Research Article

Clinical profile of acute kidney injury in a pediatric intensive care unit from Southern India: A prospective observational study

Sriram Krishnamurthy, Parameswaran Narayanan, Sivaprakasam Prabha, Nivedita Mondal, Subramanian Mahadevan, Niranjan Biswal, Sadagopan Srinivasan

Abstract

Background: Although the term acute renal failure was replaced by acute kidney injury (AKI) recently, there is a paucity of data on the incidence and profile of AKI in critically ill children from the developing world. **Objectives:** The objective of this study is to determine the incidence, etiology, short term outcome and predictors of fatality in critically ill children admitted to the pediatric intensive care unit (PICU) with AKI, aged 1 month to 13 years. **Materials and Methods:** In this prospective observational study, from June 2010 to March 2011, 215 children admitted to the PICU were screened for AKI, defined according to the AKI Network criteria. The patients with AKI were followed-up until discharge/death. Their clinical and biochemical data were recorded. **Results:** The incidence of AKI among 215 patients screened was 54 (25.1%). The common etiologies were infections, [34 (62.9%)], acute glomerulonephritis (7.6%), snake envenomation (5.7%), hemolytic uremic syndrome (3.8%) and congestive cardiac failures (3.8%). Among infections, pneumonia and septicemia constituted 26.5% each, meningoencephalitis accounted for 23.5%, and dengue, scrub typhus, tuberculosis and malaria constituted 9.3% of children with AKI. 27.8% of patients required dialysis. Overall mortality was 46.3%. On logistic regression analysis, requirement of mechanical ventilation was an independent predictor of fatality in AKI. **Conclusions:** Besides the high incidence of AKI in critically ill-children admitted to the PICU (25.1%), the condition was associated with adverse outcomes, including high mortality (46.3%) and need for dialysis (27.8%). Infections dominated the etiological profile. Requirement of mechanical ventilation predicted an adverse outcome in our patient population.

Keywords: Acute kidney injury, critically ill-children, pediatric intensive care unit

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Introduction

Acute renal failure (ARF) is associated with adverse outcomes, especially in children admitted to the pediatric intensive care unit (PICU).[1] Due to usage of multiple definitions of ARF in the literature, causing large variations in the reported incidence and outcome, the term ARF was replaced by acute kidney injury (AKI) recently,[2,3] to provide the uniformity of definition and standardize the care of patients [Figure 1]. Many studies on the incidence of AKI in critically ill-children have been conducted in developed countries and are often retrospective in nature.[4-7] Only a few retrospective studies have been conducted to determine the incidence and profile of AKI, in critically ill-children from the developing world in recent years.[8-10] This study was performed considering the paucity of data available on the incidence and determinants of AKI in Indian children and taking into account the retrospective nature of previous studies.

From:
Department of Pediatrics, Jawaharlal Institute of Postgraduate Medical Education and Research, Pondicherry, India

Correspondence:
Dr. Parameswaran Narayanan, Department of Pediatrics, Jawaharlal Institute of Postgraduate Medical Education and Research, Pondicherry - 605 006, India. E-mail: narayananp@jipmer.net

Materials and Methods

This prospective observational study was conducted over a period of 10 months from June 2010 to March

Definition of AKI

An abrupt (within 48 h) reduction in kidney function defined as an absolute increase in serum creatinine of more than or equal to 0.3 mg/dL, an increase in serum creatinine of more than or equal to 1.5-fold from baseline or a reduction in urine output (documented oliguria of less than 0.5 ml/kg/h for more than 6 h).

Classification/staging system for AKI

Stage	Serum creatinine criteria	Urine output criteria
1	Increase in serum creatinine of more than or equal to 0.3 mg/dL or increase to more than or equal to 1.5-to 2-fold from baseline	Less than 0.5 ml/kg/h for more than 6 h
2	Increase in serum creatinine to more than 2-to 3-fold from baseline	Less than 0.5 ml/kg/h for more than 12 h
3	Increase in serum creatinine to more than 3-fold from baseline or serum creatinine of more than or equal to 4.0 mg/dL with an acute increase of at least 0.5 mg/dL	Less than 0.3 ml/kg/h for 24 h or anuria for 12 h

Figure 1: Definition and classification of AKI

2011. The study was approved by the institutional ethics committee. Informed consent was obtained from the parents prior to inclusion of subjects into the study.

