



特发性膜性肾病合并急性肾损伤的危险因素分析*

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【摘要】目的 探究基于倾向性评分匹配的特发性膜性肾病合并急性肾损伤危险因素。**方法** 回顾性纳入2020年1月—2025年3月的310例特发性膜性肾病患者,分为急性肾损伤组($n=80$)和非急性肾损伤组($n=230$),从非急性肾损伤组中选择患者和急性肾损伤组患者进行PSM 1:1匹配,获得80对数据。收集其一般资料,主要结局指标为血清肌酐、估算的肾小球滤过率(estimated glomerular filtration rate, eGFR)、血尿酸、血清P-选择素、尿肾脏损伤分子1(kidney injury molecule-1, KIM-1)水平。通过Logistic回归分析确定特发性膜性肾病合并急性肾损伤危险因素,构建特发性膜性肾病合并急性肾损伤的列线图模型。**结果** 匹配后,急性肾损伤组患者的5项主要结局指标与非急性肾损伤组相比,差异均有统计学意义(均 $P<0.05$)。多因素logistic回归分析结果显示,血肌酐、eGFR、血尿酸、血清P-选择素均是特发性膜性肾病患者合并急性肾损伤的影响因素(均 $P<0.05$)。基于上述4项影响因素构建的联合预测模型(列线图)的ROC曲线下面积(AUC)为0.945(95%CI: 0.909~0.981),校准曲线显示预测曲线与理想曲线拟合良好。Hosmer-Lemeshow检验结果支持模型校准度($\chi^2=7.366$, $P=0.498$)。SHAP重要性排序显示,P-选择素是特发性膜性肾病合并急性肾损伤的重要影响因素,血尿酸、eGFR和血肌酐的重要性次之。**结论** 血肌酐、eGFR、血尿酸以及血清P-选择素是患者并发急性肾损伤的独立影响因素,且具有一定的预测价值。

【关键词】 特发性膜性肾病 急性肾损伤 倾向性评分匹配 危险因素

Risk Factors for Idiopathic Membranous Nephropathy Complicated by Acute Kidney Injury

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[Abstract] Objective To investigate the risk factors for idiopathic membranous nephropathy (IMN) complicated by acute kidney injury (AKI) based on propensity score matching (PSM). **Methods** A total of 310 IMN patients admitted between January 2020 and March 2025 were retrospectively enrolled and divided into an AKI group ($n=80$) and a non-AKI group ($n=230$). Patients in the non-AKI group were matched with those in the AKI group at a 1:1 ratio using PSM, resulting in 80 matched pairs. The general data of the patients were collected. The primary outcome measures included serum creatinine, estimated glomerular filtration rate (eGFR), uric acid, P-selectin, and urinary kidney injury molecule-1 (KIM-1) levels. Logistic regression analysis was performed to identify factors associated with risks for IMN complicated by AKI, and a nomogram prediction model was subsequently established. **Results** After PSM, all 5 primary outcome measures differed significantly between the AKI and non-AKI groups (all $P<0.05$). According to multivariate logistic regression analysis, 4 outcome measures, including serum creatinine, eGFR, serum uric acid, and serum P-selectin, were all identified as factors influencing the occurrence of AKI in patients with IMN (all $P<0.05$). A combined prediction model (a nomogram model) incorporating these 4 variables yielded an area under the curve (AUC) of the receiver operating characteristic curve (ROC) of 0.945 (95% CI: 0.909-0.981). The calibration curve demonstrated a good fit between the predicted and the ideal curves. Findings from the Hosmer-Lemeshow goodness-of-fit test supported adequate calibration of the model ($\chi^2=7.366$, $P=0.498$). Shapley additive explanations (SHAP) importance ranking analysis showed that serum P-selectin was an important influencing factor for AKI in patients with IMN, and the importance of serum uric acid, eGFR, and serum creatinine was secondary. **Conclusion** Serum creatinine, eGFR, blood uric acid, and serum P-selectin are independent influencing factors for patients with acute kidney injury, and they have certain predictive value.

