ORIGINAL ARTICLE

Acute Renal Failure Due to Jengkol Intoxication in Children

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ABSTRACT Jengkol intoxication is well-known in Indonesia. We report a series of 39 patients with jengkol intoxication admitted to the Department of Child Health, Cipto Mangunkusumo Hospital, Jakarta, during the period of 1984 through 1993. Patients ranged in age from 3 to 14 years. The male to female ratio of all cases was 1.8 to 1, but the ratio of patients sufferring from acute renal failure was 5.7 to 1. Oliguria or anuria presented in all cases with acute renal failure. Three patients underwent peritoneal dialysis which gave rapid improvement; 2 patients had died due to acute renal failure before dialysis could be performed. When compared with previous reports, it seems that the admission for jengkol intoxication has been declining. Change in the way of consuming the bean and increasing number of hospitals in Jakarta may be responsible for the decline of cases admitted. **[Paediatr Indones 1994; 34:164-169]**

Introduction

Jengkol beans *(Pithecolobium lobatum)* is widely consumed in Indonesia especially in rural areas. In certain istances, due to unknown causes, it may cause intoxication, with the cardinal symptoms of abdominal pain, vomiting, severe disuria, and gross hematuria. The mouth respiration and urine produce a typical faulty odor of the jengkol bean. If left untreated it can progress to acute renal failure. The cause of jengkol intoxication is the jengkolic acid present in the beans, which precipitates along the urinary tract and obstructs the urinary flow.

Jengkolic acid is an amino acid, isolated by Van Veen and Hyman in 1993.[1] It has an amphoteric character. It can easily precipitate in an acid melieu, forming crystals which look like needles and sometimes forming rosette, odorless, and non volatile. It is these crystals that cause laceration and obstruction of the urinary tract. Thus the renal failure found in jengkol intoxication is postulated as a post-renal type.[2,3] Since up till now reports on this complication especially in children are rare, we conducted a study of acute renal failure due to jengkol intoxication. The purpose of the study was to have a better understanding of the severity, management, and complications of this disease.

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Methods

The study was conducted during a 10-year period from January 1984 through December 1993. Twenty three children with acute renal failure due to jengkol intoxication were hospitalized in the nephrological ward of the Department of Child Health, Medical School University of Indonesia-Cipto Mangunkusumo Hospital. Diagnosis of jengkol intoxication was made on the following criteria:

1. history of consuming jengkol bean
2. complaints of abdominal pain, vomiting, disuria
3. typical odor from mouth and urine
4. microscopic and or macroscopic hematuria
5. if possible the finding of jengkol crystals in the urine.

Acute renal failure was diagnosed by the finding of elevated blood creatinine and urea concentrations with or without oliguria/anuria. Laboratory investigation to these children included urinalysis, peripheral blood examination, renal function test, electrolytes, and blood gas analysis. An ultrasonography was performed in every patient and repeated weekly. Renal biopsy was performed if possible. The children were treated immediately with either oral or intravenous sodium bicarbonate if there was severe vomiting or decrease in consciousness. Furosemide was added to maintain good diuresis.

Results

During the study period 64 patients with acute renal failure (ARF) were hospitalized at Cipto Mangunkusumo Hospital; 20 cases (31%) were due to jengkol intoxication. At the same period of time the number of children hospitalized due to jengkol intoxication were 39 cases. The sex distribution of the patients is shown in Table 2.

Males (25 patients) were more affected than females (14 patients) with a ratio of 1,8:1. Acute renal failure (ARF) was found in 20 cases (51,3%). The age and sex distribution of children suffering from ARF due to jengkol intoxication can be seen in Table 3. Males (17 patients) were affected 5.4 times more frequently than females (3 patients). The youngest child affected was 4 years old (2 cases) and the oldest 14 years (2 cases).

The clinical manifestations on admission are shown in Table 4. Oliguria and anuria were found in all cases; most of them (13 cases) showed anuria. Vomiting was found in 15 (75%), hematuria in 14 (70%), disuria in 14 (70%). Fever was present in 7 cases (35%), dyspnea in 8 (40%), decrease of consciousness in 5 (25%), and convulsion in 6 (30%). Hypertension was found in 11 cases (55%) but this eventually disappeared within two to three days.

Table 2. Number of patients with jengkol intoxication hospitalized at Cipto Mangunkusumo Hospital 1984-1993

Year	Male	Female	Total	ARF	Death
1984	7	6	13	6	1
1985	6	2	8	3	-
1986	1	2	3	2	-
1987	4	1	5	2	1
1988	-	1	1	-	-
1989	2	-	2	2	-
1990	2	2	4	2	-
1991	1	-	1	1	-
1992	2	-	2	2	-
1993	-	-	-	-	-
TOTAL	25 (64%)	14 (36%)	39 (100%)	20 (51%)	2 (5,1%)

Table 3. Age and sex distribution of children with ARF

Age (yrs)	Sex		
	Male	Female	Total
3-	2	-	2
5-	3	-	3
7-	2	2	4
9-	5	-	5
11-	3	1	4
13-	2	-	2
Total	17 (85%)	3 (15%)	20

Laboratory examination revealed hematuria either macroscopically or microscopically in all 20 cases, proteinuria in 14 of 20 cases, leukocyturia in 6 cases,

Table 4. Clinical symptoms of the patients with ARF due to jengkol intoxication

Symptoms	No. of pts	%
Vomiting	15	75
Oliguria	7	35
Anuria	13	65
Hematuria	14	70
Disuria	14	70
Fever	7	35
Dyspnea	8	40
Decrease of consciousness	5	25
Convulsion	6	30
Hypertension	11	55
Abdominal pain	6	30
Flank pain	5	25

cylinderuria in only 2 cases. The blood albumin, globulin, cholesterol were within normal limits. The blood urea ranged between 49 to 347 mg/dl, with a mean of 165 mg/dl and the blood creatinine between 1.1 mg/dl and 14.0 mg/dl with a mean of 5.12 mg/dl.