Objectives

Primary objective

To determine the incidence of AKI as defined by the Acute Kidney Injury Network (AKIN) classification in critically ill pediatric patients admitted to the PICU; aged 1 month to 13 years.

Secondary objectives

- To determine the predictors of fatality in AKI in critically ill-children
- To study the etiology and short term outcome of AKI in critically ill-children
- To compare the demographic and clinical parameters among survivors and non-survivors in AKI.

Inclusion criteria

Consecutive patients aged 1 month to 13 years, admitted to the PICU.

Exclusion criteria

- Patients with known chronic kidney disease stage 5 (estimated glomerular filtration rate < 15 ml/min/1.73 m^2)
- Bilirubin level > 5 mg/dL.

The incidence of AKI was estimated to be around 30% in PICU patients on the basis of current literature.[7,11] Assuming a variation of 7% in PICU, i.e., absolute precision d = 0.07, and the chance of this to be at least 95%, the sample size was calculated to be 165 subjects. Results were analyzed using the SPSS version 16 (IBM corporation, New York, U.S.A). Values for continuous data were expressed as mean ± SD (if normally distributed) and median (range) (if non-normally distributed). Categorical variables were reported as proportions. The incidence of AKI was defined as its occurrence as a proportion of total admissions. Continuous variables with normal distribution were compared using Student *t*-test while those not normally distributed were analyzed using Mann Whitney U test. Categorical data were analyzed using Pearson Chi-square test or Fischer exact test. Multivariate binary logistic regression models were used for multivariate analysis of statistically significant variables in univariate analysis ($P < 0.05$), to determine predictors of fatality in AKI. Several multivariate logistic regression models were constructed by evaluating various combinations of variables based on clinical and statistical significance and then the most effective model was selected. Goodness of fit of the model was ascertained by the Hosmer Lemeshow test.

As per the hospital policy, children hospitalized for any illness in the pediatric wards were transferred to the PICU if one or more of the following criteria were present: Impaired level of consciousness (Glasgow

coma scale < 7), signs suggestive of severe increase in intracranial pressure (e.g., hypertension, bradycardia, papilledema), hypoventilation or respiratory failure (oxygen saturation < 90% or arterial oxygen (PaO_2) <60 mmHg with supplemental oxygen or arterial CO2 ($PaCO_2$) >60 mmHg), uncontrollable or poorly controlled seizures, hypotension requiring inotropic support, requirement of renal replacement therapy (RRT) and fulminant hepatic failure.

The diagnosis of AKI was based on AKIN definition and classification [Figure 1].[3] Either serum creatinine or urine output was used to diagnose and stage AKI, using a criterion that led to a higher stage classification. Urine output was measured 6 hourly.

At admission, all subjects underwent serum creatinine measurement. A total of 2 ml of intravenous blood was withdrawn and centrifuged at 3000 rpm for 10 min. Serum creatinine estimation was performed by modified Jaffe method[12] using the autoanalyzer. This measured value was considered as "initial" serum creatinine. Estimation of serum creatinine was repeated every 24 ± 6 h for 3 consecutive days and daily thereafter until discharge from hospital. An absolute increase in serum creatinine of ≥0.3 mg/dL or an increase in serum creatinine of more than or equal to 1.5-fold from the initial serum creatinine was considered as AKI. Similarly, a decrease in serum creatinine of more than or equal to 0.3 mg/dL or a decrease in serum creatinine of ≥1.5-fold from the initial serum creatinine was also considered as AKI. If there was a progressive rise in serum creatinine values, re-classification and progression to maximum AKI stage during the hospital stay was recorded. Indications for RRT were as per standard hospital protocols.

The provisional diagnosis at admission and final diagnosis (at discharge/death) were recorded. The diagnosis of sepsis was made according to the International pediatric sepsis consensus conference definition.[13] Demographic parameters and short term outcomes (complete renal recovery, partial renal recovery and death) were recorded. Pediatric risk of mortality III score was used for assessment of severity of illness. Shock was defined as the presence of at least two of the following: Tachycardia (heart rate > 2 SD for age), feeble pulses, cool peripheries and hypotension (blood pressure <−2SD for age and sex) or capillary filling time > 3 s. Hypertension was defined as >95th percentile blood pressure for age, height and gender.[14] Patients were followed-up until discharge. Complete renal recovery was defined as normal serum creatinine for age (0.2-0.4 mg/dL for infants, 0.3-0.7 mg/dL for 1-12 years, 0.5-1 mg/dL for >12 years) and normal blood pressure at discharge. Partial renal recovery was defined as elevated serum creatinine for age or persistent hypertension at discharge.