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特发性膜性肾病是我国成人肾病综合征的重要致病因素,在肾小球疾病患者中占比达24.9%,发病率逐年递增,同时也是引发终末期肾病的关键诱因之一^[1-2]。该病起病隐匿,核心病理特征为肾小球基底膜上皮侧免疫复合物沉积,导致肾小球滤过功能损伤,临床多采用糖皮质激素联合生物制剂等方案干预^[3-4]。特发性膜性肾病因独特的病理改变及复杂的发病机制,较易合并急性肾损伤。若未能及时有效干预,病情可能逐步恶化至终末期肾病甚至尿毒症,显著增加患者死亡风险^[5]。因此,早期识别其并发急性肾损伤的独立危险因素,是采取有效预防措施及实施针对性治疗的重要前提。临床研究显示^[6],特发性膜性肾病患者中急性肾损伤发生率达32.9%,显著加剧终末期肾病风险及全因死亡率。现有研究已证实年龄、高血压及抗磷脂酶A2受体(phospholipase A2 receptor, PLA2R)抗体是其危险因素^[7-8];但也有研究指出上述指标与急性肾损伤之间并无明确相关性^[9]。不同研究间可能存在偏倚等干扰因素,造成结果异质性。因此,在研究设计中平衡病例组与对照组的基线特征,对控制偏倚具有关键意义。倾向性评分匹配作为一种减少选择偏倚的统计学方法,可有效均衡两组基线特征,提升回顾性研究结论的可靠性。本研究拟采用倾向性评分匹配法,综合分析特发性膜性肾病合并急性肾损伤患者的临床病理特征,确定独立危险因素,为优化风险分层及制定个体化防治策略奠定基础。

1 资料与方法

1.1 研究对象

本研究为回顾性研究。采用抽样便利法收集2020年1月—2025年3月于我院就诊的341例膜性肾病患者。采用pmsampsize函数, $n = \text{pmsampsize}(\text{type} = "b", \text{rsquared} = 0.70, \text{parameters} = 14, \text{prevalence} = 0.49)^{[10]}$, 估算样本量为240例。为了降低混淆因素的影响,最终纳入341例样本,符合最低样本量要求。纳入标准:(1)通过经皮肾穿刺活检术获取肾组织样本,经病理学检查明确诊断为膜性肾病;(2)均为首次发病;(3)随访时间 ≥ 1 年;(4)临床资料完整;(5)年龄 ≥ 18 岁。排除标准:(1)由系统性红斑狼疮、过敏性紫癜等疾病诱发的继发性膜性肾病;(2)近3个月使用过钙调磷酸酶抑制剂或生物制剂治疗者;(3)肾活检时估算的肾小球滤过率(estimated glomerular filtration rate, eGFR)(EPI公式) $< 15 \text{ mL/min} \cdot 1.73 \text{ m}^2$;(4)合并恶性肿瘤;

(5)妊娠期或哺乳期女性。以上述纳入和排除标准为依据结合相关文献^[11],从341例膜性肾病患者中选取310例特发性膜性肾病患者。根据急性肾损伤的诊断标准,将其分为急性肾损伤组($n=80$)和非急性肾损伤组($n=230$)。本研究经泉州市第一医院伦理委员会批准通过,批准号泉一伦(2026)120号(补),且符合《赫尔辛基宣言》。

为对特发性膜性肾病病例实施分组,研究团队借助logistic回归模型来计算倾向评分,所纳入的协变量涵盖年龄、体质量指数(body mass index, BMI)、糖尿病史、冠心病史、高血压病史。基于所得倾向评分,采用最近邻匹配法进行1:1配对,卡钳值设为0.02以控制匹配精度。针对数据集中的缺失值问题,为保障数据的完整性及后续分析结果的可靠性,本研究通过全数据集随机森林插补法对其进行处理。

