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BASIC RESEARCH

Portal vein embolization induces compensatory hypertrophy of remnant liver

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Abstract

AIM: To evaluate the effectiveness and safety of different portal vein branch embolization agents in inducing compensatory hypertrophy of the remnant liver and to offer a theoretic basis for clinical portal vein branch embolization.

METHODS: Forty-one adult dogs were included in the experiment and divided into four groups. Five dogs served as a control group, 12 as a gelfoam group, 12 as a coil-gelfoam group and 12 as an absolute ethanol group. Left portal vein embolization was performed in each group. The results from the embolization in each group using different embolic agents were compared. The safety of portal vein embolization (PVE) was evaluated by liver function test, computed tomography (CT) and digital subtraction angiography (DSA) of liver and portal veins. Statistical test of variance was performed to analyze the results.

RESULTS: Gelfoam used for PVE was inefficient in recanalization of portal vein branch 4 wk after the procedure. The liver volume in groups of coil-gelfoam and absolute ethanol increased 25.1% and 33.18%, respectively. There was no evidence of recanalization of embolized portal vein, hepatic dysfunction, and portal hypertension in coil-gelfoam group and absolute ethanol group.

CONCOUSION: Portal vein branch embolization using absolute ethanol and coil-gelfoam could induce atrophy of the embolized lobes and compensatory hypertrophy of the remnant liver. Gelfoam is an inefficient agent.

Key words: Portal vein embolization; Interventional therapy; Liver; Dog

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INTRODUCTION

Primary carcinoma of the liver is one of the common malignant tumors in our country. Hepatectomy remains one of the most important therapies. However, only 15-30% of diagnosed patients are suitable for hepatectomy. Although extended hepatic resection is an effective therapy, partial excision of normal and functional liver is unavoidable in this surgery. If partial excision resects a certain amount of liver, patients especially those with liver cirrhosis, would suffer from postoperative liver failure. Although limiting resection area can reduce the possibility of complications, it increases the incidence of postoperative recurrence and metastasis. These problems can be effectively solved by artificially inducing hyperplasia of normal liver tissue preoperatively. Therefore, preoperative portal vein embolization (PPVE) is an effective procedure to settle this contradiction[1-5].

In 1920, Roust Larimore discovered that ligation of rabbits' portal vein branch could contribute to the atrophy of pathological lobe and hyperplasia of non-pathological one. In 1975, Honjo *et al*[6] tried portal vein ligation in liver cancer patients not fit for hepatic resection, which resulted in atrophy of the ligated lobe of portal vein and its tumor and hyperplasia of non ligated lobe. In 1980s, Makuuchi *et al*[7] found that if the portal vein of one lobe is embolized by tumor thrombi, hyperplasia usually occurs in the other lobe of the liver, and meanwhile, patients enjoy better recovery after hepatectomy because unexpected rising of portal vein pressure could be avoided. These findings have led to the exploration of preoperative PVE prior to extensive liver hepatic resection. PVE in clinical practice[1,2,8,9] and experimental studies[10-13] has achieved success. Since foreign emboli materials are expensive and difficult to obtain, it is essential to screen for easily-obtained, convenient, cheap, and safe embolic agents to effectively induce compensatory hyperplasia. The purpose of our study was to evaluate the effectiveness and safety of three convenient and cheap agents in inducing compensatory hypertrophy of liver.

MATERIALS AND METHODS

Experimental animals

Forty-one healthy adult dogs weighing 16.2+4.2 kg (range, 12.0-20.0 kg) without obvious growth (to avoid sudden changes of weight during the process of experiment, which would affect liver volume and influence experimental results), were included in the study. All dogs were provided by Experimental Animal Center of Union Hospital attached to Fujian Medical University. Before and after the experiment, they were fed with same standard forage. Five served as the control group and the remaining 36 were divided into 3 groups at random, 12 in each group. Gelfoam, coil-gelfoam and absolute ethanol were applied to these 3 experimental groups for portal vein embolization.

