Direct puncture approach for embolization of visceral aneurysms.

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Aims and objectives

Introduction

Aneurysms along the visceral arteries can be split into 2 categories; visceral artery aneurysms (VAA) or "real" aneurysms and visceral artery pseudo-aneurysms (VAPA) or "false" aneurysms. The VAA are mainly caused by systemic disease such as atherosclerosis or collagen disorder. The VAPA are secondary to vessel wall disruption or erosion from (iatrogenic) trauma, tumor invasion, inflammation or infection. Both VAA and VAPA, but especially the latter, are at risk of rupture and potential life-threatening hemorrhage, and therefore need treatment.

Compared to surgery, transarterial embolization of aneurysms is considered the more elegant and less invasive method, but it is not always technically feasible. This may be due to either difficulties in visualizing the anatomy of the arterial feeder or due to mechanical inability to maneuver the catheter to the desired location in the feeder artery.

An alternative method can be a direct percutaneous puncture approach. Direct puncture treatment with thrombin injection under ultrasound guidance has become a main stream practice for coping with femoral artery access site pseudoaneurysms. In the Pubmed library, multiple case reports of direct puncture approach have been described for treatment of various pseudoaneurysms including visceral aneurysms. However, no single centre case series have been described on direct puncture treatment of visceral aneurysms.

Aims and objectives

The purpose of this study was to retrospectively evaluate safety, effectiveness and methods for direct percutaneous (non-endovascular) embolization as a treatment strategy for visceral aneurysms.
**Methods and materials**

*Patient selection.*

This is a retrospective analysis of visceral aneurysms treated by direct puncture (non-endovascular), as found in our searchable department database of interventional radiology procedures (2000-2014). We included aneurysms of the hepatic, pancreatic, splenic and mesenteric circulation, disregarding their etiology.

*Clinical information collection.*

Patient demographics, aneurysm properties, interventional procedure details, used materials and follow-up information were obtained from our department interventional database, the official PACS procedure reports and the hospital electronic patient documentation system. The peri-procedural images were reviewed on the PACS system.

*Procedural methods.*

Procedures were performed under angiographical, fluoroscopical, ultrasound and/or Cone-Beam CT-control. Direct puncture of a VAA or VAPA was achieved either under ultrasound guidance, or under biplanar radiofluoroscopy aiming the needle at the aneurysm that is simultaneously opacified through a selectively placed arterial catheter (Fig.1).

Direct puncture was done with either a sheathed needle 19,5G (Bard) or 21G needle (Accustick, Boston). In some cases a microcatheter (Progreat 2.7 microcatheter, Terumo) was inserted through the sheathed needle. Embolization was performed with (micro)coils (various coils, Cook Medical), glue (Glubran 2) or Tisseel (human thrombin co-injected with human fibrogen).

*Literature collection.*

The Pubmed database was searched for visceral aneurysm articles as well as direct puncture approach related articles.
Images for this section:

Figure 1. Direct puncture as performed in a biplanar angio suite. Black squares and trapzoids are the fluoroscopy panels. Contrast is injected via the microcatheter (bottom left), showing the aneurysm (centre, red). Then puncture directed at the opacified aneurysm, follows the green arrow direction and the fluoroscopy imaging direction of the coronal panels. The evaluation of the puncture needle depth is done via the lateral fluoroscopy panels. When the aneurysm is reached, a test contrast injection is performed directly through the puncture needle. Then embolization can be commenced.

**Fig. 1**

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Results

Table 1 (Figure 2) shows the patient demographics and specifics of the included patients. Patients were 1 women en 10 men, mean age 48 years, median age 46 years, range 33-72 years.

We found N=11 visceral aneurysms treated by direct puncture embolization: hepatic N=3; gallbladder = 1; splenic N=3; pancreatico-duodenal branches N=4. 10 were pseudo-aneurysms, 1 was a real splenic artery aneurysm. The mean diameter was 18.6mm (median 17mm, range 7-30mm). Etiology was either pancreatitis N=4, unknown N=3, post-surgery N=1, post-PTC N=1, trauma N=1, Ehlers-Danlos N=1. Clinical evidence of active bleeding was observed in 6 out of 11 patients. One hepatic artery pseudo-aneurysm had developed a biliary fistula. In another case the pseudoaneurysm compressed a bile duct branch, requiring biliary stenting a few days later.

Table 2 (Figure 3) shows the procedure specifics for each case. The choice for treatment by direct puncture was because either: the lesion could not be reached by transarterial catheterization N=6; or anatomical and technical problems N=2; or the feeding artery could not be identified N=2; or operator preferred to avoid endovascular approach N=1. In one case the direct puncture was performed through the stomach. Only N=4 procedures were performed under antibiotic protection, 3 of these were pancreatitis patients.

The embolic agents used were: glue N=7 ; coils N=3 ; thrombin N=1.

In N=5 cases a microcatheter was inserted through the sheathed needle and embolization done with (micro) coils in N=3 and with glue in N=2. Injection of glue consisted of mixtures with Lipiodol varying in ratios of Glubran:Lipiodol from 1:1 to 1:4.

