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# Is transarterial embolization a valuable treatment option for spontaneous rupture of hepatocellular carcinoma: experience from a tertiary care hospital of South-Asia

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## ABSTRACT

**Aim**: Transarterial embolization (TAE) has been found beneficial in treatment of ruptured Hepatocellular carcinoma (HCC) in earlier studies. So far no data is available from Pakistan. The aim of this study was to evaluate clinicopathological characteristics, outcomes of patients presented with spontaneously ruptured, unresectable HCC treated with or without TAE and to evaluate the factors associated with 30-day mortality. **Methods**: This was a cross sectional study. Patients ≥ 18 years old, presented with spontaneous rupture of unresectable HCC, were evaluated. The outcome measures were control of bleeding, in-hospital mortality, 30-day mortality and factors associated with 30-days mortality. **Results**: Out of 850 patients, 24 patients were diagnosed with spontaneously ruptured HCC. Mean age was 58.29 ± 15.26 years. A total of 11 (45.8%) patients were treated conservatively and 13 (54.2%) underwent TAE. Control of bleeding due to ruptured HCC was significantly higher for those treated via TAE as compared to those who were treated conservatively (92.3% *vs*. 36.4%, $P = 0.008$). Overall median duration for which the patients remained alive after HCC rupture was longer for TAE group (39 days *vs*. 5 days, $P = 0.03$). In-hospital mortality (30.8% *vs*.72.7%, $P = 0.04$) and 30-day mortality was also lower in TAE group (38.5% *vs*. 90.9%, $P = 0.01$). Those who underwent TAE had lower risk of mortality then conservative group [odds ratio (OR) 0.25, 95% confidence interval (CI) 0.07-0.90, $P = 0.03$). Failure to control bleeding was associated with higher 30-day mortality (OR 2.14, 95% CI 1.24-3.68, $P = 0.009$). **Conclusion**: Ruptured HCC is a life threatening complication requiring early diagnosis and treatment. TAE is an effective and well-tolerated treatment in the management of ruptured HCC.



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## INTRODUCTION

Hepatocellular carcinoma (HCC) is the fifth most common cancer and the third leading cause of cancer related mortality worldwide.[1] Relatively higher incidence rates have been reported from South Eastern Asia and sub-Saharan Africa.[2] The incidence rate of HCC in Pakistan is equivalent to 2.5 per 100,000 persons per year which is higher than the Sub-continent and Western countries.[3] Moreover, hepatitis C and B virus infection have been reported to be the major attributable factors responsible for HCC in Pakistan.[4]

While, most of the patients remain asymptomatic, HCC can manifest with right hypochondrial pain, weight loss, new onset jaundice and ascites.[5] Hemoperitoneum caused by spontaneous rupture of HCC is a rare but fatal complication associated with mortality ranging between 25-75%.[6,7] The incidence of spontaneous rupture of HCC ranges 3-15% in South-East Asian countries, which is higher as compared to the reported incidence of < 3% in Western countries.[6,8,9] Spontaneous rupture of HCC is associated with poor liver functional reserve, advanced stage of tumor and high mortality rates ranging 32-62% as seen in various studies.[10-12]

It is not only difficult to anticipate the HCC rupture; there are few therapeutic options available to treat such patients. The treatment modalities that have been employed include emergency liver resection in case of preserved liver function and resectable tumor, transarterial embolization (TAE) or transarterial chemoembolization (TACE) in case of advanced disease.[12,13] TAE has been found beneficial in the treatment of ruptured HCC in earlier studies by allowing control of bleeding and the selection of suitable patients for later liver resection. However, the utility of available treatment options is limited due to the patient's clinical condition and disease stage.[9,13,14] Moreover, most of the data available consists of studies with non-homogenous study population with variable disease stages, small sample size and limited results related to prognostic factors. No data is available from Pakistan so far.