Methods

A dog with jejunitis on operative day was given an intravenous injection of 10% pentobarbital sodium into the hind legs to induce general anesthesia. Then, the dog underwent helical CT scanning to conduct pre and post contrast-enhanced CT study of its liver. The liver volume was measured automatically by a computer with the help of SOMATOM PLUS4 spiral CT scanner (Siemens Co.). The dog was transferred into the interventional operating room and cut open for catheter placement of portal vein under general anesthesia. After a 5F Cobra catheter was inserted into the trunk of portal vein, 76% angiografin was injected at the speed of 8-10 mL/s through the catheter to study the branch pattern of portal vein. Because the right lobe was relatively fixed, left portal vein was chosen as a target vessel for embolization. Then, the catheter was inserted into the left portal vein and saline or an embolic agent was injected into the appropriate position in different experimental groups. Control group: 30-50 mL saline was injected through the catheter. Gelfoam group: gelfoam was cut into strips of 10 mm×1.0 mm, which were inserted into the embolized portal vein through the catheter with saline until complete embolization was achieved. Embolization procedure was stopped when the embolized portal vein was completely embolized and the distal portal vein branch was not opacified. The number of gelfoams used ranged between 40-60 strips. Coil-gelfoam group: gelfoam was used to embolize smaller branches of the left portal vein, then coils were used to embolize the trunk of the left portal vein until satisfactory results were achieved. The number of coils ranged between 3-5. Absolute ethanol group: Five milliliters of absolute ethanol was slowly injected through the catheter. After several minutes, an additional amount of absolute ethanol was provided based on the embolization situation of portal vein in order to achieve the result of complete embolization. The amount of absolute ethanol was generally 0.5-1.0 mL/kg. After embolization, 9-12 mL angiografin was injected through the catheter at a speed of 6-8 mL/s to carry out angiography of the portal vein, which would allow us to understand its iconography expression after embolization. At the same time, we measured the pressure of main portal vein, and the radial lines of the nonembolized portal veins including the right portal vein and main portal vein.

Table 1 Changes of the right lobe volume in gelfoam group (cm^3)

No	Pre-embolization	4W	6W	8W
Dog A1	250	265	260	263
Dog A2	338	340	334	348
Dog A3	380	395	370	398
Dog A4	273	279	277	280
Dog A5	370	368	373	378
Dog A6	320	318	334	332
Dog A7	229	245	230	237
Dog A8	342	365	344	377
Dog A9	360	353	373	374
Dog A10	362	363	370	368
Mean±SD	318±18.2	347.6±21.4	321.7±17.7	331.9±19.4

After embolization, all dogs were regularly fed. Angiography was performed 4 and 8 wk after embolization, transcatheter portal vein angiography was undertaken to understand the iconography expression of portal vein branches, and then diameter of the nonembolized portal vein was measured. Changes of portal vein pressure were monitored 4 and 8 wk after embolization, transcatheter portal vein angiography was performed to measure the portal vein pressure. Changes of liver function were detected before and after embolization, alanine aminotransferase (ALT) was assayed every 2 d until liver function became normal. Changes of liver volume were determined 4, 6, and 8 wk after embolization, liver CT scanning was performed to measure the volume of the nonembolized lobe. Dogs were killed 8 wk after embolization, pathology observation was implemented of liver tissues of the embolized and nonembolized lobes.

Statistical analysis

SPSS11.0 software package was adopted to carry out statistical analysis of experimental results.

RESULTS

Control group

During the period of embolization, all the five dogs survived. Iconography expression was the same as before, suggesting that no blood vessel was embolized. Meanwhile, there were no changes of portal vein pressure, volume of the right lobe, liver function, and pathology before and after saline injection.

