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Incidence, risk factors, and prognosis of acute kidney injury following transarterial chemoembolization in patien study

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» Abstract

Background: Transcatheter arterial chemoembolization (TACE) is an effective first-line therapy (HCC). Acute renal injury may be induced after transarterial chemoembolization because of iodir factors, and prognosis remain unclear. **Patients and Methods:** This prospective study enrolled treatments. The incidence, risk factors, and prognosis of acute kidney injury (AKI) were examine 21.84% (69/316) according to Barrett and Parfrey criteria, whereas 7.59% (24/316) according t Multivariate logistic regression analysis showed that serum total bilirubin (TB) (>13.5 μmol/L; od 1.044-3.352; *P* = 0.035) and hemoglobin (HGB) level (<120 g/L; OR: 1.823, 95% CI: 1.019-3.2 development of AKI after TACE procedure in accordance to Barrett and Parfrey criteria. Meanwhi 10.790; *P* = 0.033), post-TACE AKI history (OR: 7.108, 95% CI: 1.387-36.434, *P* = 0.019), and 95% CI: 1.792-10.906; *P* = 0.001) were associated with the development of AKI after TACE pro hospitalization cost was significantly higher (*P* = 0.034) in the patients with AKI after TACE proc post-TACE AKI diagnosis was associated with mortality in any definition used (*P* = 0.034 and *P* = respectively). **Conclusion:** The present study showed that the incidence of post-TACE AKI was h on criteria used. HGB (<120 g/L), serum TB (>13.5), and aminotransferase level (>55 U/L), age predictors of post-TACE AKI in HCC patients. The development of post-TACE AKI was associated prolonged renal insufficiency, or mortality according to AKIN criteria.

Keywords: Acute kidney injury, contrast-induced nephropathy, hepatocellular carcinoma, progn

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» Introduction

Hepatocellular carcinoma (HCC) is the sixth most common malignant cancer, affecting over 600, incidence of HCC is mainly contributed by the chronic viral hepatitis, chronic alcoholism, and met during the early stages of HCC. About 70% of patients were considered unresectable upon diagn arterial chemoembolization (TACE) is a highly selective treatment procedure for patients with int began in the early 1980s. [4] Classical TACE delivered a chemotherapeutic agent (using an oily m tumors, combined with the effect of targeted chemotherapy with ischemic necrosis caused by art

Transcatheter arterial chemoembolization procedure could be administered repeatedly and has b patients with hypervascular HCC. [6],[7] Iodinated radiocontrast medium used in angiographic pro (AKI), specifically in high-risk patients. [8]

The definition of confidence interval (CI)-AKI proposed by Barrett and Parfrey is commonly used

Acute kidney injury network (AKIN) criteria using both serum creatinine value and urine output c [10] AKIN criteria have also been widely used for CI-AKI diagnosis in non-ICU patients. [11],[12] To effect of the different criteria in the diagnosis of AKI in HCC patients receiving TACE procedure.

Several risk factors such as preoperative serum albumin and uric acid level, proteinuria level, co treatment session, and the amount and types of radiocontrast agent are reported to be associate [13],[14],[15],[16] However, no study has been reported to investigate the cost-effectiveness of HC outcome in HCC patients developing post-TACE AKI also remains unclear.

This study aims to evaluate post-TACE AKI incidence, characteristics, and prognosis in HCC patie Barrett and Parfrey criteria and AKIN definition for diagnosis.

» Patients and Methods

This prospective observational study was conducted in our intervention therapy center for 30 mo
Studies and the Research and Development committees of the General Hospital of Shenyang Mil
individual consent requirement was waived.

The patient inclusion criteria were as follows: (1) Diagnosed with HCC; (2) receive TACE treatme
form. Exclusion criteria were the following: (1) Patients with incomplete laboratory data; (2) pat
(RRT); and (3) patients with other etiologies for AKI such as new shock, cardiac arrest, or surgic

Demographic data, main admission diagnosis, HOD, cost, and mortality were recorded. Laborato
[Cr]; pre-TACE Cystatin C [CysC], blood urea nitrogen [BUN]; serum albumin, total bilirubin [TB
level [AST]; prothrombin time [PT], activated partial thromboplastin time [APTT], hemoglobin [H
platelet [PLT] count level) were also recorded.