Hence, in the current study, we report five years' experience with patients who presented with spontaneous rupture of unresectable HCC treated with or without TAE. The aim is to evaluate clinicopathological characteristics and outcomes of patients presenting with spontaneously ruptured, unresectable HCC treated with or without TAE and to evaluate the factors associated with 30-day mortality.

## METHODS

### Study population and duration

This was a retrospective cross sectional study. Patients ≥ 18 years of age, already diagnosed to have HCC and admitted to Gastroenterology ward of Aga Khan University Hospital (AKUH) during 2006-2015 were identified from our data base by using ICD code 1550. AKUH is a 563 bed, large tertiary care hospital in the metropolitan city of Karachi with a population of 18 million.[15] The medical record coders at AKUH assign numerical codes for diseases and procedures to all records in accordance with standards outlined in the International Classification of Diseases code book. Those HCC patients who presented with spontaneous rupture of unresectable HCC were studied and analyzed. However, patients with hemorrhagic ascites without HCC or where the required information was incomplete were excluded.

The information about patient's demographics, etiology of underlying cirrhosis, clinical, radiological characteristics, laboratory parameters, stage of HCC, treatment provided and follow up in days were recorded. Child-Pugh score and Model for End Stage Liver Disease (MELD) score were used to define the severity of liver disease.

The main outcome measure was control of bleeding. The other outcome measures were in-hospital mortality, 30-day mortality, overall duration of survival and factors associated with 30-day mortality.

### Diagnosis and staging of ruptured of HCC and cirrhosis

The diagnosis of HCC was made by combination of elevated alfa fetoprotein (AFP) (> 20 ng/mL) and characteristic features of HCC on triple-phase computerized tomography (CT) scan/magnetic resonance imaging (MRI); or in the absence of elevated AFP when the concurrent results were found on CT scan/MRI along with presence of background chronic liver disease, with or without histological verification. The diagnosis of cirrhosis was made either on liver biopsy or in the absence of liver biopsy by clinical and laboratory features of portal hypertension i.e. varices on upper gastrointestinal endoscopy, radiological features suggestive of cirrhosis including irregular liver margins, dilated portal vein, spleenomegaly and ascites.[16]

"Spontaneous HCC rupture" was defined when it happened without a history of recent procedure or trauma and the "diagnosis" was established by using contrast CT of the abdomen.

Modality of ruptured HCC diagnosis was defined as: (1) incidental when an asymptomatic HCC was discovered on imaging done during diagnostic procedures performed for some other disease; or (2) symptomatic when diagnosed during workup after symptom appearance. The HCC was considered as "non-advanced" if the lesion was solitary ≤ 5 cm or paucifocal ≤ 3 lesions, with the largest diameter ≤ 3 cm, in the absence of vascular invasion and distant metastases or "advanced," when the tumor exceeded these limits. Moreover, the HCC was also classified for macroscopic types as: (1) solitary; (2) paucifocal (≤ 3 nodules); (3) multifocal (> 3 nodules); (4) infiltrative (infiltrating pattern of HCC); or (5) massive (huge mass with a diameter of > 10 cm and an undefined boundaries).[17] In the presence of ≥ 2 lesions, the largest tumor was considered as representative of HCC and the diameter of the representative tumor measured in its greatest dimension was recorded as tumor size. Furthermore, information was recorded regarding hepatic lobes involved, presence of portal vein thrombosis and extra hepatic spread.

The patients were treated "conservatively" when liver reserves were poor defined by a Child class C or they were severely ill due to other comorbid conditions. TAE was performed in a well-equipped interventional radiological suite by a team of experienced interventional radiologists and Gel foam was used as embolizing agent. The study was conducted by maintaining compliance with the Helsinki Declaration and was approved by the Ethical review committee of Aga Khan University Hospital, Karachi.

