

doi: 10.13241/j.cnki.pmb.2021.22.039

硫酸镁联合小剂量阿司匹林治疗子痫前期效果及子宫动脉血流、胎盘 VEGF、MMP-9 表达影响研究*

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摘要 目的:探讨硫酸镁联合小剂量阿司匹林治疗子痫前期效果及子宫动脉血流、胎盘血管内皮生长因子(VEGF)、基质金属蛋白酶-9(MMP-9)表达影响研究。**方法:**选择 2019 年 6 月-2021 年 1 月在我院接受治疗的 125 例子痫前期患者,采用随机数表法分为试验组(n=63)和对照组(n=62)。对照组给硫酸镁治疗,试验组在对照组的基础上联合小剂量阿司匹林治疗。比较两组临床疗效、子宫动脉血流、胎盘 VEGF、MMP-9、血压水平变化情况及妊娠不良结局发生情况。**结果:**治疗后,两组总有效率比较差异显著($P<0.05$);治疗前,试验组和对照组子宫动脉指标水平比较无显著差异;治疗后,试验组和对照组 RI、PI 及 S/D 水平均随着时间的推移而升高,且试验组均高于对照组,差异显著($P<0.05$);治疗前,试验组和对照组胎盘 VEGF、MMP-9 水平比较无显著差异;治疗后,试验组和对照组胎盘 VEGF、MMP-9 水平均随着时间的推移而升高,且试验组均高于对照组,差异显著($P<0.05$);治疗前,试验组和对照组血压水平比较无显著差异;治疗后,试验组和对照组血压水平均随着时间的推移而降低,且试验组均低于对照组,差异显著($P<0.05$);试验组胎儿窘迫、宫缩乏力及新生儿窒息发生率均显著低于对照组,差异显著($P<0.05$)。**结论:**在子痫前期中应用硫酸镁联合小剂量阿司匹林治疗疗效显著,可有效改善患者子宫动脉血流、胎盘 VEGF、MMP-9 水平。

关键词:硫酸镁;小剂量;阿司匹林;子痫前期;子宫动脉血流;血管内皮生长因子;基质金属蛋白酶-9

中图分类号:R714.244 **文献标识码:**A **文章编号:**1673-6273(2021)22-4387-05

Effect of Magnesium Sulfate Combined with Low-dose Aspirin in the Treatment of Preeclampsia and Influence of Uterine Artery Blood Flow, Placental VEGF and MMP-9 Expression*

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ABSTRACT Objective: To study Effect of magnesium sulfate combined with low-dose aspirin in the treatment of preeclampsia and influence of uterine artery blood flow, placental Vascular Endothelial growth factor (VEGF), Matrix Metalloproteinase-9 (MMP-9) expression. **Methods:** 125 patients with preeclampsia who received treatment in our hospital from June 2019 to January 2021 were selected and divided into experimental group (n=63) and control group (n=62) by random number table method. The control group was treated with magnesium sulfate, and the experimental group was treated with low-dose aspirin in addition to the control group. Clinical efficacy, uterine artery blood flow, placental VEGF, MMP-9, blood pressure and adverse pregnancy outcomes were compared between the two groups. **Results:** After treatment, the total effective rate of the two groups was significantly different ($P<0.05$); Before treatment, there was no significant difference in uterine artery indexes between experimental group and control group. After treatment, RI, PI and S/D levels in experimental group and control group were increased over time, and the experimental group was higher than the control group, the difference was significant ($P<0.05$); Before treatment, there were no significant differences in placental VEGF and MMP-9 levels between experimental group and control group. After treatment, the levels of placental VEGF and MMP-9 in experimental group and control group were increased over time, and the levels in experimental group were higher than those in control group, the difference was significant ($P<0.05$); Before treatment, there was no significant difference in blood pressure between the experimental group and the control group. After treatment, the blood pressure level of both experimental group and control group decreased over time, and the experimental group was lower than the control group, the difference was significant ($P<0.05$); The incidence of fetal distress, uterine weakness and neonatal asphyxia in experimental group were significantly lower than those in control group ($P<0.05$). **Conclusion:** Magnesium sulfate combined with low-dose aspirin is effective in the treatment of preeclampsia, which can effectively improve the levels of uterine artery blood flow, placental VEGF and MMP-9.

