·专家共识·

碘对比剂诱导的急性肾损伤防治的专家共识

中华医学会临床药学分会 中国药学会医院药学专业委员会 中华医学会肾脏病学分会

通信作者：左笑丛：中南大学湘雅三医院药学部，长沙 410013，Email：zuoxc08@126.com；赵杰：郑州大学第一附属医院互联网医疗系统与应用国家工程实验室，郑州 450052，Email：zhaojie@zzu.edu.cn；张玉：华中科技大学同济医学院附属协和医院药学部，湖北省重大疾病精准用药医学研究中心，武汉 430022，Email：whxhzy@163.com；陈江华：浙江大学医学院附属第一医院肾脏病中心，浙江大学肾脏病研究所，浙江省肾脏病防治技术研究重点实验室，杭州 310003，Email：chenjianghua@zju.edu.cn

【摘要】 随着影像技术的不断发展，碘对比剂在介入治疗、血管造影等领域的应用日益增多。虽然碘对比剂的临床应用大大提高了疾病的诊疗水平，但其引起的急性肾损伤不容忽视。为降低碘对比剂诱导的急性肾损伤的发生率，中华医学会临床药学分会、中国药学会医院药学专业委员会和中华医学会肾脏病学分会组织国内专家，成立《碘对比剂诱导的急性肾损伤防治的专家共识》编写委员会，在参考国内外相关指南、共识及研究进展的基础上，结合我国的实际情况，针对碘对比剂的结构和分类、已上市的碘对比剂品种及理化性质、碘对比剂的应用现状、碘对比剂诱导的急性肾损伤的定义、流行病学、生物标志物、病理特征、危险因素与风险评估、预防措施和治疗手段等方面进行证据检索和评价，并充分讨论，制定了本共识，为临床医、药、护、技更有效、更安全地合理使用碘对比剂提供指导建议。

【关键词】 造影剂； 碘； 急性肾损伤； 对比剂肾病； 防治； 共识

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Expert consensus on prevention and treatment of iodine contrast media-induced acute kidney injury

Chinese Society of Clinical Pharmacy, Hospital Pharmacy Professional Committee of Chinese Pharmaceutical Association, Chinese Society of Nephrology

Corresponding author: Zuo Xiaocong, Department of Pharmacy, the Third Xiangya Hospital, Central South University, Changsha 410013, China, Email: zuoxc08@126.com; Zhao Jie, National Engineering Laboratory for Internet Medical Systems and Applications, the First Affiliated Hospital of Zhengzhou University, Zhengzhou 450052, China, Email: zhaojie@zzu.edu.cn; Zhang Yu, Department of Pharmacy, Hubei Province Clinical Research Center for Precision Medicine for Critical Illness, Union Hospital, Tongji Medical College, Huazhong University of Science and Technology, Wuhan 430022, China, Email: whxhzy@163.com; Chen Jianghua, Kidney Disease Center, the First Affiliated Hospital, Zhejiang University School of Medicine; Institute of Nephrology, Zhejiang University; Key Laboratory of Kidney Disease Prevention and Control Technology, Zhejiang Province, Hangzhou 310003, China, Email: chenjianghua@zju.edu.cn

前　言

为增强显影效果，提高疾病诊疗水平，碘对比剂（iodine contrast media，又称碘造影剂，简称对比剂）广泛应用于临床。然而，流行病学调查显示，11%～40%的患者在应用碘对比剂后出现急性肾损伤（acute kidney injury，AKI），即碘对比剂诱导的AKI（iodine contrast media-induced acute kidney injury），也称对比剂诱导的AKI（contrast-induced acute kidney injury，CI-AKI）或对比剂肾病（contrast-induced nephropathy，CIN）[1-4]。慢性肾脏病

表1　本专家共识的证据水平及推荐等级

级别	说明
证据水平	
A	资料来源于多项随机临床试验或荟萃分析
B	资料来源于单项随机临床试验或多项大规模非随机对照研究
C	资料来源于专家共识和/或小型临床试验、回顾性研究或注册登记
推荐等级	
Ⅰ	已证实和/或一致公认某治疗措施或操作有益、有效，应该采用
Ⅱ	某治疗措施或操作的有效性尚有争论
Ⅱa	指有关证据和/或观点倾向于有效，应用该治疗措施或操作是适当的
Ⅱb	指有关证据和/或观点尚不能充分证明有效，需进一步研究
Ⅲ	已证实和/或一致公认某治疗措施或操作无用和/或无效，并对某些病例可能有害，不推荐使用

(chronic kidney disease，CKD)患者接受碘对比剂后CI-AKI发病率高达40%，在这些患者中，CI-AKI与住院透析需求、长期肾衰竭和总死亡率(7%～31%)相关，且需要住院透析的患者中有13%可能永久依赖透析，因而CI-AKI与更长的住院时间和更高的成本相关[2]，已成为影响我国居民健康的重要疾病。因此，为了提高医务人员对CI-AKI的认识，规范碘对比剂的临床应用，降低CI-AKI的发生率，中华医学会临床药学分会、中国药学会医院药学专业委员会和中华医学会肾脏病学分会组织专家制定《CI-AKI防治的专家共识》。本共识在已有国内外相关指南和专家共识的基础上，结合CI-AKI的新证据，同时考虑我国的实际情况，参照表1中的证据水平及推荐等级，介绍了碘对比剂的结构和分类、已上市的碘对比剂品种及理化性质、碘对比剂的应用现状、CI-AKI的定义、流行病学、生物标志物、病理特征、危险因素与风险评估、预防措施和治疗手段等内容。本共识是我国最新的CI-AKI防治共识，以期为临床CI-AKI的规范防治提供参考。

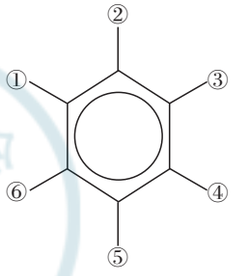
碘对比剂概述

一、基本结构和分类

1. 碘对比剂的基本结构：碘对比剂的基本结构是在苯环的①③⑤位上分别代入一个碘原子，②④⑥位分别结合三条侧链组成的三碘苯环衍生物[5-6]，具体结构如图1所示。

2. 碘对比剂分类：碘对比剂具有不同的分类方法。按照碘对比剂在溶液中是否电离出离子可分为离子型和非离子型碘对比剂；根据碘对比剂与血浆渗透浓度的大小可分为高渗、次高渗(曾称低渗碘对比剂)和等渗碘对比剂[1]；根据化学结构可分为单体型和二聚体碘对比剂[7-8]。

(1)离子型或非离子型碘对比剂：离子型与非离子型碘对比剂具有相似的苯环，苯环的①③⑤位上均有三个碘原子，但非离子型碘对比剂在苯环的②④⑥位上有多个羟基，使得亲水性的羟基分布于苯环的周围，将疏水性的碘苯基团屏蔽于其中，大大增加了化合物的水溶性并降低了化合物的毒副反应。见图1。



注：①③⑤位为碘原子；②位为羧基碱金属或葡甲胺盐或酰胺基结构；④⑥位为强亲水基团侧链，具有影响产品的亲水性和安全性等特性。结构特点：碘原子量大，吸收X线性能较强；碘与苯环键合，结构非常稳定；苯环结构具备多个有效侧链结合点，提供了不断改进整个分子结构、提高亲水性能和降低毒副作用的可能性

图1　碘对比剂的基本结构

(2)高渗、次高渗或等渗碘对比剂：高渗碘对比剂的渗透浓度高达血液渗透浓度的5倍以上(≥1 400 mmol/L)，次高渗碘对比剂的渗透浓度约为血液的2～3倍(600～800 mmol/L)，等渗碘对比剂渗透浓度与血液大致相同(约290 mmol/L)。

