

• 论著-急性胰腺炎 •

NLR 和 PLR 以及 CAR 联合血清胱抑素 C 对急性胰腺炎合并急性肾损伤的预测价值*

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[摘要] 目的:探索中性粒细胞/淋巴细胞(NLR)、血小板/淋巴细胞(PLR)、C反应蛋白/白蛋白(CAR)联合血清胱抑素 C(Cys C)对急性胰腺炎(AP)并发急性肾损伤(AKI)的预测价值。方法:回顾性分析 2016 年 4 月—2021 年 4 月期间在四川省绵阳市中心医院住院治疗的 393 例 AP 患者的临床资料,其中 328 例患者未合并 AKI(非 AKI 组),65 例患者合并 AKI(AKI 组)。收集所有患者的病例资料、临床指标并进行统计学分析。结果:性别、氯离子、血小板计数(PLT)、淀粉酶(AMY)、脂肪酶(LPS)组间相比差异无统计学意义($P>0.05$);年龄、肌酐(SCr)、血尿素氮(BUN)、尿酸(UA)、估算肾小球滤过率(eGFR)、钙离子、Cys C、C反应蛋白(CRP)、白蛋白(Alb)、白细胞计数(WBC)、中性粒细胞计数(NEUT)、淋巴细胞计数(LYM)、NLR、PLR、CAR、红细胞比容(HCT)组间比较差异均有统计学意义($P<0.05$)。二元 Logistic 回归分析发现,NLR、PLR、CAR、Cys C 是 AP 合并 AKI 的重要影响因素($P<0.05$)。ROC 曲线结果显示 NLR 预测 AP 合并 AKI 的曲线下面积为 0.867,特异度为 0.692,敏感度为 0.923。PLR 预测 AP 合并 AKI 的曲线下面积为 0.741,特异度为 0.720,敏感度为 0.677。CAR 预测 AP 合并 AKI 的曲线下面积为 0.843,特异度为 0.851,敏感度为 0.785。Cys C 预测 AP 合并 AKI 的曲线下面积为 0.954,特异度为 0.942,敏感度为 0.862。结论:Cys C、CAR、NLR、PLR 在预测 AP 患者并发 AKI 中具有重要的预测价值,有一定的临床意义。

[关键词] 急性胰腺炎;急性肾损伤;中性粒细胞/淋巴细胞;血小板/淋巴细胞;C反应蛋白/白蛋白;胱抑素 C

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The predictive value of neutrophil/lymphocyte ratio and platelet/lymphocyte ratio and C-reactive protein/albumin ratio and serum cystatin C for acute pancreatitis with acute kidney injury

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Abstract Objective: To explore the predictive value of neutrophil/lymphocyte ratio(NLR), platelet/lymphocyte ratio(PLR), C-reactive protein/albumin ratio(CAR) and serum cystatin C(Cys C) in acute pancreatitis(AP) with acute kidney injury(AKI). **Methods:** The clinical data of 393 patients with AP hospitalized in Mianyang Central Hospital from April 2018 to April 2021 were analyzed retrospectively. Among them, 328 patients did not have AKI(non-AKI group), and 65 patients had AKI(AKI group). The case data and clinical indexes of all patients were collected and statistically analyzed. **Results:** There was no significant difference between the two groups in gender, chloride ion, platelet(PLT), amylase(AMY) and lipase(LPS)($P>0.05$). There were significant differences between the two groups in age, serum creatinine(SCr), blood urea nitrogen(BUN), uric acid(UA), estimated glomerular filtration rate(eGFR), calcium ion, Cys C, C-reactive protein(CRP), albumin(Alb), white blood cell(WBC), neutrophil(NEUT), lymphocyte(LYM), NLR, PLR, CAR, and hematocrit(HCT)($P<0.05$). The analysis of binary Logistic regression found that, NLR, PLR, CAR and Cys C are the important influencing factors of AP merging AKI($P<0.05$). NLR had an area under the ROC curve of 0.867, a

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specificity of 0.692 and a sensitivity of 0.923 in predicting AKI in patients with AP. PLR had an area under the ROC curve of 0.741, a specificity of 0.720 and a sensitivity of 0.677 in predicting AKI in patients with AP. CAR had an area under the ROC curve of 0.843, a specificity of 0.851 and a sensitivity of 0.785 in predicting AKI in patients with AP. Cys C had an area under the ROC curve of 0.954, a specificity of 0.942 and a sensitivity of 0.862 in predicting AKI in patients with AP. **Conclusion:** NLR, PLR, CAR and Cys C have important predictive value and clinical significance in predicting AKI in patients with AP.

