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# Adverse events following transarterial chemoembolization for hepatocellular carcinoma and factors predicting such events

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## Abstract

**Objective:** To document the adverse events after transarterial chemoembolisation and factors predicting such events.

**Methods:** The prospective observational study was conducted at the Sindh Institute of Urology and Transplantation, Karachi, from November 2009 to November 2011. All patients diagnosed as hepatocellular carcinoma were included in this study. Complications developing within the first 6 weeks of the procedure were recorded. SPSS version 16 was used for statistical analysis.

**Results:** Of the total 80 patients, 59 (73.8%) were male. The overall mean age was 52.25±9.24 (range: 28-76 years). Most common etiology was hepatitis C related cirrhosis in 55 (68.8%). Adverse events developed in 46 (57.5%) patients. Post transarterial chemoembolisation syndrome was seen in 37 (46.3%). Of those with the syndrome, 24 (64.8%) patients had no additional complications, while 3 (8%) had renal dysfunction, 2 (5%) hypertensive crisis, and 1 (2.7%) patient each had urinary tract infection, pneumonia and sepsis. Decompensation of cirrhosis occurred in 6 (7.5%) patients of whom 3 (50%) developed sepsis and died. The syndrome was associated with tumour size >5cm (p=0.001) and higher dose of lipoidol (p=0.0001). Decompensation of cirrhosis was associated with low basal albumin (p=0.002), advanced basal child turcotte pugh (p=0.005) and model for end-stage liver disease (p=0.006) scores.

**Conclusion:** Transarterial chemoembolisation, though generally safe, may lead to serious complications in patients with advanced liver disease. Post-procedure syndrome was associated with increased tumour size and lipoidol dose.

**Keywords:** Post TACE Syndrome, Hepatocellular carcinoma, Adverse events. (JPMA 63: 239; 2013)

## Introduction

Hepatocellular carcinoma (HCC) is a common cause of cancer deaths throughout the world.[1] Various modalities, including transarterial chemoembolisation (TACE), are available to treat HCC.[2] TACE is usually employed for unresectable HCC and is largely considered to be palliative, but may be curative depending upon the stage of HCC. It has been shown to improve survival in unresectable HCC.[3]

A common adverse event is the post-TACE syndrome (PTS) which is characterised by fever, abdominal pain, nausea and vomiting, leukocytosis and elevated liver enzymes lasting for a few hours to a few days.[4] Other important complications of TACE is decompensation of cirrhosis.[1] Hepatic failure and renal failure which, although uncommon, are among the major treatment-related complications that may result in significant morbidity.[5,6] Complications associated with the use of chemotherapeutic and embolising agents include acute cholecystitis, biliary tract necrosis, pancreatitis, gastric erosions, or ulcers, if they are inadvertently injected into these organs.[7-10] Other important adverse events, including infection of the necrotic tumour presenting as liver abscess and tumour lysis syndrome, can also occur.[11,12]

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The development of complications may depend upon various risk factors.[13] The risk factors may be related to the liver disease, the patient and to the procedure itself. Factors related to the liver disease include the stage of cirrhosis as indicated by the Child Turcotte Pugh (CTP) and Model for End-stage Liver Disease (MELD) score, the morphology of HCC, including the size, location, presence or absence of thrombosis of portal vein, the baseline liver function as indicated by the prothrombin time (PT) and the albumin levels.[1,5,12] The patient-related factors may include the presence of co-morbidities and the level of immunocompetence. The dose of chemotherapeutic agent and skills of the performer of TACE procedure may also influence the outcome.[1]

The aim of this study was to identify the different adverse events following TACE and the risk factors associated with these adverse events and, hence, to enable the physician to decide which group of patients are optimal candidates for this procedure. In the West, a number of studies have been done to study the various complications occurring after TACE. In South Asia, including Pakistan, only few centres perform TACE and little work has been done in this regard. It is, therefore, essential to identify the adverse events and risk factors associated with such events in our part of the world.