### Statistical analysis

Data was entered and analyzed in SPSS version 17.0. Mean ± SD and ranges were calculated for continuous variables and proportions for categorical variables. To see the difference between two groups independent student *t*-test, Chi square or Fisher exact was used where appropriate. A univariate logistic regression analysis was conducted to assess the (crude) association of the prognostic factors for 30-day mortality. Biological significance and a value of $P \leq 0.1$ were considered as criteria for a variable to be significant at univariate analysis. Biological plausible interactions among variables and confounding were also checked. Multivariable logistic regression was done and results are expressed as odds ratio (OR), along with 95% confidence interval (CI).

## RESULTS

### Clinical characteristics of patients

The medical records of 850 patients with HCC who had visited our center during the study period were reviewed. A total of 24 patients were diagnosed to have spontaneously ruptured, unresectable HCC and were analyzed. The mean age was 58.29 ± 15.25 years (range 17-93 years) and most of them 21 (87.5%) were males. Hepatitis C was the most common cause of cirrhosis (79.2% cases). The mean Child-Pugh score was 9.96 ± 2.85 (range 7-15) and mean MELD score was 17.92 ± 6.38 (range 9-32). On presentation 62.5% had decompensated cirrhosis and many of them had prior history of hospitalization with spontaneous bacterial peritonitis (16.7%), portosystemic encephalopathy (20.8%), variceal bleed (12.5%) or hepatorenal syndrome (4.2%). The most common clinical manifestations of ruptured HCC on presentation were sudden abdominal pain (83.3%), hemoperitoneum (54.2%), symptoms of anemia (83.3%) and hypovolemic shock (25.0%). Diagnosis of ruptured HCC was confirmed on CT scan of abdomen in all cases. The mean tumor size was 7.76 ± 4.22 cm (range 1.7-17.7 cm). Almost two-third of patients had multifocal (50.0%) or massive/infiltrative (25.0%) HCC. Moreover, advanced HCC was found in 87.5% cases on presentation [Table 1].

A total of 11 (45.8%) patients were treated conservatively who either had poor general condition, impaired hepatic reserves, multiple lesions, or when patient had declined any intervention. TAE was performed in 13 (54.8%) cases of ruptured HCC. None of them underwent for emergency resection.

### Comparison of patients treated conservatively *vs.* those treated with TAE

There was no statistically significant difference in age, gender, etiology of underlying cirrhosis or symptoms and signs at presentation among those treated conservatively as compared to those who underwent TAE. The tumor size, macroscopic types, location and stage of HCC were also comparable among both groups [Table 2]. Although the prior hepatic decompensations, MELD and Child score were comparable in both groups, most of the patients in conservative group had patients with Child class C as compared to TAE group (54.5% *vs.* 15.4%, $P = 0.08$). Likewise, serum total bilirubin level (5.14 ± 3.50 *vs.* 2.15 ± 1.04, $P = 0.008$) was higher and albumin was lower (2.04 ± 0.41 *vs.* 2.63 ± 0.49, $P = 0.004$) in conservative treatment group as compared to TAE group.

The control of HCC bleeding was achieved in 66.7% cases which was significantly higher for those who were treated via TAE as compared to those who were treated conservatively (92.3% *vs.* 36.4%, $P = 0.008$). Overall median duration for which the patients remained alive after HCC rupture was longer for