Key words Acute pancreatitis; Acute kidney injury; Neutrophil/lymphocyte ratio; Platelet/lymphocyte ratio; C-reactive protein/albumin ratio; Cystatin C

急性胰腺炎(acute pancreatitis, AP)是需要急诊住院的常见胃肠道疾病,且其发病率正在上升^[1],Petrov等^[2]在全球流行病学和整体预防胰腺炎的报道称全球急性胰腺炎的发病率:每年每十万普通人群中约有34例。严重的急性胰腺炎与高死亡率相关,预后主要与其他器官功能障碍及继发感染等有关^[3],而急性肾损伤(acute kidney injury, AKI)是急性胰腺炎的常见并发症,发生率约10%,对预后具有决定性的影响,其病死率(尤其是需要透析的AKI的病死率)可以急剧上升至75%^[4-6],故及时的预测、有效的预防治疗有助于降低患者病死率,减少住院时间,减轻住院经济负担。中性粒细胞/淋巴细胞(neutrophil/lymphocyte ratio, NLR)、血小板/淋巴细胞(platelet/lymphocyte ratio, PLR)作为新的全身炎症指标,被发现与多种肿瘤、心血管疾病、炎症疾病及肾功能损伤的进展及预后等相关联^[7-15]。血清胱抑素C(cystatin C, Cys C)已被证明在预测急性肾损伤中具有较高的价值,但是目前用于预测AP患者并发AKI的报道并不多见^[16-17]。C反应蛋白/白蛋白(creactive protein/albumin ratio, CAR)是一种基本的炎症因子,与多种肿瘤、脓毒症、炎症疾病、急性胰腺炎等预后密切相关^[18-22]。本研究旨在通过分析研究NLR、PLR、CAR、Cys C等指标,探索上述指标对AP合并AKI患者的预测价值。

1 资料与方法

1.1 临床资料

回顾性分析2016年4月—2021年4月期间在四川省绵阳市中心医院住院治疗的AP患者的临床资料。AP诊断标准采用2012年修订的亚特兰大国际共识^[23],即需要符合以下三项中的两项。①与AP相符合的腹痛(持续的,严重的,上腹痛的急性发作,疼痛通常放射至背部);②血清脂肪酶(lipase, LPS)或淀粉酶(amylase, AMY)水平至少是正常上限的三倍;③影像学检查符合AP的特征性影像学表现。

AKI的诊断标准根据改善全球肾病预后组织(KDIGO)指南(2012)^[24]:①48 h内血清肌酐(SCr)升高超过 $26.5 \mu\text{mol/L}$ (0.3 mg/dL);②尿量 $<0.5 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{h}^{-1}$,且持续6 h以上;③血清肌酐(SCr)升高超过基线1.5倍。符合以上情况之

一即可诊断AKI。

纳入标准:年龄大于18岁,符合AP的诊断标准,因AP入院的患者。排除标准:临床资料不完整,慢性胰腺炎患者,其他炎症性疾病者,有慢性肾病患者,非急性胰腺炎引起的急性肾损伤者,严重的心肺功能不全者,具有严重免疫缺陷者,患有肿瘤疾病者。

本研究方案已获得绵阳市中心医院伦理委员会批准(No: S2018073)。

1.2 基本资料及临床指标

收集年龄、性别、淀粉酶(AMY)、脂肪酶(LPS)、血肌酐(serum creatinine, SCr)、血尿素氮(blood urea nitrogen, BUN)、尿酸(serum uric acid, UA)、估算肾小球滤过率(estimate glomerular filtration rate, eGFR)、胱抑素C(Cys C)、C反应蛋白(CRP)、白蛋白(Alb)、氯离子、钙离子、白细胞计数(WBC)、中性粒细胞计数(NEUT)、淋巴细胞计数(LYM)、红细胞压积(HCT)、血小板计数(PLT)、NLR、PLR、CAR等资料。记录入院后24 h内第一次外周静脉血液标本的相关实验室指标,收集所有入选患者的临床资料进行统计学分析。