Results

Overall, 215 patients were screened for AKI in the PICU. 54 children had AKI, giving incidence of 25.1%. The median age of patients with AKI was 21 months (range 1-144 months) and 53.7% of patients were boys. The mean level of maximum creatinine value during the hospital stay was 1.9 (SD 1.7) mg/dL. AKI stage 1, stage 2 and stage 3 were detected in 19 (35.2%), 14 (25.9%) and 21 (38.9%) of patients respectively. The demographic parameters of children with AKI are depicted in Table 1.

The etiology of AKI observed is summarized in Table 2. Pneumonia constituted 26.5% of all infections associated with AKI. Tropical febrile illnesses (dengue, scrub typhus, tuberculosis (TB) and malaria) constituted 9.3% of AKI patients. Sepsis (without localizing signs) was diagnosed in 9 children, 5 were culture positive. Organisms isolated were *Pseudomonas aeruginosa* (2 patients), *Escherichia coli* (1 patient), *Klebsiella pneumoniae* (1 patient) and *Streptococcus pneumoniae* (1 patient). Other common etiologies were

Table 1: Demographic parameters of critically ill-children with AKI

Parameter	Baseline characteristics (*n*=54)
Age (months) (median [range])	21 (1-144)
Sex (*N* [%])	Male-29 (53.7)
	Female-25 (46.3)
PRISM III score (mean±SD)	23.4±15
Encephalopathy (*N* [%])	19 (35.2)
Mechanical ventilation (*N* [%])	43 (79.6)
Shock (N [%])	47 (87)
Hospital stay (days) (mean±SD)	
Survivors	10.1 (5.8)
Non-survivors	6.3 (4.3)
Overall	8.4 (5.4)
Mortality (N [%])	25 (46.3)
Renal replacement therapy (*N* [%])	15 (27.8)
Common morbidites (*N* [%])	
Pneumonia	9 (16.7)
Sepsis	9 (16.7)
Meningoencephalitis	8 (14.8)
AGN	4 (7.4)
Snake envenomation	3 (5.6)
Heart disease	2 (3.7)
D+HUS	2 (3.7)
Disseminated TB	2 (3.7)
UTI	2 (3.7)
Status epilepticus	2 (3.7)

AKI: Acute kidney injury; PRISM III: Pediatric risk of mortality III; AGN: Acute glomerulonephritis; HUS: Hemolytic uremic syndrome; UTI: Urinary tract infections; TB: Tuberculosis

Table 2-Etiological profile of AKI in critically ill children (*n*=54)

Etiology	*N* (%)
Infections	**34 (62.9)**
Pneumonia	9 (16.7)
Meningoencephalitis	8 (14.8)
Sepsis	
Culture positive	5 (9.3)
Culture negative	4 (7.4)
Urinary tract infection	2 (3.8)
Tuberculosis	2 (3.8)
Scrub typhus	1 (1.9)
Malaria	1 (1.9)
Dengue	1 (1.9)
Acute watery diarrhea	1 (1.9)
Acute Glomerulonephritis	**4 (7.6)**
Acute PSGN	2 (3.8)
Rapidly progressive glomerulonephritis	
Secondary to PSGN	1 (1.9)
Secondary to Goodpasture syndrome	1 (1.9)
Snake envenomation	**3 (5.7)**
D+HUS	**2 (3.8)**
Underlying cardiac disease (congestive heart failure)	**2 (3.8)**
Congenital heart disease	1 (1.9)
Myocarditis	1 (1.9)
Underlying renal disease	**2 (3.8)**
Nephrolithiasis	1 (1.9)
Chronic glomerulonephritis	1 (1.9)
Hypoxic ischemic injury (status epilepticus)	**2 (3.8)**
Malignancy (acute lymphoblastic leukemia)	**1 (1.9)**
Postoperative	**1 (1.9)**
Poisonings	**1 (1.9)**
Intracranial hemorrhage with shock	**1 (1.9)**
Drugs	
Enalapril	1 (1.9)
Total	54

