如糖尿病肾病和高钾血症等^[19]。某些分子物质例如 PTH、瘦素和微球蛋白也会对周围神经具有毒性作用。且糖尿病患者会出现 PTH、瘦素和微球蛋白水平变化,同时糖尿病患者血液透析评率越低也会增加周围神经病变的发生率^[20];进一步结果显示:神经病变组患者的 CRP、PTH 水平明显高于非神经病变组,神经病变组患者的 Alb、Hb 水平明显低于非神经病变组,与张超等^[21]研究相似。张超等研究认为,甲状旁腺激素和慢性肾衰竭血透患者的营养具有明显相关性,周围神经病变患者 PTH 水平高于非神经病变组,这可能是因为,PTH 作为一种分子物质对于周围神经具有直接毒性作用^[22]。另外,人机体内酶活性如果受到抑制,主要与神经转酮酶及血液透析有关的并发症等因素有关。促红细胞生成素治疗能够改善尿毒症患者及其他原因所致的神经病变现象,这也是本研究病变组患者的 Hb 含量比较低的原因^[23]。CRP 在健康人体的血清中含量比较低,若组织受到肿瘤破坏、感染、炎症和损伤时血液中的 CRP 水平会积聚上升,因此尿毒症周围神经病变患者的 CRP 水平明显升高^[24,25]。患者发生尿毒症周围神经病变的时候,肾功能会受到损伤,导致营养不足,从而造成体内 Alb 水平降低,加重尿毒症周围神经病变发生^[26,27]。

本研究结果显示,两组患者 CHO、RBC、TG、SCr 水平对比无显著差异,神经病变组患者血清 PA、CO₂CP 水平低于非神经病变组,神经病变组患者血清 BUN、FBG 水平高于非神经病变组。另外,本研究还发现,除了研究中的 4 种指标的水平与尿毒症周围神经病变程度具有一定关系之外,患者血清 PA、CO₂CP、BUN、FBG 水平也与尿毒症周围神经病变的程度具有一定关系。有研究显示^[28],血清 BUN、FBG 水平变化是周围神经病变程度的独立危险因素,与本研究具有一定相关性;且发现 CRP、PTH 与尿毒症周围神经病变程度呈正相关,Alb、Hb 与尿毒症周围神经病变程度呈负相关。同时 CO₂CP 可反映血浆中呈结合状态的 CO₂,反映体内碱储备量,在尿毒症中具有一定的作用。临床上研究大多是关于尿毒症周围神经病变程度危险因素的研究,鲜少有应用相关指标与疾病发生的相关性所进行的研究,因此本文也是对以往研究的补充。最终也证明了,CRP、PTH、Alb 及 Hb 水平变化与尿毒症神经病变程度的相关性,进而临床可以考虑应用上述指标来对患者的病情发生发展进行诊断和预测^[29,30]。

综上所述,CRP、PTH、Alb 及 Hb 水平变化与尿毒症周围神经病变程度具有明显关系,可以用于判断尿毒症患者的周围神经病变程度。而且尿毒症周围神经病变患者多数原发疾病为糖尿病,血液透析频率较低,维持血液透析时间较长。

参考文献(References)