1.3 统计学方法

使用SPSS 21.0版软件进行数据分析。计量资料符合或接近正态分布,采用 $\bar{x} \pm s$ 表示,并使用Student *t*检验分析两组间的差异性。采用二元Logistic回归分析判断危险因素。根据受试者工作特征曲线(ROC曲线)评估指标的诊断效能。以 $P < 0.05$ 表示差异具有统计学意义。

2 结果

2.1 一般资料比较

本研究共纳入393例AP患者,其中男268例(68.2%),女125例(31.2%)。AP未合并AKI患者328例(非AKI组,83.5%),AP合并AKI患者65例(AKI组,16.5%)。纳入两组的临床资料进行统计学分析,其中性别、氯离子、PLT、AMY、LPS等比较,差异无统计学意义;年龄、SCr、BUN、UA、eGFR、钙离子、Cys C、CRP、Alb、WBC、NEUT、LYM、NLR、PLR、CAR、HCT等组间比较,差异均具有统计学意义($P < 0.05$),见表1。

2.2 AP合并AKI影响因素的二元Logistic回归分析

同时通过二元 Logistic 回归分析比较,发现 NLR、PLR、CAR、Cys C、钙离子均是 AP 合并 AKI 的重要影响因素($P < 0.05$),见表 2。

2.3 Scr、NLR、PLR、CAR、Cys C 对 AP 合并 AKI 的诊断价值

ROC 曲线结果显示 NIR 诊断 AP 合并 AKI 的曲线下面积(AUC)为 0.867,95%CI 为(0.823~0.910),最佳截断值为 5.995,特异度 0.692 为,敏感度为 0.923。PLR 诊断 AP 合并 AKI 的曲线

下面积(AUC)为 0.741,95%CI 为(0.677~0.806),最佳截断值为 138.880,特异度为 0.720,敏感度为 0.677。CAR 诊断 AP 合并 AKI 的曲线下面积(AUC)为 0.843,95%CI 为(0.778~0.908),最佳截断值为 0.635,特异度为 0.851,敏感度为 0.785。Cys C 诊断 AP 合并 AKI 的曲线下面积(AUC)为 0.954,95%CI 为(0.928~0.981),最佳截断值为 1.125,特异度为 0.942,敏感度为 0.862(表 3,图 1)。

表 1 两组患者临床资料指标

指标	AKI 组($n=65$)	非 AKI 组($n=328$)	P
年龄/岁	58.58±15.65	46.49±13.61	<0.01
男/女(例)	48/17	220/108	0.311
AMY/($U \cdot L^{-1}$)	632.54±751.79	552.45±1093.84	0.438
LPS/($U \cdot L^{-1}$)	990.17±1738.36	930.69±1673.81	0.795
SCr/($\mu mol \cdot L^{-1}$)	163.28±70.65	73.70±16.21	<0.01
BUN/($mmol \cdot L^{-1}$)	11.40±6.34	4.91±1.80	<0.01
UA/($\mu mol \cdot L^{-1}$)	448.41±175.10	346.67±109.56	<0.01
eGFR/($mL \cdot min^{-1} \cdot 1.73 m^{-2}$)	52.86±12.63	89.13±16.92	<0.01
CRP/($mg \cdot L^{-1}$)	81.17±84.46	15.61±28.28	<0.01
Alb/($g \cdot L^{-1}$)	38.39±6.11	44.33±4.39	<0.01
Cys C/($mg \cdot L^{-1}$)	1.64±0.60	0.88±0.16	<0.01
钙离子/($mmol \cdot L^{-1}$)	2.17±0.40	2.29±0.14	<0.01
氯离子/($mmol \cdot L^{-1}$)	103.43±7.77	104.99±53.92	0.815
WBC/($\times 10^9 \cdot L^{-1}$)	12.20±4.95	9.91±3.94	<0.01
NEUT/($\times 10^9 \cdot L^{-1}$)	10.22±4.42	7.50±3.59	<0.01
LYM/($\times 10^9 \cdot L^{-1}$)	1.06±0.42	1.67±0.96	<0.01
HCT/%	37.48±7.02	42.52±4.93	<0.01
PLT/($\times 10^9 \cdot L^{-1}$)	179.60±85.90	178.73±60.74	0.810
CAR	2.14±2.18	0.36±0.66	<0.01
NLR	10.87±6.96	5.19±3.04	<0.01
PLR	179.61±80.80	122.63±58.31	<0.01