(3)单体型或二聚体碘对比剂：单体型碘对比剂只有3个碘原子；二聚体碘对比剂通过苯环5位的(酰)氨基将两个三碘苯环连接起来形成“二聚体”，因此在相同分子数时，其碘原子含量为单体型碘对比剂的2倍。在相同碘含量时，二聚体碘对比剂的分子数比单体型碘对比剂的分子数少，渗透浓度更低，但黏度更高。

推荐意见

1. 碘对比剂为1，3，5三碘苯环衍生物，根据分类方法不同，可分为离子型或非离子型碘对比剂，高渗、次高渗或等渗碘对比剂，单体型或二聚体碘对比剂。

二、国内已上市碘对比剂品种及理化性质

目前我国已上市碘对比剂的品种及具体的理化性质

表2 我国已上市碘对比剂的品种及理化性质

分类	结构	通用名	相对分子质量	碘含量(mgI/ml)	渗透浓度(mmol/L)	黏度(mPa·s/37 ℃)
第一代(高渗碘对比剂)	离子型单体	泛影葡胺	809	306	1 530	5.0
第二代(次高渗碘对比剂)	非离子型单体	碘海醇	821	140	322	1.5
				180	408	2.0
				240	520	3.4
				300	672	6.3
				350	844	10.4
	非离子型单体	碘帕醇	777	200	413	2.0
				250	524	3.0
				300	616	4.7
				370	796	9.4
	非离子型单体	碘佛醇	807	240	502	3.0
				300	651	5.5
				320	702	5.8
				350	792	9.0
	非离子型单体	碘普罗胺	791	150	328	1.5
				240	483	2.8
				300	607	4.9
				370	774	10.0
	非离子型单体	碘美普尔	778	300	520	4.5
				350	620	7.5
				400	726	12.6
	非离子型单体	碘比醇	835	300	695	6.0
				350	915	11.4
第三代(等渗碘对比剂)	非离子型二聚体	碘克沙醇	1 550	270	290	5.8
				320	290	11.4
	非离子型二聚体	碘曲仑	1 640	320	320	8.1

见表2[7,9]。

三、碘对比剂的应用现状

随着影像技术的不断发展，碘对比剂被广泛应用于临床，如介入治疗、血管造影、电子计算机断层扫描（computed tomography，CT）等，以增加病变组织与周围正常组织的对比度，提高病变部位检出率，更清楚地显示病变部位范围，明确病变性质，有利于病变的定位、定性及鉴别诊断。目前，碘对比剂主要应用于CT增强扫描、血管造影、尿路造影、关节造影、经内窥镜胰胆管造影、疝或瘘道造影、子宫输卵管造影、涎腺造影、经皮肝胆管造影、窦道造影、胃肠道造影和“T”形管造影等[9]。

推荐意见

2. 碘对比剂广泛应用于CT增强扫描、血管造影等，可增加病变部位与周围正常组织的对比度，提高病变部位检出率和检测质量。

CI-AKI的定义

目前，碘对比剂相关的AKI并没有统一的术语和定义，临床常规使用的术语为CI-AKI。此术语最早来源于2002年欧洲泌尿生殖放射学会（ESUR）的推荐意见[10]，将CI-AKI定义为在没有手术、肾毒性药物等因素的影响下，血管内给予碘对比剂后72 h内，血肌酐水平与基线相比升高25%或44.2 μmol/L。虽然2012年改善全球肾脏病预后组织（KDIGO）沿用了CI-AKI这一术语[11]，但将其定义为在没有手术、肾毒性药物等因素的影响下，血管内给予碘对比剂后48 h内血肌酐水平与基线相比绝对值升高26.5 μmol/L或7 d内相对值升高大于50%。2018年ESUR[9]考虑到接受碘对比剂检查的患者可能合并其他一些临床问题导致AKI，首次提出用碘对比剂后AKI（post-contrast acute kidney injury，PC-AKI）替代CI-AKI，泛指血管内注射碘对比剂后48 h内肾功能的急剧下降；然而，如果明确碘对比剂的使用与肾功能的急剧下降之间存在因果关系，

仍推荐应用CI-AKI这一术语。PC-AKI或CI-AKI定义为使用碘对比剂后72 h内血肌酐升高超过26.5 μmol/L或大于基线值的1.5倍。

推荐意见

3. 使用碘对比剂后72 h内发生的急性肾功能下降统称为PC-AKI，如果明确碘对比剂的使用与急性肾功能下降存在因果关系，可称为CI-AKI；PC-AKI或CI-AKI定义为使用碘对比剂后72 h内血肌酐升高超过26.5 μmol/L或大于基线值的1.5倍。（Ⅱb，C）

流行病学

CI-AKI是除缺血性肾损伤（指肾灌注不足和大手术等因素导致肾损伤）和药物性肾损伤（除碘对比剂外药物所致肾损伤）外，医院获得性AKI的第三大病因[12-14]。CI-AKI的发生率在不同基础情况的临床患者中存在很大差异，当患者合并高龄、基础肾功能不全、糖尿病、贫血、心力衰竭，同时使用肾毒性药物以及未进行水化时，患者发生CI-AKI的风险显著升高[12,14]。在低风险的门诊患者中，CI-AKI的发生率约为11%[1]，在估算肾小球滤过率（estimated glomerular filtration rate，eGFR）< 45 ml·min^{-1}·(1.73 m^2)$^{-1}$的人群中静脉使用碘对比剂后CI-AKI的发生率可至5%～20%[15]，合并糖尿病、充血性心力衰竭或老年的高危患者中CI-AKI发生率高达40%[2]。但由于不同研究中CI-AKI的诊断标准、患者的基线特征等不一致，CI-AKI在不同人群的真实发生率仍需进一步确证。

CI-AKI与慢性肾衰竭和心血管事件等不良临床结局有关，约25%～30%的CI-AKI患者会进展为慢性肾衰竭[16-17]；虽然因肾功能下降需要肾脏替代治疗的患者只占0.06%[18-19]，但51例基础肾功能正常而行冠状动脉介入术后需要透析的CI-AKI患者的2年生存率仅18.8%，1年病死率高达54.5%[20]；CI-AKI患者的平均住院时间和社会经济负担增加5～10倍[21]。

生物标志物

血肌酐是目前指南推荐的CI-AKI诊断的生物标志物[9]，大多数患者在应用碘对比剂后24～48 h会出现一过性的血肌酐水平升高，高峰值出现在造影后3～5 d，损伤轻微者往往在1～3周后可恢复基线水平[9]。虽然血肌酐的临床应用非常广泛，但由于肌酐为水溶性、全身分布广，易受饮食、体重等因素影响，且血肌酐水平的升高往往滞后于实际的肾脏损伤，在eGFR的恢复期会出现急剧下降，近20%的患者甚至无法通过血肌酐水平诊断CI-AKI[22-23]。因此，越来越多的研究在探寻更合适的CI-AKI的诊断标志物，如胱抑素C[24]、嗜中性粒细胞明胶酶相关脂质运载蛋白（NGAL）[25-28]和肾损伤分子-1（KIM-1）[29]等。胱抑素C在碘对比剂暴露后1 h即升高，分布容积仅为血肌酐的1/3，受其他因素的影响较小，使用碘对比剂后24 h内胱抑素C水平升高10%，已在一些临床试验中用于诊断CI-AKI[24]。NGAL为远端肾单位损伤的标志物，主要表达于髓袢升支粗段、远端小管和集合管，在碘对比剂暴露后6 h就升高，该指标在重症患者中已常规用于AKI的早期诊断，但在CI-AKI中还没有得到指南的一致推荐[25-28]。KIM-1主要标记肾小管损伤，正常的肾小管没有表达，出现损伤时会表达，具有一定的特异性[29-30]。已有一些基于以上指标定义CI-AKI的临床研究[24-29]，但这些指标的临床应用仍需进一步证实。