Gelfoam group

One dog died of respiratory failure, another dog died of acute liver failure due to gelfoam back-flow which led to extensive embolization of portal vein branches in liver, and 10 dogs survived. Post embolization angiography showed that the left branch of portal vein was completely embolized, while the right branch was not obstructed, the right portal vein and main portal vein were dilated compared to pre-embolization. Four weeks after embolization the embolized left portal vein had blood flow (Figure 1). The portal vein pressure was 94.1±1.8 mm H_2O, 123.2±2.8 mm H_2O before embolization, and 94.7±2.0 mm H_2O, 95.8±6.6 mm H_2O after embolization, respectively (P>0.05). ALT value obviously increased 2 d after embolization (P<0.01), but decreased subsequently

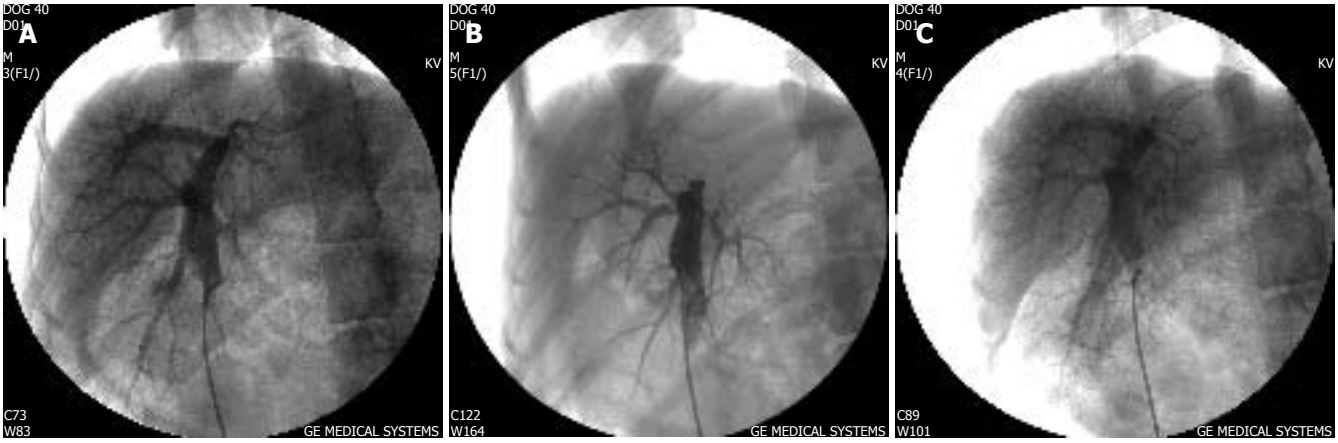

Figure 1 Portal vein angiography before (**A**) and after (**B**) embolization as well as 4 wk (**C**) after embolization with gelfoam.

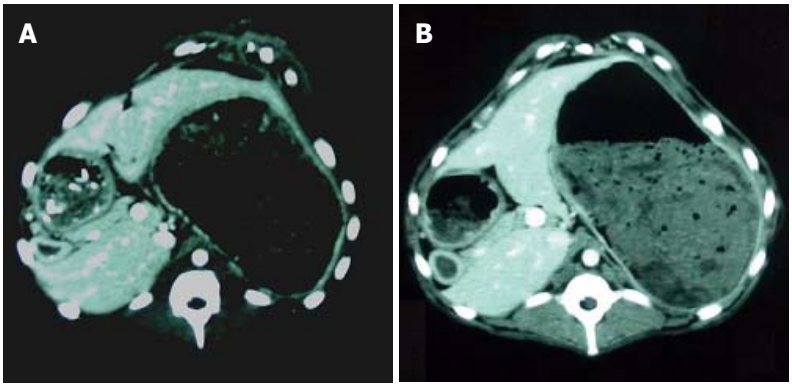

Figure 2 No obvious changes in volume of the right lobe before (**A**) and after (**B**) embolization.