## Patients and Methods

The prospective observational study was conducted at the Sindh Institute of Urology and Transplantation (SIUT),

Karachi, and comprised all patients diagnosed as HCC and found eligible for TACE from November 2009 to November 2011. Patients eligible for TACE were studied for the development of adverse events within the first 6 weeks of the procedure. All adverse events, both related or unrelated to TACE were recorded. Patients with a CTP score greater than 10 and those presenting within 6 months of any previous intervention like radiofrequency ablation (RFA) or surgical liver resection for HCC were excluded from the study.

The sampling technique employed was non-probability convenient sampling. Approval was obtained from the SIUT Ethical Review Committee. About four to five patients per month with HCC and eligible for TACE were admitted which amounted to about 100 patients over two years. Therefore, taking a population size of 100, response distribution of 50%, confidence level of 95% and a margin of error of 5%, the Raosoft sample size calculator was employed to calculate the sample size, which turned out to be 80.

TACE involved injection of a chemotherapeutic agent (doxorubicin) mixed with lipoidol into selectively or superselectively catheterised branches of the arteries feeding the tumour followed by injection of gelfoam particles to reinforce the effect of the treatment. The procedure was performed at the Radiology Department. Informed consent was taken and a structured proforma was used to collect relevant data, including the etiology and morphology of HCC (number of tumour lesions, the size(s) of the lesion(s), the lobe(s) and segment(s) of the liver affected). CTP and MELD scores of individual patients were recorded. Clinical and laboratory parametres before and after TACE were recorded on Days 0, 1, 2, 5, 7, 14, 28, 36 and 42. The clinical and laboratory parametres were recorded on an out-patient basis. Among the clinical parametres were subjective parametres (fever, abdominal pain and vomiting) and objective parametres (temperature, abdominal tenderness, level of consciousness, flapping tremors etc); while the laboratory parametres included the total leukocyte count (TLC), serum bilirubin, serum transaminases, serum PT and serum creatinine. The maximum hike in above parametres occurring within the first six weeks of the procedure was recorded. Upon discharge, patients were asked to present if any adverse event(s) developed within 6 weeks. At the end of the 6 weeks; a computed tomography (CT) scan abdomen was done as per the TACE protocol.

PTS was defined as development of fever, abdominal pain, rise in white cell count and transaminases occurring within few days of TACE procedure. Decompensation of cirrhosis was defined as the development of any of the following after the TACE procedure: ascites, variceal bleeding or portosystemic encephalopathy.

The data was statistically analysed using SPSS version 16.0. Frequency and percentages were computed for different categorical variables like gender and cause of HCC. Mean and standard deviation were computed for age. Two-sided Fisher's exact test was employed to analyse dichotomous variables before and after TACE respectively; p value of less than 0.05 was considered significant.

## Results

Of the total 80 patients studied 59 (73.8%) were males and 21 (26.2%) were females. The overall mean age was 52.25±9.24 years (range: 28-76). The mean age of the males was 52.92±9.216 (range: 38-76) and that of the females was 50.38±9.26 (range: 28-65). Besides, 74 (92.5%) patients were middle-aged to elderly, while the remaining were aged below 40. HCC was secondary to hepatitis C-related cirrhosis in 55 (68.8%) patients; secondary to co-infection of hepatitis B and C-related cirrhosis in 10 (12.5%); hepatitis B-related cirrhosis in 6 (7.5%); unknown causes in 6 (7.5%), hepatitis B and D co-infection in 2 (2.5%); and fibrolamellar carcinoma in 1 (1.3%) cases.

