Radiological aspects of portal vein embolization
van Lienden, K.P.

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Chapter 5

Intrahepatic left to right porto-portal venous collateral vascular formation in patients undergoing right portal vein ligation

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Submitted CVIR
Abstract

Background: We investigated intrahepatic vascular changes in patients undergoing right portal vein ligation (PVL) or portal vein embolization (PVE), in conjunction with the ensuing hypertrophy response and function of the left liver lobe.

Methods: Between December 2008 and October 2011, 7 patients underwent right PVL and 14 patients PVE. CT-volumetry to assess future remnant liver (FRL) and functional hepatobiliary scintigraphy were performed in all patients before and 3 weeks after portal vein occlusion. In 18 patients an intra-operative portography was performed to assess perfusion through the occluded portal branches.

Results: In all patients after initially successful PVL, reperfused portal veins were seen on CT scan three weeks after portal occlusion. This was confirmed in all cases during intra-operative portography. Intra hepatic porto-portal collaterals were identified in all patients in the PVL group and in one patient of the PVE group. In all other PVE patients, complete occlusion of the embolized portal branches was seen on CT scan and on intra-operative portography. The median increase of FRL volume after PVE was 41.6 (range 10-305) %, and after PVL only 8.1(range 0-102)% (p=0.179). There were no differences in FRL-function between both groups.

Conclusions: Preoperative PVE and PVL are both methods to induce hypertrophy of the FRL in anticipation of major liver resection. PVL seems less efficient in inducing hypertrophy of the non-occluded left lobe, compared to PVE. This is most likely caused by the formation of intrahepatic porto-portal neo-collateral vessels, through which the ligated portal branches are reperfused within 3 weeks.
Introduction

Beside portal vein embolization (PVE), the most common method to enlarge the future remnant liver (FRL) before extensive liver resection, is portal vein ligation (PVL). Both techniques are extensively described in literature. PVE is a minimally invasive procedure, in which the right portal venous system can be occluded by ipsilateral or contralateral percutaneous puncture under local anesthesia. Different embolization materials have been described, of which the majority of agents causes permanent occlusion.\(^1\),\(^2\),\(^3\) Complications consist of hematoma, septic complications and dislocation of embolization material in portal branches of liver segments to be preserved.\(^4\) PVL requires a surgical approach under general anesthesia and is mostly performed, as part of a two-stage procedure in resection of bilobar colorectal metastases in which typically during the first stage, the primary tumor is resected along with the lesser liver resection on the left side, usually a left-lateral resection of segments 2 and 3. At the same time, the right portal vein is ligated to induce hypertrophy of segment 4. Following 3-6 weeks, after sufficient volume increase has been determined, a completion right hemihepatectomy is performed in the second stage.\(^5\) The mechanism underlying both methods is occlusion of part of the portal venous system, to induce a hypertrophy response of the non-occluded liver segments.

There is however much discussion regarding the efficiency of both techniques. With PVE, the entire portal tree of the embolized side is permanently occluded, whereas occlusion caused by ligation of the portal vein is presumably short-lived, because of retrograde filling of the ligated portal branches through collateral supply from adjacent non-ligated segments. So far, it is unclear whether arterio-portal or porto-portal collaterals are responsible for this process of revascularization. Krupski et al described in a pig model, an uniform pattern of collaterals with subsequent complete recanalization of the formerly occluded portal vein, distal from the ligation.\(^6\) These collaterals had a cavernous aspect and reduced the portal blood flow.

To our knowledge, porto-portal collaterals were described in humans after portal vein ligation only by Denys et al.\(^7\) These collaterals were described as intrahepatic porto-portal collaterals arising from segment 4 and connecting with the portal venous branches of the adjacent segment 8 within the ligated portal system.

In this study we investigate the intrahepatic vascular changes in 7 patients after PVL along with the differences in hypertrophy response and function compared with 14 patients who underwent PVE in the same period.

Materials and methods

Between December 2008 and October 2011, 20 patients underwent PVE or PVL prior to extensive liver resection. One patient who initially showed no hypertrophy response after PVL, underwent PVE in second intention. In the majority of these patients (\(n=18\)), a control portography was performed intra-operatively at the time of resection, to evaluate blood
flow in the embolized or ligated portal branches. In the remaining two patients, the pre-operative CT scan performed to evaluate the volume of the FRL, was also used to evaluate liver vascularisation following PVE or PVL.

