



Original Article

Intravenous albumin for the prevention of contrast-induced nephropathy in patients with liver cirrhosis and chronic kidney disease undergoing contrast-enhanced CT



Heejung Choi, Yoonjung Kim, Soo Min Kim, Junam Shin, Hye Ryoung Jang, Jung Eun Lee, Woosong Huh, Yoon-Goo Kim, Ha Young Oh, Dae Joong Kim*

Department of Medicine, Samsung Medical Center, Sungkyunkwan University School of Medicine, Seoul, Korea

ABSTRACT

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Background: The purpose of this study was to evaluate the incidence of contrast-induced nephropathy (CIN), and the effect of intravenous albumin for prophylaxis of CIN in patients with liver cirrhosis (LC) and chronic kidney disease (CKD).

Methods: We conducted a retrospective study of 81 subjects with LC and CKD (estimated glomerular filtration rate (eGFR) < 60 mL/min/1.73 m²) who underwent contrast-enhanced computed tomography (CT). Patients received either isotonic sodium bicarbonate solution (3 mL/kg for 1 h before CT and 1 mL/kg/h for 6 h after CT) or albumin (20% albumin, 25 mL for 1 h before CT and 75 mL for 6 h after CT). CIN was defined as an increase of $\geq 25\%$ or ≥ 0.5 mg/dL in serum creatinine level.

Results: Overall, CIN developed in three patients (3.7%). Of the 81 subjects, 43 received sodium bicarbonate solution and 38 received albumin. Both groups were comparable with regard to age, sex, diabetes mellitus, and baseline eGFR. The albumin group showed a significantly poorer liver function profile. CIN incidence did not differ significantly between the groups: it occurred in one (2.3%) of the 43 subjects receiving sodium bicarbonate and two (5.3%) of the 38 subjects receiving albumin ($P=0.6$). However, the albumin group showed a significantly smaller increase in body weight ($P=0.03$).

Conclusion: The incidence of CIN in patients with LC and CKD undergoing contrast-enhanced CT after preventive measures was relatively low. The incidence of CIN was not significantly different between sodium bicarbonate and albumin groups.

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Introduction

Renal failure is a common and serious complication in patients with liver cirrhosis (LC), and is a predictor of poor outcome [1–3].

* Corresponding author. Division of Nephrology, Samsung Medical Center, Sungkyunkwan University School of Medicine, 50 Irwon-dong, Gangnam-gu, Seoul, 135-710, Korea.
E-mail address: daejoongsmc.kim@samsung.com (DJ Kim).

Patients with LC are susceptible to renal failure primarily because of disturbances in circulatory function (splanchnic vasodilation, intravascular volume depletion, and altered renal hemodynamics). Gastrointestinal bleeding, paracentesis, and administration of nephrotoxic drugs such as aminoglycosides and contrast agents also cause renal insufficiency [4,5].

Contrast-induced nephropathy (CIN), defined as a rapid deterioration in renal function after administration of contrast medium without other specific causes, is a common cause of

acute kidney injury [6]. Underlying chronic kidney disease (CKD) is known to be the most important risk factor for CIN; diabetes, heart failure, and injection of a large amount of contrast agent are also considered risk factors. Although it has been suggested that LC may be a risk factor for CIN, this has not yet been confirmed. LC patients with renal insufficiency are likely to be more vulnerable to CIN, but there has been no research on this topic in such patients.

Although the optimal therapy for prevention of CIN remains uncertain, it is widely recommended that intravenous isotonic fluid and oral *N*-acetylcysteine be administered to high-risk patients before and after they receive a radio-contrast agent. However, in patients with LC, isotonic fluid may aggravate ascites and volume overload. Albumin infusions have been used in patients with LC for volume expansion after paracentesis or in hepatorenal syndrome. Unlike crystalloid fluid, albumin increases only the intravascular volume and also has antioxidant effects, so is considered to prevent CIN. However, to the best of our knowledge, no research on the effect of intravenous albumin on prevention of CIN in patients with LC has been reported.

The aim of the present study was to examine the incidence of CIN and the effect of intravenous albumin compared with isotonic sodium bicarbonate solution for the prevention of CIN in patients with LC and CKD undergoing contrast-enhanced computed tomography (CT) in an outpatient setting.

Methods

Setting

This study was a retrospective observational study performed at a single center. Samsung Medical Center is a tertiary referral hospital in Seoul, Korea and over 40,000 contrast-enhanced CT scans are performed annually in its clinics. The five-bed short stay unit was established in October 2009 to provide short-stay treatment to prevent the occurrence of CIN in patients with CKD after contrast-enhanced CT.