推荐意见

4. 推荐将血肌酐水平用于诊断CI-AKI，其他新的生物标志物可根据临床需要进行选用。（Ⅰ，A）

病理特征

碘对比剂对肾小球的损伤不明显[31-32]。CI-AKI的基本病理变化为急性肾小管坏死，肾小管上皮细胞出现严重的颗粒和空泡变性[33]；各段肾小管上皮细胞均可见凝固性坏死和崩解脱落，细胞碎屑淤积于肾小管腔，位于肾髓质的髓袢和集合管病变尤为严重；肾间质可见弥漫性水肿，存在淋巴细胞和单核细胞浸润[34]。CI-AKI的病理机制复杂，目前尚未完全阐明[3]，主要可能与碘对比剂对肾小管上皮细胞和血管内皮细胞的直接细胞毒性作用、血管活性介质和高黏度导致的肾血流动力学改变，导致肾脏低灌注和氧化应激等有关[35-36]。经肾小球滤过的碘对比剂几乎100%以原型经肾小球滤过，并可经肾小管上皮细胞的转运体进入肾小管上皮细胞，局部高浓度的碘对比剂可致肾小管刷状缘丢失、细胞膜完整性破坏、细胞碎屑脱落进入肾小管间隙，从而使尿液中的碘对比剂进一步滞留，更多的碘对比剂进入肾小管间质间隙，形成恶性循环，加重损伤[37]。

碘对比剂可使肾脏血管网在由一氧化氮（NO）释放介导的内皮短暂依赖性舒张后持续收缩数秒钟至数分钟[38]，由此导致的肾髓质缺血缺氧对CI-AKI的病理改变至关重要[39-42]。肾脏外髓质氧气需求量大但供应量低，易受到缺血缺氧的影响[39,43]，而碘对比剂可使外髓质血流量减少40%，氧输送减少60%[44]，从而造成髓质代谢需求与血液供应之间的不匹配，导致活性氧（reactive oxygen species，ROS）的产生，使肾小管发生氧化损伤[45]。ROS可诱导内皮素、血管紧张素Ⅱ、腺苷和血栓素A2合成增加，以及NO合成减少，促进血管收缩，引起肾脏微循环改变和远端缺血，而缺血又可导致氧自由基和ROS形成增加，形成恶性循环[3]。

所有类型的碘对比剂在体外均发挥细胞毒性作用，几乎所有类型的细胞（包括血管内皮细胞和肾小管上皮

细胞）暴露于碘对比剂时均表现出严重的细胞损伤或凋亡迹象[43]。碘对比剂细胞毒性作用的分子机制可能主要涉及激活Caspase-3、Caspase-9和Bcl-2通路，直接参与凋亡信号通路，并通过钙稳态失衡引起膜蛋白的重新分配、DNA碎片化、细胞间连接的破坏、细胞增殖减少、线粒体功能障碍[43,46-47]。此外，碘对比剂可通过其强大的氧化能力刺激肾小管上皮细胞和血管内皮细胞产生有害的氧自由基和ROS[48-49]；ROS可通过影响线粒体、细胞核DNA、细胞膜脂质和细胞蛋白[48]，激活c-Jun N端激酶（JNK）和p38MAPK激酶，从而促进细胞凋亡和坏死[50]。最新文献报道，细胞自噬[51-53]、焦亡[54]、铁死亡[55]等方式也参与碘对比剂导致的细胞死亡。

推荐意见

5. CI-AKI的主要病理特点为肾小球病变不明显，肾小管急性损伤；其病理机制复杂，尚未完全阐明，可能主要与髓质缺血缺氧以及碘对比剂对肾小管的直接毒性有关。（Ⅱa，B）

危险因素与风险评估

一、危险因素

（一）患者相关的因素

1. 年龄：年龄与CI-AKI之间的相关性仍不明确[9,56-60]。2004年，Mehran等[61]将年龄＞75岁纳入CI-AKI的风险预测模型；对已发表的风险预测模型进行评估后发现，纳入了年龄变量的模型预测能力及辨别能力更好[62]。2021年美国放射学会（ACR）指南[63-64]指出年龄＞60岁的患者在接受碘对比剂前需评估患者的肾功能；然而，2018年ESUR[9]以及瑞典泌尿放射学会（SSUR）[65]并未将高龄作为CI-AKI的危险因素，并指出大多数已确定的风险因素是基于非对照研究中的多变量模型，基于对照研究的荟萃分析也没有证明年龄为CI-AKI的影响因素。CI-AKI的发生风险与高龄之间的联系，可能是随着年龄增长，患者常伴有肾功能受损或其他合并疾病，因此无法确认年龄为CI-AKI的独立影响因素。而儿童中CI-AKI的发生率很低，在一项对100例接受了碘对比剂增强的计算机断层扫描的1个月到12岁的儿童的前瞻性队列研究中，多因素Logistic回归分析结果显示年龄小于2岁与CI-AKI独立相关，但其结论有待进一步证实[66]。

推荐意见

6. 高龄与CI-AKI的相关性不明确，但考虑肾功能可能随年龄增长而减退，仍建议年龄＞60岁的患者在使用碘对比剂之前进行肾功能评估。（Ⅱa，C）

2. 性别：虽然部分研究发现女性发生CI-AKI的风险较高[9]，但在接受碘对比剂检查的9 300例患者队列中，多因素Logistic回归分析提示，男性是术后AKI的独立预测因素[56]。因此，性别对CI-AKI的影响尚未得到严格证实，国内外指南均未将其作为CI-AKI的危险因素[9,65,67-71]。

推荐意见

7. 不建议将性别作为评估CI-AKI的影响因素。（Ⅲ，C）

3. 合并疾病：

（1）基线肾功能受损：与肾功能正常的患者相比，在使用碘对比剂前即存在CKD的患者，如果接受碘对比剂检查，其发生CI-AKI的风险明显增加，且CI-AKI的风险随着CKD分期的增加而升高：eGFR≥60 ml·min^{-1}·(1.73 m^2)$^{-1}$时，CI-AKI的发生率约为5%；eGFR在45～59 ml·min^{-1}·(1.73 m^2)$^{-1}$之间时约为10%；eGFR在30～44 ml·min^{-1}·(1.73 m^2)$^{-1}$时约为15%；eGFR＜30 ml·min^{-1}·(1.73 m^2)$^{-1}$时约为30%[67]。然而各指南中推荐的CI-AKI的eGFR阈值存在差异，日本循环学会（JCS）2018年指南[72]和SSUR指南[65]分别推荐将eGFR＜60 ml·min^{-1}·(1.73 m^2)$^{-1}$和eGFR＜45 ml·min^{-1}·(1.73 m^2)$^{-1}$作为CI-AKI的危险因素，特别是在合并多种非肾脏危险因素的情况下；2018年ESUR指南[9]进一步根据碘对比剂的给药途径，提出动脉注射碘对比剂时eGFR＜45 ml·min^{-1}·(1.73 m^2)$^{-1}$或静脉注射碘对比剂时eGFR＜30 ml·min^{-1}·(1.73 m^2)$^{-1}$为CI-AKI的独立危险因素。

