Transarterial Embolization for Treatment of Symptomatic Polycystic Liver Disease: More than 2-Year Follow-up

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Abstract

Background: Currently, treatment of symptomatic polycystic liver disease (PLD) is still a challenging problem, especially for these patients who are not feasible for surgery. Minimally invasive options such as laparoscopic fenestration and percutaneous cyst aspiration with sclerotherapy demonstrated disappointing results due to multiple lesions. Because the cysts in PLD are mostly supplied from hepatic arteries but not from portal veins, transcatheter arterial embolization (TAE) of the hepatic artery branches that supply the major hepatic cysts can lead to shrinkage of the cyst and liver size, relieve symptoms, and improve nutritional status. This study aimed to evaluate the effectiveness of TAE with a mixture of N-butyl-2-cyanoacrylate (NBCA) and iodized oil for patients with severe symptomatic PLD during a more than 2-year follow-up.

Methods: Institutional review board had approved this study. Written informed consent was obtained from all patients. From February 2007 to December 2014, twenty-three patients (20 women and 3 men; mean age, 49.0 ± 14.5 years) infeasible for surgical treatments underwent TAE. Changes in the abdominal circumferences, volumes of intrahepatic cysts, hepatic parenchyma volume, and whole liver, clinical symptoms, laboratory data, and complications were evaluated after TAE.

Results: Technical success was achieved in all cases. No procedure-related major complications occurred. The median follow-up period after TAE was 48.5 months (interquartile range, 30.0–72.0 months). PLD-related severe symptoms were improved remarkably in 86% of the treated patients; TAE failed to benefit in four patients (four patients did not benefit from TAE). The mean maximum abdominal circumference decreased significantly from 106.0 ± 8.0 cm to 87.0 ± 15.0 cm (P = 0.021). The mean intrahepatic cystic volume reduction rates compared with pre-TAE were 36% at 12 months, 37% at 24 months, and 38% at 36 months after TAE (P < 0.05). The mean liver volume reduction rates were 32% at 12 months, 31% at 24 months, and 33% at 36 months (P < 0.05).

Conclusions: TAE with the mixture of NBCA and iodized oil appears to be a safe and effective treatment method for patients with symptomatic PLD, especially for those who are not good candidates for surgical treatments, to improve both hepatic volume and hepatic cysts volume.

Key words: Angiography; Autosomal Dominant Polycystic Kidney Disease; Polycystic Liver Disease; Transcatheter Arterial Embolization

INTRODUCTION

Polycystic liver diseases (PLDs) represent a group of genetic disorders in which cysts occur in the liver (autosomal dominant PLD) or in combination with cysts in the kidneys (autosomal dominant polycystic kidney disease).¹² Most patients with PLD are asymptomatic and require no special treatment. Severe symptoms such as abdominal pain, distention, and nausea can affect about 20% of patients who develop massive hepatomegaly with compression of the surrounding organs.³⁴ Conventional treatments for patients with symptomatic PLD include percutaneous cyst aspiration with or without injection of sclerosing solution, laparoscopic fenestration, open

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surgical cyst fenestration, or partial hepatectomy. However, despite these treatments, both cysts and symptoms tend to recur (cysts and symptoms would recur in spite of active treatment). In addition, these treatments may be associated with significant morbidity and even mortality. Liver transplantation is often the most suitable option for patients who experience hepatic failure and clinical deterioration as a result of the development of large cysts; however, it could only be performed in a limited number of patients because of the lack of donors and high costs. For these reasons, additional new approaches are in dire need.

In Japan, transcatheter arterial embolization (TAE) has become an accepted treatment option for patients with symptomatic PLD in several hospitals. Because the hepatic cysts patients with PLD are mostly supplied from hepatic arteries rather than portal veins, TAE of the hepatic artery branches that supply major hepatic cysts can shrink the cyst and liver size. This less invasive approach can relieve symptoms and avoid the morbidity and mortality associated with open surgery. However, this treatment has been performed only in a few hospitals and the experience and long-term outcomes are very limited in the literature reports. In addition, TAE has not been established as part of the medical treatment of PLD in Western countries. The purpose of our study was to evaluate the effectiveness of TAE using a mixture of N-butyl-2-cyanoacrylate (NBCA) and iodized oil, an embolic material different from previous reports, for patients with severe symptomatic PLD during a more than 2-year follow-up.