Patient characteristics, indications for PVE or PVL, volumetric changes, hypertrophy response, complications after PVE and PVL, and vascularisation of the embolized liver segments were evaluated.

PVE
All procedures in 14 patients were performed using the percutaneous ipsilateral approach as described by Madoff. After ultrasound-guided puncture of an anterior branch of the right portal vein, a 5 French sheath was inserted. Following portography, all right branches of the portal vein were selectively catheterized using a reverse curved catheter, and embolized with PVA particles (300-500 μg, Cook Incorporated, Bloomington, United States of America) and multiple 6 to 12 mm coils (Tornado Embolization Coils, Cook Incorporated, Bloomington, USA). The procedure was completed with a control portogram to confirm total occlusion of the right portal system and normal flow through the left future remnant portal system. Finally, the puncture tract was closed with a gelfoam plug (Spongostan Standard, Ferrosan A/S, Soeborg, Denmark).

PVL
In all 7 patients who had undergone PVL, PVL was electively performed as part of a multi-stage treatment plan in which right PVL was undertaken during initial resection of the primary tumor (n=2); during resection of metastasis in the left liver lobe in anticipation of subsequent right hemihepatectomy (n=2); during explorative laparotomy (n=2) and during an originally planned right hemihepatectomy in which the FRL appeared smaller and more cirrhotic than expected (n=1). In this patient, liver resection was performed 5 weeks after right PVL.

In all patients the portal bifurcation was dissected during intra-operative exploration and the origin of the right portal main branch was suture ligated and divided. In one patient, the portal branches arising from the left portal vein to segment 4 were also dissected and ligated.

Definition of technical and clinical success
After portal vein occlusion, technical success was defined as complete occlusion of the target branches at the end of the procedure. Clinical success was defined as adequate hypertrophy response of the FRL after portal occlusion, allowing liver resection.

Measurement of liver volume
A multiphase computed tomography (Mx 8000 or Brilliance, Philips, Eindhoven, the Netherlands) with intravenous injection of contrast medium (Ultradist-300, Bayer Schering Pharma, Bayer BV, Mijdrecht, the Netherlands) in the portal phase was performed in all
patients before PVE or PVL, and approximately three weeks after PVE or PVL to calculate the maximum hypertrophy response after occlusion of the portal vein. CT-data were processed and evaluated on an MxView – Independent Multi-Modality Diagnostic Workstation (Version 3.52 B2, August 2002, Philips Medical Systems, Eindhoven, The Netherlands). The segmental anatomy of the left and right liver segments, as well as the tumor, were manually delineated according to the Couinaud classification after which total liver volume (TLV), FRL-volume (FRLV) and tumour volume (TV) were calculated. The percentage FRL was calculated before and three weeks after PVE and PVL using the following equation:

\[
\%FRL = \frac{FRLV}{TLV - TV} \times 100\%
\]

Liver hypertrophy after PVE was defined as:

\[
1 - \frac{FRL_{prePVE}}{FRL_{postPVE}} \times 100\%
\]

Measurement of liver function with hepatobiliairy scintigraphy

Hepatobiliary scintigraphy (HBS) was performed with $^{99m}$Tc-labeled 2,4,6 trimethyl-3-bromo aminodiacetic acid ($^{99m}$Tc-mebrofenin [Bridatec]; GE Healthcare) including SPECT to evaluate liver function, and to calculate function of the FRL, as described previously. Images are obtained with a large-field-of-view (FOV) SPECT/CT camera (Infinia II; GE Healthcare) equipped with low-energy high-resolution collimators. Firstly, a dynamic acquisition, immediately after the intravenous administration of 200 MBq of $^{99m}$Tc-mebrofenin, was obtained for calculation of the hepatic uptake function. Subsequently, a fast SPECT acquisition was performed centred on the peak of the hepatic time–activity curve, which was used for the 3-dimensional assessment of liver function, and calculation of functional liver volume. Immediately following SPECT, a low-dose non-contrast-enhanced CT scan was obtained for attenuation correction and anatomic mapping on the same gantry, without moving the patient. Finally, a second dynamic acquisition was obtained to evaluate biliary excretion. Data were processed on a workstation (MultiModality; Hermes Medical Solutions). The scintigraphy was performed approximately 14 days before and three weeks after PVE or PVL.

