Doppler-Based Renal Resistive Index Can Assess Progression of Acute Kidney Injury in Patients Undergoing Cardiac Surgery

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Objectives: **The objective of this study was to test whether assessment of renal resistive index measured after cardiac surgery (RRI_{T0}) can diagnose persistent acute kidney injury (AKI). The predictive value was evaluated using a gray-zone approach.**

Design: **A prospective observational study.**

Setting: **A teaching university hospital.**

Participants: **Eighty-two patients following cardiac surgery with cardiopulmonary bypass.**

Interventions: **Measurements of hemodynamic parameters and RRI were obtained before surgery, on admission to the intensive care unit, 6 hours after admission, and on the first postoperative day. AKI was defined according to the renal risk, injury, failure, loss of kidney function, end-stage of kidney disease (RIFLE) classification during the first postoperative week. Persistent AKI was defined as AKI lasting >3 days.**

Measurements and Main Results: **Out of the 82 patients, 15 (18%) developed persistent AKI, and 6 (7%) developed transient AKI. The median value and time-course of RRI were significantly different between patients with transient AKI and persistent AKI. Doppler-based RRI_{T0} predicted persistent AKI with an area under the receiver-operating characteristic curve of 0.93 (95% confidence interval: 0.85-0.98, $p < 0.0001$). The optimal cut-off of RRI was 0.73 (95% confidence interval: 0.73-0.75). The gray-zone approach identified a range of RRI values between 0.72 and 0.75 in 14% of patients.**

Conclusions: **Doppler-based RRI can be helpful for noninvasive assessment of renal function recovery after cardiac surgery by using RRI_{T0} to predict persistent AKI. The optimal cut-off was 0.73 with a gray zone ranging between 0.72 and 0.75.**

KEY WORDS: ***acute kidney injury, cardiac surgical procedures, Doppler, postoperative period, ultrasonography***

ACUTE KIDNEY INJURY (AKI), which occurs commonly in as many as 30% of patients after cardiac surgery, is associated with increased morbidity and mortality. AKI is the consequence of an interplay of pathophysiologic mechanisms, including several factors. Preoperative administration of radiocontrast agent, cardiopulmonary bypass (CPB), ischemia-reperfusion, hemodynamic impairment, systemic inflammatory response, and red blood cell transfusion can damage the renal parenchyma.[1-3] Postoperative AKI is characterized by a progressively worsening course comprising several phases. The early phase is characterized by alterations in vasoreactivity and renal perfusion.[3] In clinical practice, the diagnosis of AKI is based on serum and urinary markers, such as serum creatinine (sCr), urinary output, and fractional excretion of sodium or urea.[4,5] These markers are insensitive, unreliable, and can be altered by several factors during the postoperative period.[6] Several authors have studied novel biomarkers for earlier detection of AKI, such as cystatine C and neutrophil gelatinase-associated lipocalin. However, bedside use of these biomarkers may be limited by their cost and predictability.[7-9] The Doppler-based renal resistive index (RRI; [peak systolic flow velocity − minimum diastolic flow velocity]/[peak systolic flow velocity]) measured by transparietal renal Doppler can assess renal perfusion. Over the past decade, some authors have emphasized the role of RRI as an earlier predictor of AKI in a wide range of clinical situations.[10-16] Early elevation of RRI has been associated with the development of acute tubular necrosis and allograft survival.[11] Platt et al suggested that RRI might distinguish acute tubular necrosis from prerenal failure.[12] Later, Darmon et al demonstrated that RRI might be helpful in predicting the reversibility of AKI in a mixed critical care population. Recently, Bossard et al demonstrated a good predictive value of RRI measured immediately after surgery to predict delayed AKI.[16] However, these authors selected a population with risk factors for AKI, and their approach did not differentiate AKI according to its duration and etiology.[16] In the area of cardiac surgery, AKI frequently presents a continuum between volume-responsive AKI and nonvolume-responsive AKI rather than 2 distinct entities. Because the duration of AKI has been associated with improved long-term survival after cardiac surgery,[17] early characterization of renal perfusion alterations associated with AKI and its duration may allow physicians to apply an appropriate treatment that could improve recovery from AKI.

The objective of this study was to assess the value of RRI measured at admission to the intensive care unit (ICU) to distinguish transient from persistent AKI. This indicator was also evaluated using a gray-zone approach.

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The authors performed this study in the course of their normal duties as full-time employees of public healthcare institutions. The study was performed at Amiens University Hospital.

This study has been approved by the IRB of Amiens University Hospital (Comité de Protection des Personnes Nord Ouest, Amiens, France).

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METHODS

Ethics

This study was approved by the Institutional Review Board (*Comité de Protection des Personnes Nord Ouest*, Amiens, France) for human subjects. Informed consent was waived, as the Institutional Review Board considered renal Doppler ultrasonography to be an existing feature without any change in treatment due to blinded evaluation.