**Methods**

**Ethics statement**

This study was approved by the Medical Ethics Committee of Chinese PLA General Hospital. Written informed consent was obtained from the patients or their guardians before the procedure.

**Subjects**

Between February 2007 and December 2014 at a single institution, a total of 37 patients with symptomatic PLD received TAE, all of them with polycystic kidney; we excluded 14 patients due to incomplete follow-up data (n = 5), TAE using particles or coils rather than NBCA-iodized oil (n = 5), and lost to follow-up (n = 4). The remaining 23 patients (20 women and 3 men; age, 49.0 ± 14.5 years, with a range of 36–68 years) met the following inclusion criteria and were enrolled in our study. The diagnosis of PLD was established using computed tomography (CT), magnetic resonance imaging (MRI), and ultrasonography (US).

Inclusion criteria for our study were as follows: (a) diagnosis of PLD with severe symptoms such as abdominal pain, distention, dyspepsia, and dyspnea not relieved with medical treatment; (b) patients with symptomatic PLD were not candidates for surgery (i.e., surgical resection or surgical fenestration) due to poor medical condition or other reasons.

Exclusion criteria included patients with abnormal liver function tests (i.e., aspartate aminotransferase [AST] or alanine aminotransferase [ALT] >80 IU/L or total bilirubin >2.0 mg/dL), serum creatinine levels >1.2 mg/dL, serum urea levels >7.1 mmol/L, allergy to contrast media, moderate-massive ascites, diffusely distributed intrahepatic cysts, liver cyst infection or other systemic infection, or uncorrectable coagulopathy.

**Procedure**

The details of technique of TAE have been described previously. All procedures were performed on an inpatient basis by two senior interventional radiologists (Wang MQ and Liu FY, with 22 and 14 years of vascular and interventional radiology experience, respectively) using a therapeutic angiography unit equipped with a digital flat-panel detector system (INNOVA 4100 IQ; GE Healthcare, Milwaukee, WI, USA) and nonionic contrast medium (Visipaque 320 mgI/mL; GE Healthcare).

After intravenous moderate sedation and local anesthesia were achieved, a 5-Fr vascular sheath (Radifocus, Terumo, Tokyo, Japan) was inserted into the right femoral artery with the Seldinger technique. Through the right femoral artery approach, selective digital subtraction angiography (DSA) of the celiac artery and superior mesenteric artery was performed using a 4-Fr catheter (RH; Cordis Corporation, Miami Lakes, Florida, USA). Portal venography was performed as the late phase of superior mesenteric artery angiography. To precisely define peripheral hepatic arterial anatomy, additional super-selective hepatic arteriographies using a 2.6-Fr microcatheter (Progreat, Terumo Corporation, Tokyo, Japan) were performed.

After super-selective catheterization of the target hepatic artery, a mixture of NBCA (Histoacryl-Blue; Braun, Melsungen, Germany) and iodized oil (Lipiodol, Laboratoire Guerbet, Roissy, France) in a ratio of 1:4 was injected under fluoroscopy. Immediately before the injection of this mixture, the microcatheter was flushed with a small amount of 5% dextrose water solution to prevent the contact of NBCA with the blood in the lumen of the microcatheter. The endpoint of TAE was stasis of feeding arterial flow. The extrahepatic collaterals (i.e., the internal thoracic artery, the right inferior phrenic artery, and the omental artery) supplying the hepatic cystic lesions were also embolized using polyvinyl alcohol (PVA) particles (300–500-μm; PVA foam embolization particles, PVA, Cook Incorporated, Bloomington, IN, USA), if necessary. An intra-arterial injection of 3–5 ml of lidocaine (Lidocaine HCl 2%; Chengdu first pharmaceutical Co., Ltd., Chengdu, China) was used for pain control during the TAE procedure. After embolization, celiac arteriography was performed to confirm the complete occlusion of the target artery.

**Postprocedural management**

The patients stayed in the hospital for 2–5 days for observation and then were discharged when there were no complications. Antibiotic therapy was not given after the
procedure. Analgesics and antiemetics were administered as needed.