Definitions

Baseline Scr was defined as the closest measurement to TACE within a timeframe of 48 h before
baseline and its maximal value within the 72 h following TACE was calculated. The development
definitions: (1) Increase in serum creatinine at least 25% from the baseline hours after TACE usi
increase of 26.4 μmol/l (0.3 mg/dl) or a relative increase of 50% in SCr level from the baseline v
postoperative hemodialysis according to the AKIN. [10],[17]

Transcatheter arterial chemoembolization treatment

The TACE was performed according to the modified Seldinger method of arterial embolization. Fe
(Terumo, Tokyo, Japan). Hepatic arteriography was performed to localize tumor nodules and eva
radiocontrast agent (Ousu iohexol, 15 g/50 ml, Yangtze River Pharmaceutical Co., Jiangsu or Vis
power injector. The arteries supplying the tumor were catheterized superselectively. Under fluor
epirubicin (Pfizer, USA), 250 mg fluorouracil injection (Jinyao, Tianjin) and 10 ml Lipiodol (Labor
of emulsion delivered to the tumor were calculated according to the size and vascularization of th

In addition, we recorded whether the operators were with experience of >10 years or not.

Hospital stay, total hospitalization cost, and death during hospitalization of the patients were als
associated with post-TACE AKI including age, laboratory data, TACE history, and post-TACE AKI
patients to identify their predictive values.

Statistical analysis

All statistical analyses were performed using the SPSS 11 (SPSS Inc., Chicago, IL, USA) software
between the two groups were determined by the Chi-square test, with the Yates' correction or th
Categorical data were expressed as percentages as appropriate, and continuous variables were e
(range). Differences in continuous variables between the two groups were determined by the an
Welch's *t*-test were used for the analysis for the presence of unequal variances between samples

used multivariate logistic regression analysis, with stepwise backward variable selection, to test
The odds ratios (ORs) from logistic regression were also presented as measures of the strength
tests, a *P* value (two-tailed) of <0.05 was considered statistically significant.

» Result

A total of 166 consecutive patients was included during the study period and received a total of
21.84% (95% CI, 26.39-17.28%) according to Barrett and Parfrey criteria, and 7.59% (95% CI,

In both definitions used, no significant difference existed between patients with or without TACE
WBC, PLT, TACE history, HOD, and whether the TACE was operated by an expert with experienc

According to Barrett and Parfrey criteria, PT-INR was significantly higher, but the level of serum
patients with AKI after TACE procedure compared with those without AKI. Meanwhile, according
the level of serum BUN and AST were significantly higher in the patients with AKI after TACE pro
patients with post-TACE AKI history was higher in the patients with AKI after TACE procedure co

Multivariate logistic regression analysis showed that TB (>13.5 μmol/L; OR: 1.871 95% CI: 1.04
1.823, 95% CI: 1.019-3.264; *P* = 0.043) were associated with the development of AKI after TAC
[Figure 1] and [Table 1]; whereas, age (>55 years; OR: 3.456, 95% CI: 1.107-10.790; *P* = 0.033)
36.434, *P* = 0.019) and AST (>55 U/L; OR: 4.420, 95% CI: 1.792-10.906; *P* = 0.001) were ass
procedure according to AKIN criteria [Figure 2] and [Table 2].