两组匹配后使用1:1匹配法,成功匹配80对,相关协变量经匹配后均达到均衡(均 $P>0.05$)。见表1。

1.2 资料收集

本研究通过医院电子病历检索系统,回顾性采集特发性膜性肾病患者肾活检时的临床资料,包括人口学特征、既往病史、实验室检查指标及肾脏病理检查分期,并对原始数据进行规范化整理与核查。基线资料收集:年龄、性别、BMI、特发性膜性肾病病程、合并急性肾损伤病程、高血压病史、冠心病史、糖尿病史、RAS阻断剂的应用情况以及病理分期。实验室指标:血肌酐、血红蛋白、白细胞数、eGFR、血尿酸、血总胆固醇、血白蛋白、血甘油三酯、D-二聚体、肾脏PLA2R阳性率、血清P-选择素、尿肾脏损伤分子1(kidney injury molecule-1, KIM-1)。

1.3 结局指标

1.3.1 主要结局指标

血肌酐、eGFR、血尿酸、血清P-选择素、尿KIM-1水平。

1.3.2 次要结局指标

性别、病理分期、血红蛋白、白细胞计数、血总胆固醇、血白蛋白、血甘油三酯、D-二聚体、肾脏PLA2R阳性率、RAS阻断剂的应用情况。

1.4 统计学方法

应用R 4.3.2软件进行匹配分析,SPSS27.0软件进行数据计算,Shapiro-Wilk检验正态分布,计量资料以 $\bar{x} \pm s$ 表示, t 检验;计数资料以例数(%)表示, χ^2 检验。使用倾向性

表 1 两组匹配前后临床特征对比

Table 1 Comparison of clinical characteristics before and after propensity score matching of the two groups

Clinical features	Before Matching				After Matching			
	Acute kidney injury group (<i>n</i> = 80)	Non-acute kidney injury group (<i>n</i> = 230)	χ^2/t	<i>P</i>	Acute kidney injury group (<i>n</i> = 80)	Non-acute kidney injury group (<i>n</i> = 80)	χ^2/t	<i>P</i>
Age/yr., $\bar{x} \pm s$	68.78 $\pm$ 7.91	66.92 $\pm$ 6.48	2.084	0.038	68.78 $\pm$ 7.91	68.31 $\pm$ 7.82	0.378	0.706
BMI/(kg/m ²), $\bar{x} \pm s$	24.88 $\pm$ 3.64	25.95 $\pm$ 3.73	2.224	0.027	24.88 $\pm$ 3.64	24.59 $\pm$ 3.48	0.515	0.607
History of diabetes/case (%)			0.008	0.931			0.108	0.742
Yes	30 (37.50)	85 (36.96)			30 (37.50)	28 (35.00)		
No	50 (62.50)	145 (63.04)			50 (62.50)	52 (65.00)		
History of coronary heart disease/case (%)			0.145	0.704			0.098	0.755
Yes	5 (6.25)	10 (4.35)			5 (6.25)	6 (7.50)		
No	75 (93.75)	220 (95.65)			75 (93.75)	74 (92.50)		
History of hypertension/case (%)			0.386	0.535			0.245	0.620
Yes	53 (66.25)	154 (66.96)			53 (66.25)	50 (62.50)		
No	27 (33.75)	76 (33.04)			27 (33.75)	30 (37.50)		

BMI: body mass index.