**Evaluations**

Before TAE, a routine physical examination, laboratory tests, and imaging studies were performed, including US, CT, or MRI. The imaging studies were performed within 2 weeks before the procedure. Follow-up was performed by two interventional radiologists (Wang MQ and Liu FY) on an outpatient basis. The subjective symptoms after TAE were based on any kind of changes (i.e., if they were better or worse or no change). The abdominal circumferences were specifically measured at the umbilicus before TAE and during the follow-up period after the procedure. Patients were followed up until loss contact, death, or December 30, 2014. All patients underwent follow-up CT at every 3 months for the 1st year after TAE and at 6-month intervals thereafter. CT was performed using a multidetector CT scanner (Brilliance 64-channel; Philips, Eindhoven, The Netherlands). Volumetric data were obtained from the portal phase images. The total intrahepatic cyst volume, hepatic parenchyma volume, and liver volume (cysts plus parenchyma) were calculated using the CT analysis system by two senior radiologists (blinded for review). Laboratory data were collected at the time of before the procedure, and 1, 7, and 14 days, 1, 3, 6, and 12 months after TAE. Laboratory data included routine blood tests, total bilirubin, AST, ALT, serum creatinine, serum urea, lactate dehydrogenase, alkaline phosphatase, and γ-glutamyl transpeptidase.

**Outcome parameters**

TAE was considered technically successful when the target hepatic arteries were complete embolized, as demonstrated by hepatic arterial angiography performed after completion of the procedure. Clinical success was defined as relief of symptoms and other invasive therapies were not required after the procedure. The total intra-hepatic cyst volume, hepatic parenchyma volume, and liver volume (cysts plus parenchyma) were also evaluated using CT analysis system by two senior radiologists (blinded for review). Complications were classified according to the guidelines of the Society of Interventional Radiology.[15] Major complications were defined as any event that resulted in additional therapy, such as an increased level of care, hospital stay beyond observational status, permanent adverse sequelae, and death. All other complications were classified as minor. Postembolization syndrome is the most common side effect of TAE, which was not considered a complication but rather an expected outcome.

**Statistical analysis**

Our study was conducted as a single-arm exploratory study. The means and standard deviations (SD) were presented if the data were normally distributed, and the medians and interquartile ranges (IQR) were presented if the data were not normally distributed. We calculated the mean and 95% confidence interval (CI) of the intrahepatic cyst volume, hepatic parenchyma volume, liver volume (cysts plus parenchyma), volume reduction rate of the liver, and circumference of the abdomen, before TAE as the baseline and at 3, 6, 12, 18, 24, and 36 months after TAE. Categorical variables were analyzed with Chi-square test or Fisher’s exact test, and continuous variables were compared using t-test. A value of P < 0.05 (two-tailed) was considered statistically significant. Statistical computer software (SPSS, version 22.0; SPSS Inc., Chicago, IL, USA) was used for data analyses.

**Results**

**Subjects**

All patients were judged not to be surgical candidates by surgeons from the Departments of Hepatobiliary and Anaesthesiologists. Before TAE, 6 (26%) patients received percutaneous cyst aspiration followed by local sclerosing agent injection with 2–4 sessions and 3 (13%) patients received laparoscopic fenestration. The patients’ backgrounds and the cystic characteristics are shown in Table 1. All patients were followed up for more than 2 years after TAE. The median follow-up period was 48.5 months (interquartile range, 30.0–72.0 months; range, 25–85 months).

**Technical success**

Technical success was achieved in all cases. TAE was performed only once in every patient, and the mean amount of NBCA used for TAE was 1.5 ± 0.5 ml (range, 1–2 ml). The mean procedure time was 75 ± 15 min, with times ranging from 55 to 95 min. No major complication related to TAE was observed. Mean length