* 基金项目:山东省中医药科技发展计划项目(2019-0590)

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(收稿日期:2021-04-28 接受日期:2021-05-23)

Key words: Magnesium sulfate; Small doses; Aspirin; Preeclampsia; Uterine artery blood flow; Vascular endothelial growth factor; Matrix metalloproteinase-9

Chinese Library Classification(CLC): R714.244 **Document code:** A

Article ID: 1673-6273(2021)22-4387-05

前言

子痫前期是妊娠期常见疾病,多发生于 20 周以后,主要表现为血压升高及头晕恶心等症状,随着疾病的进展可出现多器官功能障碍,是孕妇及围产儿死亡的主要原因,严重威胁患者的生命^[1-3]。据调查显示,国外子痫前期发病率为 7%-12%,我国发病率为 9.4%^[4]。有研究显示,胎盘 VEGF、MMP-9 在子痫前期中表达较低,参与了疾病的发展,可作为临床治疗疾病的标志物^[5]。临床多给予药物治疗缓解孕妇症状,延长产妇产前周期,提高围生儿存活率。硫酸镁是治疗子痫前期的常用药物,可控制患者血压,缓解临床症状,但单独治疗效果不佳,需联合其他药物提高临床疗效^[6]。近年来,有学者发现小剂量阿司匹林对子痫前期高危孕妇有预防作用^[7]。阿司匹林是具有抗凝活性的药物,能通过抑制前列腺素的合成,调节前列腺素,扩张血管,从而降低血压,但目前缺乏关于硫酸镁联合小剂量阿司匹林对胎盘 VEGF、MMP-9 影响的相关报道^[8,9]。因此,本研究旨在探讨硫酸镁联合小剂量阿司匹林治疗子痫前期效果,并分析其对子宫动脉血流、胎盘 VEGF、MMP-9 表达影响。

1 资料与方法

1.1 一般资料

选择 2019 年 6 月 -2021 年 1 月在我院接受治疗的 125 例子痫前期患者,采用随机数表法分为 2 组,试验组 63 例,年龄 21~34 岁,平均(26.64±3.58)岁,体重 53~74 Kg,平均(61.21±4.25)Kg,孕周 23~38 周,平均(29.87±1.23)周。对照组 62 例,年龄 20~32 岁,平均(26.58±3.57)岁,体重 51~73 Kg,平均(61.15±4.21)Kg,孕周 23~37 周,平均(29.84±1.20)周。两组一般资料无显著差异($P>0.05$),可比较。

参照《妊娠期高血压疾病诊治指南》^[10]中标准:新发蛋白

尿;蛋白尿突然增加;血压增高。

纳入标准:(1)符合相关标准确诊;(2)无生命危险;(3)无恶性肿瘤;(4)知情同意。排除标准:(1)免疫低下者;(2)中途退出者;(3)严重肝肾疾病者;(4)抗凝、激素治疗者;(5)恶性肿瘤;(6)妊娠期妇女;(7)全身感染者;(8)合并卵巢肿瘤者;(9)合并血液系统疾病者。

1.2 方法

对照组给予硫酸镁(规格:10 mL:2.5 g;生产厂家:北京益民药业有限公司;国药准字:H11020318)20 g,加入葡萄糖静脉滴注,1 d 1 次。试验组在对照组的基础上给予小剂量阿司匹林:(规格:100 mg,厂家:拜耳医药保健有限公司,国药准字 H20160684)50 mg,口服,每日 1 次。

1.3 观察指标

采用蛋白-过氧化物酶连接法和免疫组化链霉素抗生物检测法测定胎盘 VEGF、MMP-9 水平,试剂盒购于英国 Abcam 公司;采用彩色多普勒超声检测子宫动脉血流;记录患者血压变化情况;记录妊娠不良结局。

疗效评定标准:(1)显效:SBP 平均下降 15 mmHg 以上,24 h 尿蛋白定量持续减少;(2)有效:SBP<140 mmHg,临床症状有所改善;(3)无效:临床症状无明显改善甚至加重。

1.4 统计学分析

以 spss22.0 软件包处理,符合正态分布计量资料用均数±标准差($\bar{x} \pm s$)表示,组间比较使用独立样本 t 检验,计数资料以率表示, χ^2 检验, $P<0.05$ 表示差异具有统计学意义。

2 结果

2.1 两组临床效果评价

治疗后,两组总有效率比较差异显著($P<0.05$)见表 1。

表 1 两组临床效果评价[n(%)]
Table 1 Clinical effect evaluation of the two groups[n(%)]