表 2 AP 合并 AKI 影响因素的二元 Logistic 回归分析

指标	B	SE	Wals	95%CI	P
年龄	-0.032	0.028	1.293	0.917~1.023	0.255
UA	0.002	0.004	0.283	0.995~1.009	0.595
钙离子	-4.626	2.011	5.293	0.000~0.504	<0.05
NLR	0.364	0.100	13.144	1.182~1.752	<0.01
PLR	0.009	0.004	4.334	1.001~1.017	<0.05
CAR	0.999	0.288	12.015	1.544~4.779	0.001
Cys C	11.822	2.246	27.710	1669.555~11114409.860	<0.01
HCT	-10.302	6.484	2.524	0.000~11.103	0.112

表3 Scr、NLR、PLR、CAR、Cys C对AP合并AKI的诊断价值

指标	最大约登指数	临界值	敏感度	特异度	AUC	95%CI
Scr	0.942	106.750	0.969	0.973	0.955	0.990~1.000
NLR	0.615	5.995	0.923	0.692	0.867	0.823~0.910
PLR	0.396	138.880	0.677	0.720	0.741	0.677~0.806
CAR	0.635	0.635	0.785	0.851	0.843	0.778~0.908
Cys C	0.804	1.125	0.862	0.942	0.954	0.928~0.981

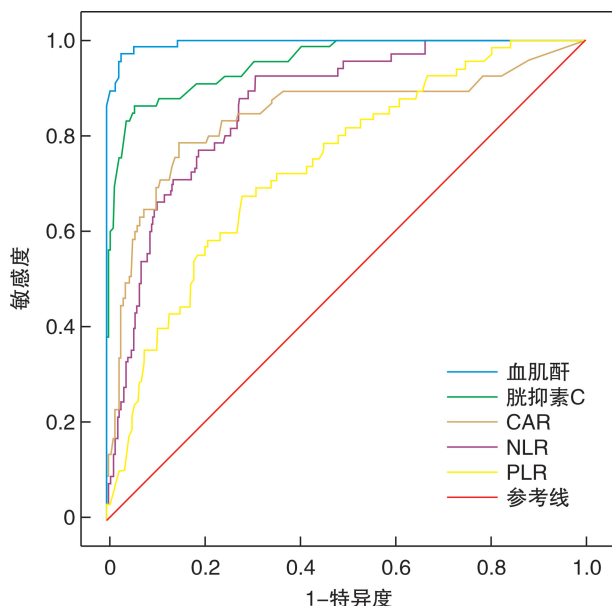


图1 Scr、Cys C、CAR、NLR、PLR对AP合并AKI的ROC曲线图

3 讨论

AP是一种以胰腺腺泡细胞功能障碍、胰蛋白酶激活不当、胰腺自我防御机制破坏为特征,进一步加剧胰腺细胞损伤和损伤的疾病,是一种快速发展的胰腺炎症过程。作为住院患者最常见的胃肠道疾病之一,也是住院死亡的主要原因之一,其严重程度从轻度自限性疾病到以全身并发症和多器官衰竭为特征的重症急性坏死性胰腺炎^[24]。而AKI是急性胰腺炎的常见并发症,预后非常差,其病死率(尤其是需要透析的AKI的病死率)可超过75%^[4-6],因此早期诊断、及时治疗是尤为重要的。目前,AKI的诊断依据是血肌酐动态升高和尿量减少,血清肌酐的显著增加已经是AKI的晚期迹象,因此我们需要寻找合适(高诊断敏感度和特异度)的生物标志物,在发生不可逆转的变化之前对AKI进行早期诊断。

