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IgA 肾病合并高尿酸血症患者的危险因素分析 *

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摘要 目的：探讨 IgA 肾病合并高尿酸血症患者的危险因素。**方法：**回顾性分析 2018 年 1 月至 2021 年 1 月于我院进行治疗的 IgA 肾病患者 149 例的病理资料，根据高尿酸血症发生情况分为高尿酸血症组（n=65），正常尿酸组（n=84）。比较两组病理特征，收集患者年龄、性别、BMI、性别、高血压、血肌酐、尿素氮、血白蛋白、血胆固醇、甘油三酯、24 h 尿蛋白定量、IL-6、IL-1 及胱抑素 C 及 CRP 等资料，分析高尿酸血症发生的危险因素。**结果：**两组患者年龄、BMI、CRP 差异无统计学意义（*P*>0.05）；性别、高血压、血肌酐、尿素氮、血白蛋白、血胆固醇、甘油三酯、24 h 尿蛋白定量、IL-6、IL-1 及胱抑素 C 与 IgA 肾病患者发生高尿酸血症相关（*P*<0.05）；高尿酸组患者球性硬化、节段硬化、新月体形成、小管萎缩、间质炎性浸润及间质纤维化发生率均显著高于正常尿酸组，差异显著（*P*<0.05）；多因素非条件 Logistic 分析显示，性别、高血压、血肌酐、尿素氮、血白蛋白、血胆固醇、甘油三酯、24 h 尿蛋白定量、IL-6、IL-1 及胱抑素 C 均是 IgA 肾病患者发生高尿酸血症的独立危险因素（*P*<0.05）。**结论：**患者性别、高血压、血肌酐、尿素氮、血白蛋白、血胆固醇、甘油三酯、24 h 尿蛋白定量、IL-6、IL-1 及胱抑素 C 均是 IgA 肾病患者发生高尿酸血症的危险因素，临床上对于具有危险因素的患者引起重视，提高治疗效果。

关键词：IgA；肾病；高尿酸血症；危险因素

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Analysis of the Risk Factors in IgA Nephropathy with Hyperuricemia*

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ABSTRACT Objective: To study Analysis of theRisk factors in IgA nephropathy with hyperuricemia. **Methods:** The pathological data of 149 patients with IgA nephropathy treated in our hospital from January 2018 to January 2021 were retrospectively analyzed. According to the occurrence of hyperuricemia, they were divided into hyperuricemia group (n=65) and normal uric acid group (n=84). The pathological characteristics of the two groups were compared. The data of patients' age, gender, BMI, gender, hypertension, serum creatinine, urea nitrogen, serum albumin, blood cholesterol, triglyceride, 24 h urine protein, IL-6, IL-1, cystatin C and CRP were collected to analyze the risk factors of hyperuricemia. **Results:** There were no significant differences in age, BMI and CRP between 2 groups (*P*>0.05). Gender, hypertension, serum creatinine, urea nitrogen, serum albumin, serum cholesterol, triglyceride, 24 h urinary protein, IL-6, IL-1 and cystatin C were correlated with hyperuricemia in patients with IgA nephropathy (*P*<0.05). The incidence of bulbar sclerosis, segmental sclerosis, crescent formation, tubule atrophy, interstitial inflammatory infiltration and interstitial fibrosis in high uric acid group was significantly higher than that in normal uric acid group, the difference was significant(*P*<0.05); Multivariate Logistic analysis showed that gender, hypertension, serum creatinine, urea nitrogen, serum albumin, blood cholesterol, triglyceride, 24 h urinary protein level, IL-6, IL-1 and cystatin C were all independent risk factors for hyperuricemia in patients with IgA nephropathy (*P*<0.05). **Conclusion:** Patients' gender, hypertension, serum creatinine, urea nitrogen, serum albumin, blood cholesterol, triglyceride, 24h urine protein, IL-6, IL-1 and cystatin C are all risk factors for hyperuricemia in patients with IgA nephropathy. Clinical attention should be paid to patients with risk factors to improve the treatment effect.

