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# Impact of contrast exposure from computed tomography angiography on acute kidney injury after neonatal cardiopulmonary bypass surgery

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## Abstract

**Objective:** Acute kidney injury (AKI) is a frequent complication after cardiopulmonary bypass (CBP) for cardiac surgery in neonates. It is unclear if exposure to computed tomography angiography (CTA) in the preoperative period increases the risk of AKI. We hypothesized a short interval between CTA and CPB surgery would be associated with higher rates of AKI in infants.

**Design:** In this single center retrospective review of patients between 2012 and 2015, neonates less than one month old were analyzed if they had CTA prior to cardiac surgery with CPB. Baseline, demographic, fluid balance, and laboratory data was analyzed. AKI was staged according to KDIGO criteria.

**Results:** Fifty-six neonates were analyzed. AKI developed in 42 (75%) of patients; severe AKI (KDIGO stages 2 and 3) occurred in 18 (32%). Patient characteristics were similar at baseline and at time of CTA between those with and without severe AKI. Patients with severe AKI had longer CPB time, lower postoperative urine output, higher peak serum creatinine, and longer hospital length of stay. When considering intervals between CTA and CPB surgery $\leq$1 day (n = 19), $\leq$3 days (n = 28), and >3 days (n = 28); there was no difference in AKI incidence nor postoperative outcomes among these three interval cohorts.

**Conclusion:** Routine exposure to CTA and CPB surgery in close succession does not appear to increase the risk of AKI after neonatal cardiac surgery. Though other risks need to be weighed (eg, sedation, intubation, radiation exposure), this result may enable more liberal utilization of CTA for preoperative surgical planning of congenital heart operations in patients with unclear or complex anatomy.

KEYWORDS

acute kidney injury, congenital heart disease, congenital heart surgery

## 1 | INTRODUCTION

Computed tomography angiography (CTA) can help delineate complex cardiovascular anatomy and has become an important diagnostic tool for management of pediatric congenital heart disease.[1] Contrast-induced nephropathy (CIN) has been reported in adults undergoing cardiopulmonary bypass (CPB) following contrast media exposure.[2,3] It is unclear if contrast exposure prior to cardiac surgery with CPB is a risk factor for acute kidney injury (AKI) in neonates.

AKI occurs in up to 64% of neonates undergoing cardiac surgery with CPB.[4–7] Risk factors for AKI in these studies was multifactorial, but preoperative exposure to contrast was not examined as a possible risk factor. In this study, we reviewed our institutional experience with CTA preceding neonatal CPB surgery and sought to determine the impact of contrast exposure on postoperative AKI and related outcomes. We hypothesized AKI occurred at a higher rate as the interval between CTA and cardiac surgery decreased.

  

**TABLE 1** Neonatal AKI KDIGO classification

| Stage | Serum creatinine | Urine output |
|---|---|---|
| 0 | No change in SCr or rise <0.3 mg/dL | ≥0.5 mL/kg/h |
| 1 | SCr rise ≥0.3 mg/dL within 48 h or SCr rise ≥1.5–1.9 × reference SCr within 7 d | <0.5 mL/kg/h for 6–12 h |
| 2 | SCr rise ≥2.0–2.9 × reference SCr | <0.5 mL/kg/h for ≥12 h |
| 3 | SCr rise ≥3 × reference SCr or SCr ≥2.5 mg/dL or receipt of dialysis | <0.3 mL/kg/h for ≥24 h or anuria for ≥12 h |

This table is adapted from Selewski et al.[8] Because our center routinely uses prophylactic peritoneal dialysis, presence of dialysis was not independently classified as KDIGO stage 3.
Abbreviations: AKI, acute kidney injury; KDIGO, Kidney Disease: Improving Global Outcomes; SCr, serum creatinine.