The result of ultrasonography of the urinary tract showed variable hydronephrosis and hydroureter, which might be unilateral and bilateral, but disappeared on subsequent ultrasonography.

Biopsy was done in only 2 patients due to difficulty in having parental consent, because the children usually became better quickly with disappearance of all symptoms after 2 to 3 days, except in patients who had complication or who underwent dialysis. The results showed that in one case there was a tubular epithelial damage, which could be due to

obstruction of the urinary tract. The results of treatment showed that in 17 patients the renal failure could be overcome conservatively with the combination of diuretic and sodium bicarbonate either orally or intravenously.

Three patients underwent peritoneal dialysis due to the advanced renal failure on admission i.e., the blood urea was 347 mg/dl and creatinine 10,92 mg/dl in the first case and 317 mg/dl and 14 mg/dl in the second case and in the third case 261 mg/dl and 6,2 mg/dl respectively. Two out of 20 patients (10%) died with ARF or 5% out of the total number of 39 jengkol intoxication due to advanced renal failure before dialysis could be done (Table 2).

Discussion

Jengkol intoxication is well-known in Indonesia. The first report in Indonesia was published by de Jong in 1930.[4] Unfortunately since then not many publications were written in the Indonesian literature.[1-3,5-7] Suharjono and Sadatun[5] reported 50 cases of jengkol intoxication at Cipto Mangunkusumo Hospital in a period of 8 years (1959-1967). Most of them were boys (90%) with a mortality of 6%. Tambunan in 1979 reported 15 children with jengkol intoxication in a period of 1,5 years, most of them were also boys (86,6%).[8] Sjamsudin et al. reported 24 cases of jengkol intoxication among 125 children hospitalized due to all kinds of intoxication in 13 hospitals in Jakarta between 1977-1978.[9] Ramayati et al.[10] reported 14 cases of jengkol intoxication in children at Dr. Pirngadi Hospital Medan in a period of 5 years (1982-1986), while Sekarwana and Singadipoera in 1990 reported 6 cases of acute renal failure due jengkol intoxication and a period of 4 years, 3 of them were dialyzed but all patients survived.[11]

In this report 39 jengkol intoxication cases were hospitalized at Cipto Mangunkusumo Hospital in a period of 10 years (1984-1993). Twenty (51.3%) of them had acute renal failure. The male to female ratio in this study is 1,8:1. It is less prominant than the report of Suharyono and Sadatun[5] or Tambunan[8] who found a ratio 9:1, but in this study the male to female ratio in children who had acute renal failure complication is higher, namely 5.4:1 (Table 3).

The number of jengkol intoxication patients hospitalized in this last 10 years was less than previously reported by Tambunan et al. in 1979 and also by Suharyono and Sadatun between (1959-1967). It seems that in the last years there is a decline in the number of children hospitalized with this intoxication. Also if we observe the number of cases in this report, more than half were hospitalized in the first 2 years (1984-1985), while in the last eight years the number of cases was smaller, and in 1993 no single case was admitted. The reason of this is difficult to answer since the consumption of jengkol among the population is still high. It could be due to the change in the way of consuming the bean since the concentration of the acid is much lower if the bean is roasted and eaten as chips than when it is boiled.[12] On the other hand it could be due to the increased number of new hospitals build in Jakarta, so that only severe cases

were referred to Cipto Mangunkusumo Hospital as the top referral Hospital in Jakarta. An epidemiological survey thus should be done to answer this question.

The jengkolic acid crystals could be dissolved in an alkaline solution. Mild cases of jengkol intoxication could be managed with oral sodium bicarbonate, but in severe cases where vomiting is one of the prominent symptoms (Table 4), sodium bicarbonate should be given by infusion. To dissolve crystals in the bladder and in the urethra which sometimes could be seen with the bare eye in the preputium or orificium urethra externa the bladder should be irrigated with sodium bicarbonate solution.

If the acute renal failure is severe, peritoneal dialysis should be performed. In this report 3 patients underwent dialysis for only a few days, because diuresis was than increased progessively following alkalization of the blood and urine. Two cases died due to advanced renal failure before dialysis could be done. Suharyono and Sadatun reported a mortality of 6% among 50 jengkol intoxication cases that were also due to acute renal failure.[5]

The cause of oliguria and anuria in these patients was postulated previously to be due to obstruction of the urinary tract by deposits of jengkolic acid crystals.[2,3] The finding of hydronephrosis and hydroureter in this report and also the result of renal biopsy showing damage of the tubuli although only in 1 case could support the hypothesis of urinary tract obstruction as the cause of acute renal failure. The result of beta$_2$ micro globulin (β_2m) examination as a specific parameter of renal proximal tubular dysfunction showed that the β_2m level in the urine is high, meaning that the reabsorptive mechanism of the proximal tubule is impaired.[13] This finding further supports the role of tubular damage due to the obstruction of the urinary tract. Whether this damage is due to cris- talization of jengkolic acid in the tubular area or back pressure due to obstruction in the ureter or pyelum has not been solved yet, since up till now no crystals could be demonstrated in the histological picture either in patients or in experimental animals.[14] This is due to the fact that the jengkolic acid crystals are dissolved in the preparation or staining of the histological specimen.

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