基线肾功能对评估患者的CI-AKI风险具有重要意义，但在临床实践中并非所有患者在使用碘对比剂前都能获得肾功能检测的结果。一项回顾性分析显示，非CKD的卒中患者在没有基础肾功能检测结果时即进行碘对比剂检查是安全的[73]。我国《碘对比剂使用指南（第2版）》[5]建议，对于择期检查的患者，应当在检查前7 d内检测血肌酐；在不立即进行检查就会对患者造成危害的紧急情况下，可不进行血肌酐检测。2018年ESUR指南[9]同样推荐，对于因急性疾病或慢性疾病急性发作住院的患者，建议在进行碘对比剂检查前7 d内进行肾功能检测；对于急诊患者，在能推迟检查的情况下，可先检测患者肾功能，无法推迟检查的患者可以使用行碘对比剂检查前3个月内的肾功能检测结果。对CI-AKI高危患者，接受碘对比剂检查后48 h内应复查肾功能，若48 h内诊断为CI-AKI，应尽早采取干预措施，并随访监测患者肾功能至少30 d[9]。

推荐意见

8. 基线肾功能受损为CI-AKI的独立危险因素。（Ⅰ，A）

9. 动脉注射碘对比剂时eGFR＜45 ml·min^{-1}·(1.73 m^2)$^{-1}$或静脉注射碘对比剂时eGFR＜30 ml·min^{-1}·(1.73 m^2)$^{-1}$可作为CI-AKI的危险因素。（Ⅱb，C）

10. 非紧急情况下，建议在行碘对比剂检查前7 d内进行肾功能检测；紧急情况下，若能推迟碘对比剂检查，建议先行肾功能检测；在不立即行碘对比剂检查就会对患者造成危害的情况下，仍建议先行肾功能检测，但可在没有肾功能检测结果的情况下紧急行碘对比剂检查。（Ⅱa，C）

11. 对于CI-AKI高危的患者，接受碘对比剂检查后48 h内应复查肾功能，若48 h内诊断为CI-AKI，需尽早采取干预措施，并随访监测患者肾功能至少30 d。（Ⅱa，C）

（2）糖尿病：糖尿病是CI-AKI常见的危险因素，通过评估接受冠状动脉造影或受冠状动脉介入治疗患者的CI-AKI风险，发现合并糖尿病患者的CI-AKI发生率较高[74]。但糖尿病患者合并疾病较多，因此糖尿病是否为CI-AKI的独立危险因素并不明确。2020年一项纳入1 023例冠心病患者的前瞻性开放队列研究发现，21.2%的CI-AKI患者合并糖尿病，12.9%的患者同时患有糖尿病和肥胖症，但糖尿病并不是CI-AKI的独立危险因素[75]；而2021年一项纳入84项研究的荟萃分析结果提示糖尿病是CI-AKI的独立危险因素，但肾功能正常的糖尿病患者发生CI-AKI的风险明显下降[76]。因此，糖尿病与CI-AKI的相关性可能与患者的合并疾病、基础肾功能等相关，尚需要在更大规模的研究中进一步评估。

推荐意见

12. 糖尿病是CI-AKI的危险因素，但不推荐将其作为评估CI-AKI风险的独立危险因素。（Ⅱb，C）

（3）高尿酸血症：目前已有一些研究发现高血清尿酸水平可能与CI-AKI的发生有关。一项纳入1 440例患者的队列研究结果显示，血清尿酸水平≥8.0 mg/dl与CI-AKI风险增加有关，相比于血清尿酸水平＜8.0 mg/dl，血清尿酸水平≥8.0 mg/dl的患者CI-AKI的发生风险更高[77]。几项荟萃分析结果也同样显示高尿酸血症与CI-AKI的发生独立相关[78-80]。

推荐意见

13. 高尿酸血症可能与CI-AKI的风险增加相关。（Ⅱb，C）

（二）碘对比剂的类型

由于碘对比剂的物理化学性质（主要是渗透浓度和黏度）在其肾毒性中起重要作用，因此降低碘对比剂渗透浓度可减少CI-AKI的发生率[58]。然而，次高渗或等渗碘对比剂在降低渗透浓度的同时，黏度会增加，从而导致肾超滤液黏度增加，肾小管血流阻力也随之增加，最终引起肾小管损伤。使用离子型高渗碘对比剂的患者发生CI-AKI的风险明显增加，在31项研究的荟萃分析中，使用非离子型次高渗碘对比剂的患者血肌酐水平升高超过44 μmol/L的发生风险是离子型高渗碘对比剂的61%（95% *CI* 0.48～0.77）[81]。国内外指南一致推荐患者使用非离子型次高渗或等渗碘对比剂，不推荐使用离子型高渗碘对比剂[9，65，67-71]。

然而，次高渗碘对比剂与等渗碘对比剂导致CI-AKI的风险是否不同以及不同的次高渗或等渗碘对比剂发生CI-AKI的风险是否存在差异并不明确。多项临床试验和荟萃分析均显示，没有证据表明使用等渗碘对比剂的患者CI-AKI发生风险显著低于非离子型的次高渗碘对比剂，且不同次高渗或等渗碘对比剂导致CI-AKI的风险差异不显著[82-86]。但ACR和美国肾脏基金会（NKF）关于肾病患者静脉使用碘对比剂的共识声明[67]中提到，有间接证据表明，与其他次高渗碘对比剂相比，碘海醇可能有更高的CI-AKI风险，但潜在的风险差异尚未得到证实。2009年美国心脏病学会（ACC）/美国心脏协会（AHA）ST段抬高心肌梗死患者处理指南[87]建议使用除碘海醇以外的等渗碘对比剂或次高渗碘对比剂。但2011年美国心脏病学会基金会（ACCF）/AHA不稳定型心绞痛/非ST段抬高心肌梗死患者的治疗指南[68]和2011年ACCF/AHA/美国心血管造影和介入学会（SCAI）经皮冠状动脉介入（percutaneous coronary intervention，PCI）治疗指南[69]同样指出目前的数据不足以证明在次高渗和等渗碘对比剂中哪些品种的CI-AKI风险更低。

推荐意见

14. 次高渗和等渗碘对比剂发生CI-AKI的风险低于高渗碘对比剂，推荐使用非离子型次高渗或等渗碘对比剂，不推荐使用离子型高渗碘对比剂。（Ⅰ，A）

15. 不同品种的等渗碘对比剂或次高渗碘对比剂发生CI-AKI的风险是否不同，目前还没有明确的结论。（Ⅱb，C）

（三）碘对比剂的剂量

ESUR[9]和SSUR[65]都认为动脉内给予大剂量碘对比剂是CI-AKI的重要危险因素。碘对比剂的肾毒性效应可能与使用的剂量成正比，使用更高剂量的碘对比剂与CI-AKI发生率和死亡率增加相关[59]，碘对比剂剂量小于100 ml可显著降低冠状动脉造影术后CI-AKI的发生率[88]。因此，碘对比剂的剂量是CI-AKI的影响因素，临床上应该尽可能选择满足诊疗需求的最低剂量[89]。

根据体重和基线血肌酐水平可估算碘对比剂剂量的上限（最大碘对比剂剂量）[90]，公式如下：碘对比剂限值=[5 ml/kg×体重（kg）]/Scr（mg/dl）（即使计算出的剂量＞300 ml，最大碘对比剂剂量也不能超过300 ml）[89]。在未超过最大碘对比剂剂量时碘对比剂诱导的肾功能紊乱（血肌酐水平升高≥1 mg/dl）和CI-AKI的发生率分别为2.00%和0.18%，超过最大碘对比剂剂量时分别为21.0%和2.4%[90-91]。在接受冠状动脉介入治疗的患者中，碘对比剂剂量超过最大剂量被确认为CI-AKI和死亡的重要预测因子[9，92-93]。高剂量的碘对比剂为CI-AKI的第三大危险因素，纳入碘对比剂剂量作为变量的CI-AKI风险预测模型显示预测性能良好[62]。