评分匹配法降低急性肾损伤组和非急性肾损伤组的基线差异,利用斯皮尔曼等级相关系数(Spearman's rank correlation coefficient)对倾向性评分匹配后的两组基线数据进行筛选,并行特发性膜性肾病合并急性肾损伤的单因素分析,筛选出的指标进行多重共线性检验;将无共线性的 $P<0.1$ 的单因素数据纳入多因素logistic回归分析;构建特发性膜性肾病合并急性肾损伤的列线图模型。

$P<0.05$ 为差异有统计学意义。

2 结果

2.1 主要结局指标

急性肾损伤组患者的血肌酐、eGFR、血尿酸、血清P-选择素、尿KIM-1与非急性肾损伤组相比,差异均有统计学意义(均 $P<0.05$)。见表2。

表 2 两组患者的主要结局指标对比

Table 2 Comparison of the main outcome indicators between the two groups of patients

Baseline data	Acute kidney injury group (<i>n</i> = 80)	Non-acute kidney injury group (<i>n</i> = 80)	X^2/t	<i>P</i>
Serum creatinine/($\mu\text{mol/L}$), $\bar{x} \pm s$	94.66 $\pm$ 11.84	71.28 $\pm$ 9.44	13.810	< 0.001
eGFR/(mL/min $\cdot$ 1.73 m ⁻²), $\bar{x} \pm s$	83.42 $\pm$ 11.09	113.54 $\pm$ 13.96	15.110	< 0.001
Blood uric acid/($\mu\text{mol/L}$), $\bar{x} \pm s$	429.68 $\pm$ 36.97	342.55 $\pm$ 24.11	17.657	< 0.001
Serum P-selectin/($\mu\text{g/L}$), $\bar{x} \pm s$	17.71 $\pm$ 2.30	10.72 $\pm$ 2.11	20.321	< 0.001
KIM-1/(ng/mL), $\bar{x} \pm s$	1.83 $\pm$ 0.23	0.90 $\pm$ 0.18	28.481	< 0.001

eGFR: estimated glomerular filtration rate; KIM-1: kidney injury molecule-1.

2.2 次要结局指标

除性别($P<0.05$)外,其余次要结局指标在两组间差异无统计学意义。见表3。

2.3 特发性膜性肾病合并急性肾损伤的单因素分析和多因素分析

以单因素分析结果 $P<0.05$ 的变量为自变量,以是否发生急性肾损伤为因变量(非急性肾损伤=0,急性肾损伤=1),赋值:性别(女=0,男=1)、血肌酐(实测值)、eGFR(实测值)、血尿酸(实测值)、血清P-选择素(实测

值)、尿KIM-1(实测值)。

单因素分析显示,性别、血肌酐、eGFR、血尿酸、血清P-选择素与特发性膜性肾病患者合并急性肾损伤相关(均 $P<0.05$)。见表4。为排除单因素分析结果中5个差异指标间的多重共线性,进行共线性检验。结果显示,性别、血肌酐、eGFR、血尿酸、血清P-选择素的容差均 >0.1 、VIF均 <5 。因此,变量间无共线性,可进行下一步研究。见网络资源附件附表1。

Logistic回归分析结果显示,血肌酐、eGFR、血尿酸、

表 3 两组患者的次要结局指标对比
Table 3 Comparison of secondary outcome indicators between the two groups of patients

Indicator		Acute kidney injury group (n = 80)	Non-acute kidney injury group (n = 80)	χ^2/t	P
Sex	Male	66 (82.50)	47 (58.75)	10.876	0.001
	Female	14 (17.50)	33 (41.25)		
Pathological staging	Phase I	50 (62.50)	47 (58.75)	1.402	0.705
	Phase II	18 (22.50)	24 (30.00)		
	Phase III	6 (7.50)	7 (8.75)		
	Phase IV	6 (7.50)	4 (5.00)		
Hemoglobin/(g/L)		102.77 ± 17.41	105.07 ± 15.68	0.878	0.381
White blood cell count/(×10 ⁹ L ⁻¹)		7.46 ± 1.08	7.80 ± 1.13	11.946	0.054
Total blood cholesterol/(μmol/L)		7.71 ± 2.02	7.52 ± 1.49	0.677	0.499
Blood albumin/(g/L)		23.89 ± 5.74	24.31 ± 4.36	0.521	0.603
Blood triglycerides/(μmol/L)		2.02 ± 0.57	1.99 ± 0.64	0.313	0.755
D-dimer/(mg/L)		0.79 ± 0.35	0.72 ± 0.34	1.283	0.201
The positive rate of PLA2R in the kidneys		57 (71.25)	66 (82.50)	2.848	0.092
RAS blocker	Use	51 (63.75)	55 (68.75)	0.447	0.504
	Not used	29 (36.25)	25 (31.25)		

PLA2R: phospholipase A2 receptor. RAS: renin-angiotensin system. Data are presented as case (%) or $\bar{x} \pm s$.