| Table 1: Patient characteristics before TAE (n = 23) |
|---------------------------------|
| Parameters | Values |
| Age (years) | 49.5 ± 14.5 (36–68)* |
| >50 | 8 (35) |
| ≤50 | 15 (65) |
| Gender | Male 3 (13) Female 20 (87) |
| Symptom | Abdominal pain 23 (100) Abdominal distention 23 (100) Dyspepsia 21 (91) Dyspnea 17 (74) |
| Proportion of cysts in the liver | 50–70% 15 (65) >70% (71–85%) 8 (35) |
| Abdominal circumference (cm) | 106.0 ± 8.0 (90–112)* |
| Previous treatment | Cyst aspiration 6 (26) Laparoscopic fenestration 3 (13) |
| Blood sample data | Hemoglobin (g/dl) 11.5 ± 1.5 (9.1–12.5)* Total protein (g/dl) 6.9 ± 1.5 (5.6–7.5)* Albunin (g/dl) 3.3 ± 0.4 (2.9–3.9)* |

Data are shown as n (%). *Data are the mean ± SD, with the range in parentheses; †Data are calculated from CT or MRI. TAE: Transcatheter arterial embolization; MRI: Magnetic resonance imaging; CT: Computed tomography; SD: Standard deviation.
of hospital stay was 5.5 ± 2.0 days (range, 4–9 days). During the interventional procedure, the extrahepatic arterial blood supply to the cystic lesions was observed in 11 patients (48%); among these patients, 9 received other treatments (needle cyst aspiration or laparoscopic fenestration) before TAE. In total, 20 collateral vessels that fed the cysts were demonstrated on DSA, including the right inferior phrenic artery (n = 11), internal thoracic artery (n = 4), omental artery (n = 2), left inferior phrenic artery (n = 2), and the right renal capsular artery (n = 1). All these collateral vessels were successfully embolized [Figure 1].

Clinical outcomes
Symptoms improvement
At the end of follow-up, 21 (91%) of 23 patients remained alive. Two patients died during the follow-up: cardiovascular disease and cerebrovascular disease at 25 and 36 months after TAE, respectively, led to the mortalities. During the follow-up, these PLD-related symptoms (i.e., abdominal pain, distention, dyspepsia, and dyspnea) have notably improved in 19 (83%) of 23 patients, and these patients experienced good quality of life. The time of symptoms improvement after TAE was 4.5 ± 2.0 months (range, 2–6 months) and no recurrent symptoms were reported during the follow-up. Of these patients, the mean maximum abdominal circumference decreased significantly from 106.0 ± 8.0 cm (90–112 cm) to 87.0 ± 15.0 cm (80–105 cm; P = 0.021). TAE failed to benefit in 4 (17%) patients: the PLD-related symptoms were the same as pre-TAE; however, no patient complained of worsening of the symptoms after TAE.

Changes in volumes of total liver, liver cysts, and parenchyma before and after transcatheter arterial embolization
The median total liver volume pre-TAE was 8070 cm³ (interquartile range, 4650–10200 cm³), representing marked hepatomegaly. At follow-up CT at 3 months, the volumes of liver cysts, normal parenchyma, and total liver showed no statistically significant differences compared to pre-TAE (P > 0.05). At 6–36-month follow-up, the volumes of liver cysts and total liver decreased significantly (P < 0.05) compared to pre-TAE; the volumes of liver parenchyma increased significantly (P = 0.024) [Table 2].

The mean liver cystic volume reduction rates in comparison with pre-TAE value (with 95% CIs) were 7% (0, 8.5; P > 0.05) at 3 months, 23% (10.0, 35.5; P < 0.05) at 6 months, 36% (24.6, 46.3; P < 0.05) at 12 months, 37% (29.0, 51.2; P < 0.05) at 24 months, and 38% (25.0, 48.7; P < 0.05) at 36 months after TAE [Figure 2]. The mean liver volume reduction rates compared with pre-TAE value were 5% (0, 9.0; P > 0.05) at 3 months, 19% (12.0, 37.5; P < 0.05) at 6 months, 32% (25.0, 44.5; P < 0.05) at 12 months, 31% (27.0, 54.0; P < 0.05) at 24 months, and 33% (26.0, 58.5; P < 0.05) at 36 months after