## 2 | METHODS

### 2.1 | Patients

Institutional review board approval and waiver of informed consent was obtained. All neonates less than 1 month undergoing CTA preceding CPB for congenital heart surgery were retrospectively identified from radiology and congenital heart surgery databases at Children's of Alabama from 2012 to 2015. Daily serum creatinine, urine output (UOP), and fluid balance data were collected between 2 days prior and 5 days after the operation as well as the day before and after CTA. Basic demographic and cardiac surgery variables, and postoperative outcomes were collected from the cardiac intensive care unit (CICU) clinical databases. AKI was defined using only serum creatinine (SCr) criteria of the Kidney Disease: Improving Global Outcomes (KDIGO, Table 1); severe AKI was defined as KDIGO stage 2 or 3.[8] As our center routinely performs prophylactic peritoneal dialysis (PD) in neonates undergoing CPB for prevention of fluid overload,[9] presence of PD was not independently classified as stage 3 KDIGO. Maximum postoperative SCr is limited to the first 5 days after surgery.

### 2.2 | CTA protocol

CTA is performed at our institution when echocardiography fails to delineate cardiovascular anatomy pertinent to the planned operation. Most patients are sedated, intubated, and paralyzed for the procedure. We perform no routine CIN prevention measures, such as volume loading, sodium bicarbonate containing fluids, or *N*-acetylcysteine. Pharmacologic heart rate reduction beyond sedation is not employed. CTA is performed on a 256 slice helical CT scanner (Philips iCT Brilliance). Prospective gating is typically utilized. Settings for this population include 80 kV tube voltage, 115 mA tube current, rotation time 0.27 seconds and individualized windowing and bolus tracking. Nonionic, low osmolality contrast agent ioversol (320 mg I/mL, Optiray, Mallinckrodt Imaging) is given at 2 mL/kg.

### 2.3 | Intraoperative and postoperative management

Methylprednisolone (10 mg/kg) was given at eight and one hour before transfer to the operating room; no intraoperative steroids were given. The CPB circuit was primed with 25% albumin, mannitol, sodium bicarbonate, and Normosol-R (Hospira Inc., Lake Forest, IL). Fresh frozen plasma, 20 mL/kg, was added to the prime for patients weighing less than 5 kg. Packed red blood cells were added to the CPB circuit to maintain the desired hematocrit based on physiologic characteristics.

All patients received zero-balance ultrafiltration during CPB and modified ultrafiltration after CPB. Those requiring aortic cross clamp were cooled to 22°C and received one dose of DelNido cardioplegia. During arch reconstruction, continuous low flow cerebral perfusion was employed. Postoperative management was protocolized to target age- and physiology-specific hemodynamic and respiratory goals via inotrope titration, colloid boluses, and protocolized ventilator adjustments. All complex neonatal repairs received prophylactic PD within 6 hours of admission to the CICU; all other patients received furosemide infusions on postoperative day 1.

### 2.4 | Statistical analysis

Categorical variables were compared using Fisher's exact test. Continuous variables were compared using Mann–Whitney *U* test. Median is reported with interquartile range for nonnormally distributed continuous variables. Bivariate correlation was performed with Spearman coefficient for nonnormally related variables. Receiver operator curve was performed to assess whether timing between CTA and surgery predicted development of severe AKI. $P < .05$ was considered statistically significant. Statistical analysis was performed using SPSS software version 22 (IBM, Chicago, IL).

## 3 | RESULTS

### 3.1 | Patients

Fifty-six neonates underwent CTA prior to cardiac surgery during the study period and were included in the analysis; cardiac diagnoses are listed in Table 2. Patient and surgical characteristics are included in Table 3. Forty-two (75%) patients developed AKI in this study. Eighteen (32%) developed severe AKI (KDIGO 2, 3). To ensure inclusion of *clinically significant* AKI, we chose to primarily focus this study on development of severe AKI. KDIGO 1 AKI may include patients with only isolated mild postoperative serum creatinine increase and has inconsistent correlation with outcomes.

### 3.2 | AKI risk factors and outcomes

There were no differences in patient characteristics and renal variables at time of CTA between those that develop severe AKI after cardiac

**TABLE 2** Cardiac diagnoses in patients undergoing CTA

| Diagnosis | n |
|---|---|
| Complex CHD with pulmonary atresia | 15 |
| Tetralogy of Fallot | 8 |
| d-Transposition of great arteries | 7 |
| Aortic arch obstruction with VSD | 7 |
| Aortic arch obstruction without VSD | 4 |
| Total anomalous pulmonary venous return | 6 |
| Truncus arteriosus | 3 |
| Hypoplastic left heart syndrome | 3 |
| Pulmonary atresia, intact ventricular septum | 1 |
| Pulmonary vein stenosis | 1 |
| Cardiomyopathy | 1 |

surgery and those that did not (Table 3), except the cohort that developed severe AKI had significantly longer CPB times. Table 4 shows the results of univariate analysis comparing clinical outcomes in patients with or without severe AKI. Not surprisingly, the severe AKI group had lower urine output in the first 24 hours after surgery and higher maximum postoperative SCr. PD and mechanical ventilation utilization were not statistically different, though lack of power may have belied important clinical differences in mechanical ventilation shown in severe AKI patients. Hospital length of stay was longer in the severe AKI group.