表 4 特发性膜性肾病合并急性肾损伤的单因素分析
Table 4 Univariate analysis of acute kidney injury in patients with idiopathic membranous nephropathy

Factor	β	SE	P	OR	95% CI	
					Lower limit	Upper limit
Sex	1.904	0.372	< 0.001	6.714	3.240	13.912
Serum creatinine	0.189	0.029	< 0.001	1.208	1.143	1.278
eGFR	-0.209	0.034	< 0.001	0.811	0.759	0.867
Blood uric acid	0.091	0.016	< 0.001	1.096	1.062	1.130
Serum P-selectin	1.084	0.360	0.003	2.957	1.461	5.982
KIM-1	1.148	0.823	0.163	3.164	0.628	15.831

eGFR: estimated glomerular filtration rate; KIM-1: kidney injury molecule-1. β : partial regression coefficient; SE: standard error; OR: odds ratio; CI: confidence interval.

血清P-选择素均是特发性膜性肾病患者合并急性肾损伤的影响因素(均 $P < 0.05$)。见表5。

2.4 列线图预测模型的构建

基于上述4项影响因素构建特发性膜性肾病合并急性肾损伤的列线图模型,结果显示:特发性膜性肾病合并急性肾损伤的概率为0.1 ~ 0.9,见图1。联合预测模型(列线图)的ROC曲线下面积(AUC)为0.945(95%CI: 0.909 ~ 0.981),见图2。校准曲线显示预测曲线与理想曲线拟合良好。见图3。Hosmer-Lemeshow检验结果支持模型校准度($\chi^2 = 7.366, P = 0.498$)。决策曲线分析结果显示,当阈

值概率处于0.50 ~ 0.98区间时,本模型的临床净获益均优于“全部干预”与“不干预”两种极端策略。见图4。采用SHAP算法对特发性膜性肾病合并急性肾损伤的列线图模型展开可解释性分析,SHAP重要性排序;其中,血清P-选择素是特发性膜性肾病合并急性肾损伤的重要影响因素,血尿酸、eGFR和血肌酐的重要性次之。

3 讨论

倾向性评分匹配由Rosenbaum和Rubin于1983年提出,可有效消除非随机分组带来的混杂偏差,均衡组间影

表 5 特发性膜性肾病合并急性肾损伤的多因素分析
Table 5 Multivariate analysis of acute kidney injury in patients with idiopathic membranous nephropathy

Factor	β	Wald	SE	<i>P</i>	OR	95% CI	
						Lower limit	Upper limit
Sex	1.463	3.167	0.822	0.075	4.321	0.862	21.654
Serum creatinine	0.185	5.971	0.076	0.015	1.203	1.037	1.396
eGFR	−0.220	6.759	0.085	0.009	0.802	0.680	0.947
Blood uric acid	0.104	5.555	0.044	0.018	1.110	1.018	1.210
Serum P-selectin	1.383	24.986	0.277	< 0.001	3.988	2.318	6.859
Constant	−33.814	15.747	4.611	0.032	0.000	-	-

eGFR: estimated glomerular filtration rate. β : partial regression coefficient; SE: standard error; OR: odds ratio; CI: confidence interval.