No complications were observed in 34 (42.5%) patients, while 3 (3.75%) died within six weeks of the procedure. PTS occured in 37 (46.3%) patients (Figure-1), among whom 24 (64.8%) patients developed PTS alone and 13 (35.1%) had other complications too. Decompensation of cirrhosis occured in 6 (7.5%): 1 (16.6%) patient developed urosepsis followed by portosystemic encephalopathy and death; 1 (16.6%) became septic and expired 10 days after the procedure; 1 (16.6%) who had undergone renal transplant 20 years ago, developed pneumonia followed by portosystemic encephalopathy and expired 2 weeks after TACE, 1 (16.6%) developed worsening of ascites and variceal bleeding, and 2 (33.3%) patients had worsening of ascites alone. Among infectious complications, 3 (3.75%) patients had sepsis: 1 (33.3%) had pneumonia, 1 (33.3%) developed multi-drug resistant urinary tract infection which was manifested by persistent and high-grade, intermittent fever which continued for four weeks, while 1 (33.3%) had septic phlebitis. Three (3.75%) patients had a transient renal dysfunction manifested by decreased urinary output and rising serum creatinine level. Renal function of all these patients returned to normal within 2 weeks of the procedure. Three (3.75%) patients had hypertensive crisis, requiring intravenous administration of nitrates to control the blood pressure immediately after the procedure. Two (2.75%) patients had isolated abdominal pain and isolated fever each without other features of PTS. One (1.25%) patient developed haematoma at the site of femoral artery catheterisation. One (1.25%) patient had melena secondary to gastric stress ulcer.

PTS was the commonest complication observed. Factors such as age, gender, co-morbids, size of HCC, dose of

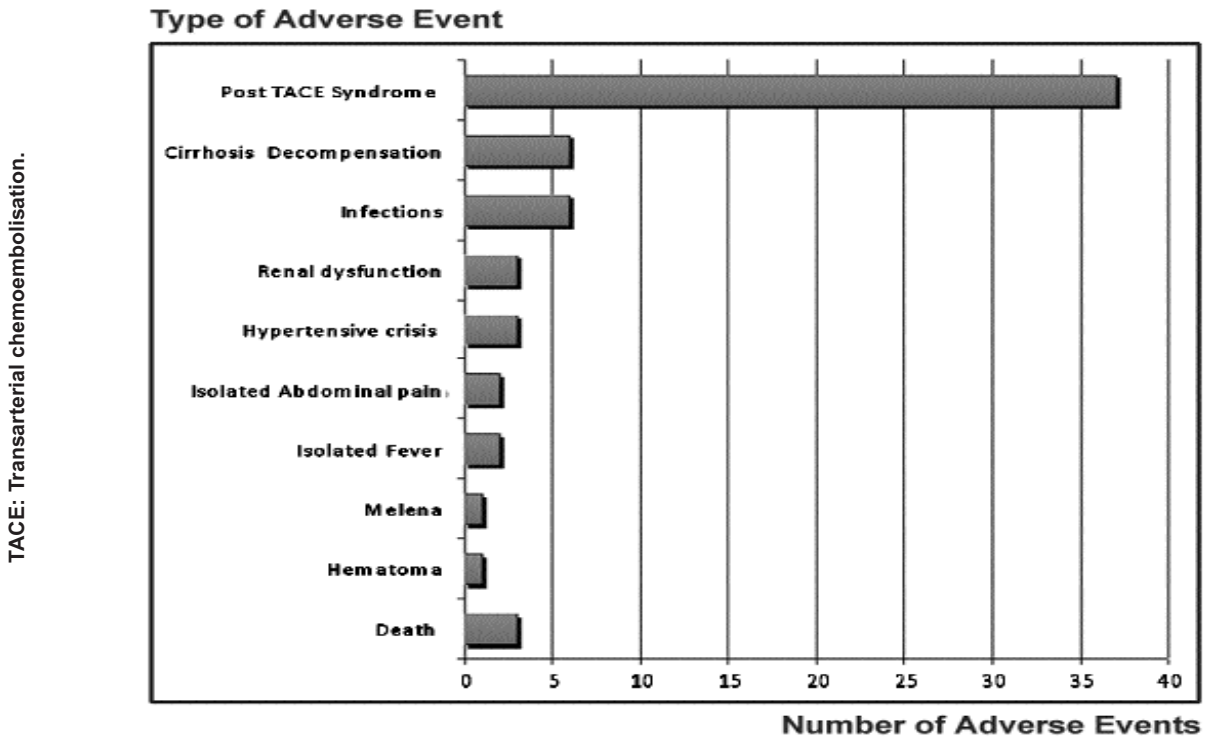
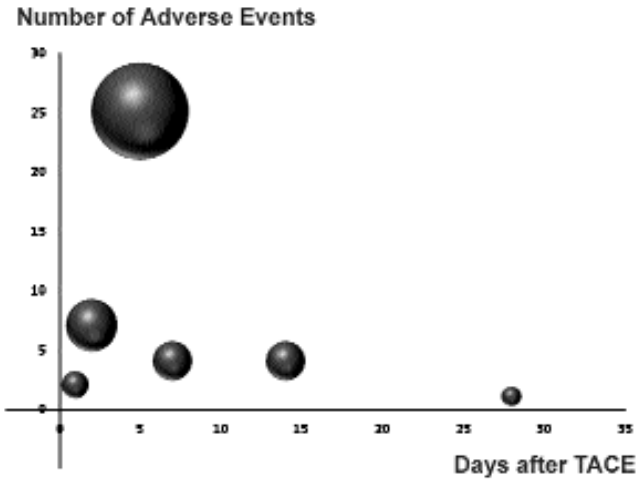