AKI: Acute kidney injury; PSGN: Post-streptococcal glomerulonephritis; HUS: Hemolytic uremic syndrome

Table 3: Comparison of survivors and deaths in critically ill-children with AKI (*n*=54)

Parameter	Survivors (*n*=29)	Deaths (*n*=25)	*P*
Age (months) (median [range])	30 (3-132)	10 (1-144)	0.1
Sex (*N* (%)	M-16, F-13	M-13, F-12	1
PRISM III score (mean±SD)	16.6±12.3	31.2±14.1	<0.0001*
Morbidities (*N* (%)			
Pneumonia	3 (10.3)	6 (24)	0.28
Sepsis	4 (13.8)	1 (4)	0.36
Meningoencephalitis	3 (10.3)	5 (20)	0.45
AGN	4 (13.8)	0	0.12
Snake envenomation	3 (10.3)	0	0.24
Encephalopathy (*N* [%])	9 (31)	10 (40)	0.343
Mechanical ventilation (*N* [%])	19 (65.5)	24 (96)	0.007*
Shock (*N* [%])	22 (75.9)	25 (100)	0.01*
Renal replacement therapy (*N* [%])	8 (27.6)	7 (28.0)	0.61
Maximum creatinine value (mean±SD)	2.13±2.14	1.64±1.08	0.28

**P* value significant. AKI: Acute kidney injury; PRISM III: Pediatric risk of mortality III; AGN: Acute glomerulonephritis

acute post-streptococcal glomerulonephritis (PSGN), snake envenomation, hemolytic uremic syndrome (HUS) and congestive cardiac failure.

Mortality rate in children with AKI was 46.3%. In AKI stage 1, 7 (36.8%) patients died while in stage 2 and stage 3, 8 (57.1%) and 10 (47.6%) patients died respectively (differences not significant). Mortality was nil in PSGN, diarrhea, snake envenomation and TB, but highest in pneumonia (66.7%). Mortality in babies aged less than 10 months was 65% while it was 35.3% above 10 months (P = 0.049).

The age, etiological profile and maximum serum creatinine values were not different among the survivors and non-survivors [Table 3]. The length of hospital stay was 10.1 ± 5.8 days among survivors. Among the non-survivors, the corresponding value was 6.3 ± 4.3 days.

A total of 23 (79.3% of survivors) children with AKI had complete renal recovery while 6 (20.7% of survivors) had partial renal recovery at discharge. In AKI stage 1, out of the survivors, 11 (91.7%) had complete renal recovery while 1 (8.3%) had partial renal recovery at discharge. In AKI stage 2, 4 (66.7%) had complete renal recovery while 2 (33.3%) had partial renal recovery at discharge. In AKI stage 3, 8 (72.7%) had complete renal recovery, while 3 (27.3%) had partial renal recovery at discharge (differences not significant).

A total of 15 patients (27.8%) required dialysis. Nine (60%) underwent peritoneal dialysis while 6 (40%) underwent hemodialysis. The mortality among children requiring RRT was similar to children not requiring RRT (46.7% vs. 46.2%). Requirement of RRT was not related to age or the etiology of AKI.

The complications and co-morbidities observed among the study subjects included severe metabolic acidosis in 32 (59.3%), hyponatremia in 14 (25.9%), hypernatremia in 8 (14.8%), hyperkalemia in 13 (24.1%), hypertension in 8 (14.8%), encephalopathy in 19 (35.2%), thrombocytopenia in 21 (38.9%), mechanical ventilation in 43 (79.6%) and shock in 47 (87%) children. There was no correlation between stages of AKI and etiological profile, mortality or length of hospital stay.

The predictors of mortality on univariate analysis were: Age less than 10 months, shock and requirement of mechanical ventilation [Table 4]. In the multivariate model, requirement of mechanical ventilation was found to be an independent predictor of fatality (R^2 = 24.3%; odds ratio 9.7; 95% confidence interval [CI]: 1.1 – 85.5; P value 0.041).