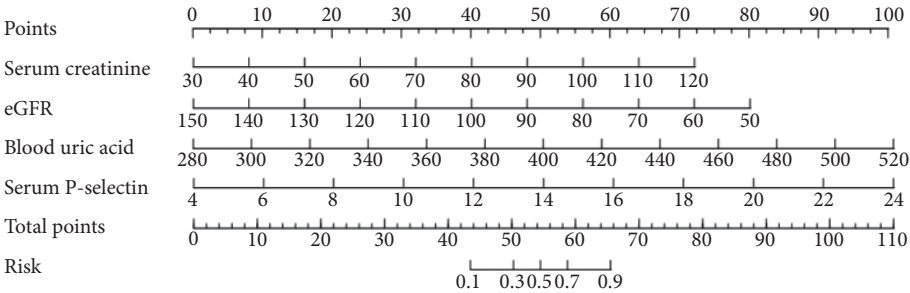


图 1 列线图
Fig 1 Nomogram

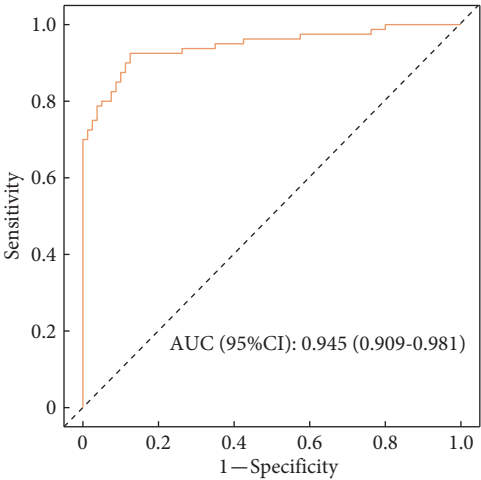


图 2 ROC曲线
Fig 2 ROC curve

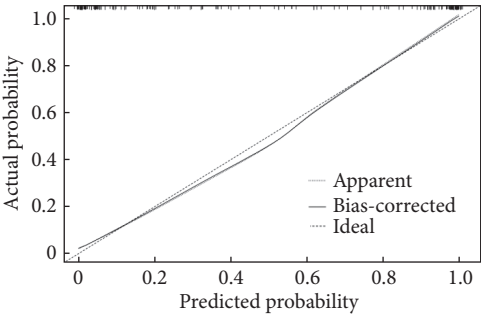


图 3 校准曲线
Fig 3 Calibration curve

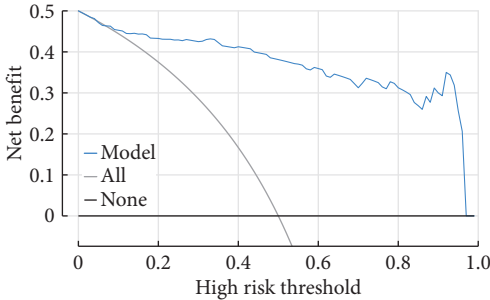


图 4 决策分析曲线
Fig 4 Decision analysis curve

响因素分布,提高研究因素的真实性^[2]。本研究中急性肾损伤组与非急性肾损伤组样本量差异较大,为最大限度保留原始样本信息,采用1:1匹配^[12]。本研究经倾向性评分匹配对2020-2025年间两组患者的临床基线资料进行匹配后,两组基线特征达到均衡且具备可比性,进而分析了急性肾损伤的独立危险因素。

性别是否影响特发性膜性肾病患者合并急性肾损伤尚无定论。相关指南指出女性可能是医院获得性急性肾损伤的危险因素之一^[11],而另有研究证实男性为独立危险因素^[13-14]。本研究结果显示,虽然男性患者占比更高,但性别并非其影响因素,具体机制仍有待进一步阐明。

急性肾小管坏死、肾静脉血栓以及多种肾毒性药物等是肾病综合征并发急性肾损伤的主要致病因^[15]。本次研究结果显示,血肌酐是特发性膜性肾病患者合并急性

肾损伤的影响因素,与Li等^[16]的研究结果一致。究其原因主要是,特发性膜性肾病患者因肾小球基底膜上侧免疫复合物沉积,导致肾小球滤过膜受损,大量蛋白尿漏出,进而引起GFR下降,肌酐排泄减少,同时,特发性膜性肾病合并急性肾损伤患者常伴随严重的小管间质病变,进一步影响肾小管重吸收和排泄功能,导致肌酐清除能力下降,血肌酐升高^[17]。