The contrast agents used for CT scans are low-osmolality contrast agents. For CT scans other than coronary CT angiography, one of the following four contrast agents is randomly given to patients in specified amounts, depending on the area to be scanned: iobitridol (Xenetix 300; 300 mg iodine/mL, 695 mOsm/kg; Guerbet, Aulnay S. Bois, France), iomeprol (Iomeron 300; 300 mg iodine/mL, 520 mOsm/kg; Ilsung Pharmaceuticals, Seoul, Korea), iohexol (Omnipaque 300; 300 mg iodine/mL, 672 mOsm/kg; GE Healthcare, Little Chalfont, UK), iopromide (Ultravist 300; 300 mg iodine/mL, 586 mOsm/kg; Bayer Healthcare, Berlin, Germany). For coronary CT angiography, iomeprol (Iomeron 350; 350 mg iodine/mL, 620 mOsm/kg; Ilsung Pharmaceuticals) is used and one of the subjects of this study received this agent.

Standard CIN prophylaxis protocol

The standard CIN prophylaxis protocol in our institution is as follows. Patients receive an intravenous injection of 3 mL/kg/h and of isotonic sodium bicarbonate fluid for 1 h before the CT scan and 1 mL/kg/h for 6 h after the CT scan. The patients take *N*-acetylcysteine 1200 mg orally twice daily on the day before and on the day of CT. Serum creatinine is measured when the patient comes to the outpatient clinic 2–5 days after leaving the hospital.

Patients with LC who consent receive an intravenous injection of 100 mL of 20% albumin before and after the CT scan. They are injected with 25 mL for 1 h before the CT scan and 75 mL for 6 h after the CT scan (albumin group) and take *N*-acetylcysteine according to the same protocol as for the group receiving isotonic sodium bicarbonate. If patients do not agree to the infusion of albumin, isotonic sodium bicarbonate is injected (bicarbonate group).

Patients

The study subjects were patients with LC who stayed in the short-stay unit of the hospital between October 2009 and February 2011 and who underwent a CT scan after the CIN prophylaxis protocol. We included those patients whose estimated glomerular filtration rate (eGFR) was below 60 mL/min/1.73 m² on the day of their CT scan. The diagnosis of LC was made on the basis of the usual clinical, laboratory, and radiologic findings. The study was approved by the Institutional Review Board of the hospital.

Measures

The medical records and electronic data for patients included in the study were reviewed retrospectively to check clinical characteristics such as age, sex, body weight, diabetes mellitus, hypertension, causes of LC, existence of hepatocellular carcinoma, and medications (diuretics, angiotensin-converting enzyme inhibitor or angiotensin receptor blocker, statin). Child–Pugh classifications were assessed to examine the severity of LC. Baseline serum creatinine and total CO₂ were measured before the CT scan on the same day, and were measured again at outpatient clinics 2–5 days after the CT scan to assess the development of CIN. CIN was defined as an increase of $\geq 25\%$ or ≥ 0.5 mg/dL in serum creatinine level. Estimated GFR was based on the abbreviated Modification in Diet and Renal Disease Study equation and calculated as follows: $eGFR$ (mL/min/1.73 m²) = $175 \times (\text{serum creatinine})^{-1.154} \times (\text{age})^{-0.203} \times (0.742, \text{ if female})$ [7].

Statistical analysis

Data are expressed as mean \pm SD. Categorical variables for the two groups were compared using a χ^2 test or Fisher's exact test. Continuous variables were compared using an unpaired *t* test if the data from both groups had a normal distribution, but the Mann–Whitney test was applied if data from either of the two groups did not have a normal distribution. Two-sided *P* values are reported, with 0.05 as the level of significance. All statistical analyses were performed using SPSS 12.0 (SPSS, Chicago, IL, USA).

Results

Baseline characteristics and CIN incidence

A total of 67,302 contrast-enhanced CT scans were performed in our outpatient clinics from October 2009 to February 2011. Some 1111 of these were performed after implementation of preventive treatments in the short-stay unit, and 107 patients who underwent such treatment had LC. Excluding the 13 patients with eGFR ≥ 60 mL/min/1.73 m² on the day of the CT scan and 13 patients who were not followed up at outpatient clinics after their CT scan, 81 patients were included.