Discussion

Detection of incidence, etiological profile and outcome of AKI is important for the institution of appropriate management as well as comparison of epidemiological

Table 4: Predictors of fatality in AKI on univariate analysis

Parameter	Death	Survived	Odds ratio	95% CI	*P* value
Age					
<10 months	13	7	1.842	1.055-3.216	0.049*
>10 months	12	22			
Gender					
Male	13	16	0.93	0.53-1.66	1
Female	12	13			
Etiology of AKI					
Pneumonia	6	3	1.58	0.89-2.80	0.27
Hemolytic uremic syndrome	1	1	1.08	0.26-4.47	1
Congestive cardiac failure	1	1	1.08	0.26-4.47	1
Stage of AKI					
Stage 1	7	12	0.72	0.37-1.40	0.4
Stage 2	8	6	1.35	0.75-2.40	0.37
Stage 3	10	11	1.05	0.58-1.88	1
Oliguria	11	10	1.24	0.70-2.18	0.58
Metabolic complications					
Metabolic acidosis	18	14	1.77	0.89-3.50	0.1
Hyponatremia	8	6	1.35	0.75-2.40	0.37
Hypernatremia	5	3	1.44	0.77-2.70	0.45
Hyperkalemia	5	8	0.79	0.37-1.68	0.54
Hypertension	1	7	0.24	0.04-1.53	0.056
Encephalopathy	10	9	1.23	0.69-2.18	0.57
Thrombocytopenia	9	12	0.88	0.48-1.62	0.78
Mechanical ventilation	24	19	6.14	0.93-40.56	0.007*
Shock	25	22	-	-	0.012*
Underlying renal disease	2	3	0.85	0.28-2.60	1
Requirement of RRT	7	8	1.01	0.53-1.915	1
Type of AKI					
Prerenal	1	2	0.71	0.14-3.60	1
Renal	22	26	0.92	0.39-2.16	1
Postrenal	2	1	1.48	0.63-3.48	0.59

**P* value significant. AKI: Acute kidney injury; RRT: Renal replacement therapy; CI: Confidence interval

studies for improved clinical decision making. The present prospective observational study from a tertiary center in southern India found the incidence of AKI to be 25.1% in critically ill-children admitted to the PICU. 27.8% of patients required RRT. Some recent pediatric studies on AKI using risk, injury, failure, loss, end-stage criteria or its modifications have reported the incidence of AKI to be widely varying from 10% to 82%, highlighting the heterogeneity of patient populations, diverse regional differences, sample sizes and study designs.[4-7,15] One of these studies was a retrospective analysis of prospectively collected clinical data in 3396 critically ill-children; 15.7% had some degree of AKI at admission and 10% had AKI develop during hospital course.[6] Another study from Texas in 150 mechanically ventilated children found the incidence of AKI to be 82%.[15] Of these children, 11 required dialysis. In another retrospective study from the Netherlands, among 103 children requiring mechanical ventilation, 58% developed AKI; 6 patients received RRT.[4] In yet another study from California, in 123 children with burn injury of 10% or more of body surface area, incidence of AKI was 45.5%.[5]

A wide spectrum of etiologies for AKI has been found in studies across the world. While sepsis, glomerulonephritis, HUS and acute tubular necrosis predominate in developing countries, these have been replaced by hemato oncologic complications and pulmonary failure as causes of AKI in the west.[10,16-21] One study from Turkey on 100 children with AKI described the most common causes as bone marrow transplantation, renal disease, dehydration, nephrotoxic medication and cardiac surgery. In another study from the same country, on 472 children with AKI (including 32.6% neonates), hypoxic ischemic injury and sepsis were leading causes of AKI.[8,22] At Kolkata, India, glomerulonephritis and snake bite were the two most important causes of AKI in 37 children, making up 70% of all cases.[23]