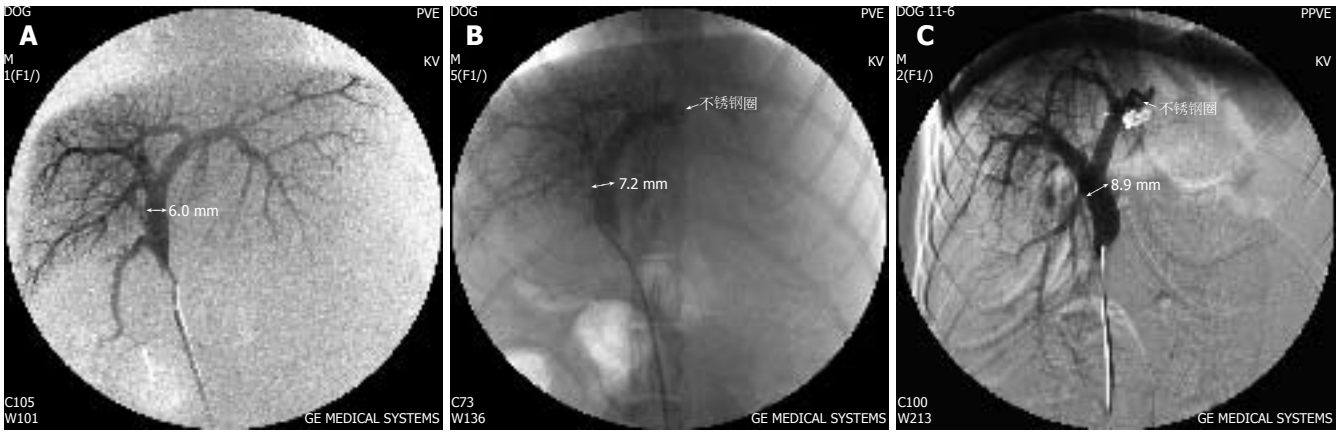

Figure 3 Portal vein angiography before (**A**) and after (**B**) embolization as well as 8 wk (**C**) after embolization with coil-gelfoam.

and then became normal in the following 3 d. No obvious changes were found in the volume of the right lobe before and after embolization (Table 1). Contrast-enhanced CT scanning revealed that the left branch of portal vein was recanalized (Figure 2). Ten dogs were killed 8 wk after embolization, no obvious differences were found between the embolized and nonembolized lobes.

Coil-gelfoam group

One dog died of respiratory failure due to narcosis, the rest had successful surgery (91.7%). After embolization, the left branch was completely embolized while the right branch was patent. Compared to pre-embolization situations, the main portal vein and right portal vein were expanded to a certain extent (Figure 3). The portal vein pressure was 96.5±5.1 mm H_2O, 133.9±10.4 mm H_2O before embolization and 97.0±6.3 mm H_2O, 97.6 ±4.7 mm H_2O after embolization. ALT in experimental dogs increased 2 d after embolization ($P<0.01$), and returned to the pre-embolization level in the following 3 d ($P>0.05$). The right lobes of all the survived dogs after embolization were expanded to varying extent. The liver volume was 344.9+38.3, 374.8+43.0 before embolization and 431.5+50.9, 434.0+50.4 after embolization (Table

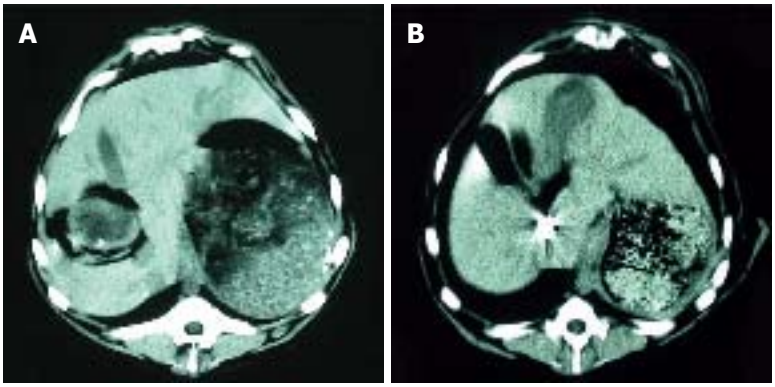

Figure 4 Changes in volume of the right lobe before (**A**) and after (**B**) embolization.