## 3.3 | Impact of timing of CTA and AKI

Median time between CTA and cardiac surgery for the entire cohort was 3.5 days with a range of 0–15 days. No patient developed AKI between CTA and cardiac surgery. Table 5 demonstrates results of univariate analysis comparing postoperative AKI prevalence and outcomes with respect to different time intervals between CTA and cardiac surgery. The analysis is performed using two separate interval cut-off points: 1 and 3 days. There was no association of temporal relationship of the two procedures and any SCr related variable or UOP.

Nineteen patients had less than 1 day between CTA and surgery; this cohort did not have increased prevalence of severe AKI compared to all others. Conversely, there was nonstatistically significant higher UOP during the first 24 postoperative hours in those patients that received CTA and cardiac surgery less than 1 day apart compared to all others. Figure 1 demonstrates postoperative changes in SCr for patients that had ≤3 days between CTA and surgery, compared with those >3 days; there was no significant difference in SCr at any postoperative time point, and severe AKI occurred at the same rate in these two groups (32% and 29%, $P = 1.0$). Time between CTA and CPB was

**TABLE 3** Baseline variables

| | All (n = 56) | Severe AKI (n = 18) | No/mild AKI (n = 38) | *P* value |
|---|---|---|---|---|
| Age at surgery (days) | 8 (5, 13.8) | 9 (4.8, 15) | 8 (5, 13) | .5 |
| Age at CTA (days) | 4 (2, 7) | 3 (2, 7) | 4 (2, 8) | .9 |
| Weight at surgery (kilograms) | 3.2 ± 0.6 | 3.1 ± 0.4 | 3.2 ± 0.6 | .9 |
| Lowest (baseline) SCr | 0.5 (0.4, 0.6) | 0.4 (0.3, 0.5) | 0.5 (0.4, 0.6) | .2 |
| STAT category, n (%) | | | | .2 |
| 2 | 5 (8.9) | 0 (0) | 5 (13.2) | |
| 3 | 9 (16.1) | 3 (16.7) | 6 (15.8) | |
| 4 | 38 (67.9) | 14 (77.8) | 24 (63.2) | |
| 5 | 3 (5.4) | 0 (0) | 3 (7.9) | |
| CTA timing (preoperative), n (%) | | | | |
| ≤1 day | 19 (34) | 6 (33) | 13 (34) | 1.0 |
| >3 days | 28 (50) | 8 (44) | 20 (53) | .8 |
| ≤3 days | 28 (50) | 10 (56) | 18 (47) | .8 |
| Medications at CTA, n (%) | | | | |
| Vancomycin/gentamycin | 11 (21) | 4 (25) | 7 (19) | .7 |
| Catechlamines | 2 (4) | 1 (6) | 1 (3) | .5 |
| Diuretics | 24 (46) | 9 (56) | 15 (41) | .4 |
| Cardiopulmonary bypass (minutes) | 114 ± 65 | 150 ± 95 | 97 ± 32 | <.01 |
| Aortic cross clamp (minutes) | 66 ± 40 | 83 ± 55 | 59 ± 30 | .08 |
| Circulatory arrest, n (%) | 6 (11) | 3 (17) | 3 (8) | .4 |

Data presented as mean with standard deviation or medians with interquartile range.
Abbreviations: AKI, acute kidney injury; CTA, computed tomography angiography; SCr, serum creatinine; STAT, society of thoracic surgeons - European association for cardiothoracic surgery. Congenital heart surgery mortality categories.