**Table 1: Demographic and clinic-pathological characteristics of all HCC patients at baseline (*n* = 24)**

| Characteristics | Data, mean ± SD or *n* (%) |
|---|---|
| Age (years) | 58.29 ± 15.26 (range 17-93) |
| Etiology of CLD | |
| HCV | 19 (79.2) |
| HBV | 3 (12.5) |
| NBNC | 2 (8.3) |
| Child class | |
| A | 0 (0) |
| B | 16 (66.7) |
| C | 8 (33.3) |
| Abdominal pain | |
| Yes | 20 (83.3) |
| No | 4 (16.7) |
| Abdominal distension | |
| Yes | 16 (66.7) |
| No | 8 (33.3) |
| Anemia | |
| Yes | 20 (83.3) |
| No | 4 (16.7) |
| Hypovolemic shock | |
| Yes | 6 (25) |
| No | 18 (75) |
| Hemoperitonium | |
| Yes | 13 (54.2) |
| No | 11 (45.8) |
| Mean hemoglobin (g/dL) | 8.4 ± 3.0 |
| Platelet count ($10^9$/L) | 202.58 ± 176.50 |
| Total lecucocyte count ($10^9$/L) | 10.96 ± 4.17 |
| Prothrombin time (s) | 17.38 ± 5.64 |
| Mean creatinine (mg/dL) | 1.35 ± 0.57 |
| Serum total bilirubin (mg/dL) | 3.52 ± 2.87 |
| Alanine transaminase (IU/L) (median) | 50.00 (range 13-768) |
| Alkaline phosphatase (IU/L) | 210.13 ± 158.07 |
| Albumin (g/dL) | 2.36 ± 0.54 |
| Tumor size (size of largest lesion in cm) | 7.76 ± 4.22 (1.7-17.7) |
| AFP (IU/mL) (median) | 52.00 (range 1.00-100000) |
| Macroscopic types | |
| Solitary | 3 (12.5) |
| Paucifocal (≤ 3 nodules) | 3 (12.5) |
| Multifocal (> 3 nodules) | 12 (50.0) |
| Massive (huge diameter > 10 cm, undefined boundaries)/infiltrative | 6 (25.0) |
| Hepatic lobes (location of rupture) | |
| Right | 12 (50.0) |
| Left | 1 (4.2) |
| Both | 11 (45.8) |
| Stage of HCC | |
| Non-advanced | 3 (12.5) |
| Advanced | 21 (87.5) |
| PVT | |
| Yes | 10 (41.7) |
| No | 14 (58.3) |
| Extra hepatic spread | |
| Yes | 9 (37.5) |
| No | 15 (62.5) |

HCC: hepatocellular carcinoma; CLD: chronic liver disease; HCV: hepatitis C virus; HBV: hepatitis B virus; NBNC: non-B, non-C; MELD: Model for End Stage Liver Disease; AFP: alfa fetoprotein; PVT: portal vein thrombosis

TAE group (39 days, interquartile range 88 days) as compared to conservatively treated group (5 days, interquartile range 10 days) (*P* = 0.03). In addition, in-hospital mortality was significantly lower in TAE group as compared to patients treated conservatively (30.8% *vs*. 72.7%, *P* = 0.04). Moreover, 30-day mortality was also lower in patients treated with TAE (38.5% *vs*. 90.9%, *P* = 0.01) [Table 3].

### Predicting factors for 30-day mortality

To find out the predicting factors for 30-day mortality, biologically plausible variables were tested on univariate analysis [Table 4]. The only factors which were found significant on univariate and multivariate analysis were TAE to control HCC bleed and control of bleeding. Those who underwent TAE had lower risk of mortality than conservatively treated group (OR 0.25, 95% CI 0.07-0.90, *P* = 0.03). Failure to control bleeding was associated with higher 30-day mortality (OR 2.14, 95% CI 1.24-3.68, *P* = 0.009).

## DISCUSSION

In this study, we have evaluated the clinicopathological characteristics, treatment outcomes and survival of patients presenting with spontaneously ruptured HCC who were treated conservatively or with TAE. Success rate for control of bleeding via TAE was higher than with conservative treatment. Overall median duration of survival after HCC rupture was longer for patients treated with TAE. In-hospital and 30-day mortality were significantly lower in TAE group.

The reported prevalence of spontaneously ruptured HCC ranges 5-15%.[18] The exact mechanism and risk factors for spontaneous rupture are not well known. However, subcapsular localization, rapid growth with tumor necrosis, portal hypertension and regional increase of venous pressure due to tumor thrombi or direct invasion could be responsible for HCC rupture.[19]

Sudden abdominal pain, hemoperitoneum and hypovolemic shock have been reported as the typical clinical features of ruptured HCC.[6,9,20] Moreover, hemoperitoneum ascertained by performing abdominal paracentesis has been considered a reliable test to confirm the diagnosis in up to 86% of clinically suspected HCC rupture.[21] Consistent with the results of other studies most of our patients were male, presented with abdominal pain and distention, hemoperitoneum and shock.