碘对比剂剂量/肌酐清除率可能与碘对比剂血药浓度-时间曲线下面积密切相关，与碘对比剂剂量绝对值相比，这一指标是CI-AKI的一个更强的预测因子[88]。

在3 179例接受PCI的非选择性患者中，碘对比剂剂量/肌酐清除率≥3.7%能够识别出术后血肌酐异常升高的高危患者[94]。碘对比剂剂量与eGFR比值（g-I/eGFR）≥1.0的患者发生CI-AKI的风险为25%，显著高于g-I/eGFR < 1.0的患者（3%）[89]。虽然短时间内（48～72 h）重复使用碘对比剂增加CI-AKI的发生风险[95]，但在没有其他高危因素时，只要g-I/eGFR不超过1.0，即使重复注射碘对比剂，患者的CI-AKI风险也较低[96]；在有CI-AKI危险因素的患者中，反复注射碘对比剂最好间隔至少48 h，并在此之前检测肾功能[95]。

推荐意见

16. 碘对比剂的剂量为CI-AKI的危险因素，建议在满足临床诊疗需求的前提下尽量减少碘对比剂的剂量。（Ⅱb，C）

17. PCI患者短时间内（48～72 h）重复使用碘对比剂为CI-AKI的危险因素，当没有其他危险因素存在时，只要g-I/eGFR不超过1.0，可重复注射碘对比剂；在有其他危险因素时，反复注射碘对比剂最好间隔至少48 h，并在此之前检测肾功能。（Ⅱb，C）

（四）碘对比剂的给药途径

碘对比剂的主要给药途径包括静脉给药和动脉给药，尽管2018年ESUR[9]提出动脉注射碘对比剂较静脉注射导致CI-AKI的风险更高，但没有前瞻性随机对照试验（randomized-controlled trials，RCT）证实这种关联。动脉注射碘对比剂导致的高CI-AKI风险可能与静脉系统和动脉系统对氧化应激的反应性不同有关[97]，与动脉注射相比，静脉注射的碘对比剂在到达动脉系统之前经过全身血液稀释，到达肾脏的浓度较低，从而降低CI-AKI的风险[9]。然而目前评估不同的给药途径导致CI-AKI风险的临床试验结论不一致，2016年一项纳入1 969例患者的队列研究结果显示，虽然两种给药途径的AKI总发生率相似，但eGFR < 30 ml·min^{-1}·(1.73 m^2)$^{-1}$的患者静脉注射碘对比剂的AKI发生率高于动脉注射[98]；2011年一项多中心回顾性研究[99]表明，动脉注射碘对比剂和静脉注射碘对比剂的AKI的总体发生率分别为7.6%和8.7%，差异无统计学意义；2013年一项回顾性研究比较了动脉注射碘对比剂和静脉注射碘对比剂后患者CI-AKI的发生风险，发现无论使用何种标准来定义CI-AKI，其结果均表明，两种给药途径的CI-AKI的发生风险相似[100]。

推荐意见

18. 静脉注射碘对比剂与动脉注射碘对比剂导致的CI-AKI发生风险差异不显著。（Ⅱb，C）

（五）合并药物

既往研究报道，非选择性非甾体抗炎药、选择性环氧合酶-2（Cox-2）抑制剂、抗菌药物和化疗药物等均可影响肾功能，诱发AKI[101]；在合并使用肾毒性药物时，CI-AKI的风险明显升高[102]，因此2018年ESUR提出应在临床可能的情况下尽量减少肾毒性药物的使用[9]。2020年ACR和NKF共识[67]同样提出，合并使用肾毒性药物（如氨基糖苷类药物或化疗药物等）的患者在使用碘对比剂前后应检测血肌酐水平；对于已经发生AKI或eGFR < 30 ml·min^{-1}·(1.73 m^2)$^{-1}$的患者，使用碘对比剂前后48 h内不推荐使用非必需的肾毒性药物及可影响肾脏功能的药物（如非甾体抗炎药、利尿剂、氨基糖苷类抗菌药物、两性霉素B、唑来膦酸盐、铂类药物和甲氨蝶呤等抗肿瘤药物）。

推荐意见

19. 合并使用可影响肾功能的药物及肾毒性药物（如非甾体抗炎药、利尿剂、氨基糖苷类抗菌药物、两性霉素B、唑来膦酸盐、铂类药物和甲氨蝶呤等抗肿瘤药物）的患者推荐在使用碘对比剂前后检测肾功能。（Ⅱb，C）

长期使用血管紧张素转换酶抑制剂（ACEI）或血管紧张素Ⅱ受体阻断剂（ARB）由于对肾小球出球小动脉的扩张作用强于对入球小动脉的作用，可能会引起血肌酐水平的升高，但是目前并没有循证证据或指南推荐应在造影期间停药，只需要检测肾功能，如果2个月内肌酐上升超过30%才考虑停药。

二甲双胍为临床一线降糖药物，与碘对比剂合并使用具有潜在的乳酸酸中毒风险，但在使用碘对比剂前后是否需要停用二甲双胍、如何停用以及停用之后如何重启在说明书及不同的指南推荐方案中存在差异，见表3。

推荐意见

20. 使用ACEI/ARB的患者与碘对比剂合用时无证据证明需要停药，但需检测肾功能；使用二甲双胍的患者需根据患者的肾功能以及碘对比剂的给药途径进行个体化停用及重启。（Ⅱb，C）

二、风险评估方法

1. 预测冠状动脉造影或冠状动脉介入术围术期患者CI-AKI风险的评估方法：目前预测CI-AKI风险的评估模型中最多的是基于冠状动脉造影或冠状动脉介入术围术期患者构建的。2004年Mehran等[61]构建了CI-AKI评分模型，是目前临床上使用最广泛的CI-AKI风险预测模型，该模型纳入了美国8 357例PCI治疗患者，共包括9个变量（见表4），对应的风险见表5。

虽然在一些外部验证队列中显示Mehran评分具有较好的CI-AKI预测能力[104-110]，但Mehran评分具有术中变量，如碘对比剂用量、是否进行主动脉内球囊反搏（intra-aortic balloon pump，IABP）等，并不适用于在使用碘对比剂前进行风险预测。因此，有多项研究探索了新的评分模型：Ni模型[111]、Yao模型[112]、AGEF评分[113]、ACEF-MDRD评分[114]、SYNTAX评分[115]、CHADS2评分[116]、Zwolle评分[117]、