- [1] Puri I, Shirazi NM, Yap E, et al. Intestinal dialysis for conservative management of Uremia [J]. *Curr Opin Nephrol Hypertens*, 2020, 29 (1): 64-70
- [2] Cozzolino M. Uremic Patients with Increased Vascular Calcification Score Have Serum with High Calcific Potential: Role of Vascular Smooth Muscle Cell Osteoblastic Differentiation and Apoptosis[J]. *Blood Purif*, 2019, 48(2): 142-149
- [3] Raina R, Krishnappa V, Blaha T, et al. Atypical Hemolytic-Uremic Syndrome: An Update on Pathophysiology, Diagnosis, and Treatment [J]. *Ther Apher Dial*, 2019, 23(1): 4-21
- [4] Hamed SA. Neurologic conditions and disorders of uremic syndrome of chronic kidney disease: presentations, causes, and treatment strategies[J]. *Expert Rev Clin Pharmacol*, 2019, 12(1): 61-90
- [5] Thome T, Salyers ZR, Kumar RA, et al. Uremic metabolites impair skeletal muscle mitochondrial energetics through disruption of the electron transport system and matrix dehydrogenase activity[J]. *Am J Physiol Cell Physiol*, 2019, 317(4): C701-C713
- [6] Manrique-Caballero CL, Peerapornratana S, Formeck C, et al. Typical and Atypical Hemolytic Uremic Syndrome in the Critically Ill[J]. *Crit Care Clin*, 2020, 36(2): 333-356
- [7] Demeulenaere M, Devreese K, Vanbelleghem H, et al. Thrombomodulin and Endothelial Dysfunction: A Disease-Modifier Shared between Malignant Hypertension and Atypical Hemolytic Uremic Syndrome[J]. *Nephron*, 2018, 140(1): 63-73
- [8] Lim YJ, Sidor NA, Toniai NC, et al. Uremic Toxins in the Progression of Chronic Kidney Disease and Cardiovascular Disease: Mechanisms and Therapeutic Targets[J]. *Toxins (Basel)*, 2021, 13(2): 142
- [9] Arai Y, Shioji S, Tanaka H, et al. A Novel Uremic Score Reflecting Accumulation of Specific Uremic Toxins More Precisely Predicts One-Year Mortality after Hemodialysis Commencement: A Retrospective Cohort Study[J]. *Toxins (Basel)*, 2020, 12(10): 634
- [10] Moulton MN, Kay CL, Romain JT, et al. Clostridioides difficile-Associated Atypical Hemolytic-Uremic Syndrome Successfully Treated With Eculizumab: A Case Report and Literature Review[J]. *Crit Care Explor*, 2021, 3(7): e0475
- [11] Khalid M, Andreoli S. Extrarenal manifestations of the hemolytic uremic syndrome associated with Shiga toxin-producing Escherichia coli (STEC HUS)[J]. *Pediatr Nephrol*, 2019, 34(12): 2495-2507
- [12] Rapp N, Evenepoel P, Stenvinkel P, et al. Uremic Toxins and Vascular Calcification-Missing the Forest for All the Trees[J]. *Toxins (Basel)*, 2020, 12(10): 624
- [13] Wu CL, Tarng DC. Targeting Uremic Toxins to Prevent Peripheral Vascular Complications in Chronic Kidney Disease [J]. *Toxins (Basel)*, 2020, 12(12): 808
- [14] Sridharan M, Go RS, Willrich MAV. Atypical hemolytic uremic syndrome: Review of clinical presentation, diagnosis and management[J]. *J Immunol Methods*, 2018, 461(15): 15-22
- [15] Chao CT, Lin SH. Uremic Toxins and Frailty in Patients with Chronic Kidney Disease: A Molecular Insight[J]. *Int J Mol Sci*, 2021, 22(12): 6270
- [16] Zipfel PF, Wiech T, Stea ED, et al. CFHR Gene Variations Provide Insights in the Pathogenesis of the Kidney Diseases Atypical Hemolytic Uremic Syndrome and C3 Glomerulopathy [J]. *J Am Soc Nephrol*, 2020, 31(2): 241-256
- [17] Hayden MR, Tyagi SC, Kolb L, et al. Vascular ossification-calcification in metabolic syndrome, type 2 diabetes mellitus, chronic kidney disease, and calciphylaxis-calcific uremic arteriopathy: the emerging role of sodium thiosulfate [J]. *Cardiovasc Diabetol*, 2005, 4 (2): 4