Doppler ultrasound and CT are useful modalities for the diagnosis of HCC rupture.[22] The CT scan demonstrate HCC rupture by showing the vascular tumor, extent of the bleed and by showing serial density changes with the age of the hematoma.[23,24] Triphasic contrast enhanced CT scan was done for all

**Table 2: Comparison of baseline characteristics of patients treated conservatively *vs*. those treated with TAE**

| Characteristics | Conservative treatment, mean ± SD or *n* (%), *n* = 11 | TAE, mean ± SD or *n* (%), *n* = 13 | *P* value |
|---|---|---|---|
| Age (years) | 61.36 ± 16.13 | 55.69 ± 14.60 | 0.37 |
| Gender | | | 0.45 |
| Male | 9 (81.8) | 12 (92.3) | |
| Female | 2 (18.2) | 1 (7.7) | |
| Etiology of cirrhosis | | | 0.72 |
| HCV | 8 (72.7) | 11 (84.6) | |
| HBV | 2 (18.2) | 1 (7.7) | |
| NBNC | 1 (9.1) | 1 (7.7) | |
| Decompensated cirrhosis | 6 (54.5) | 9 (62.5) | 0.67 |
| Prior history of SBP | 3 (27.3) | 1 (7.7) | 0.30 |
| Prior history of PSE | 3 (27.3) | 2 (15.4) | 0.63 |
| Prior history of variceal bleed | 2 (18.2) | 1 (7.7) | 0.57 |
| Prior history of HRS | 0 (0) | 1 (7.7) | 1.0 |
| Abdominal pain | 8 (72.7) | 12 (92.3) | 0.22 |
| Anemia | 9 (81.8) | 11 (84.6) | 0.85 |
| Hypovolemic shock | 2 (18.2) | 4 (30.8) | 0.64 |
| Ascites | 10 (90.9) | 9 (69.2) | 0.32 |
| Hemoperitonium | 7 (63.6) | 6 (46.2) | 0.39 |
| Child class | | | 0.08 |
| A | 0 (0) | 0 (0) | |
| B | 5 (45.5) | 11 (84.6) | |
| C | 6 (54.5) | 2 (15.4) | |
| Child score | 11.0 ± 2.90 | 9.08 ± 2.60 | 0.105 |
| MELD score | 19.27 ± 7.17 | 16.77 ± 5.67 | 0.361 |
| Prothrombin time (s) | 19.83 ± 5.50 | 15.32 ± 5.07 | 0.51 |
| Serum total bilirubin (mg/dL) | 5.14 ± 3.50 | 2.15 ± 1.04 | 0.008 |
| Albumin (g/dL) | 2.04 ± 0.41 | 2.63 ± 0.49 | 0.004 |
| AFP (IU/mL) | | | 0.99 |
| ≤ 20 | 4 (36.4) | 5 (38.5) | |
| > 20 | 7 (63.6) | 8 (61.5) | |
| Tumor size (cm) | 7.64 ± 4.14 | 7.88 ± 4.45 | 0.892 |
| Macroscopic type | | | 0.84 |
| Solitary | 2 (18.2) | 1 (7.7) | |
| Paucifical | 1 (9.1) | 2 (15.4) | |
| Multifocal | 5 (45.5) | 7 (53.8) | |
| Infiltrative | 3 (27.3) | 3 (23.1) | |
| Stage of HCC | | | 0.99 |
| Non-advanced | 1 (9.1) | 2 (15.4) | |
| Advanced | 10 (90.9) | 11 (84.6) | |
| Hepatic lobes (location of rupture) | | | 0.53 |
| Right | 5 (45.5) | 7 (53.8) | |
| Left | 0 (0) | 1 (7.7) | |
| Both | 6 (54.5) | 5 (38.5) | |
| PVT | | | 0.69 |
| Yes | 4 (36.4) | 6 (46.2) | |
| No | 7 (63.6) | 7 (53.8) | |
| Extra hepatic spread | | | 0.42 |
| Yes | 3 (27.3) | 6 (46.2) | |
| No | 8 (72.7) | 7 (53.8) | |