表3　说明书及国内外指南推荐的二甲双胍在造影前后的停用和重启时机

来源	患者	造影前	造影后
欧洲泌尿生殖放射学会指南（2018年版）[9]	二级碘对比剂暴露，如果eGFR＞30 ml·min^{-1}·(1.73 m^2)$^{-1}$且无AKI的证据	不停用	
	二级碘对比剂暴露，如果eGFR＜30 ml·min^{-1}·(1.73 m^2)$^{-1}$；动脉内注射碘对比剂的一级碘对比剂暴露；已有AKI	注射碘对比剂时停用	停药48 h，复查肾功能无恶化后重启
二甲双胍临床应用专家共识（2018年版）[103]	eGFR＞60 ml·min^{-1}·(1.73 m^2)$^{-1}$	造影前或造影时停用	至少48 h且复查肾功能无恶化后重启
	eGFR 45～59 ml·min^{-1}·(1.73 m^2)$^{-1}$	术前48 h停用	停药48～72 h，复查肾功能无恶化后重启
	eGFR＜45 ml·min^{-1}·(1.73 m^2)$^{-1}$	禁用二甲双胍	禁用二甲双胍
中国食品药品监督管理局说明书	eGFR＞60 ml·min^{-1}·(1.73 m^2)$^{-1}$	造影前或造影时停用	至少48 h且复查肾功能无恶化后重启
	eGFR 45～59 ml·min^{-1}·(1.73 m^2)$^{-1}$	术前48 h停用	停药48 h，复查肾功能无恶化后重启
	eGFR＜45 ml·min^{-1}·(1.73 m^2)$^{-1}$	禁用二甲双胍	禁用二甲双胍

注：一级碘对比剂暴露：经左心、胸主动脉、肾动脉等处注射的碘对比剂未经血液循环稀释，到达肾动脉的碘对比剂为未稀释的高浓度；二级碘对比剂暴露：经右心、肺动脉、颈动脉、锁骨下动脉、肱动脉、冠状动脉、肠系膜动脉等处注射的碘对比剂经肺循环和外周循环稀释后，到达肾动脉时为较低浓度

表4　Mehran评分内容[61]

危险因素	评分
低血压	5
IABP	5
充血性心力衰竭	5
年龄＞75岁	4
贫血	3
糖尿病	3
碘对比剂用量	每100 ml记1分
肾功能[a]	
血肌酐＞1.5 mg/dl	4
eGFR 40~60	2
eGFR 20~40	4
eGFR＜20	6

注：低血压：收缩压≤80 mmHg至少持续1 h，需要药物或者动脉内正性肌力支持；IABP：主动脉内球囊反搏，术前24 h内进行IABP；充血性心力衰竭：充血性心力衰竭等级Ⅲ/Ⅳ（纽约心脏病协会分类）或者肺水肿史；贫血：男性基线血细胞比容＜39%，女性基线血细胞比容＜36%；eGFR：估算肾小球滤过率，单位为ml·min^{-1}·(1.73 m^2)$^{-1}$；a：肾功能评分可根据血肌酐水平或eGFR水平，两者不叠加

表5　Mehran评分对应风险[61]

风险评分	CI-AKI风险	透析风险
0~5分	7.5%	0.04%
6~10分	14.0%	0.12%
11~16分	26.1%	1.09%
16分以上	57.3%	12.6%

Chen评分[118]、CAC评分[119]、CI-AKI模型[120]等，与Mehran评分模型相比，这些均显示出了相似或者更好的性能；Liu等[121]在422例接受冠状动脉介入治疗的ST段抬高型心肌梗死患者中前瞻性比较了Mehran等6种模型预测CI-AKI的准确性，结果显示所有的模型对CI-AKI发生的预测表现良好，但ACEF风险评分对患者院内和3年全因死亡率及主要不良临床事件的预测表现更佳；而在另一项纳入了1 247例行择期或急诊冠状动脉介入治疗患者的研究中[122]，17个模型都只显示弱至中度的判别能力，并没有哪一个模型显示出极佳的优越性；Yin等[123]在中国人群中验证比较了8个CI-AKI术前风险评分的预测能力，当CI-AKI定义为血肌酐升高44.2 μmol/L时，Maioli评分的辨别和校准能力最好；而当CI-AKI定义为血肌酐升高44.2 μmol/L或25%和定义血肌酐升高44.2 μmol/L或50%时，所有的预测模型的准确性均不能达到统计要求。

2. 预测总体人群CI-AKI风险的评估方法：临床上碘对比剂的使用并不仅仅局限于冠状动脉造影或冠状动脉介入治疗的患者，因此，近年来也有多个研究开发了针对总体人群CI-AKI的风险评分模型。Yin等[124]基于8 800例使用碘对比剂的中国患者开发了一个仅包含术前变量的风险预测模型，模型显示出较好的预测能力（ROC曲线下面积为0.907，预测精确度为80.8%，敏感性为82.7%，特异性为78.8%）；Wilhelm-Leen等[125]、Zhang等[126]、Kulkarni等[127]构建的模型在预测CI-AKI风险方面具有较好的敏感性和准确性；此外针对癌症患者[128]、危重患者[129]、卒中患者[130]、心肌梗死患者[131]的CI-AKI风险预测模型均显示有较好的预测能力，但以上模型的准确性还缺乏可靠的外部验证，临床实用性有待进一步评价。

推荐意见

21. 接受冠状动脉造影或冠状动脉介入治疗的患者可选择Mehran评分评估CI-AKI风险，其他模型的预测能力仍需大规模的外部验证进行证实。（Ⅱb，C）

预防措施

一、水化

水化疗法是目前公认的预防CI-AKI的有效措施，其可通过扩容改善肾血流量、稀释肾小管内的碘对比剂浓度诱导利尿、减少肾素-血管紧张素系统的激活、减少抗利尿激素的分泌等降低CI-AKI的风险。一项大型RCT的AMACING研究表明，大于18岁的高风险患者[eGFR 30～59 ml·min^{-1}·(1.73 m^2)$^{-1}$]，采用静脉补液预防CI-AKI与没有任何预防措施组的CI-AKI发生率没有明显差别，且没有任何预防措施组的成本更低[4]。更多的RCT研究表明静脉水化可以预防PCI患者[132-135]和CT-肺动脉造影患者[136]的CI-AKI发生，其发生率显著低于安慰剂组，且可减少紧急透析次数[135]；荟萃分析同样表明水化对预防CI-AKI有较好的效果[137-143]。目前未发现有研究报道水化会导致死亡率或其他不良事件发生增加。因此，2012年KDIGO AKI临床实践指南[71]和2018年ESUR的CI-AKI防治指南[144]均推荐采用水化疗法预防CI-AKI。

推荐意见

22. 患者如果没有扩容的禁忌证，推荐水化预防CI-AKI。（Ⅰ，A）

1. 口服水化与静脉水化的选择：无论是静脉水化还是口服水化均可以降低CI-AKI的风险。虽然目前研究表明静脉水化与口服水化在预防CI-AKI方面无明显差异，但研究受到样本量小、异质性和缺乏确切临床结果的限制[134, 145-155]。相比静脉水化，口服水化预防CI-AKI因难以监测或控制水化速度，2018年EUSR的CI-AKI防治指南[144]不建议使用口服水化作为CI-AKI的首选或唯一的预防策略，除了建议首选静脉水化外，也不限制进行口服水化。

推荐意见

23. 不推荐口服水化作为首选或唯一的预防策略。（Ⅰ，A）

2. 水化晶体的选择：生理盐水（0.9% NaCl）和碳酸氢钠溶液（1.4%或154 mmol/L $NaHCO_3$）是目前研究最多的静脉水化溶液。但是生理盐水和碳酸氢钠溶液在预防CI-AKI有效性的差异方面并未有统一定论：最初的研究倾向于使用碳酸氢盐，碳酸氢盐可以碱化尿液并减少活性氧的形成[48]，许多RCT研究[156-160]和荟萃分析[143, 161-173]均表明静脉注射碳酸氢钠预防CI-AKI比氯化钠更有效；但在后续的一些RCT研究[174-182]和荟萃分析[140, 183]中并没有得到相同的结论；最近的PRESERVE试验研究[181]表明在接受血管造影的高风险患者中，静脉注射碳酸氢钠与静脉注射氯化钠对预防死亡、需要透析或90 d肾功能持续下降或CI-AKI没有明显获益。因此，静脉注射碳酸氢盐水化与生理盐水水化具有相似的有效性，但在临床应用时需考虑碳酸氢盐溶液成本较高、可能导致碱中毒等，为患者选择合适的水化晶体。