Key words: IgA; Kidney disease; Hyperuricemia; Risk factors

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前言

IgA 肾病是常见的原发性肾小球疾病，临床表现为反复发作性肉眼血尿，还伴有不同程度蛋白尿，是导致终末期肾病的

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主要原因，有研究显示，15%~40%的患者在 20 年内进展为终末期肾脏病，严重威胁人们的生命[1,2]。因此，及时发现疾病的危险因素可减少疾病的进展。该病的发病原因较多，相关研究证实，高血压、血胆固醇、肾小球硬化等已成为 IgA 肾病进展的危险因素[3]。近年来有研究认为高尿酸血症与 IgA 肾病的进展有关[4]。随着人们生活水平的提高，高尿酸血症的发病率逐渐上升，高尿酸增高可导致肾脏损伤，进一步导致肾小管及肾间质纤维化[5-8]。有研究显示，尿酸与高血压、心血管疾病等密切相关[9,10]。近年来，高尿酸血症作为新型危险因素引起广大学者的关注。本研究旨在分析 IgA 肾病合并高尿酸血症患者的危险因素，为预测疾病的发生提供依据。

1 资料与方法

1.1 一般资料

回顾性分析 2018 年 1 月至 2021 年 1 月于我院进行治疗的 IgA 肾病患者 149 例的病理资料。我院伦理委员会批准本研究。据纳入标准：(1)病理学活检确诊；(2)血尿为尿沉渣每高倍镜视野 RBC ＞3 个。排除标准：(1)有严重感染，其他器官严重疾病；(2)神经精神类疾病患者；(3)临床资料不完整患者。根据高尿酸血症发生情况分为高尿酸血症组和正常尿酸组，高尿酸血症组中男 35 例，女 30 例，年龄 30~67 岁，平均（42.38± 11.05）岁；正常尿酸组男 28 例，女 56 例，年龄 31~66 岁，平均（42.41± 10.89）岁。两组患者一般资料无显著差异(P>0.05)，存在可比性。

1.2 方法

采用自制调查问卷收集患者临床资料，采用单因素及多因素 Logistic 回归分析 IgA 肾病合并高尿酸血症患者发生的危险因素。

1.3 观察指标

对所有患者资料进行整理，包括病理特征，年龄、性别、BMI、性别、高血压、血肌酐、尿素氮、血白蛋白、血胆固醇、甘油三酯、24 h 尿蛋白定量、IL-6、IL-1 及胱抑素 C 及 CRP 等资料；分析 IgA 肾病患者发生高尿酸血症的危险因素。

1.4 统计学分析

以 SPSS24.0 软件包处理，计量资料表示采用均数± 标准差($\bar{x}$± s)，组间比较用独立样本 t 检验，计数资料率表示，x^2 检验，将 IgA 肾病患者发生高尿酸血症单因素分析结果中具有统计学差异的变量带入 Logistic 回归模型进行危险因素的多元分析，P＜0.05 表示差异具有统计学意义。

2 结果

2.1 IgA 肾病合并高尿酸血症患者和正常尿酸患者临床特征比较

两组患者年龄、BMI、CRP 差异无统计学意义(P>0.05)；性别、高血压、血肌酐、尿素氮、血白蛋白、血胆固醇、甘油三酯、24 h 尿蛋白定量、IL-6、IL-1 及胱抑素 C 与 IgA 肾病患者发生高尿酸血症相关(P<0.05)见表 1。

表 1 IgA 肾病合并高尿酸血症患者和正常尿酸患者临床特征比较

Table 1 Comparison of clinical features between IgA nephropathy patients with hyperuricemia and normal uric acid patients