**Figure-1:** Post TACE Adverse Events.

chemotherapeutic agent, serum transaminases, albumin level, international normalised ratio (INR), CTP and MELD scores of patients with and without PTS were compared. The basal values of these parametres as well as the values at follow-up were compared between patients who developed




**X axis = Days after TACE procedure. Y axis = Number of Adverse Events. The bubble size indicates the number of complications occurring on the particular day after TACE. TACE: Transarterial chemoembolisation.**

**Figure-2:** Frequency of Adverse Events during follow-up period.

PTS and those who did not. Similar comparison was done for patients who developed decompensation of cirrhosis with those who did not. Analysis of the factors predicting PTS (Table-1) revealed a significant association with tumours having dimension more than 5cm (p= 0.001) and increased dose of the chemotherapeutic/lipoidol mixture (p= 0.0001). No statistically significant association was observed with age, gender, presence of co-morbids, basal total bilirubin, platelet levels, transaminases, albumin, sodium, creatinine, alfa fetoprotein, Okuda stage, CTP score or MELD score.

An analysis of the follow-up parametres (Table-2) showed significant association of PTS with abdominal pain (p= 0.000), rise in temperature (p= 0.000), white cell count (p= 0.000), total bilirubin (p= 0.000), serum glutamate pyruvate transminase (SGPT) (p= 0.000) and serum glutamic oxatoacetic transminase (SGOT) (p= 0.000) which are the part of PTS itself. However, rise in serum creatinine was also found to be associated with PTS (p= 0.045), suggesting a trend of renal dysfunction in patients with PTS. Rise in the CTP score (p= 0.038) and MELD score (p= 0.002) at day five post-procedure suggested a tendency towards worsening of the liver disease in patients who developed PTS.

Decompensation of cirrhosis was the second most common adverse event observed. An analysis of factors predicting cirrhosis decompensation was performed (Table-3).

Table-1: Predictive factors of Post-TACE Syndrome (PTS).

| Clinical Characteristic | PTS | No PTS | p value |
|---|---|---|---|
| **Age** | | | |
| Young | 4 | 2 | 0.407 |
| Middle aged to elderly | 33 | 41 | |
| **Gender** | | | |
| Male | 29 | 30 | 0.450 |
| Female | 8 | 13 | |
| **Co-morbids** | | | |
| Present | 17 | 21 | 0.826 |
| Absent | 20 | 22 | |
| **Size of tumour** | | | |
| <5cm | 8 | 29 | 0.001* |
| >5cm | 25 | 18 | |
| **Dose of Lipoidol** | | | |
| <10 ml | 21 | 40 | 0.0001* |
| >10 ml | 16 | 3 | |
| **Basal platelet count** | | | |
| < 100,000/mm3 | 11 | 22 | 0.069 |
| > 100,000/mm3 | 26 | 21 | |
| **Basal PT INR** | | | |
| < 1.4 | 27 | 26 | 0.343 |
| > 1.4 | 10 | 17 | |
| **Basal SGPT (ALT)** | | | |
| < 50 U/L | 23 | 22 | 0.371 |
| > 50 U/L | 14 | 21 | |
| **Basal SGOT (AST)** | | | |
| < 50 U/L | 13 | 15 | 1.00 |
| > 50 U/L | 24 | 28 | |
| **Basal Albumin** | | | |
| < 2.8gm/dl | 16 | 15 | 0.495 |
| > 2.8gm/dl | 21 | 28 | |
| **Basal Sodium** | | | |
| ≤ 133 meq/L | 7 | 12 | 0.433 |
| > 133 meq/L | 30 | 31 | |
| **Basal Creatinine** | | | |
| < 1.2mg/dl | 30 | 40 | 0.174 |
| > 1.2mg/dl | 7 | 3 | |
| **AFP** | | | |
| < 200 ng/ml | 24 | 29 | 0.817 |
| > 200 ng/ml | 13 | 14 | |
| **Basal CTP score** | | | |
| CTP A | 22 | 23 | 0.655 |
| CTP B/C | 15 | 20 | |
| **Basal MELD score** | | | |
| < 10 | 22 | 22 | 0.505 |
| > 10 | 15 | 21 | |