推荐意见

24. 生理盐水和碳酸氢钠溶液均可作为水化的晶体溶液，可根据临床需要选择合适的水化晶体。（Ⅰ，A）

3. 水化方案的选择：目前对于最佳的水化方案（静脉补液的速度、体积、时间等）仍没有共识。住院患者和门诊患者由于CI-AKI的风险、水化条件等存在差异，在进行水化时应进行个体化调整。而大多数研究都是在因冠状动脉造影或冠状动脉介入治疗住院的心脏病患者中进行，少纳入门诊、CT增强或eGFR小于30 ml·min^{-1}·(1.73 m^2)$^{-1}$的患者[156, 180, 184-190]，且很少评估不同的给药方案之间的有效性[136, 191-192]。目前国内外指南推荐的水化治疗方案见表6。

没有数据表明患有严重肾功能损害（CKD 4～5期）或严重心力衰竭（NYHA 3～4级）的患者应该接受不同的水化治疗方案。然而，AMACING试验研究表明大量静脉水化可加重急性心力衰竭，引起肺水肿[4]。2018年ESUR的CI-AKI防治指南[144]提出，这些患者的水化方案应该根据类型、体积和持续时间进行个体化调整，但具体如何调整仍有待进一步的研究。

推荐意见

25. 不同水化方案的优劣仍不明确，个体化的最佳水化方案（静脉补液的速度、体积、时间等）仍需进一步的研究。（Ⅱb，A）

二、抗氧化治疗

1. N-乙酰半胱氨酸（N-acetylcysteine，NAc）：NAc是自由基的直接清除剂，并通过NO介导的途径改善血液流动、扩张血管[197]，被认为对CI-AKI有保护作用[198-200]，但临床研究并未证实NAc在预防CI-AKI方面具有确切的效果。虽然荟萃分析显示，NAc无论是否加用高剂量他汀类药物，在水化作用下都能有效预防CI-AKI[142, 183, 197, 201]，与对照组相比，NAc能显著降低造影后CI-AKI的发生风险（风险比：0.78，95%*CI* 0.68～0.90）[202]，但最近的RCT或荟萃分析并未显示NAc对冠状动脉或周围血管造影术[186, 203-211]或行增强CT患者[212-213]的CI-AKI具有预防作用。PRESERVE试验研究也表明进行血管造影的肾脏病高风险患者，口服NAc无法降低死亡、需要透析、90 d后的持续肾功能下降或CI-AKI的发生风险[181]。生理盐水或碳酸氢钠是否联合使用NAc时比较，未发现联合使用NAc有任何附加的保护作用[178, 214-218]。

表6　国内外指南推荐的住院患者以及门诊患者的水化方案

患者	指南	造影前	造影后
住院患者	2010年预防CI-AKI的快速手册[193]	扩容对eGFR < 60 ml·min^{-1}·(1.73 m^2)$^{-1}$的患者尤为重要；造影前12 h以1 ml·kg^{-1}·h^{-1}予以0.9%氯化钠溶液或154 mmol/L碳酸氢钠溶液	维持12 h
	2017年美国放射学会指南[194]	6~12 h以100 ml/h的速度输注0.9%氯化钠溶液	继续输注4~12 h
	2018年欧洲心肌血管重建指南[195]	对于慢性肾脏病3b和4期的患者，如估计碘对比剂剂量 > 100 ml，造影前12 h以1 ml·kg^{-1}·h^{-1}的速度输注0.9%氯化钠溶液（如果左心室射血分数 < 35%或纽约心脏病协会分级 > 2速度为0.5 ml·kg^{-1}·h^{-1}）	继续输注至24 h
	2018年欧洲泌尿生殖放射学会指南[144]	静脉注射碘对比剂或二级暴露的动脉注射碘对比剂：	
		造影前1 h以3 ml·kg^{-1}·h^{-1}的速度输注1.4%碳酸氢钠（154 mmol/L融入5%葡萄糖溶液）	无推荐
		造影前3~4 h以1 ml·kg^{-1}·h^{-1}的速度输注生理盐水	继续输注4~6 h
		一级暴露的动脉注射碘对比剂：	
		造影前1 h以3 ml·kg^{-1}·h^{-1}的速度输注1.4%碳酸氢钠（154 mmol/L融入5%葡萄糖溶液）	以1 ml·kg^{-1}·h^{-1}继续输注4~6 h
		造影前3~4 h以1 ml·kg^{-1}·h^{-1}的速度输注生理盐水	继续输注4~6 h
门诊患者	2010年预防CI-AKI的快速手册[193]	造影前1 h以3 ml·kg^{-1}·h^{-1}予以0.9%氯化钠溶液或154 mmol/L碳酸氢钠溶液	维持6 h
	2014年欧洲心脏病学会心胸手术指南[196]	无法预先水化的患者先静脉注射250 ml生理盐水（如左心室功能不全降至150 ml），后静脉注射0.25~0.50 mg/kg呋塞米，当患者尿量 > 300 ml/h可进行冠状动脉操作	维持4 h
	2018年欧洲心肌血管重建指南[195]	造影前1~3 h输注0.9%氯化钠溶液	继续输注至6 h

推荐意见

26. NAc预防CI-AKI的有效性仍不明确。（Ⅱb，A）

2. 他汀类药物：3-羟基-3-甲基戊二酰辅酶A还原酶抑制剂，又称他汀类药物，广泛用于降低血清胆固醇水平[219]。已有部分RCT研究[220~225]和荟萃分析[142, 183, 226~235]证明他汀类药物可以降低CI-AKI的发生率，但也有一些相反的结果[199~201, 226, 236~243]。荟萃分析显示，与对照组相比，使用大剂量、短期他汀类药物治疗的患者，CI-AKI总体发生率更低[142, 183, 226~235]。尽管有许多积极的结果，但很难对他汀类药物作出普遍的推荐[244]，因为这些研究的患者都是心脏病患者，并且使用了多种他汀类药物和标准的水化治疗方案，eGFR < 45 ml·min^{-1}·(1.73 m^2)$^{-1}$的患者纳入不多，大多数接受冠状动脉造影或行冠状动脉介入治疗的患者已经在长期服用他汀类药物，这些混杂因素导致结果存在不确定性[221, 232~233, 245]。因此，尽管短期大剂量他汀类药物可能具有潜在预防作用，但不推荐在无其他适应证的情况下，使用他汀类药物预防CI-AKI[144]。

推荐意见

27. 短期大剂量他汀类药物可能具有潜在的CI-AKI预防作用，但不推荐他汀类药物作为CI-AKI的常规预防策略。（Ⅱb，A）

3. 维生素C：有研究报道维生素C对CI-AKI具有预防作用[246]，部分荟萃分析也证明维生素C联合生理盐水可以显著降低CI-AKI的发生风险[183, 199, 247]。在行冠状动脉介入治疗的患者中，维生素C的使用还可能与额外的临床效益相关，如心脏保护作用[248]。但大多数RCT研究或荟萃分析并未证明维生素C可降低接受冠状动脉造影CKD患者的CI-AKI发生风险[213, 249~252]。