Patients

A prospective, observational study was conducted over a period of 6 months at the Amiens University Hospital surgical ICU, admitting all patients after cardiac surgery. Inclusion criteria were patients aged >18 years at the time of cardiac surgery with cardiopulmonary bypass. Exclusion criteria were poor abdominal echogenicity, frequent ectopic beats, arrhythmia, and known renal artery stenosis.

Preoperative Period

Anesthesia procedure and CPB management were standardized for all patients. CPB with a heart-lung machine (Stockert Sorin S5 Heart-Lung) was established at a target systemic blood flow of 2.4 L/min/m^2, and mean arterial pressure (MAP) was maintained at >50 mmHg by first increasing pump flow or, if the blood pressure did not improve, by a bolus administration of phenylephrine or norepinephrine. During CPB, normothermia (bladder temperature $>36^\circ$C) was maintained with a perfusion temperature of 37°C.

Postoperative Procedures

After surgery, all patients were sedated with propofol and were mechanically ventilated until stabilization of hemodynamic parameters, normothermia, and bleeding control were achieved. Circulatory support was guided by the authors' local protocol to achieve predefined endpoints: mean arterial pressure (MAP) >70 mmHg, cardiac index >2.2 L/min/m^2, urinary output >0.5 mL/kg/h. Hypotension (MAP ≤ 60 mmHg) without symptoms of cardiogenic shock (all patients had cardiac index >2.2 L/min/m^2) defined postcardiac surgery vasoplegia, and was treated with continuous intravenous administration of norepinephrine to restore MAP >70 mmHg.

Measurements

RRI was measured using a transparietal 5 MHz pulsed-wave Doppler probe on a Philips Envisor ultrasound system (Philips Medical System, Suresnes, France). Two trained physicians measured velocities in the interlobar arteries of the upper, median, and lower segments of each kidney. For each artery, RRI was calculated as RRI = (peak systolic velocity − end-diastolic velocity)/peak systolic velocity. All values were the average of 3 measurements. RRI then was calculated as the average RRI for each kidney. Physicians were blinded to the subjects' characteristics. The reproducibility of RRI measurement was tested before the study; the intraobserver variability and interobserver variability of RRI measurements were 4% (3-6) and 7% (6-10), respectively.

Study Protocol

Demographic, intraoperative, and postoperative data were recorded prospectively. RRI was measured on the day before

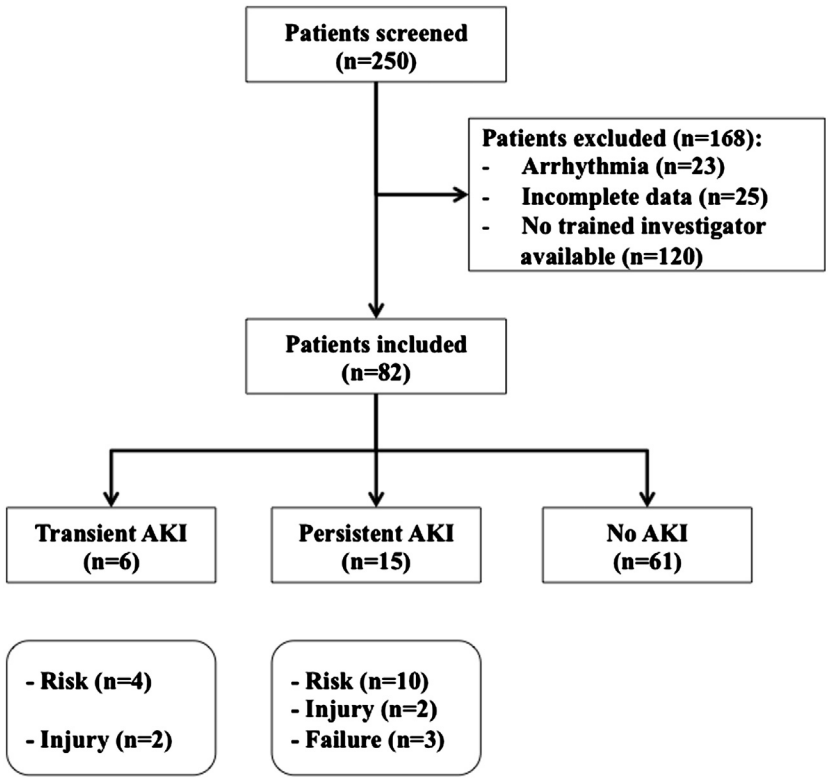

Fig 1. Flow chart diagram. Class R (risk): sCr $\geq$1.5-fold increase or eGFR $>$25% decrease compared with baseline; Class I (injury); sCr $\geq$2-fold increase or eGFR $>$50% decrease compared with baseline; Class F (failure): sCr $\geq$3-fold increase or eGFR $>$75% decrease compared with baseline, or sCr increase to $\geq$4 mg/dL in the setting of an increase of $\geq$0.5 mg/dL. AKI, acute kidney injury; sCr, serum creatinine; eGFR, estimated glomerular filtration rate.