本次研究结果显示,eGFR是特发性膜性肾病患者合并急性肾损伤的影响因素,与朱贺等^[18]的研究结果相似。eGFR水平降低往往提示肾脏在宏观与微观结构上已出现改变,而对于特发性膜性肾病患者来说,其肾脏所承受的损伤通常更为显著。eGFR作为衡量肾功能的关键指标,能够反映肾脏的基础功能状态及代偿潜能,其数值的降低通常意味着肾损害发生的可能性增大,且数值越低,肾损害进展的风险也相应越高^[19]。

本次研究结果显示,血尿酸是特发性膜性肾病患者合并急性肾损伤的影响因素,与Chen等^[20]研究结论一致。高尿酸诱发急性肾损伤的机制主要涉及三方面:一是对肾脏微血管及血流动力学产生全身性影响;二是在肾小管局部促成结晶形成;三是促进活性氧自由基及炎症介质的生成与释放,导致肾脏结构与功能损伤^[21]。尿酸作为嘌呤代谢终产物,以可溶性尿酸与尿酸盐结晶两种形态存在。研究表明^[22],其可通过促进炎症反应、加剧氧化应激、诱导线粒体功能异常及激活肾素-血管紧张素系统等多重途径引发肾脏损伤。在脓毒症状态下,高尿酸水平会进一步加重细胞氧化应激与炎症反应,并引发肾脏微循环障碍,提高急性肾损伤发生风险。因此,对尿酸水平较高的患者及时干预以降低血尿酸,有助于减轻肾脏微血管及肾小管损伤、减少活性氧自由基和炎症介质释放,从而降低急性肾损伤的发生风险。

P-选择素是一种分布于血管内皮细胞及血小板颗粒膜表面的糖蛋白分子。既往研究表明^[23],敲低P-选择素糖蛋白配体1可升高小鼠体内炎症因子表达。本研究结果显示,血清P-选择素是特发性膜性肾病患者合并急性肾损伤的影响因素,与张静等^[24]研究结果一致。P-选择素作为黏附分子选择素家族成员,主要在血小板与血管内皮细胞表面高表达,参与炎症反应调控及免疫识别,是介导炎症细胞早期向受损组织迁移的关键信号分子。组织损伤时,血管内皮内膜下P-选择素表达显著上升,通过充当白细胞“滚动”介质,加速白细胞聚集及与单核细胞、中性粒细胞、血小板的黏附,诱发组织结构破坏及炎症损伤。急性肾损伤发生时,肾小球毛细血管内皮细胞表达的P-选择素通过与活化血小板表面PSGL-1相互作用,引

发内皮细胞损伤,增加急性肾损伤发生风险。此外,急性肾损伤还涉及肾小球与肾小管滤过功能下降,临床可表现为24 h Pro升高及白蛋白水平降低等特征性变化^[25]。

最后,本研究聚焦特发性膜性肾病患者这一合并急性肾损伤高发人群,基于血肌酐、eGFR、P-选择素5项临床易获取的指标构建了特发性膜性肾病患者合并急性肾损伤风险预测列线图模型,模型AUC达0.945(95%CI: 0.909 ~ 0.981),且经检验显示校准度与临床实用性良好,可为临床提供量化、可视化的风险分层工具。

综上所述,血肌酐、血红蛋白以及血尿酸是患者并发急性肾损伤的独立危险因素,具有一定的临床预测价值。但由于本研究为单中心设计,该预测模型的有效性 & 普适性尚需在多中心、大样本的临床研究中进一步验证,以明确其在不同人群及诊疗环境中的应用价值。

* * *

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利益冲突 所有作者均声明不存在利益冲突

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