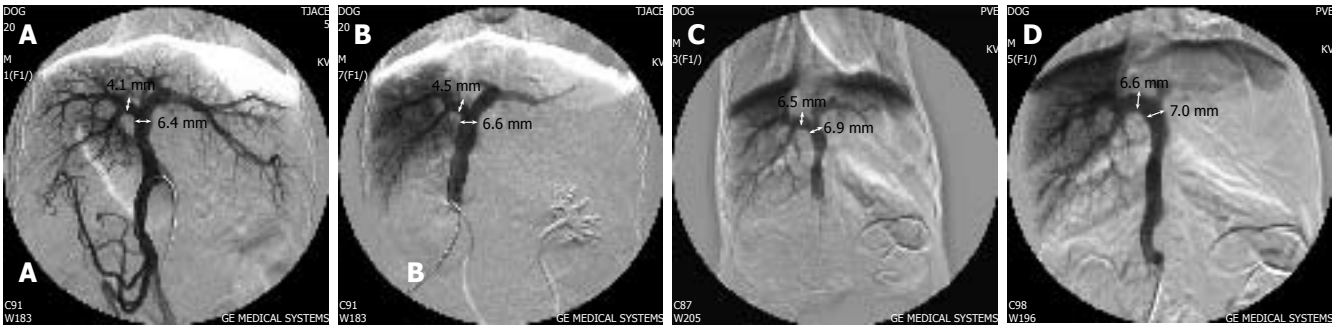

Figure 5 Portal vein angiography before (**A**) and after (**B**) embolization as well as 4 wk (**C**) and 8 wk (**D**) after embolization with absolute ethanol.

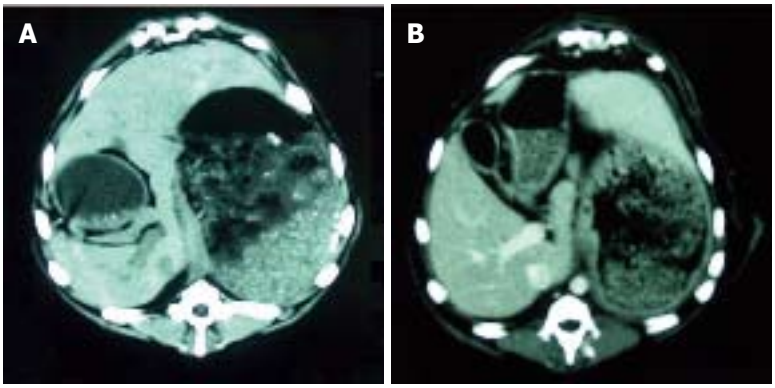

Figure 6 Obvious changes in volume of the right lobe before (**A**) and after (**B**) embolization.

Table 2 Changes of the right lobe volume in coil-gelfoam group (cm^3)

No	Pre-embolization	4W	6W	8W
Dog B1	380	414	474	478
Dog B2	298	325	369	370
Dog B3	376	412	470	472
Dog B4	332	362	417	420
Dog B5	360	392	450	451
Dog B6	294	310	363	365
Dog B7	310	340	379	382
Dog B8	388	426	491	495
Dog B9	300	326	378	385
Dog B10	376	413	473	474
Dog B11	380	403	482	482
Mean±SD	344.9±38.3	374.8±43.0	431.5±50.9	434.0±50.4

Table 3 Changes of the right lobe volume in absolute ethanol group (cm^3)

No.	Pre-embolization	4W	6W	8W
Dog C1	360	392	495	495
Dog C2	272	301	363	363
Dog C3	356	390	492	498
Dog C4	319	350	438	437
Dog C5	329	359	447	449
Dog C6	326	357	430	430
Dog C7	289	293	387	392
Dog C8	328	352	437	439
Dog C9	303	329	412	413
Dog C10	400	409	470	473
Mean±SD	328.2±39.3	353.3±40.3	437.1±45.0	438.9±45.6

2). No further hyperplasia was found (Figure 4). The embolized lobe was dark red with a sharp edge, while the nonembolized lobe was slightly bright red and comparatively plump. Coils were surrounded by fibrin and combined forming the tight emboli. Under microscope, the embolized lobe showed structural changes of hepatic lobules, atrophy and degeneration of liver cells, and congestion of blood vessel, while the nonembolized lobe

revealed cell hypertrophy and obvious hyperplasia.