Groups	n	Excellent	valid	Invalid	Total effective rate
Experimental group	63	35(55.56)	22(34.92)	6(9.52)	57(90.48)
Control group	62	25(40.32)	19(30.65)	18(29.03)	44(70.97)
χ^2 value					7.666
P value					0.006

2.2 两组子宫动脉指标水平比较

治疗前,试验组和对照组子宫动脉指标水平比较无显著差异;治疗后,试验组和对照组 RI、PI 及 S/D 水平均随着时间的推移而升高,且试验组均高于对照组,差异显著($P<0.05$),见表 2。

2.3 两组胎盘 VEGF、MMP-9 水平比较

治疗前,试验组和对照组胎盘 VEGF、MMP-9 水平比较无显著差异;治疗后,试验组和对照组胎盘 VEGF、MMP-9 水平

均随着时间的推移而升高,且试验组均高于对照组,差异显著($P<0.05$),见表 3。

2.4 两组血压水平比较

治疗前,试验组和对照组血压水平比较无显著差异;治疗后,试验组和对照组血压水平均随着时间的推移而降低,且试验组均低于对照组,差异显著($P<0.05$),见表 4。

表 2 两组子宫动脉指标水平比较($\bar{x} \pm s$)Table 2 Comparison of uterine artery indexes between the two groups($\bar{x} \pm s$)

Groups	n	RI		PI		S/D	
		Before the intervention	After the intervention	Before the intervention	After the intervention	Before the intervention	After the intervention
Experimental group	63	0.50± 0.02	0.59± 0.08	1.32± 0.32	1.59± 0.24	0.72± 0.12	0.86± 0.41
Control group	62	0.51± 0.07	0.52± 0.02	1.34± 0.33	1.39± 0.35	0.73± 0.13	0.75± 0.12
t value		1.089	6.687	0.344	3.731	0.447	2.029
P value		0.278	0.000	0.731	0.000	0.656	0.045

表 3 两组胎盘 VEGF、MMP-9 水平比较($\bar{x} \pm s$)Table 3 Comparison of placental VEGF and MMP-9 levels between the two groups($\bar{x} \pm s$)

Groups	n	VEGF		MMP-9	
		Before the intervention	After the intervention	Before the intervention	After the intervention
Experimental group	63	105.26± 7.45	133.41± 6.09	0.15± 0.08	0.28± 0.11
Control group	62	106.05± 7.51	115.26± 7.65	0.16± 0.07	0.20± 0.12
t value		0.590	14.687	0.743	3.886
P value		0.556	0.000	0.459	0.000

表 4 两组血压水平比较($\bar{x} \pm s$, mmHg)Table 4 Comparison of blood pressure between the two groups($\bar{x} \pm s$, mmHg)

Groups	n	Systolic blood pressure		Diastolic blood pressure	
		Before the intervention	After the intervention	Before the intervention	After the intervention
Experimental group	63	163.51± 10.61	131.52± 7.51	98.68± 6.61	81.05± 5.61
Control group	62	164.05± 11.16	147.56± 7.68	98.69± 6.73	89.96± 6.58
t value		0.277	11.806	0.008	8.151
P value		0.782	0.000	0.993	0.000

2.5 两组妊娠不良结局比较

于对照组,差异显著($P < 0.05$),见表 5。

试验组胎儿窘迫、宫缩乏力及新生儿窒息发生率均显著低

表 5 两组妊娠不良结局比较[n(%)]

Table 5 Comparison of adverse pregnancy outcomes between the two groups[n(%)]

Groups	n	Fetal distress	Contractions are weak	Neonatal asphyxia
Experimental group	63	7(11.11)	7(11.11)	5(7.94)
Control group	62	18(29.03)	20(32.26)	19(30.65)
χ^2 value		6.272	8.252	10.387
P value		0.012	0.004	0.001

3 讨论

子痫前期是孕期常见并发症之一,具有起病急、病情快等特点,患者主要表现为血压持续升高,对孕妇的神经系统、内分泌系统等造成损伤,是引发新生儿死亡的一个重要因素^[11]。其发病机制较为复杂,可能是由于胎盘滋养细胞缺血,导致血管内皮损伤,使胎盘释放的多种因子发生改变,从而引起子痫前期一系列临床表现^[12]。据调查显示,我国子痫前期围产儿死亡率高达 10%以上,是其他妊娠疾病的 4 倍以上,因此,给予患者有效治疗对改善不良妊娠结局具有重要意义^[13]。