**TABLE 4** Outcomes in patients with or without severe acute kidney injury

| Outcome variable | All (n = 56) | Severe AKI (n = 18) | No/mild AKI (n = 38) | *P* value |
|---|---|---|---|---|
| Urine output (mL/kg/h) | | | | |
| 24 h pre-CTA | 4.2 ± 1.6 | 4.3 ± 1.9 | 4.2 ± 1.4 | .8 |
| 24 h post-CTA | 4.2 ± 1.9 | 4.7 ± 2.1 | 4 ± 1.7 | .3 |
| 24 h pre-op | 4.4 ± 2.1 | 5.1 ± 2.9 | 4.1 ± 1.6 | .2 |
| 24h post-op | 0.9 (0.5, 1.5) | 0.6 (0.2, 0.9) | 1.1 (0.6, 1.6) | <.01 |
| Serum creatinine | | | | |
| At CTA, % of baseline | 102 ± 7 | 100 ± 0 | 104 ± 9 | .1 |
| At surgery, % of baseline | 94 ±24 | 100 ± 25 | 91 ± 25 | .3 |
| At surgery, % of CTA value | 91 ± 30 | 94 ± 36 | 89 ± 25 | .6 |
| Maximum post-op (mg/dL) | 0.9 ± 0.3 | 1.2 ± 0.3 | 0.7 ± 0.2 | <.01 |
| Maximum post-op (% of baseline) | 189 ± 91 | 280 ± 100 | 142 ± 33 | <.01 |
| Discharge | 0.3 (0.3, 0.4) | 0.3 (0.2, 0.4) | 0.3 (0.3, 0.4) | .8 |
| Peritoneal dialysis, n (%) | 28 (50) | 9 (50) | 19 (50) | 1.0 |
| Peritoneal dialysis duration (days) | 3 (1, 6) | 3 (1.3, 5.8) | 3 (1, 5) | 1.0 |
| Mechanical ventilation (days) | 3 (1, 6) | 4 (1.8, 7.8) | 2 (0.7, 5.4) | .2 |
| Prolonged ventilation > 3 day, n (%) | 19 (36) | 9 (50) | 11 (29) | .1 |
| Hospital length of stay (days) | 24 (14, 41) | 31 (25, 90) | 21 (11, 33) | .01 |
| Mortality, n (%) | 3 (5) | 2 (11) | 1 (3) | .2 |

Data presented as means with standard deviations or medians with interquartile ranges.
Abbreviations: AKI, acute kidney injury; CTA, computed tomography angiography.

not predictive of severe AKI as seen by ROC analysis (AUC 0.48, $P = .8$). There was no significant correlation between days from CTA to CPB surgery and peak postoperative change from baseline SCr. There was also no correlation between CTA timing and UOP in first 24 hours. There was a very weak relationship between peak postoperative serum creatinine and CTA timing. ($r = -0.3$, $P = .03$). Days between CTA and cardiac surgery also had no correlation with other clinical outcomes, including postoperative PD ultrafiltration duration, and duration of postoperative mechanical ventilation, or postoperative length of stay.

## 3.4 | Mortality

Three patients died (5.4%). One patient was a 33-week premature infant who underwent complete repair of d-transposition of the great arteries with ventricular septal defect (VSD) and interrupted aortic arch. This patient required extracorporeal membrane oxygenation support immediately after surgery. Another patient experienced severe hemolytic anemia after repair of truncus arteriosus. Both patients had severe AKI related to postoperative complications. A third patient, who had multiple congenital anomalies and underwent complete repair of