The clinical and laboratory characteristics of all subjects are shown in Tables 1 and 2. The mean age was 65.7 years and 62 patients (76.5%) were male. Diabetes mellitus was found in 39 patients (48.1%) and hypertension in 31 patients (38.3%). The mean baseline serum creatinine was 1.73 mg/dL and mean eGFR was 40.5 mL/min/1.73 m². Hepatitis B was the most common cause of LC and hepatocellular carcinoma was identified in 70 of 81 patients (86%).

Overall, CIN developed in three patients, representing an incidence of 3.7% (Table 3). Patients with ascites were at increased risk of developing CIN ($P=0.02$; Table 4). In two of the three patients with CIN, renal function improved to the baseline serum creatinine level during their outpatient

follow-up. The remaining patient, who had baseline eGFR of 8.9 mL/min/1.73 m², refused further aggressive treatment including hemodialysis because of advanced hepatocellular carcinoma. He was discharged from the hospital and died 26 days after his CT scan (Table 5).

Effect of intravenous albumin on CIN prevention

Of the 81 study patients, 43 received sodium bicarbonate and 38 received albumin. The clinical and laboratory characteristics of the two groups are shown in Table 1. There were no significant differences between the groups in age, sex, diabetes mellitus, and baseline renal function. The albumin

Table 1. Baseline clinical and biochemical characteristics of the study patients

Characteristics	Total (n=81)	Sodium bicarbonate (n=43)	Albumin (n=38)	P
Age (years)	65.7 ± 8	66.2 ± 8.4	65.1 ± 7.6	0.4
Male	62 (76.5)	34 (79.1)	28 (73.7)	0.6
Diabetes mellitus	39 (48.1)	17 (39.5)	22 (57.9)	0.1
Hypertension	31 (38.3)	19 (44.2)	12 (31.6)	0.2
Cause of chronic kidney disease				
Diabetes mellitus	31 (38.3)	17 (39.5)	14 (36.8)	
Hypertension	20 (24.7)	13 (30.2)	7 (18.4)	
Glomerulonephritis	15 (18.5)	8 (18.6)	7 (18.4)	
Unknown	15 (18.5)	5 (11.6)	10 (26.3)	
Serum creatinine (mg/dL)	1.73 ± 0.6	1.6 ± 0.3	1.8 ± 0.9	0.9
eGFR (mL/min/1.73 m ²)	40.5 ± 10	41.3 ± 8.1	39.7 ± 11.8	0.5
Serum total CO ₂ (mEq/L)	19.5 ± 3.1	19.6 ± 3.1	19.5 ± 3.2	0.8
Use of diuretics	25 (30.9)	9 (20.9)	16 (42.1)	0.04
Use of ACE inhibitor/ARB	27 (33.3)	18 (41.9)	9 (23.7)	0.08
Use of statins	8 (9.9)	1 (2.3)	7 (18.4)	0.02

Data are expressed as mean ± SD or number (percentage).

ACE, angiotensin-converting enzyme; ARB, angiotensin receptor blocker; eGFR, estimated glomerular filtration rate.

Table 2. Baseline liver function tests and cirrhosis etiology for the study patients

Characteristics	Total (n=81)	Sodium bicarbonate (n=43)	Albumin (n=38)	P
Bilirubin (mg/dL)	0.9 ± 0.7	0.7 ± 0.5	1.2 ± 0.8	0.004
Albumin (g/dL)	3.7 ± 0.6	4.0 ± 0.4	3.4 ± 0.6	< 0.001
Prothrombin time (INR)	1.2 ± 0.2	1.1 ± 0.2	1.3 ± 0.2	< 0.001
Child–Pugh class (A/B/C)	59/20/2	39/4/0	20/16/2	< 0.001
Etiology of cirrhosis				
Alcoholism	14 (17.3)	9 (20.9)	5 (13.2)	
Hepatitis B	51 (63)	26 (60.5)	25 (65.8)	
Hepatitis C	5 (6.2)	3 (7.0)	2 (5.3)	
Primary biliary	1 (1.2)	1 (2.3)	0 (0.0)	
Cryptogenic	10 (12.3)	4 (9.3)	6 (15.8)	
Hepatocellular carcinoma	70 (86.4)	33 (76.7)	37 (97.4)	0.007
Ascites on CT scan	22 (27.1)	6 (14.0)	16 (42.1)	0.007

Data are expressed as mean ± SD or number (percentage).

CT, computed tomography; INR, international normalized ratio.