子痫前期发病较为复杂,单一药物治疗效果不佳,较多学者提高联合用药的治疗方案^[14]。硫酸镁属于神经系统抑制类药物,具有镇静、抗痉挛等作用,能抑制神经-肌肉接头处的乙酰胆碱的释放,缓解血管痉挛,扩张外周血管,降低血压,能较好缓解子痫前期的临床症状^[15,16]。阿司匹林是抗凝药物,能抑制前列腺素的合成、血小板聚集,调节前列腺素与血栓素比值,起到抗血小板凝集和血栓形成,从而改善血液高凝状态^[17]。国内外较多学者对阿司匹林预防用药的剂量尚有争议,但已明确了小剂量阿司匹林对子痫前期有预防的效果,能降低子痫前期的发病率^[18-20]。本研究结果显示,联合小剂量阿司匹林治疗的患者总

有效率较单独使用硫酸镁治疗的患者高,血压水平也低于对照组,且胎儿窘迫、宫缩乏力及新生儿窒息发生率也低于对照组,结果提示,硫酸镁联合小剂量阿司匹林能提高子痫前期的临床疗效,降低患者血压,减少不良妊娠结局。Katmini K^[21]等研究也显示,硫酸镁联合阿司匹林治疗妊娠期高血压效果显著,能降低患者血压及剖宫产率,且胎儿窘迫、早产等机率也有所降低,对改善母婴结局有一定影响。分析其原因可能是因为硫酸镁可抑制中枢神经系统,扩张外周血管,降低血压;阿司匹林能够抑制血小板内环氧化酶,减少血小板的聚集,两种药物联合治疗降低患者血压,提高临床疗效。

近年来,子宫动脉血流在子痫前期发生中得到广泛关注,子痫前期患者病理改变可导致患者血管内皮细胞功能发生障碍,使血流灌注减少,滋养细胞侵袭力减弱,胎盘呈现浅着床状态,导致胎盘血流阻力增加,引起灌注不足,使胎盘缺血缺氧,而子宫动脉血流可通过 RI、PI 及 S/D 评估^[22-24]。其中 RI 反映血流阻力的大小,还能反映舒张末期血流是否存在,当 RI 大于 1 时,说明舒张期出现反向血流;PI 值主要反映收缩期峰值流速和舒张末期流速,同时还能反映整个周期的平均流速,其水平高时说明血管阻力高;S/D 值反映收缩期峰值流速和舒张末期流速^[25]。本研究结果显示,治疗后患者 RI、PI 及 S/D 水平明显降低,且硫酸镁联合小剂量阿司匹林治疗的患者低于对照组,结果提示,硫酸镁联合小剂量阿司匹林可增加胎盘的灌注,降低患者子宫动脉的血流阻力。相关研究显示,子痫前期的发生可使滋养胎盘的细胞分化异常,深入浸润滋养胎盘,使肌层的小动脉不能进行生理重铸过程,从而导致患者胎盘病理发生变化^[26]。VEGF 属于血管内皮生长因子中的一种,可促进相关血管的生长、增殖,使细胞深入基层,修复受损的血管,当其水平降低时,其血管增殖相对较差,血管损伤修复功能受损,胎盘功能状态也较差^[27]。MMP 是滋养细胞层在浸润过程中分泌的一类蛋白水解酶,MMP-9 是滋养细胞分泌的最有效的蛋白水解酶,对内膜细胞外基质具有消化作用,其分泌和滋养细胞侵袭能力相关^[28]。本研究观察在不同治疗方案中胎盘 VEGF、MMP-9 水平变化,结果显示,治疗后,患者胎盘 VEGF、MMP-9 水平明显上升,且硫酸镁联合小剂量阿司匹林治疗的患者高于对照组,结果提示,硫酸镁联合小剂量阿司匹林在子痫前期的治疗中可控制血管损伤。分析其原因可能是因为子痫前期可导致患者体内活化的血小板使得扩张,使 VEGF、MMP-9 水平异常降低,而硫酸镁是子痫前期治疗的首选药物,能减轻心脏负荷、促进血液循环,有效控制患者血压;而小剂量阿司匹林可通过中和环氧酶的活性来发挥作用,抑制血栓素的形成、减少血小板聚集,使血管舒张、血流增多,从而改善患者子宫动脉血流、胎盘 VEGF、MMP-9 水平。

综上所述,在子痫前期中应用硫酸镁联合小剂量阿司匹林治疗疗效显著,可有效改善患者子宫动脉血流、胎盘 VEGF、MMP-9 水平。

参考文献(References)