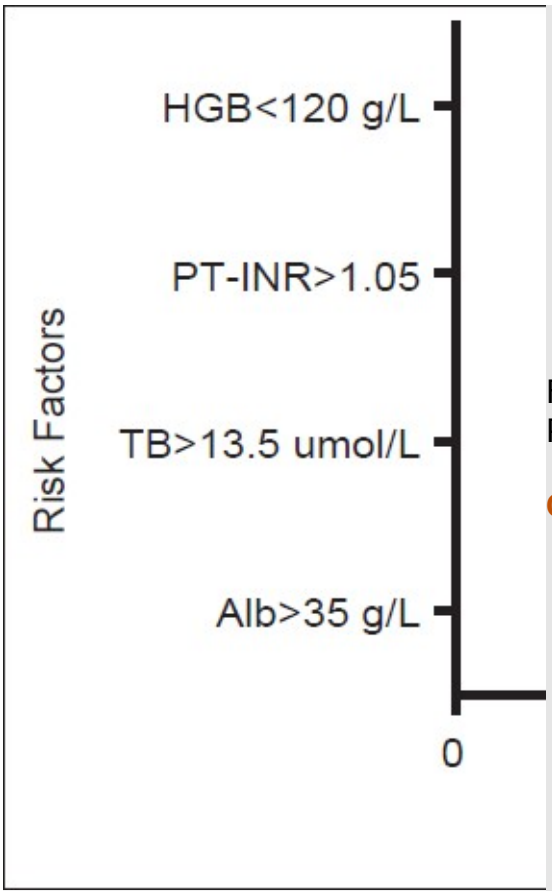

Figure 1: Predictors of post - transcatheter arterial chemoembo
Parfrey criteria in multivariate logistic regression analysis

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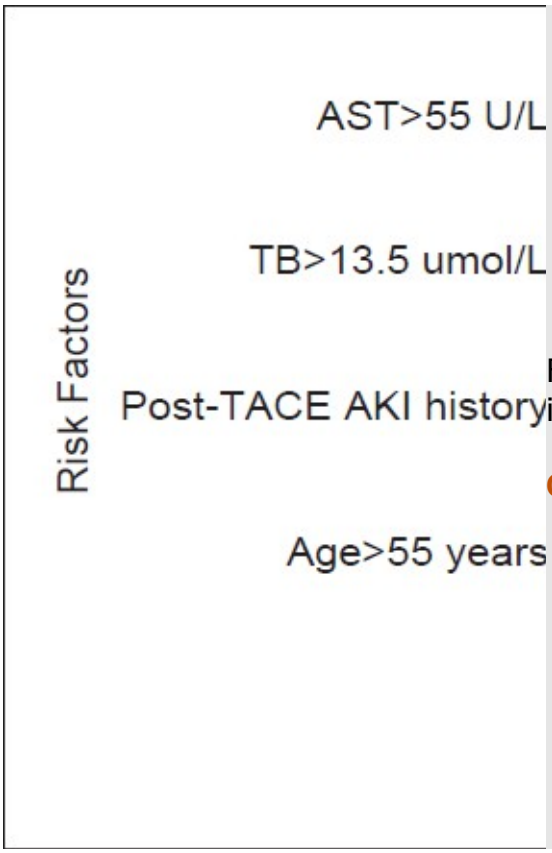

Figure 2: Predictors of post - transcatheter arterial chemoembo
injury network criteria in multivariate logistic regression analys

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	Over all (*n*=316)
Male, *n* (%)	241.00 (76.27)
Female, *n* (%)	75.00 (23.73)
Age (year)	57.00 (51.00-64.00)
CysC (mg/L)	0.78 (0.65-1.00)
BUN (mmol/L)	4.76 (4.00-6.00)
Alb (g/L)	37.58±4.36
TB (μmol/L)	14.10 (10.60-18.80)
DB (μmol/L)	5.50 (3.93-7.50)
AST (U/L)	38.00 (27.00-59.75)
PT (s)	13.50 (12.80-14.38)
APTT (s)	38.20 (34.93-41.58)
PT-INR	1.04 (0.98-1.11)
HGB (g/L)	128.00 (114.25-139.00)
WBC (×10⁹/L)	5.00 (3.90-6.28)
PLT (×10⁹/L)	126.00 (88.25-170.00)
TACE-AKI history B and P/AKIN *n* (%)	44.00 (13.92)/8 (2.5)
TACE history *n* (%)	150.00 (47.47)
Expert *n* (%)	62.00 (19.62)
Hospital stay (day)	12.00 (10.00-15.00)
Cost (¥)	23570.19 (20462.12-26956.44)
Death, *n* (%)	4.00 (1.27)