Figure 1: Images in a 44-year-old woman presented with severe symptomatic polycystic liver disease. (a) A portal phase computed tomography image obtained before transcatheter arterial embolization shows marked hepatomegaly and that almost the entire right hepatic lobe is replaced by multiple cysts (arrows). The asterisks indicate the normal liver parenchyma. (b) A celiac arteriography before transcatheter arterial embolization shows that the right hepatic arterial branches are stretched (arrows), representing cystic regions. (c) Selective right inferior phrenic angiography before transcatheter arterial embolization shows the multiple small branches supplying the right lobe liver cysts (arrows). (d) Pretranscatheter arterial embolization, portal venography obtained at the late phase of superior mesenteric artery angiography, shows that the right portal vein branches are obstructed (arrows), which correspond to the right hepatic region replaced by multiple cysts; the left portal vein is well patent (arrowheads), which corresponds to the preserved intact hepatic parenchyma. (e) Celiac arteriography obtained at posttranscatheter arterial embolization shows the right hepatic artery branches supplying the cystic regions embolized by the mixture of N-butyl-2-cyanoacrylate and iodized oil (arrows). (f) Computed tomography image at the same level as in (a), obtained at 1 month after transcatheter arterial embolization, shows the iodized oil deposited in the cystic regions (arrows) with intact the normal parenchyma (asterisks). (g) Computed tomography image at the same level as in (a) obtained at 36 months after transcatheter arterial embolization shows marked decrease in the intra-hepatic cystic volume (arrows) and increase in the hepatic parenchyma volume (asterisks).
The volume of mean liver parenchyma increase rates compared with pre-TAE value were 6% (0, 9.0; P > 0.05) at 3 months, 25% (15.0, 38.5; P < 0.05) at 6 months, 37% (26.5, 48.0; P < 0.05) at 12 months, 34% (28.0, 50.0; P < 0.05) at 24 months, and 35% (25.0, 55.7; P < 0.05) at 36 months after TAE [Figure 2].

**Complications**

No procedure-related major complications occurred. Posttreatment syndrome, characterized by some degree of abdominal pain, low-grade fever, loss of appetite, nausea, and leukocytosis developed in all patients within 1 week after TAE; these symptoms were self-limited and reversible and some patients received antiemetics, nonopioid analgesic, and antipyretics.

**Laboratory data**

An increase in liver enzymes (AST, ALT) to nearly twice the normal levels was observed 24 h after TAE. In all cases, the enzyme levels returned to the baseline levels after 1–2 weeks without specific treatment. Values of serum creatinine and urea were unchanged in all patients after TAE. At the end of follow-up, the blood sample data were obtained in 17 patients and these patients had significant changes in the total protein, albumin, and hemoglobin levels, increased from 6.9 ± 1.6 g/dl to 7.5 ± 0.7 g/dl (P < 0.05), 3.2 ± 0.5 g/dl to 3.5 ± 0.6 g/dl (P < 0.05), and 11.0 ± 1.7 g/dl to 12.0 ± 1.7 g/dl (P < 0.05) after TAE, respectively, suggesting improvement in their general nutritional status.

**Discussion**

Our study was designed to relieve the severe compression symptoms caused by PLD in patients in whom other medical therapy has failed, and who are not good candidates for surgical treatments due to poor medical conditions. At follow-up of 25–85 months, PLD-related highly symptoms were improved remarkably in 85.7% of our treated patients.
and these patients experienced sustained effectiveness without recurrence and had excellent quality of life. On CT follow-up, the mean liver cystic volume reduction rates compared with pre-TAE were 23% at 6 months, 36% at 12 months, 37% at 24 months, and 38% at 36 months after TAE; the results seem to be better than those previous reports,\[9,10,11\] In an early study by Takei et al.,\[10\] they treated 30 patients with TAE for PLD, with a follow-up period of 18–37 months; 80% (24/30) of these patients benefited from TAE. Of these patients, the mean volume of intrahepatic cysts decreased significantly from \(6677 \pm 2978 \text{ cm}^3\) to \(4625 \pm 2299 \text{ cm}^3\), with a mean reduction rate of 31%; the mean liver volume decreased significantly from \(7882 \pm 2916 \text{ cm}^3\) to \(6041 \pm 2282 \text{ cm}^3\), with a mean reduction rate of 23%. Recently, Yang et al.\[5\] reported that the failure rate of TAE for PLD was relatively high (69.6%). Of 18 TAE patients, only 6 (33%) required no further treatment, the others required a second treatment for uncontrolled symptoms or hepatic failure following treatment, and 5 of these patients required surgical treatments eventually.\[17\] However, the authors did not provide the technical details of TAE. Better results in our study might come from using different embolic agent: mixture of NBCA and iodized oil, as we reported previously.\[14\]