- [1] Ambusaidi A, Rogado N L, Hashmi Z A. Preeclampsia Masquerading as Catatonic Relapse in Schizophrenia: A Case Report[J]. Middle East Journal of Psychiatry and Alzheimers, 2018, 9(2): 21-24
- [2] Alrahmani L, Willrich M. The Complement Alternative Pathway and Preeclampsia[J]. Current Hypertension Reports, 2018, 20(5): 40
- [3] Qu H M, Qu L P, Li X Y, et al. Overexpressed HO-1 is associated with reduced STAT3 activation in preeclampsia placenta and inhibits STAT3 phosphorylation in placental JEG-3 cells under hypoxia[J]. Archives of Medical Science Ams, 2018, 14(3): 597
- [4] Nordqvist M, Jacobsson B, Brantsæter A L, et al. Timing of probiotic milk consumption during pregnancy and effects on the incidence of preeclampsia and preterm delivery: a prospective observational cohort study in Norway[J]. Bmj Open, 2018, 8(1): e018021
- [5] Sheng C, Zhao Y, Zhu L. Down-regulation of EDN1 gene expression by circulating miR-206 is associated with risk of preeclampsia [J]. Medicine, 2020, 99(22): e20319
- [6] Granger J P, Spradley F T, Bakrania B A. The Endothelin System: A Critical Player in the Pathophysiology of Preeclampsia [J]. Current Hypertension Reports, 2018, 20(4): 32
- [7] Rajaa A, Louise B, Daniel V, et al. Oxidative Stress in Preeclampsia and Placental Diseases [J]. International Journal of Molecular Ence, 2018, 19(5): 1496
- [8] Li B, Yang H, Zhang W, et al. Fatty acid-binding protein 4 predicts gestational hypertension and preeclampsia in women with gestational diabetes mellitus[J]. Plos One, 2018, 13(2): e0192347
- [9] Li G, Ma L, Lu H, et al. Transactivation of Met signalling by semaphorin4D in human placenta: implications for the pathogenesis of preeclampsia[J]. Journal of Hypertension, 2018, 36(11): 2215-2225
- [10] Gestation hypertension Group of Obstetrics and Gynecology Society of Chinese Medical Association. Guidelines for diagnosis and treatment of hypertension during pregnancy (2020)[J]. Chinese Journal of Obstetrics and Gynecology, 2020, 55(4): 277-238
- [11] Huppertz B. The Critical Role of Abnormal Trophoblast Development in the Etiology of Preeclampsia [J]. Current Pharmaceutical Biotechnology, 2018, 19(10): 771-780
- [12] Liong S, Barker G, Lappas M. Bromodomain protein BRD4 is increased in human placentas from women with early-onset preeclampsia[J]. Reproduction, 2018, 155(6): 573-582
- [13] J Müller-Deile, Schrder P, Beverly-Taggs L, et al. Overexpression of preeclampsia induced microRNA-26a-5p leads to proteinuria in zebrafish[J]. Scientific Reports, 2018, 8(1): 3621
- [14] D Mrema, Lie R T, Østbye Truls, et al. The association between pre pregnancy body mass index and risk of preeclampsia: a registry based study from Tanzania[J]. BMC Pregnancy & Childbirth, 2018, 18(1): 56
- [15] Benschop L, Duvekot J J, Versmissen J, et al. Blood Pressure Profile 1 Year After Severe Preeclampsia Novelty and Significance[J]. Hypertension, 2018, 71(3): 491
- [16] Li J L, Li R, Gao Y, et al. LncRNA CCAT1 promotes the progression of preeclampsia by regulating CDK4[J]. European review for medical and pharmacological sciences, 2018, 22(5): 1216-1223
- [17] Akbari S, Khodadadi B, Ahmadi S, et al. Association of vitamin D level and vitamin D deficiency with risk of preeclampsia: A systematic review and updated meta-analysis[J]. Taiwanese Journal of Obstetrics