CysC=Cystatin C; BUN=Blood urea nitrogen; A
partial thromboplastin time; PT-INR=Prothromb
TACE=Transcatheter arterial chemoembolizatior

Table 1: Comparison of clinical characteristics, laboratory data,

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AKI for barrett and parfrey criteria
Alb >35 g/L
TB >13.5 μmol/L
PT-INR >1.05
HGB >120 g/L
AKI for AKIN criteria
Age >55 years
Post-TACE AKI history
TB >13.5 μmol/L
AST >55 U/L

AKI=Acute kidney injury; TACE=Transc
AKIN=Acute kidney injury network; OR=
Alb=Albumin; TB=Total bilirubin; PT-INR=Pr
ratio; HGB=Hemoglobin; AST=Aspartate an

Table 2: Risk factors for the development of AKI after TACE pr
AKIN criteria

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In any definition used, no significant difference in hospital stay existed between patients with or
significantly higher (P = 0.034) in the patients with AKI after TACE procedure according to Barre
was associated with mortality in any definition used (P = 0.034 and P = 0.001 for Barrett and Pa
(12.50%) patients developed chronic renal insufficiency (P = 0.000), whereas 2 (8.33%) receive
according to the physician's decision (P = 0.006).

» Discussion

In the present prospective cohort study, incidence of post-TACE, AKI was high in HCC patients, r
the criteria used. Post-TACE AKI ranged from 6.64% to 9.05% according to the AKIN criteria wh
in the previous study. TACE treatment on HCC patients may increase the risk of developing AKI
used. For the general population receiving iodine contrast, induced AKI ranged from 3% to 14%,
cause of hospital-acquired AKI. [19]

Three major pathways are recognized in the pathophysiology of contrast-induced AKI: Hemodyn
tubular cell toxicity, and increase in oxygen free radicals. These three interacting pathways lead
CM are eliminated through glomerular filtration in 24 h after intravascular administration in patie
administration of contrast shows a biphasic impact on renal hemodynamics: A transient increase
25% below baseline. [20],[22] PO_2 of outer medullary declines by 50-67% (9-15 mmHg) after CM
microcirculatory blood flow and the increased oxygen demand of renal tubular cells caused by CM

Ischemia/reperfusion injury leads to increased formation of cytotoxic substance, including oxyge
When the concentration of these molecules exceeds the cellular scavenging capacities, cellular ir
imbalance between vasoconstrictive and vasodilative mediators, including an increase in angiote
[25]

The alteration of these vasoactive mediators induces renal vasoconstriction and aggravates hypo
leads to intracellular imbalance between oxidants and antioxidants. This imbalance increased cel
membrane lipids, and cellular proteins, eventually increasing oxygen free radicals and ROS. [25]

Apoptosis, redistribution of membrane proteins, and DNA fragmentation of renal cell cultures ind
study. [26] Apoptosis is associated with increased levels of oxygen free radicals *in vitro* studies. T
by CM has also been demonstrated *in vivo* studies. [27] Iodine is toxic to human cells, [28] but th
responsible for tubular cell toxicity remains unknown. [29]

The key points of renal vascular affected by CM seem to be the afferent arterioles and descendin
vasoconstriction of the afferent arterioles and reduction in glomerular filtration rate. [29] Descend
endothelial damage and dysfunction. [30] Advanced cirrhosis is a frequent concomitant of HCC du
vasoconstrictor system, induce peripheral vasodilatation, and decrease renal perfusion. [31],[32] C
renal function.

Serum albumin constitutes 60% of the total plasma protein and contributes about 70% of the to
oncotic pressure in the vascular system and has antioxidant properties in the body. [34] Serum a
contrast-induce AKI by vascular expansion and antioxidant properties. [35] The level of serum alb
after TACE procedure, compared with those without AKI according to Barrett and Parfrey criteria
the low albumin is responsible for the development of post-TACE AKI. Further studies are manda
the association between post-TACE AKI and low albumin precisely.