Absolute ethanol group

One dog died during surgery and 1 dog died in 24 h after surgery due to respiratory failure and acute liver failure caused by the back-flow of absolute ethanol. The rest 10 dogs had successful surgery. The left branch of portal vein was completely embolized, while the right branch was patent. Meanwhile, the main portal vein and right portal vein compared to pre-embolization, were dilated to a certain degree. The left branch of portal vein was occluded without recanalization 8 wk after embolization, while blood flow was seen in the right portal vein and further dilatation was seen in the main portal vein and right portal vein (Figure 5). The portal vein pressure was 95.2±5.2 mm H_2O, 125.2±6.4 mm H_2O before embolization and 96.2± 5.2 mm H_2O, 95.8±6.6 mm H_2O after embolization. ALT was 25.2±1.2, 62.2±3.2, 28.8±1.6, and 26.1±1.3 IU/L after 1, 3, 5, and 7 d of embolization, respectively. ALT values increased by different degrees according to the different injection amount of absolute ethanol, decreased dramatically 3-5 d after embolization and then returned to the pre-embolization level after 1 wk. The volume of right lobe was 328.2±37.1, 353.2±37.9 before embolization and 436.7±42.5, 438.9±45.6 after embolization. No further liver hypertrophy was found (Figure 6). Experimental dogs were killed 8 wk after embolization for pathological observation. The embolized lobe was dark red with a sharp edge, while the nonembolized lobe was slightly bright red and comparatively plump. Under microscope, the embolized lobe showed structural changes of hepatic lobules, liver atrophy, degeneration of liver cells and congestion of fiber tissue, while the nonembolized lobe revealed cell hypertrophy and obvious hyperplasia (Table 3).

DISCUSSION

All our experimental groups with coil-gelfoam and absolute ethanol as embolic agents for left portal vein had right lobe hypertrophy, but the control group and gelfoam group failed to embolize the left portal vein, suggesting that there was no compensatory hypertrophy in the right lobe. The increasing blood flow to the nonembolized portal vein is one of the important factors for liver regeneration. After PVE, dilatation of the nonembolized portal vein branch with obviously increased blood flow could lead to hyperplasia of the nonembolized lobe. These changes were found in our study. Nutritious substances can stimulate regeneration of liver cells. As we know, portal vein blood contains nutritious substances which stimulate regeneration of liver cells. In clinical practice, patients with liver cirrhosis or portal hypertension after shunt operation would suffer from liver atrophy due to decreased blood flow of portal vein. Lindroos *et al*[14] reported that hepatocyte growth factor (HGF) could increase 17 times after two-thirds of rat liver were resected and 13 times after being treated with carbon tetrachloride. Kinoshita *et al*[15] reported that the amount of HGF mRNA in rat liver interstitial cells increased obviously after being treated with carbon tetrachloride, indicating that the increase of HGF can lead to the regeneration of liver cells.

The increase of portal vein pressure and abnormal liver function after PVE, were transient and returned to normal after 4 wk and 1 wk, respectively[16], indicating that transient ischemia of liver portal vein branch has a limited impact on liver function. In our study, five dogs died of accidental extensive embolization of liver portal vein or respiratory failure related to narcosis overdose or absolute ethanol. If PVE is applied in clinical practice, overdose of narcosis can be avoided. The levels of ALT and total bilirubin increased after PVE and returned to normal in 1-2 wk[17,18], demonstrating that the abnormal liver function secondary to PVE is transient and reversible, and does not result in permanent damage to liver function. The portal vein pressure of the three experimental groups was measured at different time points after embolization. We found that the portal vein pressure after embolization was usually higher after embolization, and then returned to normal within 4 wk. Our result is consistent with the study of Baere *et al*[2]. Portal hypertension has not been found in long-term follow-up[19].