**TABLE 5** Development of acute kidney injury based on interval between CT angiography and surgery

| | | Analysis using 1 day interval cut point | | | Analysis using 3 day interval cut point | | |
|---|---|---|---|---|---|---|---|
| | All (n = 56) | ≤1 day (n = 19) | >1 day (n = 37) | *P* value | ≤3 day (n = 28) | >3 day (n = 28) | *P* value |
| AKI stage, n (%) | | | | 1.0 | | | .9 |
| none | 14 (25) | 5 (26) | 9 (24) | | 7 (25) | 7 (25) | |
| 1 | 26 (46) | 8 (42) | 16 (43) | | 11 (39) | 13 (46) | |
| 2 | 13 (23) | 4 (21) | 9 (24) | | 7 (25) | 6 (21) | |
| 3 | 5 (9) | 2 (11) | 3 (8) | | 3(11) | 2 (6) | |
| Any AKI, n (%) | 42 (75) | 14 (74) | 28 (76) | 1.0 | 21 (75) | 21 (75) | 1.0 |
| Severe AKI, n (%) | 18 (32) | 6 (32) | 12 (32) | 1.0 | 9 (32) | 8 (29) | 1.0 |
| Peak serum creatinine | 0.8 (0.6, 1) | 0.9 (0.7, 1) | 0.7 (0.6, 1.1) | .3 | 0.9 (0.7, 1.1) | 0.7 (0.6, 1) | .1 |
| Change in serum creatinine | 0.3 (0.2, 0.6) | 0.3 (0.2, 0.5) | 0.3 (0.2, 0.6) | .8 | 0.3, (0.2, 0.7) | 0.3 (0.2, 0.5) | .9 |
| UOP, 24 h postsurgery (ml/kg/day) | 0.9 (0.5, 1.5) | 1.2 (0.7, 1.4) | 0.6 (0.4, 1.4) | .3 | 1 (0.4, 1.5) | 0.7 (0.5, 1.5) | .8 |
| Duration of PD, hours | 3 (1, 5) | 3 (1, 6.5) | 3 (1.5, 5) | 1.0 | 3 (1.5, 5) | 2.5 (1, 6) | .7 |

Data presented as median with interquartile range.
Abbreviations: AKI, acute kidney injury; PD, peritoneal dialysis; UOP, urine output.

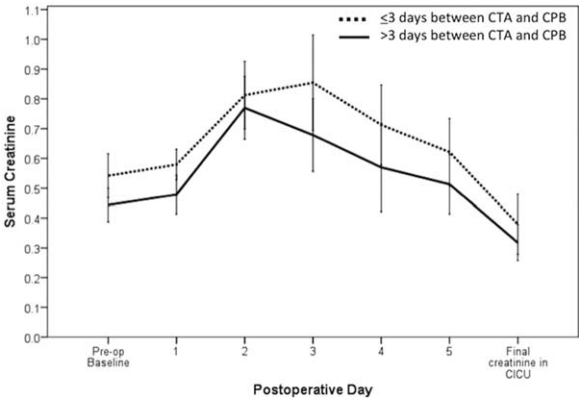


**FIGURE 1** Change in postoperative serum creatinine in patients with interval of ≤3 days or >3 days between computed tomography angiography and cardiopulmonary bypass. No statistically significant difference at any time point

pulmonary atresia with VSD, experienced stage 1 AKI and died from complications of severe pulmonary vein stenosis 1 month later.

## 4 | DISCUSSION

Contrary to our hypothesis, in this study we found that decreased time interval between CTA and cardiac surgery was not associated with increased rate of postoperative AKI. Importantly, this includes greater than one third of our cohort receiving cardiac surgery within 24 hours of CTA contrast exposure. No AKI developed between CTA and cardiac surgery. Postoperative severe AKI occurred at a similar rate in these contrast-exposed neonates compared to a contemporary neonatal cohort from our institution (32% vs 35%, respectively),[10] and the severe AKI rate in other studies of neonatal AKI (21%-44%).[4–7] Severe AKI was associated with increased postoperative morbidity, including decreased UOP, however shorter interval between CTA and cardiac surgery was not. This data suggests CTA can be utilized to define complex anatomy for neonatal surgical planning just prior to CPB with minimal concern of increasing the risk of clinically important postoperative AKI.

AKI is a common complication after neonatal cardiac surgery that is associated with increased morbidity and mortality; multiple patient and clinical risk factors have been described in this cohort, including increased surgical complexity.[4–7] Neonates undergoing cardiac surgery after CTA may have increased risk for postoperative AKI via potential double insult of direct renal toxicity from contrast followed by CPB-induced renal ischemia and inflammation. Possibility of increasing the morbidity from AKI may lead some clinicians to be hesitant about using CTA for surgical planning or delay CPB after CTA is performed. Preoperative contrast exposure has been described as a risk factor for AKI in adult patients, but contribution of CIN to postoperative AKI has not been clearly delineated in pediatric patients. Previous reports have focused on cardiac catheterization and cardiac surgery in close succession as source of potential contrast induced nephropathy[8,10]; our study is the first in pediatric congenital heart surgery to investigate whether preoperative CTA may worsen cardiac surgery-induced AKI. Furthermore, we focus entirely on neonates which have the highest burden of postoperative AKI. Though we do not have a true control group of noncontrast exposed neonates, we do report a similar rate of severe AKI in our high risk CTA cohort compared to that seen in other neonatal AKI epidemiologic studies and a contemporary institutional neonatal cohort (21%-44%)[4–7]; this is despite the fact our cohort was presumably at higher risk for AKI, given the predominance of higher surgical complexity in our study. Thus, we surmise in this high risk population of neonates that preoperative contrast exposure (even within 24 hours) does not appear to increase the risk for development of clinically important AKI above the baseline rate. These results parallel a study of Glenn and Fontan patients, in which the combination of cardiac catheterization with angiography and CPB within 48 hours was not an additional risk factor for AKI.[11]