INR= International Normalised Ratio, AFP= Alfa fetoproteins. CTP= Child Turcotte Pugh. MELD= Model for end-stage liver disease. *Statistically significant values. SGPT: Serum glutamate pyruvate transminase. SGOT: Serum glutamic oxaloacetic transminase. TACE: Transrterial chemoembolisation.

Table-2: Follow up parameters in patients with Post-TACE Syndrome (PTS).

| Clinical Characteristic | PTS | No PTS | p value |
|---|---|---|---|
| **Rise in Temperature** | | | |
| > 1010 F | 33 | 13 | 0.000* |
| < 1010 F | 4 | 30 | |
| **Abdominal pain** | | | |
| Yes | 31 | 10 | 0.000* |
| No | 6 | 33 | |
| **Rise in WBC** | | | |
| < 3000/mm3 | 8 | 34 | 0.000* |
| > 3000/mm3 | 29 | 9 | |
| **Rise in Total bilirubin** | | | |
| < 1mg/dl | 9 | 30 | 0.000* |
| > 1mg/dl | 28 | 13 | |
| **Rise in SGPT (ALT)** | | | |
| <200 U/L | 14 | 39 | 0.000* |
| >200 U/L | 23 | 4 | |
| **Rise in SGOT (AST)** | | | |
| <200 U/L | 10 | 37 | 0.000* |
| >200 U/L | 27 | 6 | |
| **Rise in PT INR** | | | |
| > 3sec | 15 | 10 | 0.146 |
| < 3sec | 22 | 33 | |
| **Rise in Creatinine** | | | |
| > 1mg/dl | 6 | 1 | 0.045* |
| < 1mg/dl | 31 | 42 | |
| **Rise in CTP score** | | | |
| ≥ 2 | 8 | 2 | 0.038* |
| < 2 | 29 | 41 | |
| **Rise in MELD score** | | | |
| ≥ 2 | 26 | 15 | 0.002* |
| < 2 | 11 | 28 | |
| **Decompensation** | | | |
| Yes | 5 | 1 | 0.090 |
| No | 32 | 42 | |
| **Death** | | | |
| Yes | 3 | 0 | 0.095 |
| No | 34 | 43 | |

INR= International Normalised Ratio. CTP= Child Turcotte Pugh score; MELD= Model for end-stage liver disease. *Statistically significant values. SGPT: Serum glutamate pyruvate transminase. SGOT: Serum glutamic oxaloacetic transminase. PT: Prothombin time. TACE: Transrterial chemoembolisation.

Decompensation was found to be associated with basal serum albumin levels less than 2.8gm/dl (p= 0.002), basal CTP score of ≥ 7 (p= 0.005), and basal MELD score >10 (p= 0.006). A rise in CTP score ≥ 2 (p= 0.024), and rise in MELD score ≥ 2 (p= 0.026) was observed on day 5 in patients who decompensated. However, no statistically significant association of cirrhosis decompensation was observed with size of the tumour, dose of chemoembolisation agents, basal PT INR, transaminases or alfa fetoprotein levels. Most of the complications occurred in the first two weeks (Figure-2) following TACE. Hypertensive crisis was observed during the first two days, PTS duing the first week, and decompensation of cirrhosis during the second week after TACE.