All types of CM have negative effects on cell cultures *in vitro* studies. High-osmolar CM is more t
Regarding the effect on descending vasa recta constriction, no significant difference exists amon
low-osmolar iopromide, and the iso-osmolar iodixanol CM amidotrizoate. [36] The use of iodixano
no positive association with renal outcomes, compared with low-osmolar CM; high-osmolar CM ir
cytotoxicity of hyperosmolar solutions. [37] Compared with low-osmolar CM, iso-osmolar nonionic
viscosity, which leads to a relatively long contact time with renal tubular cells, [29] decrease of o
injury markers, and formation of vacuoles in the renal tubular epithelium of the proximal and dis
the risk of renal cell apoptosis between iso-osmolar and low-osmolar contrast agents, [40] and ra
between the iodixanol and different low-osmolar contrast agents. [41],[42],[43]

Low-osmolar iohexol is reported to induce a higher risk of AKI in CKD patients. [44] CM isoosmola
contrast-induced AKI in DM or CKD patients compared with low-osmolar iohexol. [8],[44] Neverthe
consistent; no significant advantage of iohexol over iodixanol was observed in the prevention of
of coronary angiography may prefer to use iodixanol for radiography in high-risk subjects, hence
selection bias, the comparison of the risk for post-TACE AKI between iohexol and iodixanol was r

Apart from contrast osmolarity, other mechanisms such as the clinical comorbidities and hemody

[45] Older age, cardiac or liver failure, diabetic nephropathy, and preexisting chronic kidney disea
induced nephropathy in the general population. [18] Several risk factors for post-TACE AKI in HC
Preoperative serum albumin and uric acid level, proteinuria level, coexisting hypertension and di
amount and types of radiocontrast agent are important predisposing factors. [13],[14],[15],[16] In t
level (<120 g/L) were independent risk factors of post-TACE AKI according to Barrett and Parfre
history, and serum aminotransferase level (>55 U/L) were independent risk factors of post-TACE
logistic regression analysis.

Inpatient hospital mortality, renal replacement treatment, and hospital length of stay are increas
[46],[47],[48]

Statistically significant associations were found between post-TACE AKI and inpatient hospital m
AKIN criteria, 3 (12.50%) patients developed chronic renal insufficiency (P = 0.000), whereas 2
hemofiltration treatment according to the physician's decision (P = 0.006).

No statistically significant difference in the development of post-TACE AKI existed between TACE
of >10 years and those operated by doctor with intervention experience of <10 years, in both de
operation of the TACE procedures on patients with critical or complex concomitant diseases by e

The increase of creatinine values are associated with adverse renal outcomes, hence the AKIN d
creatinine level. [10]

Increase of serum creatinine level in iodine CIN patient occurs in 48 h after angiography, reache
decreases during days 7-21. [18]

The baseline creatinine value is not necessary for Barrett and Parfrey and the AKIN definition. AK
mortality in critically ill patients. [49]

Using the AKIN criteria, the ratio of RRT and mortality was more frequent in patients with post-T
renal insufficiency and renal replacement treatment according to the AKIN criteria used. Hence,
the post-TACE AKI in our study. Further studies are mandatory to determine the role of AKIN cri
AKI in HCC patients precisely.

Most of the post-TACE AKI were transient and reversible, usually not needing of any special trea
history (OR: 7.108, 95% CI: 1.387-36.434, P = 0.019) were associated with the development o
To the best of our knowledge, the impact of post-TACE AKI history on the development of post-T
previous studies. We suppose that aside from the three main pathways and CM acting on renal,
post-TACE AKI. Nevertheless, the factors and mechanism remain unclear. Clinical and experimer
investigation.

The present study has few limitations. First, it focused on the data from a single center and was
other etiologies of AKI may not be ruled out completely, despite the exclusion criteria that were
follow-up data to assess the long-term prognosis.

» Conclusion

The present study showed that the incidence of post-TACE AKI was high in HCC patients, ranging criteria used. The serum aminotransferase level (>55 U/L), age (>55 years), and post-TACE AKI patients. The development of post-TACE AKI was associated with the risk of renal replacement t according to AKIN criteria. Further studies are mandatory to investigate the prevention of post-T

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