Table 3. Contrast volumes and biochemical responses in patients receiving either sodium bicarbonate or albumin

Characteristics	Total (n=81)	Sodium bicarbonate (n=43)	Albumin (n=38)	P
Contrast medium volume by body weight (mL/kg)	1.9 ± 0.3	2.0 ± 0.3	1.9 ± 0.3	0.07
Change in serum creatinine (mg/dL)	0.03 ± 0.2	0.01 ± 0.2	0.06 ± 0.3	0.3
Change in eGFR (mL/min/1.73 m ²)	0.1 ± 5.1	0.67 ± 5.1	-0.54 ± 5.1	0.3
Change in serum total CO ₂ (mEq/L)	2.6 ± 2.5	2.9 ± 2.2	2.2 ± 2.7	0.2
Incidence of CIN	3 (3.7)	1 (2.3)	2 (5.3)	0.6
Change in body weight (kg)	0.3 ± 0.8	0.5 ± 0.7	0.1 ± 0.9	0.03

Data are expressed as mean ± SD or number (percentage).

CIN, contrast induced nephropathy; eGFR, estimated glomerular filtration rate.

group contained significantly more patients who were taking diuretics ($P=0.04$) or statins ($P=0.02$).

The albumin group had a significantly poorer liver function profile than the sodium bicarbonate group: higher bilirubin (0.7 mg/dL for sodium bicarbonate and 1.2 mg/dL for albumin; $P=0.004$) and prothrombin time (international normalized ratio 1.1 for sodium bicarbonate and 1.3 for albumin; $P<0.001$) and lower albumin (4.0 g/dL for sodium bicarbonate and 3.4 g/dL for albumin; $P<0.001$). In terms of the Child–Pugh classification, the albumin group had a significantly higher proportion of patients in the Child–Pugh class B or C ($P<0.001$). Six patients (14%) in the sodium bicarbonate group and 16 (42.1%) in the albumin group had ascites ($P=0.007$).

CIN developed in one (2.3%) of the 43 patients receiving sodium bicarbonate and two (5.3%) of the 38 patients receiving albumin; this difference was not significant ($P=0.6$). The albumin group showed a significantly smaller increase in body weight; the average weight of the group increased by 0.1 kg compared with 0.5 kg for the sodium bicarbonate group ($P=0.03$; Table 3).

None of the patients who received sodium bicarbonate or albumin showed any complication related to fluid administration.

Table 4. Clinical characteristics of patients with and without contrast-induced nephropathy

Characteristics	No CIN ($n=78$)	CIN ($n=3$)	P
Age (years)	65.4 ± 7.9	73 (66–80)	0.1
Diabetes mellitus	36 (46.2)	3 (100)	0.1
Use of diuretics	25 (32.1)	0 (0.0)	0.5
Baseline eGFR (mL/min/1.73 m ²)			0.1
≥ 45 to < 60	29 (37.2)	0 (0.0)	
≥ 30 to < 45	39 (50.0)	2 (66.7)	
< 30	10 (12.8)	1 (33.3)	
Ascites	19 (24.4)	3 (100)	0.02

Data are expressed as mean ± SD or median (range) for nonparametric variables, or number (percentage).

CIN, contrast induced nephropathy; eGFR, estimated glomerular filtration rate.

Table 5. History and clinical outcome for patients with contrast-induced nephropathy

Patient	Sex	Age (y)	Fluid	DM	Baseline Cr (mg/dL)	Baseline eGFR (mL/min/1.73 m ²)	Child–Pugh class	Ascites	Peak serum Cr (mg/dL)	Outcome
1	M	66	Albumin	Yes	1.94	34.8	B	Yes	2.99	Improved
2	M	80	Albumin	Yes	6.11	8.9	B	Yes	6.73	Death
3	M	72	Sodium bicarbonate	Yes	1.9	34.9	A	Yes	2.39	Improved

Cr, creatinine; DM, diabetes mellitus; eGFR, estimated glomerular filtration rate.

Table 6. Literature review of contrast-induced nephropathy in patients with liver cirrhosis after contrast-enhanced computed tomography.

Study	LC patients	LC patients with CKD (CKD definition)	Baseline serum Cr (mg/dL)	CIN incidence in LC patients	CIN incidence in LC patients with CKD
Najjar et al. [12]	72	2 (Cr > 1.5 mg/dL)	0.87 ± 0.06	2/72 (2.8%)*	1/2
Guevara et al. [11]	31	5 (GFR < 50 mL/min)	0.9 ± 0.02 (0.3–1.9)	0/31	0/5
Lodhia et al. [13]	216	3 (Cr > 1.5 mg/dL)	Not reported	53/216 (25%)†	1/3

* No clear definition of CIN was provided.