We found that the common etiologies were infections, PSGN, snake envenomation, hemolytic uremic syndrome (HUS) and congestive cardiac failure. Pneumonia, sepsis and meningoencephalitis accounted for the majority of all infections. Pneumonia constituted one-fourth of all infections associated with AKI and was associated with high mortality. Mortality was not seen in PSGN, diarrhea, snake envenomation and TB, but was quite common in pneumonia (66.7%). Increased risk of developing AKI has been mentioned with pneumonia, but seems to have been under-reported in children.[24] In a prospective study from Scotland, out of 1241 adults with pneumonia, 18% had AKI.[25] Tropical febrile illnesses have been significantly associated with AKI, especially in adults.[26] In our study too, tropical febrile illnesses (dengue, scrub typhus, TB and malaria) constituted 9.3% of children with AKI. Mechanisms for AKI in tropical illnesses include direct invasion by the micro-organism leading to acute interstitial nephritis (in dengue and TB), hemodynamic perturbation and renal ischemia (in malaria).[27-31] Severe diarrhea and PSGN form a large proportion of children with AKI in India.[32,33] In our study, acute glomerulonephritis (predominantly PSGN) accounted for 7.6% of patients. Diarrhea leading to AKI was uncommonly encountered. Probably, awareness regarding the usage of oral rehydration solution has led to fewer cases of severe dehydration and thereby AKI, being referred to our institute. Three patients developed AKI owing to snake envenomation in our study, which is an important problem in some regions of India.[23]

The mortality in AKI in children also has been reported to vary widely from 16% to 43.8%.[4,8,10,15-18] In our study, it was 46.3%, which is comparable to a recent study from Kuwait reporting 43.8% mortality.[18] A retrospective study of 311 children with ARF over a 22 year period

from Thailand reported mortality as 41.5%.[10] These studies used different definitions and criteria.[10,18] Much of the data on the incidence and mortality of AKI are limited to the developed world. Apart from mortality, 20.7% of children with AKI who survived, had partial renal recovery at discharge (majority being AKI stage 3), pointing toward significant morbidity resulting from AKI.

In the present study, requirement of mechanical ventilation was found to be an independent predictor of fatality in children with AKI. However, the wide CI of the odds of death indicates that the estimate lacks precision since the study was not powered to look at the predictors of fatality. Though age less than 10 months and shock predicted fatality on univariate analysis, they were eliminated on multivariate logistic regression analysis. Mortality in AKI is primarily related to etiology; PSGN and gastroenteritis having a much better outcome than sepsis, malignancy or major surgery.[1] Multiple factors have been described as predictors of outcome in AKI, again reflecting heterogeneity of patient populations. These include etiology of AKI and duration of symptoms before presentation.[16] Sepsis and HUS with prolonged anuria have been associated with poor outcome. In rapidly progressive glomerulonephritis, the outcome is related to prompt immunosuppressive therapy and histopathological findings. In contrast, acute tubular necrosis generally has a favorable outcome. Delay in seeking health-care, infections and cardiovascular/respiratory complications result in poor outcome.[34] In critically ill-patients with AKI undergoing hemodialysis, cardiovascular co-morbidities, metabolic acidosis and acute respiratory distress syndrome led to poor outcome.[35] Age below 2 years, shock, fluid overload, need for mechanical ventilation, multi-organ failure and late referral predicted poor outcomes in a study from Kuwait.[18]

The present study has some limitations. We examined only short term outcomes of hospitalized children with AKI. Children with AKI may have long-term residual renal injury e.g., microalbuminuria, hypertension or elevated creatinine levels.[23,36] Lack of information on the long-term outcome does not permit evaluation of the impact of mild AKI on renal function. Secondly, the study was conducted at a tertiary hospital; the clinical profile of patients would be affected by a referral bias. We did not compare children with AKI and those without AKI as this was not the objective of our study. Thirdly, data regarding non AKI patients was not collected and hence increased odds of mortality with AKI could not be analyzed. Finally, the study was not powered to examine predictors of mortality in AKI as this was not the primary outcome variable; and larger studies would be required.

In summary, we emphasize that the incidence of AKI is high in critically sick hospitalized children. It is associated with adverse outcomes, including high mortality. A clearer understanding of the long-term outcomes of this condition would allow optimization of follow-up strategies.

Conclusions

The present prospective observational study from a tertiary center in Pondicherry, southern India found the incidence of AKI to be 25.1% in critically ill-children admitted to the PICU. The mortality was 46.3% in children with AKI admitted to the PICU and 20.7% of children with AKI who survived (majority being AKI stage 3), had partial renal recovery at discharge. Overall, 27.8% of patients required RRT. The etiological profile of AKI was dominated by infections, including pneumonia, sepsis, meningoencephalitis and tropical febrile illnesses. PSGN, snake envenomation and HUS also contributed significantly. On multivariate logistic regression analysis, requirement of mechanical ventilation was found to be an independent predictor of fatality in children with AKI.

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