- and Gynecology, 2018, 57(2): 241-247
- [18] Leavey K, Wilson S L, Bainbridge S A, et al. Epigenetic regulation of placental gene expression in transcriptional subtypes of preeclampsia [J]. Clinical Epigenetics, 2018, 10(1): 28
- [19] Quan L M, Xu Q L, Zhang G Q, et al. An analysis of the risk factors of preeclampsia and prediction based on combined biochemical indexes [J]. The Kaohsiung journal of medical sciences, 2018, 34(2): 109-112
- [20] Dorien R, Liu C C, Xu X, et al. Celecoxib restores angiogenic factor expression at the maternal-fetal interface in the BPH/5 mouse model of preeclampsia[J]. Physiological Genomics, 2018, 50(5): 385-392
- [21] Katmini K, Murti B, Td O, et al. Path Analysis on the Effect of Social Capital on the Empowerment of Pregnant Women in Preeclampsia Prevention Using Precede-Proceede in Kediri East Java, Indonesia [J]. Asian Journal of Pharmaceutical & Clinical Research, 2018, 11(4): 271
- [22] Ameh E A, Mshelbwala P M, Sabiu L . Maternal and Neonatal Outcomes of Induction of Labor Compared with Planned Cesarean Delivery in Women with Preeclampsia at 34 Weeks' Gestation or Longer [J]. Am J Perinatol, 2018, 35(01): 095-102
- [23] Zhang X H, Zhang H Y, Lu S, et al. MMP-14 aggravates onset of severe preeclampsia by mediating soluble endoglin release[J]. European Review for Medical & Pharmacological Sciences, 2018, 22(5): 1209
- [24] IA Brussé, Berg C, Duvekot J J, et al. Visual evoked potentials in women with and without preeclampsia during pregnancy and postpartum[J]. Journal of Hypertension, 2018, 36(2): 1
- [25] Ferhi F, Khelifi A, Hachani F, et al. Ultrasound assessment of visual loss during severe preeclampsia: a case report [J]. Critical Ultrasound Journal, 2018, 10(1): 6
- [26] You S H, Cheng P J, Ting-Ting C, et al. Population-based trends and risk factors of early- and late-onset preeclampsia in Taiwan 2001-2014[J]. Bmc Pregnancy & Childbirth, 2018, 18(1): 199
- [27] Valero L, Alhareth K, Gil S, et al. Nanomedicine as a potential approach to empower the new strategies for the treatment of preeclampsia[J]. Drug Discovery Today, 2018, 23(5): 1099-1107
- [28] Contini C, Jansen M, Kötzig B, et al. Lipoprotein turnover and possible remnant accumulation in preeclampsia: insights from the Freiburg Preeclampsia H.E.L.P.-apheresis study[J]. Lipids in Health & Disease, 2018, 17(1): 49

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- [28] Jebali H, Ghabi H, Mami I, et al. Prognostic value of the Oxford classification and the Oxford score in IgA nephropathy: A Tunisian study [J]. Saudi journal of kidney diseases and transplantation: an official publication of the Saudi Center for Organ Transplantation, Saudi Arabia, 2020, 31(6): 1366
- [29] Pei G Q, Qin A Y, Wang S Q, et al. Effect of serum IgA/C3 and glomerular C3 staining on clinical prognosis in patients with IgA nephropathy[J]. Zhonghua Yi Xue Za Zhi, 2020, 100(30): 2372-2377
- [30] Chen X, Molyneux K, Barratt J. SAT-386 Galactosylation of Serum IgA Antibodies Against Alimentary and Systemic Antigens in IgA Nephropathy in Ethnic Groups [J]. Kidney International Reports, 2020, 5(3): S161-S162
- [31] Yu A H, Ji W M, Ha M A, et al. The Impact of Obesity on the Severity of Clinicopathologic Parameters in Patients with IgA Nephropathy[J]. Journal of Clinical Medicine, 2020, 9(9): 2824
- [32] Jiang X, Xu Z, Du Y, et al. Bioinformatics analysis reveals novel hub gene pathways associated with IgA nephropathy[J]. European Journal of Medical Research, 2020, 25(1): 40
- [33] He J W, Zhou X J, Lv J C, et al. Perspectives on how mucosal immune responses, infections and gut microbiome shape IgA nephropathy and future therapies[J]. Theranostics, 2020, 10(25): 11462-11478
- [34] Tang F, Hu H, Xu R, et al. Association Between Serum IgG Concentrations and Prognosis in IgA Nephropathy[J]. Iranian Journal of Kidney Diseases, 2020, 14(6): 454-462
- [35] Lingaraj U, Aralapuram K, Chikkanayakanhalli S, et al. Successful treatment of a patient with posttransplant IgA nephropathy with targeted release formulation of budesonide [J]. Saudi journal of kidney diseases and transplantation: an official publication of the Saudi Center for Organ Transplantation, Saudi Arabia, 2020, 31(2): 521