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EDITORIAL COMMENT

Acute Kidney Injury After Carotid Artery Stenting*

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Percutaneous carotid artery stenting for significant stenoses has considerably advanced over the years with improved catheters, guidewires, and embolic protection devices. However, the aorta from the bifurcation to the carotids is crossed with wires and catheter exchanges, and the vessels are imaged with iodinated contrast that has both a hemodynamic/ischemic and ischemic mechanism of injury to the renal tubular cells, mainly in the outer medulla. Contemporary outcomes reported for 985,737 consecutive patients undergoing percutaneous coronary intervention (PCI) at 1,253 sites participating in the National Cardiovascular Data Registry (NCDR) Cath-PCI registry highlighted an overall rate of contrast-induced acute kidney injury (CI-AKI) of 7.1% (1). Although much has been published concerning the epidemiology of CI-AKI in the setting of percutaneous coronary intervention (PCI), little is known about the risks of CI-AKI in patients undergoing carotid artery stenting (2,3).

SEE PAGE 1506

In this issue of *JACC: Cardiovascular Interventions*, Donahue et al. (4) report on 126 patients with pre-existing chronic kidney disease (CKD) who underwent carotid artery stenting at a mean age of ~76 years with an estimated glomerular filtration rate of ~42 ml/min/1.73 m^2 receiving a contrast load of 121 ml. The rate of CI-AKI of 17% was more than double that of patients undergoing PCI (1,5). The finding of intraprocedural hypotension as a major determinant of CI-AKI during carotid artery stenting is novel and suggests transient well-documented decreases in systolic blood pressure (<90 mm Hg) or bradycardia (<60 beats/min) have an additive impact on CI-AKI in addition to the relatively modest contrast volumes used in this series. Both hemodynamic instability and CI-AKI were independent predictors of clinical outcomes at 30 days with large measures of association, suggesting that even more detailed accounting of confounders would not adjust away these relationships.

Approximately two-thirds of those who became hemodynamically unstable needed intravenous vasopressors. Hypotension and bradycardia appeared to be more likely in cases in which there was balloon pre-dilation. The number of catheter exchanges was not reported, and it is unclear whether there were any procedural factors that could have caused cholesterol microembolism to the kidneys. Because the contrast volumes were relatively low and were not significant predictors of CI-AKI, hemodynamic and other mechanisms appear to be more important in this procedure compared with PCI.

Avoidance of bradycardia and hypotension is an important part of the management of patients during carotid artery stenting (**Figure 1**). The key steps in practice include pre-treatment with atropine and cessation of antihypertensive drugs and phosphodiesterase inhibitors on the day of the procedure. To anticipate the possibility of hypotension or bradycardia, intravenous dopamine should be prepared and ready for infusion, and isoproterenol should be available for bradycardia that occurs despite anticholinergic pre-medication. If hypotension occurs, gentle volume administration can also