† CIN was defined as a decrease of ≥ 25% in creatinine clearance using the Cockcroft–Gault equation.

CIN, contrast-induced nephropathy; CKD, chronic kidney disease; Cr, creatinine; GFR, glomerular filtration rate; LC, liver cirrhosis.

Discussion

In the present study, patients with LC and renal insufficiency (eGFR < 60 mL/min/1.73 m²) received CIN prophylaxis of isotonic sodium bicarbonate or albumin before and after a CT scan; CIN occurred in 3.7%. There was no significant difference in CIN incidence between the sodium bicarbonate and albumin groups, but the albumin group showed a smaller increase in weight. Underlying renal insufficiency is the most important risk factor for CIN, and CIN incidence after a CT scan in patients with CKD can range from 2.5% to 12% [8–10]. Considering that the patients surveyed for this study had both renal insufficiency and LC, the CIN incidence observed seems to be relatively low.

LC has not been verified as a risk factor for CIN and only three small studies on this issue have been reported (Table 6). Guevara et al. prospectively followed the renal function of 31 patients with LC after administering a contrast agent [11]. The glomerular filtration rate was measured by ¹²⁵I-iothalamate clearance and no significant changes in renal function were observed. As patients with serum creatinine of 2 mg/dL or higher were excluded, the mean baseline serum creatinine was 0.9 ± 0.02 mg/dL. Only five patients with GFR < 50 mL/min were included. In a retrospective case-control study of 72 patients with LC performed by Najjar et al., the CIN incidence was not higher than for patients without LC [12]. Although CIN was not clearly defined in this study, CIN developed in two (2.8%) of 72 LC patients. Two patients with CKD (serum creatinine > 1.5 mg/dL) were included among the 72 patients, and one suffered from CIN. Lodhia et al. also carried out a retrospective study of 216 patients with LC in 2009 in which CIN developed in 25% of patients [13]. CIN was defined as a decrease in creatinine clearance of 25% or more and patients with baseline serum creatinine of 1.8 mg/dL or higher were excluded. Only three patients with serum creatinine of 1.5 mg/dL or higher were included, and CIN occurred in one.

All three studies were based on a limited number of patients, and the study by Lodhia et al. [13] had no control group because it was a retrospective study. The three studies were also based on hospitalized patients with LC, only a few patients had renal insufficiency, and no preventive treatment was performed before and after the CT scan.

Renal failure in hospitalized patients with LC is common and the reported incidence ranges from 23% [5,14] to 44% [15]. The most common causes of renal insufficiency are prerenal, including infection, hypovolemia, vasoconstriction caused by nonsteroidal anti-inflammatory drugs or radiocontrast agents, and hepatorenal syndrome. In addition to these, intrinsic renal factors such as acute tubular necrosis and glomerulonephritis and post-renal obstruction are considered to be causes. Since the three studies described above were based on hospitalized patients with LC, some patients may have suffered from renal failure caused by factors other than CIN. However, renal insufficiency caused by factors other than CIN can be ruled out in our study, because participants were outpatients. Prakash et al. reported that many patients with renal insufficiency and LC could be categorized as having CKD (63/178, 35%) [15]. Because CKD is the most important factor that increases susceptibility to CIN and the prognosis of LC patients who have acute renal failure is poor, it is important to prevent CIN when a radiocontrast agent is injected into a patient with LC and renal insufficiency.

Albumin is the most abundant plasma protein and is responsible for 70% of the plasma oncotic pressure [16]. Albumin plays various roles in the human body, and its most important role is to maintain intravascular volume and colloid oncotic pressure. Its other physiological functions are to bind ligands, carry various materials including hormones, lipids, and drugs, and act as an antioxidant [17].

Indications for the infusion of albumin have not been clearly defined, and the procedure is still controversial. It has not been demonstrated to be better than crystalloid fluid for volume expansion in acute hypovolemia [18], and has not been shown to have benefits for patients with hypoalbuminemia [19]. The use of albumin infusion is recommended in LC patients with spontaneous bacterial peritonitis, hepatorenal syndrome, and large-volume paracentesis [20].