Intra-operative portography

After laparotomy, the hepatoduodenal ligament was dissected and the main portal vein was identified. A 18G venflon needle was inserted into the portal vein main branch after a purse-string suture (5-0 Prolene) allowing advancement of the catheter in distal direction. Subtraction portographies were performed in several directions after manual injection of 10 ml of contrast material (Ultravist-300, Bayer Schering Pharma, Bayer BV, Mijdrecht, the Netherlands). The degree of occlusion of the ligated or embolized portal branches and the presence of collateral flow was evaluated.
Statistical analysis

Statistical analysis was performed with Statistical Package for Social Sciences (SPSS 18.0). CT volumetry data were compared using a mixed model analysis based on ranked data. The independent sampled T-test was used for continuous data. Non-parametric data were compared by the Wilcoxon Mann-Whitney U test. All statistical differences were considered significant at a p-value of $\leq 0.05$. Data were expressed as means ± SD, unless otherwise stated.

Results

Patients

In 20 patients (12 male and 8 female, mean age 59.6 ± 10.2 years), 21 procedures have been performed to occlude the right portal vein. Seven patients initially underwent a PVL. In one patient, ligation of the right portal vein caused no hypertrophy response at all. The CT-scan, performed 4 weeks after ligation, showed a completely patent right portal vein, despite adequate ligation, without any increase of the FRL volume. In this patient, additional PVE was performed 6 weeks after PVL. In the other 13 patients, PVE was primarily performed. There were no significant differences in patient characteristics. Also, the number of patients receiving chemotherapy or radiotherapy prior to the procedure, or the occurrence of compromised livers, was not significantly different between both groups. (Table 1)

Indications for liver resection consisted of colorectal liver metastasis (CRLM, n=17), cholangiocarcinoma (CC, n=2) or hepatocellular carcinoma (HCC, n=1).

<table>
<thead>
<tr>
<th>Table 1: Patient characteristics</th>
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<tr>
<td></td>
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<tr>
<td>Age</td>
</tr>
<tr>
<td>Male/ Female</td>
</tr>
<tr>
<td>BMI (19.9-32.0)</td>
</tr>
<tr>
<td>Liver cirrhosis (36%)</td>
</tr>
<tr>
<td>Cholestasis (14%) (0%)</td>
</tr>
<tr>
<td>Radiotherapy (29%) (14%)</td>
</tr>
<tr>
<td>Chemotherapy (64%) (71%)</td>
</tr>
</tbody>
</table>

Success rate portal vein occlusion

All PVL and PVE procedures were technically successful. Except for mild abdominal discomfort in some cases (n=4), no other complications were seen in the PVE group. The ligation procedures were also conducted without complications. All PVE procedures were clinically successful showing adequate hypertrophy response of the FRL, three weeks after PVE. In one patient of the PVL group, no hypertrophy response was seen at all on CT scan 4 weeks after PVL, resulting in a clinical success rate of 86% in the PVL group. Six weeks after
PVL, an additional PVE was performed inducing enough hypertrophy response to make liver resection possible. (Figure 1)

CT scan and liver volumetry

CT scans were performed with an average of 25 (range 1-79) days before PVE and 35 (range 25-41) days prior to PVL. Control CT scans were performed with an average of 22 (range 18-40) days after PVE and 22 (range 20-38) days after PVL. There were no significant differences between the groups. Liver volumes are listed in Table 2. There were no significant differences in base-line liver volumes and percentage FRL between the PVE and PVL groups.

In the PVE group, portal occlusion led to a significant increase of the FRL from 25.8±7.5% to 37±6.4% (p=0.001). The median percentage of increase of the FRL was 41.6 (range 10.4-304.6) %.

In the PVL group, the FRL significantly increased from 27.2±7.0% to 36.1±5.0% (p=0.016). The median percentage of increase of the FRL after PVL was 8.1 (range 0-102) %. Although the percentage of increase of the FRL after PVL is obviously less than after PVE, this is statistically not significant (p=0.179).

Evaluation of the vascular structures after PVE demonstrated complete occlusion of the embolized right portal branches in all cases but one, where flow was seen in a segment 7 branch of the right portal system. Complete patency was seen of the hepatic artery, hepatic veins and left portal vein branches. Evaluation of portal occlusion on CT-scan was however, difficult since severe artefacts caused by coils in the proximal segmental branches of the right portal vein hampered definition of vascular structures.