According to our experience, TAE using the mixture of NBCA and iodized oil for patients with symptomatic PLD is a safe treatment option.\[14,16\] No major complications were observed in our study. Currently, NBCA has rarely been used for hepatic arterial embolization due to its liquid property.\[17,18\] Huang et al. reported that TAE with liquid embolic agents for the treatment of hepatic hemangioma could cause severe bile duct injuries and infarction.\[19\] Theoretically, the rate of TAE related complications should be higher in using the mixture of NBCA and iodized oil than that of using coils embolization.\[10,11\] However, these liver TAE-related complications such as hepatic infarction, biloma, abscess, and bile duct injuries, reported by others,\[17,18\] did not occur in our study. We speculate that absence of the bile duct structure in the hepatic cystic regions is an important reason for the absence of the bile duct injury complications. In addition, TAE should be performed superselectively to target hepatic cystic regions and to preserve remaining intact hepatic parenchyma. In our study, post-treatment syndrome and transient increase in liver enzymes slightly were observed in all patients within 1–2 weeks after TAE, which were comparable with those reported in other studies;\[19\] these symptoms were usually self-limited and reversible without special treatment.

Technically, TAE for PLD should be performed as completely as possible in embolization of the feeders supplying the cystic lesions.\[10,14\] Based on our experience, the liver cystic lesions are usually fed by multiple feeding arteries, not only through the hepatic arterial branches but also through extrhepatic collaterals, and these collaterals may be one of negative influence factors on the long-term efficacy of TAE. For more effective treatment of PLD with TAE, not only the hepatic arterial branches but also these collaterals should be adequately embolized. In our study, the extrhepatic arterial collaterals supplying the liver cysts were demonstrated in 48% of patients, mainly through the right inferior phrenic artery and internal thoracic artery. We embolized these collaterals using PVA successfully without any complications.

Our study has several limitations. First, it was a single-center study, and the number of patients is limited. Further experiences are essential to establish the safety and effectiveness of TAE with the mixture of NBCA and iodized oil. Second, this study did not have control group of patients – which is not practicable – because all patients had severe conditions and were not candidates for surgery (i.e., surgical resection or surgical fenestration) due to poor medical condition or other reasons. Third, only the mixture of NBCA and iodized oil was used and did not use the other embolic materials (i.e., microcoils, particles) as a control. Finally, there might be a selection bias because only a part of our patients with severe symptomatic PLD infeasible for surgery were referred for TAE.

In conclusion, TAE with the mixture of NBCA and iodized oil appears to be a safe and effective treatment method for patients with symptomatic PLD, especially for those who are not good candidates for surgical treatments, with improvement of hepatic volume and shrinkage of cysts volume.

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Conflicts of interest
There are no conflicts of interest.