推荐意见

28. 维生素C可能具有潜在的CI-AKI预防作用，但仍需临床研究证实。（Ⅱb，A）

4. 肾素-血管紧张素-醛固酮系统（RAAS）抑制剂：RAAS抑制剂包括ACEI或ARB，对CI-AKI的预防作用并无定论。据报道，ACEI/ARB可通过影响血流动力学对冠心病和糖尿病患者的肾功能起保护作用[253]，显著降低CI-AKI的发生风险[254]，然而最近的荟萃分析显示RAAS抑制剂对CI-AKI的发生率没有显著影响[213, 255~256]，甚至与CI-AKI风险增加相关[257~258]。由于目前的研究存在较大的异质性，尚缺少大型的临床试验来验证RAAS抑制剂预防CI-AKI的有效性。

推荐意见

29. RAAS抑制剂对CI-AKI的预防作用不明确，不推荐使用RAAS抑制剂作为CI-AKI的常规预防策略。（Ⅱb，A）

三、其他干预手段

关于前列地尔[259-262]、曲美他嗪[263-264]、茶碱[265-268]和重组人C1酯酶抑制剂[269]等药物对CI-AKI作用的结果并不确定，也不推荐单独使用这些药物来降低CI-AKI的风险。也有文献报道[270-271]，别嘌呤醇可能是潜在CI-AKI预防药物，可降低CI-AKI发生风险，且这种获益在CI-AKI高危患者中更显著，但仍需大规模临床试验进行验证。最新的荟萃分析[272]显示，在水化预防的基础上联合曲美他嗪可进一步降低行冠状动脉造影CKD患者发生CI-AKI的风险(6.6%比20.0%)，为联合使用多种干预措施预防CI-AKI提供了依据。也有一些临床研究显示，非药物的干预手段，如远端缺血预处理[273]，可有效预防CI-AKI，但透析等其他手段并未显示可预防CI-AKI[274]，这些干预措施的临床有效性仍需要进一步研究。

治疗措施

一、透析

碘对比剂主要通过肾小球滤过，对于肾功能不全的患者，碘对比剂的排泄会延迟，而血液透析或腹膜透析可有效清除碘对比剂[275]。对于已经行维持性透析治疗的患者，如果患者不存在容量负荷过重包括由于高渗透浓度引起的任何循环血容量增加，均可使用碘对比剂检查[275]。尽管透析可以促进碘对比剂的清除，但由于导管放置和感染等相关风险，CI-AKI患者肾功能存在进一步恶化的风险[276]。现有临床试验关于透析是否可改善CI-AKI患者肾功能预后或降低死亡率的结论并不一致[277]，开始肾脏替代治疗的时机也不确定。因此，除非必需，如患者出现尿量及频次减少等症状并危及生命时，临床不推荐采用透析治疗CI-AKI[274,278]。

推荐意见

30. 不推荐常规采用透析治疗CI-AKI，仅在病情严重危及生命、有透析指征的情况下可考虑透析治疗。(Ⅲ，C)

二、其他治疗手段

水化虽然可有效预防CI-AKI，但治疗CI-AKI的有效性尚缺乏证据支持，并不推荐将其用于除血容量不足的CI-AKI患者的治疗[274]。利尿剂[279]、多巴胺[280]、利钠肽[281]等药物虽有助于对患者进行容量管理[274]，促进碘对比剂的排出，但治疗CI-AKI的证据并不确定，因此也不推荐使用这些药物治疗CI-AKI。

利益冲突 所有作者声明无利益冲突

共识编写专家组

顾问：余学清(广东省人民医院/中山大学附属第一医院)

组长：赵杰(郑州大学第一附属医院)、张玉(华中科技大学同济医学院附属协和医院)、陈江华(浙江大学医学院附属第一医院)

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（中南大学湘雅三医院）、沈爱宗（中国科学技术大学附属第一医院）、史琛（华中科技大学同济医学院附属协和医院）、宋燕青（吉林大学第一医院）、隋忠国（青岛大学附属医院）、孙脊峰（空军军医大学第二附属医院）、孙晶（山东省立医院）、孙林（中南大学湘雅二医院）、孙洲亮（厦门大学附属第一医院）、索郎欧珠（西藏自治区第二人民医院）、童俊容（中国人民解放军南部战区总医院）、童荣生（四川省人民医院）、万建新（福建医科大学附属第一医院）、王彩丽（内蒙古科技大学包头医学院第一附属医院）、王家伟（北京同仁医院）、王建华（新疆医科大学第一附属医院）、王俭勤（兰州大学第二医院）、王晋文（昆明市延安医院）、汪年松（上海交通大学附属第六人民医院）、文爱东（第四军医大学西京医院）、文友民（宁夏医科大学总医院）、吴东方（武汉大学中南医院）、吴广礼（中国人民解放军联勤保障部队第九八〇医院）、吴永贵（安徽医科大学第一附属医院）、武新安（兰州大学第一附属医院）、夏培元（陆军军医大学第一附属医院）、谢娟（贵州省人民医院）、邢昌赢（南京医科大学第一附属医院）、许杜鹃（安徽医科大学第一附属医院）、许钟镐（吉林大学第一医院）、徐钢（华中科技大学同济医学院附属同济医院）、徐贵丽（成都军区昆明总医院）、徐珽（四川大学华西医院）、胥婕（北京大学第三医院）、闫铁昆（天津医科大学总医院）、颜小峰（浙江大学医学院附属第二医院）、杨宏昕（内蒙古自治区人民医院）、杨敏（广东省人民医院）、杨婉花（上海交通大学附属瑞金医院）、杨向东（山东大学齐鲁医院）、姚丽（中国医科大学附属第一医院）、姚毅（江苏省中医院）、尹东锋（新疆军区总医院）、尹文俊（中南大学湘雅三医院）、于倩（吉林大学中日联谊医院）、余学清（广东省人民医院/中山大学附属第一医院）、泽碧（西藏自治区人民医院）、查艳（贵州大学人民医院）、翟所迪（北京大学第三医院）、张毕奎（中南大学湘雅二医院）、张波（中国医学科学院北京协和医院）、张春（华中科技大学同济医学院附属协和医院）、张浩（中南大学湘雅三医院）、张惠娟（天津市人民医院）、张健（上海交通大学医学院附属新华医院）、张鉴（山东省立医院）、张景红（海军第九〇五医院）、张抗怀（西安交通大学第二附属医院西北医院）、张克勤（重庆医科大学附属第二医院）、张兰（首都医科大学宣武医院）、张伶俐（四川大学华西第二医院）、张伟（河南省人民医院）、张晓坚（郑州大学第一附属医院）、张幸国（浙江大学医学院附属第一医院）、张艳华（北京肿瘤医院）、张永军（石河子大学医学院第一附属医院）、张玉（华中科技大学同济医学院附属协和医院）、张志清（河北医科大学第二医院）、张志仁（哈尔滨医科大学附属肿瘤医院）、赵杰（郑州大学第一附属医院）、赵明辉（北京大学第一医院）、赵庆春（沈阳军区总医院）、赵荣生（北京大学第三医院）、赵志刚（北京天坛医院）、肇丽梅（中国医科大学第二临床学院）、郑志昌（贵阳医学院附属医院）、周凌云（中南大学湘雅三医院）、周巧玲（中南大学湘雅医院）、周晓玲（宁夏医科大学总医院）、周玉生（南华大学附属第二医院）、周云曙（江苏恒瑞医药股份有限公司）、朱珠（中国医学科学院北京协和医院）、庄永泽（中国人民解放军联勤保障部队第九〇〇医院）、邹洪斌（吉林大学第二医院）、左笑丛（中南大学湘雅三医院）

执笔人：左笑丛（中南大学湘雅三医院药学部）；韩飞（浙江大学医学院附属第一医院）；周凌云（中南大学湘雅三医院药学部）

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