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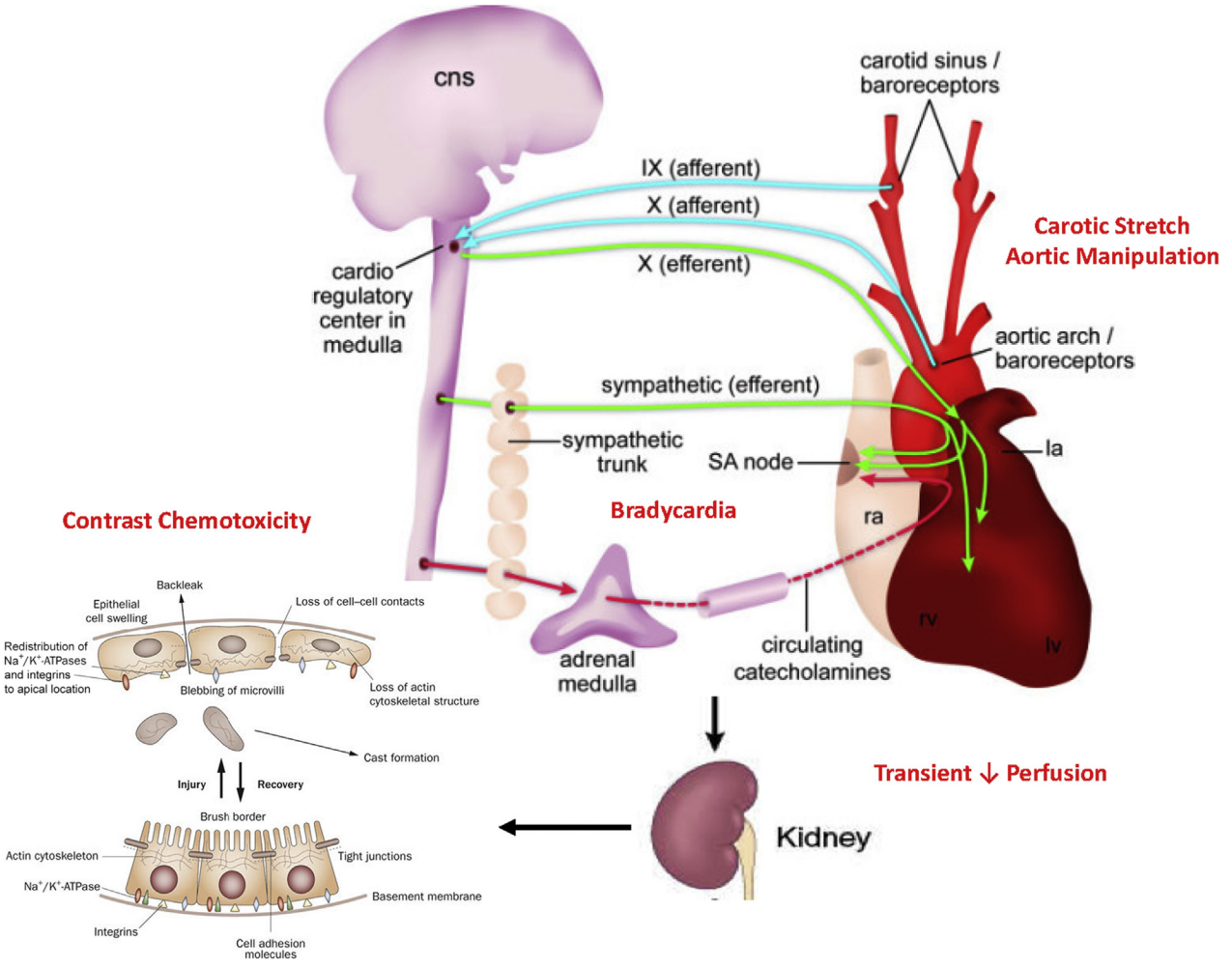
FIGURE 1 The Pathophysiology of CI-AKI in the Setting of Carotid Stenting Involves Both Transient Hemodynamic and Chemotoxic Injury to the Kidneys



Potential mechanisms contributing to contrast-induced acute kidney injury in patients undergoing carotid artery stenting. cns = central nervous system; la = left atrium; lv = left ventricle; ra = right atrium; rv = right ventricle; SA = sinoatrial.

be used with care not to overhydrate, which can lead to bladder distention and reflex vagal stimulation.

In the future, it is possible that newer vascular interventional techniques or the use of novel vasopressor support may lessen the frequency of bradycardia and hypotension and translate into a reduced risk of CI-AKI. Additionally, because CI-AKI during carotid artery stenting appears to be amplified by hemodynamic instability, it could conceivably be ameliorated by remote ischemic pre-conditioning using transient arm ischemia with a blood pressure cuff, as recently reported in several trials (6,7). As in studies of PCI, it is most desirable to avoid CI-AKI after carotid artery stenting to be at the lowest risk of associated clinical complications in the short and intermediate term.

In summary, it appears that CI-AKI is more common after carotid artery stenting than PCI and that the unique risk feature is intraprocedural hypotension. More research is needed on the potential vascular-renal mechanisms of injury at play in this patient population brought to light by the present study.

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