TAE: transarterial embolization; SBP: spontaneous bacterial peritonitis; PSE: porto systemic encephalopathy; HRS: hepatorenal syndrome; HCV: hepatitis C virus; HBV: hepatitis B virus; NBNC: non-B, non-C; MELD: Model for End Stage Liver Disease; AFP: alfa fetoprotein; HCC: hepatocellular carcinoma; PVT: portal vein thrombosis

of our patients and was found very useful in our study to confirm HCC rupture in all cases.

For spontaneously ruptured HCC, emergency hepatic resection with hepatic artery ligation has been used as preferred method of treatment in past. However, the procedure was found to be associated with high mortality of 44-73%. Moreover, it is technically difficult to perform in decompensated liver disease and in palliative setting for advance disease where it could be associated with high likelihood of peritoneal seeding and poor outcome after resection.[6,12,18,25] The majority of our patients had advanced HCC, with large tumor size (mean diameter 7.76 cm) and multifocal disease. Hence, none of our patients had emergency hepatic resection.

**Table 3: Comparison of outcome among patients treated conservatively *vs*. those treated with TAE**

| Outcomes | Overall | Conservative treatment, *n* (%) or median ± range | TAE, *n* (%) or median ± range | *P* value |
|---|---|---|---|---|
| Control of bleeding | | | | 0.008 |
| Yes | 16 (66.7) | 4 (36.4) | 12 (92.3) | |
| No | 8 (33.3) | 7 (63.6) | 1 (7.7) | |
| In hospital mortality | | | | 0.04 |
| No | 12 (50.0) | 3 (27.3) | 9 (69.2) | |
| Yes | 12 (50.0) | 8 (72.7) | 4 (30.8) | |
| 30-day mortality | | | | 0.01 |
| No | 9 (37.5) | 1 (9.1) | 8 (61.5) | |
| Yes | 13 (54.2) | 10 (90.9) | 5 (38.5) | |
| Median survival (days) | 11.5 (interquartile range 53 days) | 5 (interquartile range 10 days) | 39 (interquartile range 87.5 days) | 0.03 |

TAE: transarterial embolization

**Table 4: Univariate analysis for predicting factors for 30-day mortality**

| Factors | OR (95% CI) | *P* value |
|---|---|---|
| Age (years) | 1.1 (0.95-1.06) | 0.76 |
| Gender | 1.23 (0.09-15.87) | 0.87 |
| Abdominal pain | 2.0 (0.17-22.79) | 0.57 |
| Hypovolemic shock | 0.20 (0.30-13.06) | 0.46 |
| Child score | 1.04 (0.80-1.47) | 0.58 |
| Child's class | 2.33 (0.35-15.30) | 0.37 |
| MELD score | 1.01 (0.87-1.14) | 0.98 |
| INR | 2.2 (0.30-16.16) | 0.43 |
| Serum total bilirubin (mg/dL) | 1.42 (0.85-2.38) | 0.17 |
| Albumin (g/dL) | 0.43 (0.08-2.23) | 0.31 |
| AFP (IU/mL) | | |
| ≤ 20 | 1 | |
| > 20 | 1.33 (0.23-7.51) | 0.74 |
| Tumor size (cm) | 0.90 (0.73-1.11) | 0.34 |
| Stage of HCC | | |
| Non-advanced | 1 | |
| Advanced | 1.23 (0.09-15.87) | 0.87 |
| PVT | | |
| No | 1 | |
| Yes | 2.5 (0.45-13.64) | 0.29 |
| Extra hepatic spread | | |
| No | 1 | |
| Yes | 0.75 (0.13-4.22) | 0.74 |
| Intervention for control of bleeding (TAE) | | |
| No | 1 | |
| Yes | 0.25 (0.07-0.90) | 0.03 |
| Control of bleeding | | |
| Yes | 1 | |
| No | 2.14 (1.24-3.68) | 0.009 |