Item	High uric acid group(n=65)	Normal uric acid group(84)	Statistics	P value
Age (years)	42.38± 11.05	42.41± 10.89	0.017	0.987
Gender (male/female)	35/30	28/56	6.318	0.012
BMI(kg/m^2)	24.53± 9.31	22.31± 10.14	1.373	0.172
Hypertension	30(46.15)	18(21.43)	10.259	0.001
Serum creatinine(μmol/L)	126.45± 38.12	93.41± 40.12	5.094	0.000
Urea nitrogen(mmol/L)	9.87± 4.41	5.51± 2.51	7.611	0.000
Blood albumin(g/L)	30.51± 10.41	34.88± 8.24	2.861	0.005
Blood cholesterol(mmol/L)	5.73± 1.24	4.69± 1.68	4.185	0.000
TG(mmol/L)	2.17± 0.63	1.74± 0.26	5.667	0.000
24 h urine protein quantification(g/L)	1.70± 0.46	1.34± 0.51	4.458	0.000
IL-6(pg/mL)	59.84± 13.81	52.53± 14.17	3.158	0.002
CRP(mg/mL)	11.65± 2.56	10.59± 4.62	1.662	0.099
IL-1(pg/mL)	7.51± 1.18	6.98± 1.83	2.030	0.044
Cystatin C(mg/L)	2.41± 0.67	1.65± 0.13	10.162	0.000

2.2 IgA 肾病合并高尿酸血症患者和正常尿酸患病理特征比较

高尿酸组患者球性硬化、节段硬化、新月体形成、小管萎缩、间质炎性浸润及间质纤维化发生率均显著高于正常尿酸组，差异显著(P<0.05)。见表 2。

2.3 IgA 肾病发生高尿酸血症的危险因素 Logistic 分析

将以上有统计学意义的的因素作为自变量，以发生高尿酸血症为因变量，进行多因素非条件 Logistic 分析。性别、高血压、血肌酐、尿素氮、血白蛋白、血胆固醇、甘油三酯、24 h 尿蛋白定量、IL-6、IL-1 及胱抑素 C 均是 IgA 肾病患者发生高尿酸血症的独立危险因素(P<0.05)。见表 3。

表 2 IgA 肾病合并高尿酸血症患者和正常尿酸患病理特征比较[n(%)]

Table 2 Comparison of pathological characteristics between IgA nephropathy patients with hyperuricemia and normal uric acid[n(%)]

Item	High uric acid group(n=65)	Normal uric acid group(84)	x^2 value	*P* value
The ball sclerosis	51(78.46)	49(58.33)	6.727	0.009
Segmental sclerosis	33(50.77)	26(30.95)	6.017	0.014
Crescent formation	32(49.23)	25(29.76)	5.880	0.015
Tubular atrophy	54(83.08)	54(64.29)	6.488	0.011
Interstitial inflammatory infiltration	52(80.00)	51(60.71)	6.386	0.012
Interstitial fibrosis	36(55.38)	31(36.90)	5.057	0.025

表 3 IgA 肾病发生高尿酸血症的危险因素 Logistic 分析

Table 3 Logistic analysis of risk factors for hyperuricemia in IgA nephropathy

Variable	Regression coefficient	Wald x^2	*P* value	ORvalue	95%CI
Gender	0.998	9.586	0.000	2.314	1.396~3.235
Hypertension	0.897	10.115	0.011	1.899	1.528~3.037
Serum creatinine	1.051	11.233	0.029	0.724	0.458~0.879
Urea nitrogen	1.001	11.441	0.027	0.855	0.689~1.379
Blood albumin	1.263	12.521	0.015	1.546	1.045~2.325
Blood cholestero	1.291	10.269	0.0078	1.613	1.068~2.475
TG	0.697	9.584	0.036	1.177	0.829~1.777
24 h urine protein quantification	0.796	9.983	0.005	2.012	1.258~3.587
IL-6	0.998	10.115	0.017	2.115	1.597~4.582
IL-1	0.999	15.635	0.018	1.958	1.448~3.697
Cystatin C	0.759	11.147	0.024	2.056	1.569~5.241