- [18] Iddings AC, Shenoi AN, Morales Pozzo A, et al. Hemolytic uremic syndrome complicated by *Clostridium septicum* bacteremia and new-onset type 1 diabetes mellitus. Report of a case[J]. Clin Nephrol, 2017, 87(4): 207-211
- [19] Senthil B, Shanmugam S, Elangovan S, et al. Comparative study: Oral mucosal lesions, signs and symptoms in diabetes mellitus patients with end stage renal disease with analogous findings in diabetes mellitus patients with non-end stage renal disease[J]. Indian J Dent Res, 2017, 28(4): 406-412
- [20] Napolitano P, Capuano A, D'Apice L, et al. Arteriolopatia calcifica uremica: un'entità clinica rara? Report di tre casi [Calcific uremic arteriopathy (Calcyphilaxis): a rare disease? Report of three cases] [J]. G Ital Nefrol, 2015, 32(1): 323-326
- [21] 张超, 魏锦曦, 朱伦美, 等. 高通量血液透析对慢性肾衰竭尿毒症患者甲状腺激素, β_2 -微球蛋白, 血肌酐水平的影响[J]. 中国药业, 2021, 30(S2): 59-60
- [22] Bransky N, Iyer NR, Cannon SM, et al. Three Rare Concurrent Complications of Tertiary Hyperparathyroidism: Maxillary Brown Tumor, Uremic Leontiasis Ossea, and Hungry Bone Syndrome [J]. J Bone Metab, 2020, 27(3): 217-226
- [23] 骆强, 卿山林, 尧鹏. 血液透析, 高通量血液透析及血液透析滤过治疗尿毒症患者的效果及对血清炎症因子, 氧化应激指标的影响[J]. 临床误诊误治, 2020, 33(4): 6
- [24] Urban A, Kowalska D, Stasiłojć G, et al. Gain-of-Function Mutations R249C and S250C in Complement C2 Protein Increase C3 Deposition in the Presence of C-Reactive Protein[J]. Front Immunol, 2021, 12(2): 724361
- [25] Zhao JH, Zhu QS, Li YW, et al. Determinants of the intensity of uremic pruritus in patients receiving maintenance hemodialysis: A cross-sectional study[J]. PLoS One, 2021, 16(1): e0245370
- [26] Savira F, Magaye R, Hua Y, et al. Molecular mechanisms of protein-bound uremic toxin-mediated cardiac, renal and vascular effects: underpinning intracellular targets for cardiorenal syndrome therapy[J]. Toxicol Lett, 2019, 308(12): 34-49
- [27] 葛伟, 王海峰, 张长江, 等. 高通量血液透析联合肾衰宁颗粒对尿毒症患者钙磷代谢, 炎症反应以及营养状况的影响[J]. 现代生物医学进展, 2021, 21(9): 5
- [28] 田茜, 李宝新, 李娜, 等. 2型糖尿病肾脏病患者血清长正五聚蛋白3和 α -klotho蛋白水平的变化及意义[J]. 安徽医学, 2020, 41(8): 7
- [29] Tetta C, Gallieni M, Panichi V, et al. Vascular calcifications as a footprint of increased calcium load and chronic inflammation in uremic patients: a need for a neutral calcium balance during hemodialysis[J]. Int J Artif Organs, 2012, 25(1): 18-26
- [30] 段皓, 刘国亭, 孙宏武, 等. 红细胞沉降率, C反应蛋白, 糖尿病周围神经病变与糖尿病足溃疡严重程度的相关性研究[J]. 足踝外科电子杂志, 2021, 8(1): 22-26

(上接第474页)

- [21] 张旭红, 王少霞. Kt/V 评估血液透析患者高血压与透析充分性临床应用[J]. 中国保健营养, 2017, 27(28): 179-180
- [22] Mace ML, Olgaard K, Lewin E. New Aspects of the Kidney in the Regulation of Fibroblast Growth Factor 23 (FGF23) and Mineral Homeostasis[J]. Int J Mol Sci, 2020, 21(22): 8810
- [23] 王云. 活性维生素D联合西那卡塞治疗血液透析继发性甲旁亢的疗效[J]. 临床肾脏病杂志, 2018, 18(9): 564-567
- [24] 张颖, 王会苗, 陈超, 等. MHD患者血浆 ox-LDL、Scr 及 ALB 水平与生活质量及心功能关系的研究[J]. 国际检验医学杂志, 2017, 38(23): 3318-3320
- [25] Zhou J, Yang T. The efficacy of L-carnitine in improving malnutrition in patients on maintenance hemodialysis: a meta-analysis[J]. Biosci Rep, 2020, 40(6): BSR20201639
- [26] Chen X, Gui G, Ji W, et al. The relationship between obesity subtypes based on BMI and cardio-cerebrovascular disease [J]. Hypertens Res, 2019, 42(6): 912-919
- [27] Qiao W, Zhang X, Kan B, et al. Hypertension, BMI, and cardiovascular and cerebrovascular diseases [J]. Open Med (Wars), 2021, 16(1): 149-155
- [28] 程全周, 陈爱莲, 赵振凯. 心血管病高危人群中体质特点及与 BMI、IMT 的相关性分析[J]. 中西医结合心脑血管病杂志, 2020, 18(9): 1406-1408
- [29] Parati G, Stergiou GS, Dolan E, et al. Blood pressure variability: clinical relevance and application [J]. J Clin Hypertens (Greenwich), 2018, 20(7): 1133-1137
- [30] 于冠宇, 宋超, 冯翔, 等. 血压波动性对主动脉夹层发生发展影响的研究进展[J]. 现代生物医学进展, 2017, 17(4): 769-772