OR: odds ratio; CI: confidence interval; MELD: Model for End Stage Liver Disease; INR: international normalized Ratio; AFP: alfa fetoprotein; HCC: hepatocellular carcinoma; PVT: portal vein thrombosis; TAE: transarterial embolization

TAE has been found to be associated with many complications including bleeding, post-embolization syndrome, implanted peritoneal metastases and mortality rate up to 30%. However, considering TAE as minimally invasive and effective in achieving immediate hemostasis in patients with ruptured HCC as compared to resection, TAE could be a procedure of choice to achieve hemostasis without surgery for ruptured HCC.[26,27] In the past it has been suggested that TAE should only be administered only in the presence of a patent portal vein.[28] However, in our study we did not find any significant difference in the control of bleeding and 30-day mortality between patients having a patent or a thrombosed portal vein, its success in both conditions is comparable.

In a series of 62 patients with ruptured HCC, control of bleeding was achieved in 91% (57/62) cases after TAE. Moreover, 30-day mortality was 38% and overall median survival time was 39 days.[29] In another study, 3 out of 4 patients treated with TAE died within 30 days but most of them had Child's class C cirrhosis.[30] Likewise, TAE was found effective for control of bleeding in all 14 patients with HCC rupture, without significant impairment in liver function or treatment related deaths. However, only 3 patients survived for more than 6 months.[31] A success rate of 83% has been reported in series from Hong Kong.[9] Contrary to that conservative treatment has been reported to carry 100% mortality.[32] Our results are consistent to the existing evidence. We found higher rates for control of bleeding after TAE as compared to conservative treatment (92.3% *vs*. 36.4%, $P = 0.008$). In hospital mortality was 72.7% for those treated conservatively as compared to 30.8% after TAE. Moreover, our 30-day mortality rate was lower among our patients after TAE (38.5%) as compared to what has been reported in previous studies.[6,9,33] None of our patients had procedure related complications.

Severity of underlying cirrhosis, tumor size, vascular and extrahepatic spread, serum creatinine and hypovolemic shock have been reported as prognostic factors influencing survival after spontaneously ruptured HCC.[13,14,29,34] Although majority of patients who underwent TAE had Child's class A or B and the conservative group had many patients with Child's class C; no significant difference was found in Child score or MELD score between the two groups. In our study, the only variables that were found to be associated with 30-day mortality were TAE and control of HCC bleed. This might be due to small sample size in our study.

Our study had certain limitations; this is a retrospective, single-center study and our sample size was small. Moreover, none of our study patients had further loco-regional therapy, or chemotherapy later on that could improve their life expectancy. However, considering ruptured HCC is an uncommon, life threatening complication, our study could provide some information about its manifestations and treatment options from this part of the world. The primary aim of managing patients with ruptured HCC is control of bleeding which could be an important factor in determining early mortality. Considering high success rate in control of bleeding, lower mortality rates and improvement in survival as well as quality of life, TAE could be used as procedure of choice to achieve hemostasis at presentation for ruptured HCC. Larger studies would be required to support currently available evidence in favor of TAE.

In conclusion, ruptured HCC is a life threatening complication requiring early diagnosis and treatment. TAE is an effective and well-tolerated treatment in the management of unresectable, ruptured HCC in patients with liver cirrhosis.

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## Conflicts of interest
There are no conflicts of interest.

## Patient consent
Consent forms were obtained from the patients.

## Ethics approval
The study was conducted by maintaining compliance with the Helsinki Declaration and was approved by the Ethical review committee of Aga Khan University Hospital, Karachi.

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