3 讨论

IgA 肾病是世界范围内常见原发性肾小球疾病，指以 IgA 沉积为主，伴有其他免疫球蛋白在肾小球系膜区沉积的疾病，部分患者会出现了渐进性肾功能衰竭，威胁人们的生命[11-13]。其发病机制尚不明确，有学者认为可能与上呼吸道感染、遗传因素地等相关[14,15]。IgA 肾病发病率较高，有研究显示，中国原发性 IgA 肾病占原发性肾小球疾病的 20%～45%[16,17]。高尿酸血症是 IgA 肾病常见并发症，近年来逐渐受到人们重视。尿酸是机体嘌呤代谢的终产物，而嘌呤主要来源于外界摄入及核酸分解代谢产生，其中约 30%的尿酸经胃肠道分解排出，其余经肾脏排泄，而人体内尿酸排泄障碍可影响血尿酸水平[18-21]。有研究显示，尿酸是一把双刃剑，在人体内参与多个病理过程[22-24]。近年来随着人们生活方式的改变，高尿酸血症的患病率以高达 13.3%，相关报道称，IgA 肾病患者发生高尿酸血症的发生率为 36.6%，明显高于普通人群[25-28]。

有研究显示，代谢综合征与 IgA 肾病合并高尿酸血症关系密切，而高血压患者高尿酸血症发病率较高，可作为高尿酸血症的危险因素[29,30]。本研究结果显示，性别、高血压、血肌酐、尿素氮、血白蛋白、血胆固醇、甘油三酯、24 h 尿蛋白定量、IL-6、IL-1 及胱抑素 C 与 IgA 肾病患者发生高尿酸血症相关，与上述文献报道相似。本研究还显示，高尿酸组患者球性硬化、节段硬化、新月体形成、小管萎缩、间质炎性浸润及间质纤维化发生率均显著高于正常尿酸组，提示，IgA 肾病合并高尿酸血症患者病理损伤程度较正常血尿酸正常者严重。分析其原因可能是因为血尿酸可促进内皮细胞增殖，诱发微炎症反应，从而激活肾素血管紧张素系统，加重肾小球损伤。Yu A H[31]等研究也显示，高尿酸升高可引起动脉收缩，发生动脉闭塞，导致缺血性病理损伤。

本研究进一步多因素非条件 Logistic 分析显示，性别、高血压、血肌酐、尿素氮、血白蛋白、血胆固醇、甘油三酯、24 h 尿蛋白定量、IL-6、IL-1 及胱抑素 C 均是 IgA 肾病患者发生高尿酸血症的独立危险因素。Jiang X[32]等研究也显示，高尿酸血症可导致肾脏小动脉调节受损，导致肾小球压力升高，血尿酸在高血压中起重要作用。分析其原因可能是因为高尿酸血症抑制血管内皮细胞 - 氧化氮的合成，刺激血管平滑肌细胞增殖，导致血管内皮功能障碍，促进血管平滑肌细胞增殖，引起血压升高，因此高血压与血尿酸相互作用可能与 IgA 肾病的发生密切相关。而血肌酐、尿素氮、血白蛋白、血胆固醇、甘油三酯可导致高三酰甘油血症，血胰岛素可提高肾脏局部钠潴留减少尿酸排泄，最终引起高尿酸血症的发生。He J W[33]等研究也显示，高甘油三酯可增加游离脂肪酸产生及肾小管对尿酸重吸收，加速三

磷酸腺苷的分解，导致嘌呤代谢增加，最终引起血尿酸增加。尿蛋白的产生主要是足细胞损伤所致，而高尿酸可诱导足细胞损伤，导致蛋白尿。有研究显示，白细胞计数与高尿酸血症的发生呈正相关[34,35]。主要是因为白细胞可用来预测体内的炎症反应，而尿酸是一种促炎因子，可诱导体内产生炎症反应，从而导致炎症因子如 IL-6、IL-1 等增高。

综上所述，患者性别、高血压、血肌酐、尿素氮、血白蛋白、血胆固醇、甘油三酯、24 h 尿蛋白定量、IL-6、IL-1 及胱抑素 C 均是 IgA 肾病患者发生高尿酸血症的危险因素，临床上对于具有危险因素的患者引起重视，提高治疗效果。

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