It has been hypothesized that the two major mechanisms underlying the pathogenesis of CIN are renal vasoconstriction and direct cytotoxic effects of contrast agents. Renal blood flow decreases by 30–45% within 2–4 h after injection of a contrast agent [21,22], which could lead to a deterioration in renal function because of medullary hypoxia. Another possible cause is the direct cytotoxic effect of oxygen free radicals created after injection of a radiocontrast agent, which cause renal tubular injury [23,24]. It has been shown the hydration with isotonic fluid is an effective strategy for CIN prevention, and addition of oral *N*-acetylcysteine, which is expected to have an antioxidant effect, is recommended for high-risk patients.

If albumin is infused before and after a CT scan, it has volume expansion and antioxidant effects and is thus expected to help prevent the occurrence of CIN. First, its effect on volume expansion is assessed. Although each gram of albumin theoretically binds approximately 18 g of water, a study of plasma volume expansion in patients injected with 50 g of albumin after surgery showed that this increased plasma volume by approximately 500 mL (or 11 mL/g of retained albumin), which was less than expected [25]. If a patient weighing 60 kg is injected with isotonic sodium bicarbonate in accordance with our protocol, the total volume of fluid injected before and after the CT scan amounts to 540 mL (180 mL before the CT scan, 360 mL after the CT scan). Assuming that a quarter of the isotonic sodium bicarbonate solution is accounted for by fluid in the intravascular space,

this equals 135 mL (45 mL before and 90 mL after the CT scan). The total injection of albumin in this study was 20 g. Assuming that 1 g of albumin was combined with 11 mL of water, this amounts to 220 mL (55 mL before and 165 mL after the CT scan). According to our study results, injection of albumin increased expansion of the intravascular volume, but induced a limited weight increase after fluid injection. Second, with respect to the antioxidant effects of albumin, it has been reported that a physiological solution of human serum albumin restricts the creation of oxygen free radicals by polymorphonuclear leukocytes [17,26]. In a clinical study that measured endotoxin, nitric oxide products, tumor necrosis factor (TNF)- α , and interleukin (IL)-6 in patients with spontaneous bacterial peritonitis and LC, a group injected with albumin showed a significant decrease in TNF- α and IL-6 compared with a group without albumin injection [27]. Patients with acute lung injury had an increased antioxidant capacity after injection of albumin [28]. If albumin is injected before and after injection of a contrast agent, it is expected to scavenge oxygen free radicals, a major cause of CIN.

Comparison of patients with and without CIN showed no significant difference in age or frequency of diabetes mellitus, both of which are known to be major risk factors for CIN. Although the incidence of CIN increased as the baseline eGFR decreased [0% (0/29) for $45 \leq \text{eGFR} < 60$, 4.9% (2/41) for $30 \leq \text{eGFR} < 45$, 9.1% (1/11) for $\text{eGFR} < 30$], the correlation was not significant ($P=0.1$). This may be because our study involved a limited number of patients and CIN cases. All patients with CIN had ascites according to CT imaging, and thus patients with ascites had a significantly higher incidence of CIN ($P=0.02$). The study by Lodhia et al. showed similar results [13], which suggests that ascites can cause intravascular volume depletion and increase the incidence of CIN.

Our study has several limitations. First, it is a retrospective study. Because of the lack of randomization and differences in baseline characteristics between the sodium bicarbonate and albumin groups, the effect of preventive measures could not be clearly defined. Second, since only patients who agreed to use the short-stay unit were included in the study, the incidence of CIN among patients who underwent a CT scan without preventive treatment could not be assessed. Third, CIN was defined on the basis of serum creatinine, but serum creatinine in patients with LC is an unreliable marker of renal function [29]. Inulin clearance or 24-h urine collection can be used for more accurate assessment of renal function, but these methods were not feasible in this retrospective study of outpatients who were hospitalized for a short period. Furthermore, because the creatinine was measured once for each patient at variable time intervals after the CT scan, we may have missed some patients who developed CIN.

In conclusion, in patients with LC and CKD, the incidence of CIN after contrast-enhanced CT was relatively low after preventive therapy with intravenous volume expansion and oral *N*-acetylcysteine. The incidence of CIN after intravenous administration of isotonic sodium bicarbonate or albumin did not significantly differ. However, the group administered albumin showed a smaller increase in weight and patients with ascites were found to be more vulnerable to CIN. Further large randomized controlled trials are warranted to investigate the preventive effect of albumin on CIN.

Conflict of interest

The authors declare that there were no conflicts of interest in this study.

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