All kinds of materials such as absolute ethanol, polydocanol, and gelfoam can be used for portal vein embolization[20-22]. Park *et al*[23] used percutaneous puncture of portal vein to inject Embol into the left branches of pigs' portal vein, and then killed these pigs at different time points to show the safety and effect of Embol. Ko *et al*[24] employed Embol-78 to embolize portal vein of patients with hepatocellular carcinoma or with nonhepatocellular carcinoma to evaluate its effect and safety. Wu *et al*[25] carried out selective embolization experiment of portal vein branches in SD rats with α-cyanoacrylate (DTH) and found compensatory hyperplasia of the nonembolized lobe, suggesting that DTH can effectively induce compensatory hypertrophy of nonembolized lobes. Brown *et al*[26] adopted transcatheter portal vein operation to embolize portal vein branches of liver-cancer patients with polyvinyl alcohol particles (PVA). The above PVA embolization materials have many disadvantages, such as high price, deficient specifications and lack of supply. Gelfoam, coils and absolute ethanol with different characteristics have been used as embolic agents in our clinical practice. Gelfoam can last for a moderate length of time and is safe, non-poisonous and cheap. However, during the process of portal vein embolization, a large amount of gelfoam should be used to completely embolize distal branches of the portal vein, because it easily results in back-flow of gelfoam and accidental embolization of non-target portal vein. We also found that recanalization occurred in gelfoam-embolic portal vein, which failed to effectively induce compensatory hypertrophy of the nonembolized lobe. Therefore, it is not suitable and safe to use gelfoam as an embolic agent for PVE. On the other hand, after being inserted into the target blood vessel, coils lead to mechanical embolization and neointimal hyperplasia, forming permanent embolization. However, coils cannot embolize small blood vessels and easily result in collateral vessels, limiting its use as a PVE embolic agent for inducing hypertrophy of liver. Therefore, combination of gelfoam and coils is a kind of safe and effective mode to achieve complete embolization and to avoid displacement and accidental embolization. In addition, absolute ethanol

can instantly produce permanent embolization in target blood vessels without collateral vessel formation after embolization, thus preventing recanalization of blood vessels and achieving better embolized results. The above results show that absolute ethanol is better than coil-gelfoam for PVE. However, when absolute ethanol is used as an embolic agent, back-flow easily occurs. Once back-flow occurs, it results in serious consequences. It is safe and effective to settle back-flow of absolute ethanol using a balloon catheter[27,28]. In absolute ethanol group, two dogs died of injection of large absolute ethanol doses, while the rest of the dogs which received an injection of small absolute ethanol doses survived. A larger amount of absolute ethanol can achieve good embolic effect, but death rate of experimental animals increases. A smaller amount of absolute ethanol cannot achieve satisfactory results. Thus, further research is needed to determine the safe amount of absolute ethanol injected. Absolute ethanol is a peripheral embolic agent. Compared to coil-gelfoam, it can reach distal branches of portal vein. After embolization, collateral vessels are difficult to form. At the same time, absolute ethanol exerts stronger influence on embolized portal vein branches and even smaller distal branches. Absolute ethanol causes certain damage to neointimal cells inside small branches of hepatic arteries and hepatic cells. The event enhances embolic effect, prevents recanalization of blood vessel, thus stimulating and inducing compensatory hypertrophy of nonembolized lobes more significantly.

In conclusion, gelfoam alone cannot effectively induce compensatory hypertrophy. Coil-gelfoam and absolute ethanol can be used as selective embolic agents to induce compensatory hypertrophy of liver, but absolute ethanol is better than coil-gelfoam for inducing compensatory hypertrophy. However, the safety of absolute ethanol is inferior to that of coil-gelfoam.

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