## Discussion

This study analysed the various adverse events following

Table-3: Predictive factors of cirrhosis decompensation following TACE.

| Clinical Characteristic | Cirrhosis decompensation | No cirrhosis decompensation | p value |
|---|---|---|---|
| **Age** | | | |
| Young | 0 | 6 | 1.00 |
| Middle aged to elderly | 6 | 68 | |
| **Sex** | | | |
| Male | 3 | 56 | 0.182 |
| Female | 3 | 18 | |
| **Comorbids** | | | |
| Present | 2 | 36 | 0.678 |
| Absent | 4 | 38 | |
| **Size of tumour** | | | |
| < 5cm | 3 | 30 | 0.687 |
| > 5cm | 3 | 44 | |
| **Dose of Lipoidol** | | | |
| < 10ml | 6 | 55 | 0.327 |
| > 10ml | 0 | 19 | |
| **Basal platelet count** | | | |
| < 100,000/mm3 | 4 | 29 | 0.224 |
| > 100,000/mm3 | 2 | 45 | |
| **Basal PT INR** | | | |
| < 1.4 | 2 | 51 | 0.172 |
| > 1.4 | 4 | 23 | |
| **Basal SGPT (ALT)** | | | |
| < 50 U/L | 5 | 40 | 0.223 |
| >50 U/L | 1 | 34 | |
| **Basal SGOT (AST)** | | | |
| < 50 U/L | 1 | 27 | 0.659 |
| > 50 U/L | 5 | 47 | |
| **Basal Albumin** | | | |
| < 2.8gm/dl | 6 | 25 | 0.002* |
| > 2.8gm/dl | 0 | 49 | |
| **AFP** | | | |
| < 200 ng/ml | 5 | 48 | 0.658 |
| < 200 ng/ml | 1 | 26 | |
| **Basal CTP score** | | | |
| CTP A | 0 | 45 | 0.005* |
| CTP B/C | 6 | 29 | |
| **Rise in CTP score** | | | |
| ≥ 2 | 3 | 7 | 0.024* |
| < 2 | 3 | 67 | |
| **Basal MELD score** | | | |
| < 10 | 0 | 44 | 0.006* |
| > 10 | 6 | 30 | |
| **Rise in MELD score** | | | |
| ≥ 2 | 6 | 35 | 0.026* |
| < 2 | 0 | 39 | |

INR= International Normalised Ratio, AFP= Alfa fetoprotein level. CTP= Child Turcotte Pugh score; MELD= Model for end stage liver disease. *Statistically significant values. SGPT: Serum glutamate pyruvate transminase. SGOT: Serum glutamic oxaloacetic transminase. TACE: Transrterial chemoembolisation. PT: Prothombin time.

TACE and the factors predicting such events. The majority of earlier series published to date reported relatively few side-effects, with TACE being well tolerated.[14-16] A study from Europe reported that approximately 60% of patients had at least one episode of acute liver failure within few days of TACE.[17] In contrast, in a series of 132 patients at the University of Hong Kong, the occurrence of liver failure was reported to be only 1.5%.[18]

The most common complication is usually PTS which consists of various symptoms, including fever, abdominal pain, nausea and vomiting, leukocytosis, and elevated liver enzymes lasting for a few hours to a few days.[4] This syndrome is usually self-limited and is treated symptomatically. In most patients it decreases in severity with subsequent procedures. In our study too, PTS was the commonest complication.