Although all PVL procedures were technically successful, the right portal branches were patent in 100% of patients on CT scan three weeks after PVL. The site of central ligation

![Figure 1](image)
of the right portal system could be identified in all cases. In one patient, CT scan revealed ligation of the right anterior portal branches only, although ligation of the complete right portal system was planned. This ligated sectorial right portal branch also showed retrograde flow distal to the ligature. In the latter case, partial right portal vein ligation however caused sufficient hypertrophy of the left liver lobe to allow liver resection. Intrahepatic collateral formation from the left to the right hemi liver could be identified on CT scan in 5/7 (71%) of patients after PVL.

No extrahepatic collaterals were seen.

Table 2. Liver volumes before and after PVE / PVL

<table>
<thead>
<tr>
<th>Liver volumes</th>
<th>PVE</th>
<th>PVL</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Pre-intervention:</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total liver volume (ml)</td>
<td>1588 (1054-2587)</td>
<td>2065 (1122-3495)</td>
<td>0.218</td>
</tr>
<tr>
<td>Tumor volume (ml)</td>
<td>40 (0.7-225)</td>
<td>124 (4.8-695)</td>
<td>0.146</td>
</tr>
<tr>
<td>FRL volume (ml)</td>
<td>399 (294-517)</td>
<td>467 (303-851)</td>
<td>0.146</td>
</tr>
<tr>
<td>% FRL</td>
<td>25.8 ± 7.5</td>
<td>27.2 ± 7.0</td>
<td>0.685</td>
</tr>
<tr>
<td><strong>Post-intervention:</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total liver volume (ml)</td>
<td>1586 (1132-3610)</td>
<td>1881 (1277-3021)</td>
<td>0.314</td>
</tr>
<tr>
<td>Tumor volume (ml)</td>
<td>30.1 (1.390)</td>
<td>157 (9-788)</td>
<td>0.146</td>
</tr>
<tr>
<td>FRL volume (ml)</td>
<td>589 (368-1412)</td>
<td>542 (436-945)</td>
<td>0.576</td>
</tr>
<tr>
<td>% FRL</td>
<td>37.0 ± 6.4</td>
<td>36.1 ± 5.0</td>
<td>0.792</td>
</tr>
<tr>
<td>Increase (%)</td>
<td>41.6 (10.4-304.6)</td>
<td>8.1 (0-102)</td>
<td>0.076</td>
</tr>
</tbody>
</table>

Hepatobiliary scintigraphy and functional liver volumetry

In the PVE-group, HBS was performed 5 (range 1-21) days before, and 22 (range 18-40) days after PVE. In the PVL group HBS was performed 3 (range 1-41) days before, and 23 (range 20-35) days after ligation. There were no significant differences between the groups.

Before intervention, total uptake of the complete liver was 15.8 (range 5.7-25.0) %/min in the PVE group and 17.2 (range 11.6-19.6) in the PVL group. The percentage functional-FRL was 24.8 ± 4.5 % in the PVE group and 27.0±1.9 in the PVL group. The estimated FRL-function before intervention was 3.6(1.4-7.0) %/min/m² in the PVE group and 4.9(3.0-5.7) %/min/m² in the PVL group. After portal vein occlusion, total uptake of the complete liver was 13.7 (8.9-18.3) %/min in the PVE group and 17.6 (9.4-22.6) %/min in the PVL group. The percentage functional-FRL increased from 24.8 ± 4.5 to 35.7 ± 9.9% in the PVE group and from 27.0 ± 1.9 to 38.8 ± 7.6 % in the PVL group. The estimated FRL-function after intervention increased to 4.5 (3.4-7.3) %/min/m² in the PVE group and 5.6 (4.2-9.0) %/min/m² after PVL. The increase in functional volume of the FRL was 44.0% after PVE and 43.7% after PVL (p=0.003).

Data are listed in table 3. No significant differences between the groups were found.
Portography before resection

Of all patients who underwent PVE, one patient was found unresectable because of tumor progression as evidenced on the post-PVE CT-scan. Therefore no portography was performed. Complete occlusion of the right portal system was however observed in this patient on CT-scan performed 3 weeks after PVE.