Our study does not have the capability to directly determine if CTA causes or exacerbates AKI in this population. Numerous studies have demonstrated the presence of renal injury after contrast exposure in children with and without CPB.[11–14] These studies have demonstrated subclinical AKI biomarker elevation after contrast exposure, which may or may not be associated with development of creatinine elevation and oliguria.[13,15] Creatinine may not be sensitive enough to detect mild acute renal injury, and CTA may cause subclinical renal injury, when combined with other nephrotoxic/injury exposures (CPB, cyanosis, medications, hypotension, etc.)—may contribute to clinically important AKI. While we have no AKI biomarker data to rule out subclinical renal injury, utilizing the KDIGO creatinine criteria, we provide indirect evidence that small dose contrast exposure (2 mg/kg) from CTA does not lead to increased frequency of severe clinical AKI after cardiac surgery. Additionally, decreased time interval between CTA and CPB is not associated with worse kidney function as evidenced by negligible impact on UOP and duration of PD. We hypothesize from this retrospective study that CTA in close proximity to CPB does not exacerbate post-CPB AKI.

Our CT scan protocol and imaging results appear comparable to other institutions.[1,16] It must be noted that we did not provide any management for prevention of CIN. Despite the fact, we saw no AKI develop between CTA and CPB, we cannot eliminate the possibility that volume loading, bicarbonate fluids, and/or *N*-acetylcysteine could decrease subclinical renal injury as measured by AKI biomarkers. Alternatively, our study seems to suggest that prophylaxis for CIN prevention may be unnecessary in this population, and in fact, practices such as volume loading with saline may be counterproductive, given the clear association of fluid overload and morbidity in the cardiac surgical neonate.

Our study has important limitations led by those inherent with any small, single-center retrospective study; this study is not powered to detect small increases in AKI. Second, we did not include a control group unexposed to CTA, limiting our ability to definitively conclude that CTA exposure does not increases postoperative AKI frequency. However, for this study, using a control group of neonates unexposed to CTA would introduce additional confounding variables because such patients would inherently be different with regards to cardiac diagnosis

and clinical condition. We did provide an indirect control group, as AKI rate in this cohort is the same as another high risk contemporary neonatal cohort undergoing CPB without CTA at our institution. Third, our care protocols related to preoperative and postoperative cardiac management as well as CTA may differ from other centers and may have affected the risk of AKI. Fourth, we cannot exclude the possibility that clinical decision making impacted the interval between CTA and CPB with patients felt to be at higher risk for AKI having their operation delayed, which may have ameliorated AKI risk in those patients. Clinicians may have also foregone CTA in some patients deemed at high risk for AKI. Finally, potential risks of CTA in this population (eg, sedation, intubation, transport, radiation exposure) need to be considered before adopting CTA as routine imaging modality, and these were not examined in this study.

Despite these limitations, we conclude that decreased time interval between CTA and CPB does not appear to significantly increase the rate or severity of AKI, or decrease UOP in a high risk neonatal cohort. Postoperative severe AKI rate in this cohort is similar to other neonatal cohorts reported in the literature. The results of this study may impact clinical practice by suggesting CTA can be used in the preoperative period with reasonable confidence that it will not importantly increase the risk of postoperative severe AKI, and that there does not need to be a minimum interval between CTA contrast exposure and CPB.

## CONFLICT OF INTERESTS

The authors disclose no conflict of interest.

## AUTHOR CONTRIBUTIONS

All authors participated in data analysis and approval of article.

*Concept/design*, *statistics*, *drafting*: Carlo, Alten, Borasino

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