A number of studies have been performed internationally with variable results. In a prospective study regarding the complications of TACE in patients with HCC,[1] elevation of bilirubin after TACE was found to be associated with the dosage of chemotherapeutic agent and stage of cirrhosis. In our study, the development of PTS was also found to be associated with high doses of chemotherapeutic agent, but not associated with the stage of cirrhosis as reflected by the basal CTP score. However, in our study, cirrhosis decompensation was found to be closely associated with the stage of liver disease as reflected by the basal CTP score and basal albumin levels. In another study, severe complications after TACE were found to correlate with poor hepatic function and portal hypertension before therapy, and to overdose and reflux of chemotherapeutic agents.[19] It was concluded that these complications could be prevented through careful selection of cases before treatment with close observation, and protection of the hepatic function. In our study, the most severe complication of TACE was septicaemia which resulted in deaths of three patients, two of whom were those whose liver disease was fairly advanced and belonged to early Child class C, whereas one was on immunosuppression, after renal transplant that was performed 20 years back, who developed sepsis after TACE. Cirrhosis decompensation was found to be related to advanced liver disease, which is in conformity with an earlier study.[19]

In a Taiwanese study, the incidence of fever after TACE for HCC was analysed and risk factors for the development of fever were studied.[20] It was found that fever after TACE was common and chemoembolisation dosage and tumour size were predictive of fever. In our study, the frequency of PTS (which also involves rise in temperature) was analysed instead of fever alone. PTS was also related to the dose of chemotherapeutic agent and the size of tumour. Another study in Korea, studied the incidence and risk factors of acute hepatic failure after transcatheter arterial chemoembolisation for HCC.[5] The incidence of acute hepatic failure after TACE was 12.0%; and elevated bilirubin level and portal vein thrombosis (PVT) were considered as the predictive factors for acute hepatic failure

after TACE in HCC patients. However, in our study, we did not study acute hepatic failure separately, as insult by TACE is acute on chronic injury which ends up in PTS or decompensation of cirrhosis. Decompensation as a result of TACE was seen in 7.5% cases and no association was found with PVT (p= 1.00). Also, we chose to chemoembolise only those selected patients who had no or partial PVT and not those with main PVT. In a study in Wisconsin, USA, chemoembolisation in patients at high risk was studied for results and complications.[21] It was concluded that patients with advanced disease and decreased hepatic reserve who were treated with TACE exhibited no significant increase in morbidity or mortality and no significant decrease in survival, and that TACE could be performed safely in patients with the relative risk factors that may classify them in high-risk groups. In our study, though no complications were observed in 42.5% patients, three deaths that occurred were all cases of advanced liver disease. Also, a statistically significant association was observed between the advanced CTP and MELD scores and development of cirrhosis decompensation. Hence, cases that are supposed to undergo TACE should be carefully selected. As most of the complications occurred within the first two weeks after TACE, we concluded that patients should be kept under close observation for this period and that the time period for PTS should be reduced to two weeks. Moreover, as there was significant increase in the MELD score in patients who developed PTS, we suggest that increase in MELD score may be included in the definition of PTS.

## Conclusion

TACE is a useful modality for both palliative and curative management of HCC. It is generally safe with few, if any, complications in patients with early cirrhosis. However, serious complications like sepsis and death may ensue if this procedure is performed in patients with advanced liver disease with minimal hepatic reserve. The study showed that most of the adverse events occur within the first two weeks of TACE and that the development of PTS is associated with the size of the tumour and dose of chemotherapeutic mixture. Development of decompensation of cirrhosis is related to low basal serum albumin levels, and advanced stage of liver disease as reflected by advanced CTP and MELD scores.