In the other 13 patients, complete occlusion of the total right portal system after PVE was seen in 11 (84.6%) patients on portography performed during laparotomy before liver resection. In one patient, normal hepatopetal flow was seen in the portal branch of segment 7, which was missed during the initial portal vein embolization procedure. In the second patient with incomplete occlusion, a right segment 8 portal branch was patent and filled with contrast through left to right collateral flow from segment 4 to segment 8, although the portal branch was centrally, adequately occluded with coils. (Figure 2) Both patients had a normal hypertrophy response.

In the patient who underwent PVE in addition to PVL, no intra-operative portography was performed. In all other patients who primarily underwent PVL, normal flow was seen through the left portal venous system. Although adequate right central occlusion was demonstrated, complete or partial patency of the ligated right portal system was seen in all patients after PVL. All portograms confirmed the re-established, right portal venous perfusion as demonstrated on the pre-resection CT-scans. Porto-portal collateral flow was seen from segment 4 to segment 5 or 8 (Figure 3). During resection, no extrahepatic collaterals were found.

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### Table 3. Outcome HIDA scan pre- and post-occlusion of the portal vein

<table>
<thead>
<tr>
<th>Liver-uptake</th>
<th>PVE</th>
<th>PVL</th>
<th>P-value</th>
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<tbody>
<tr>
<td><strong>Pre-intervention:</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total liver uptake (%/min)</td>
<td>15.8 (5.7-25.0)</td>
<td>17.2 (11.6-19.6)</td>
<td>0.741</td>
</tr>
<tr>
<td>% FRL</td>
<td>24.8 ± 4.5</td>
<td>27.0 ± 1.9</td>
<td>0.193</td>
</tr>
<tr>
<td>Estimated FRL function (%/min/m²)</td>
<td>3.6 (1.4-7.0)</td>
<td>4.9 (3.0-5.7)</td>
<td>0.229</td>
</tr>
<tr>
<td><strong>Post-intervention:</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total liver uptake(%/min)</td>
<td>13.7 (8.9-18.3)</td>
<td>17.6 (9.4-22.6)</td>
<td>0.122</td>
</tr>
<tr>
<td>% FRL</td>
<td>35.7 ± 9.9</td>
<td>38.8 ± 7.6</td>
<td>0.482</td>
</tr>
<tr>
<td>Estimate FRL function (%/min/m²)</td>
<td>4.5 (3.4-7.3)</td>
<td>5.6 (4.2-9.0)</td>
<td>0.160</td>
</tr>
<tr>
<td>Increase functional FRL (%)</td>
<td>44.0</td>
<td>43.7</td>
<td>0.976</td>
</tr>
</tbody>
</table>
Discussion

In literature, different techniques for portal vein occlusion are described. Right portal vein ligation can be performed during staging laparotomy or during a two-stage procedure in which local tumor resection on the left side is combined with right portal vein portal ligation prior to right hemihepatectomy. This procedure was already described in 1965. Alternatively, PVE can be performed during laparotomy using an ileocolic venous approach although this approach is less popular among surgeons.

Since the introduction of percutaneous PVE, the need for invasive techniques as PVL is strongly reduced. In our institution, PVL procedures are now only performed in selected cases.
as in two-stage resection for bilobar tumors. PVE by the percutaneous transhepatic approach is an effective method to induce hypertrophy of the FRL, with only minor complications.4

The exact mechanism behind the hypertrophy response is not exactly known, but for many years it has been believed that redirecting the flow through partial portal vein occlusion caused hypertrophy of the un-affected liver lobe. Goto et al described in 1990 that the hypertrophy rate of the non-embolized hepatic segments after embolization, was predictable from the extent of increase in portal blood flow in the non-embolized liver.11 This is in line with the theory that hypertrophy of the non-occluded lobe is influenced by increased supply of hepatotropic substances as growth factors, nutrients and gastrointestinal hormones.12 There is also evidence from experimental models that indicate the hemodynamic influences

Figure 3. Control portogram during laparotomy, performed before liver resection, demonstrates complete reperfusion of the ligated right portal venous system through collateral flow from segment 4 to segment 8 (white arrows).
after portal vein ligation, to be the crucial factor in regulating volume of the un-affected liver after resection.\textsuperscript{13,14}