References

1. Abu‑Wasel B, Walsh C, Keough V, Molinari M. Pathophysiology, epidemiology, classification and treatment options for polycystic liver diseases. World J Gastroenterol 2013;19:5775‑86. doi: 10.3748/wjg.v19.i35.5775.
2. Drenth JP, Chrispijn M, Nagorney DM, Kamath PS, Torres VE. Medical and surgical treatment options for polycystic liver disease. Hepatology 2010;52:2223‑30. doi: 10.1002/hep.24036.
3. Gevers TJ, Drenth JP. Diagnosis and management of polycystic liver disease. Nat Rev Gastroenterol Hepatol 2013;10:101‑8. doi: 10.1038/nrgastro.2012.254.
4. Khan S, Demison A, Garcea G. Medical therapy for polycystic liver disease. Ann R Coll Surg Engl 2016;98:18‑23. doi: 10.1308/rcsann.2016.0023.
5. Yang J, Ryu H, Han M, Kim H, Hwang YH, Chung JW, et al. Comparison of volume‑reductive therapies for massive polycystic liver disease in autosomal dominant polycystic kidney disease. Hepatol Res 2016;46:183‑91. doi: 10.1111/hepr.12560.
6. Tseng J, Orloff SL. Management of symptomatic polycystic liver disease with hepatic resection. JAMA Surg 2015;150:81‑2. doi: 10.1001/jamasurg.2014.307.
7. Antonacci N, Ricci C, Taffurelli G, Casadei R, Minni F. Systematic review of laparoscopic versus open surgery in the treatment of non‑parasitic liver cysts. Updates Surg 2014;66:231‑8. doi: 10.1007/s13304‑014‑0270‑3.
8. Ogawa K, Fukunaga K, Takeuchi T, Kawagishi N, Ubara Y, Kudo M, et al. Current treatment status of polycystic liver disease in Japan. Hepatol Res 2014;44:1110‑8. doi: 10.1111/hepr.12286.
9. Baber JT, Hiatt JR, Busuttil RW, Agopian VG. A 20-year experience with liver transplantation for polycystic liver disease: Does previous palliative surgical intervention affect outcomes? J Am Coll Surg 2014;219:695-703. doi: 10.1016/j.jamcollsurg.2014.03.058.

10. Takei R, Ubara Y, Hoshino J, Higa Y, Suwabe T, Sogawa Y, et al. Percutaneous transcatheter hepatic artery embolization for liver cysts in autosomal dominant polycystic kidney disease. Am J Kidney Dis 2007;49:744-52. doi: 10.1053/j.ajkd.2007.03.018.

11. Hoshino J, Ubara Y, Suwabe T, Sumida K, Hayami N, Mise K, et al. Intravascular embolization therapy in patients with enlarged polycystic liver. Am J Kidney Dis 2014;63:937-44. doi: 10.1053/j.ajkd.2014.01.422.

12. Hoshino J, Suwabe T, Hayami N, Sumida K, Mise K, Kawada M, et al. Survival after arterial embolization therapy in patients with polycystic kidney and liver disease. J Nephrol 2015;28:369-77. doi: 10.1007/s40620-014-0138-0.

13. Ubara Y, Takei R, Hoshino J, Tagami T, Sawa N, Yokota M, et al. Intravascular embolization therapy in a patient with an enlarged polycystic liver. Am J Kidney Dis 2004;43:733-8. doi: 10.1053/j.ajkd.2003.12.035.

14. Wang MQ, Duan F, Liu FY, Wang ZJ, Song P. Treatment of symptomatic polycystic liver disease: Transcatheter super-selective hepatic arterial embolization using a mixture of NBCA and iodized oil. Abdom Imaging 2013;38:465-73. doi: 10.1007/s00261-012-9931-1.

15. Brown DB, Gould JE, Gervais DA, Goldberg SN, Murthy R, Millward SF, et al. Transcatheter therapy for hepatic malignancy: Standardization of terminology and reporting criteria. J Vasc Interv Radiol 2009;20 7 Suppl:S425-34. doi: 10.1016/j.jvir.2009.04.021.

16. Duan F, Wang MQ, Liu FY, Wang ZJ, Song P. Transcatheter hyper-selective hepatic arterial embolization for the treatment of polycystic liver disease (in Chinese). Chin J Hepatol 2011;19:67-8. doi: 10.3760/cma.j.issn.1007-3418.2011.01.021.

17. Guiu B, Deschamps F, Aho S, Munck F, Dromain C, Boige V, et al. Liver/biliary injuries following chemoembolisation of endocrine tumours and hepatocellular carcinoma: Lipiodol vs. drug-eluting beads. J Hepatol 2012;56:609-17. doi: 10.1016/j.jhep.2011.09.012.

18. de Baere T, Arai Y, Lencioni R, Geschwind JF, Rilling W, Salem R, et al. Treatment of liver tumors with lipiodol TACE: Technical recommendations from experts opinion. Cardiovasc Intervent Radiol 2016;39:334-43. doi: 10.1007/s00270-015-1208-y.

19. Huang XQ, Huang ZQ, Duan WD, Zhou NX, Feng YQ. Severe biliary complications after hepatic artery embolization. World J Gastroenterol 2002;8:119-23.