## References


1. Chan AO, Yuen MF, Hui CK, Tso WK, Lai CL. A prospective study regarding the complications of transcatheter intraarterial lipiodol chemoembolization in patients with hepatocellular carcinoma. Cancer 2002; 94: 1747-52.
2. Caturelli E, Siena DA, Fusilli S. Transcatheter arterial chemoembolization for hepatocellular carcinoma in patients with cirrhosis: evaluation of damage to nontumourous liver tissue - long-term prospective study. Radiology 2000; 215: 123-8.
3. Josep M, Llovet J B. Systematic review of randomized trials for unresectable hepatocellular carcinoma: Chemoembolization improves survival. Hepatology 2003; 37: 429-42.
4. Leung DA, Goin JE, Sickles C, Raskay BJ, Soulen MC. Determinants of postembolization syndrome after hepatic chemoembolization. Vasc Interv Radiol 2001; 12: 321-6.
5. Jeon SH, Park KS, Kim YH, Shin YS, Kang MK, Jang BK, et al. Incidence and risk factors of acute hepatic failure after transcatheter arterial chemoembolization for hepatocellular carcinoma. Korean J Gastroenterol 2007; 50: 176-82.
6. Huo TI, Wu JC, Lee PC, Chang FY, Lee SD. Incidence and risk factors for acute renal failure in patients with hepatocellular carcinoma undergoing transarterial chemoembolization: a prospective study. Liver Int 2004, 24: 210-5.
7. Wagnetz U, Jaskolka J, Yang P, Jhaveri KS. Acute ischemic cholecystitis after transarterial chemoembolization of hepatocellular carcinoma: incidence and clinical outcome. J Comput Assist Tomogr 2010; 34: 348-53.
8. Kim HK, Chung YH, Song BC, Yang SH, Yoon HK, Yu E, et al. Ischemic bile duct injury as a serious complication after transarterial chemoembolization in patients with hepatocellular carcinoma. J Clin Gastroenterol 2001, 32: 423-7.
9. Beyza Özç?nar, Koray Güven, Arzu Poyanl?, Ilg?n Özden. Necrotizing pancreatitis after transcatheter arterial chemoembolization for hepatocellular carcinoma. Diagn Interv Radiol 2009, 15: 36-8.
10. Hirakawa M, Iida M, Aoyagi K, Matsui T, Akagi K, Fujishima M. Gastroduodenal lesions after transcatheter arterial chemo-embolization in patients with hepatocellular carcinoma. Am J Gastroenterol 1988; 83: 837-40.
11. VanderWalde A, Marx H, Leong L. Liver Abscess as a Complication of Hepatic Transarterial Chemoembolization: A Case Report, Literature Review, and Clinical Recommendations. Gastrointest Cancer Res 2009; 3: 247-51.
12. Hsieh PM, Hung KC, Chen YS. Tumour lysis syndrome after transarterial chemoembolization of hepatocellular carcinoma: case reports and literature review. World J Gastroenterol 2009; 15: 4726-8.
13. Chung J W, Park J H, Han J K. Hepatic tumours: predisposing factors for complications of transcatheter oily chemoembolization. Radiology 1996; 198: 33-40.
14. Sasaki Y, Imaoka S, Kasugai H. A new approach to chemoembolization therapy for hepatoma using ethiodized oil, cisplatin, and gelatin sponge. Cancer 1987; 60: 1194-203.
15. Venook AP, Stagg RJ, Lewis BJ. Chemoembolisation for hepatocellular carcinoma. J Clin Oncol 1990, 8: 1108-14.
16. Bruix J, Castells A, Montanya X. Phase II study of transarterial embolization in European patients with hepatocellular carcinoma: need for controlled trials. Hepatology 1994; 20: 643-50.
17. Groupe d'Etude et de treatmentt du carcinoma hépatocellulaire. A comparison of lipiodol chemoembolization and conservative treatment for unresectable hepatocellular carcinoma. N Eng J Med 1995; 11: 1256-61.
18. Ngan H, Lai CL, Fan ST, Lai EC, Yuen WK, Tso WK. Transcatheter arterial chemoembolisation in inoperable hepatocellular carcinoma: four-year followup. J Vascu Interv Radiol 1996; 7: 419-25.
19. Liang SN, Liu LL, Su HY, Feng B, Zhao GS, Xu K. Analysis of severe complications after transcatheter arterial chemoembolization for primary hepatocellular carcinoma. Zhonghua Zhong Liu Za Zhi 2008; 30: 790-2.
20. Li CP, Chao Y, Chen LT, Lee RC, Lee WP, Yuan JN, et al. Fever after transcatheter arterial chemoembolization for hepatocellular carcinoma: incidence and risk factor analysis. Scand J Gastroenterol 2008; 43: 992-9.
21. Kiely JM, Rilling WS, Touzios JG, Hieb RA. Chemoembolization in patients at high risk: results and complications. J Vasc Interv Radiol 2006; 17: 47-53.