Currently the theory is exposed, that the hypertrophy response is regulated by the amount of parenchymal loss. Liver mediating cytokines, including TNF-\textalpha, interleukin IL-1\beta and IL-6, are released from Kupffer cells and activate hepatocytes to proliferate.\textsuperscript{15} Furrer et al\textsuperscript{15} showed in a rat-model that hepatocyte proliferation after PVL was more pronounced than after PVE, suggesting that PVL is the more effective technique. Wilms et al\textsuperscript{12} however, studied PVL and PVE in a pig model, and concluded that PVE was more effective in increasing the future liver remnant, owing to more durable occlusion of the portal venous branches. This was also demonstrated by our group (vd Esschert et al\textsuperscript{16}) in a rabbit PVE-model, in which beside a greater increase of the FRL volume after PVE, also a greater amount of proliferating hepatocytes was demonstrated, without any difference in cytokine levels between PVE and PVL.

Human studies comparing the hypertrophy response after PVE and PVL, are also ambiguous. Aussilhou et al\textsuperscript{17} reported no significant difference in hypertrophy response between PVE and PVL. However in this study, PVL was undertaken in the setting of a two-stage liver resection. The combination of PVL with partial liver resection introduces an advantage because post-resectional liver regeneration is probably augmenting post-PVL regeneration. Broering et al\textsuperscript{18} on the other hand, reported a significantly better result in hypertrophy response after PVE compared to PVL. Also, the time between portal occlusion and operation, to achieve the necessary hypertrophic reaction was significantly longer in patients after PVL. In this study however, there was a large proportion of PVE procedures in which additional segment 4 branches were embolized. This may cause an overrating of the hypertrophy results in comparison with PVL in which only the right portal branches are ligated.

None of the PVE patients in our study, underwent additional embolization of segment 4. In one patient of the PVL group, segment 4 branches arising from the left portal vein were selectively ligated. There were obviously substantial differences in hypertrophy response of the FRL in the PVL and PVE groups, however these were not statistically significant, as may be explained by the small number of patients in the PVL group. The percentage functional FRL was significantly increased after portal vein occlusion whereas no significant differences were seen between the PVE-group and PVL-group. The reason for this may be that increase in function evolves well ahead of increase in volume after portal vein occlusion, as was previously demonstrated by de Graaf et al\textsuperscript{8}

In literature, the difference in hypertrophy response between PVE and PVL is in part attributed to the formation of collaterals after PVL. The first to describe these collaterals in humans after PVL, were Denys et al\textsuperscript{7} in 1999. In their case report, intrahepatic porto-portal collaterals from segment 4 branches were shown to drain into segment 8 branches, causing complete reperfusion of the ligated right system. Hypertrophy response of the FRL, was only achieved after embolization of these collaterals.

Few animal studies have been published on this topic. Wilms et al\textsuperscript{12} in a pig model of PVE reported revascularization of the ligated portal branches in all cases, by intrahepatic arterio-
portal collaterals coming from adjacent non-occluded liver segments. This was confirmed by Ferko et al\textsuperscript{19} who reported patent intrahepatic portal branches after truncal portal vein occlusion caused by rapidly developed hepatopetal collaterals in mini-pigs.

Our study provides the first evidence of collateral flow and reperfusion of the ligated portal venous system in a group of patients after PVL. As soon as 3 weeks after PVL, complete patency of the ligated portal branches was seen on all CT-scans. This was also confirmed by the portograms performed intra-operatively, showing intrahepatic collateral connections between the branches of segment 4 and the branches of the adjacent right segments 5 and 8. No extrahepatic collaterals were detected. This phenomenon could very well explain the difference in hypertrophy response between PVE and PVL. This theory was particularly demonstrated in one patient who showed no hypertrophy response after PVL but finally showed increase of the FRL volume after complete secondary percutaneous embolization with PVA particles distal of the central ligation.

The lack of statistical significance does not support the conclusion of this study as probably caused by the relatively small sample sizes. Because PVE has become the method of choice in inducing hypertrophy of FRL before extensive liver resection, the number of PVL-procedures has become limited. We demonstrated neo-collateral vessel formation after PVL and a trend of restricted hypertrophy corroborating the notion of PVE being superior to PVL.

In summary, we conclude that PVE and PVL are both useful methods to induce hypertrophy of the FRL before major liver resection. PVL seems less efficient in inducing hypertrophy, compared to PVE. This is most likely caused by the formation of intrahepatic porto-portal neo-collateral vessels, through which the ligated portal branches are reperfused in spite of an appropriate ligation procedure.
References