AP并发AKI的确切病理生理机制尚不清楚,关键的病理生理机制可能与胰腺腺泡细胞内胰酶的过早激活有关,胰酶和蛋白酶大量释放导致血管内皮损伤、胰腺及周围组织自身消化,引起一系列反应,从而导致AKI^[24]。有研究表明^[17,25] AP并发AKI原因大致如下:低有效循环血量导致肾脏灌注

不足、全身炎症反应导致肾损伤、缺血缺氧导致肾组织损害、在AP后期,细菌感染和脓毒症成为器官衰竭发展的重要因素、微循环功能障碍、肾毒性药物的使用、可能的其他因素。

SCr的释放受年龄、性别、饮食、肌肉质量、药物、肝功能等影响较大,是AKI的相对不敏感和晚期指标,而Cys C是一种非糖基化的13-kDa蛋白,是半胱氨酸蛋白酶抑制剂cystatin超家族的成员,受年龄、性别、种族或肌肉质量的影响相对较小^[26]。Cys C已被证实是一种优越的GFR生物标志物^[27],研究发现^[26],Cys C在预测急性肾损伤及肾功能恢复中具有较高的价值,并且优于SCr,但目前国内缺乏多中心研究数据,用于预测AP患者并发AKI的报道并不多见。Nakhjavan-Shahraki等^[28]在Cys C预测儿童急性肾损伤的准确性的研究中总纳入24篇文章(来自Medline, Embase, ISI Web of Science, Cochrane library and Scopus)进行meta分析(1302例非AKI患者和645例AKI患者),最终得出结论入院后24h内Cys C的水平可以预测随后几天AKI的发生,并且无论是入院后24h内还是在24h后测量其浓度,其预后价值都没有显著差异,因此在入院后24h内测量Cys C水平可提供预测AKI的最高价值。

越来越多的实验和临床研究表明,炎症反应在AKI病理生理学中起着不可否认的作用,而AKI期间出现的全身炎症反应可能是由肾脏组织的局部炎症引起的^[29]。尽管局部胰腺损伤驱动全身炎症反应的病理生理学尚未完全明确,但研究表明先天免疫系统(包括中性粒细胞、单核细胞和巨噬细胞)和适应性免疫系统(主要由淋巴细胞组成)都在疾病进展中起着关键的作用^[12,30],NLR代表炎症激活因子中性粒细胞和炎症调节淋巴细胞之间的平衡,数值越高,炎症状态越不平衡^[31-32]。NLR、PLR作为新的全身炎症标志物被熟知^[33],是很容易获得、低成本的参数,已经在多种肿瘤、心脑血管疾病、炎性疾病及脓毒症中被证实具有预测诊断及预后评估价值^[7-15]。

CRP和ALB是评估炎症状态的常用参数,被称为阳性和阴性急性期反应物,CAR是通过将CRP除以ALB测量值来确定的,它是一个既定的评分系统,被认为是一种新的基于炎症的评分,用

于确定炎症疾病的程度和活跃度,被认为是比单独的 CRP 或 ALB 更有价值的炎症指标^[34],在本研究中也得到相同的结论,但目前几乎没有相关研究报道过 AP 患者的 CAR 与并发 AKI 之间的关联。

本研究发现 CAR 和 Cys C 在预测 AP 合并 AKI 中的特异度、敏感度均较高,均可作为重要的预测因子。NLR 在预测 AP 合并 AKI 中的特异度偏低,但敏感度高,有利于尽早发现 AKI 的发生,而 PLR 在预测 AP 合并 AKI 中的敏感度稍低,但特异度较高,两者均具有重要的预测价值,当 NLR、PLR 值均在临界值以上时,需高度警惕 AP 合并 AKI 的发生。

综上所述,Cys C、CAR、NLR、PLR 作为易获得、低成本的参数,在 AP 患者合并 AKI 中具有重要的预测价值,有一定的临床意义。我们应尽早地预测 AKI 的发生,及早地有效的干预治疗,降低患者病死率,减少住院时间,减轻住院经济负担。

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