All aneurysms were successfully treated. In N=1 case, the largest pseudoaneurysm, there was a recurrence that was diagnosed and treated 2 days later by endovascular coiling of the main hepatic artery. In N=5 cases minor technical complications were spill of glue into the feeding artery and from thereon into the periphery. In all cases without clinical symptoms during the procedure or during follow-up. No other complications were retrospectively found.

Follow-up imaging was performed in 9 of 11 patients, in timespans ranging from 3 days to 1,5 years. Figures 4 and 5 are examples of direct puncture embolizations with glue and coils respectively.
Patient 8, example of a glue embolization. (a) Hilar pseudo-aneurysm diagnosed on CT. (b) AP Angio showing coeliac trunk occlusion and tortuous collateral gastro-duodenal artery with cystic artery pseudo aneurysm (c) Tortuous trajectory catheterized (d) Direct puncture with opacification of the aneurysm (e) Control blanc CT showing puncture trajectory and embolized pseudoaneurysm.

Fig. 4

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Patient 1, example of coil embolization. (a) CT shows pseudoaneurysm at middle of pancreas corpus. (b) AP Angio from mesenteric artery with aneurysm at pancreatrico-dorsal branch. (c) Detail. (d) Transcatheter aneurysm opacification with scalpel projection during localization of the puncture site. (e) Angiographic control during coil placement. Horizontal faint shadow of the silicone sheath of the sheathed needle. (f) Control CT showing successful embolization.

Fig. 5

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<table>
<thead>
<tr>
<th>Indication direct puncture</th>
<th>Needle type</th>
<th>Microcatheter</th>
<th>Material</th>
<th>Material specifics</th>
<th>Technical complications</th>
<th>Initial Result</th>
<th>Follow-up</th>
</tr>
</thead>
<tbody>
<tr>
<td>1  tortuous feeder</td>
<td>Sheated needle 19,5G</td>
<td>yes</td>
<td>coils</td>
<td>10 microcoils</td>
<td>none</td>
<td>occlusion</td>
<td>After 1,5 years occlusion on CT</td>
</tr>
<tr>
<td>2  tortuous feeder</td>
<td>Sheated needle 19,5G</td>
<td>no</td>
<td>glue</td>
<td>Glubran/Lipiodol 1:3, 1,5ml</td>
<td>Glue spill</td>
<td>occlusion</td>
<td>During 5 days observation stable</td>
</tr>
<tr>
<td>3  feeding artery not identifiable</td>
<td>Sheated needle 19,5G</td>
<td>yes</td>
<td>coils</td>
<td>1 coil en 5 microcoils</td>
<td>none</td>
<td>occlusion</td>
<td>After 5 days and 5 months occlusion on CT</td>
</tr>
<tr>
<td>4  Primary non endovascular Eiters-Danlos</td>
<td>Sheated needle 19,5G</td>
<td>no</td>
<td>glue</td>
<td>Glubran/Lipiodol 1:2</td>
<td>Glue spill</td>
<td>occlusion</td>
<td>After 9 days occlusion on CT</td>
</tr>
<tr>
<td>5  anatomical and technical problems</td>
<td>Sheated needle 19,5G</td>
<td>yes</td>
<td>coils</td>
<td>16 microcoils, various types</td>
<td>none</td>
<td>occlusion</td>
<td>After 1 day recanalisation between coils, portal flow normal to endovascular hepatic artery embolisation</td>
</tr>
<tr>
<td>6  anatomical and technical problems</td>
<td>Sheated needle 19,5G</td>
<td>yes</td>
<td>glue</td>
<td>Glubran/Lipiodol 1:3, 1ml</td>
<td>Glue spill, minimal</td>
<td>occlusion</td>
<td>After 3 months occlusion on CT</td>
</tr>
<tr>
<td>7  tortuous feeder</td>
<td>21 G needle</td>
<td>no</td>
<td>thrombin</td>
<td>0,6 ml</td>
<td>none</td>
<td>occlusion</td>
<td>After 3 weeks occlusion on CT</td>
</tr>
<tr>
<td>8  tortuous feeder</td>
<td>Sheated needle 19,5G</td>
<td>no</td>
<td>glue</td>
<td>Glubran/Lipiodol, amount unknown</td>
<td>Glue spill, partial occlusion right hepatic artery, sufficient collateral flow</td>
<td>occlusion</td>
<td>After 5 days and after 3 months occluded on CT</td>
</tr>
<tr>
<td>9  feeding artery not identifiable</td>
<td>21 G needle</td>
<td>no</td>
<td>glue</td>
<td>Glubran/Lipiodol 1:2, 2ml</td>
<td>Glue spill minimal</td>
<td>occlusion</td>
<td>After 3 and 9 months occluded on ultrasound and CT</td>
</tr>
<tr>
<td>10 tortuous feeder</td>
<td>Sheated needle 19,5G</td>
<td>yes</td>
<td>glue</td>
<td>Glubran/Lipiodol 1:4, 0,5ml</td>
<td>none</td>
<td>occlusion</td>
<td>During 5 days observation stable and no complaints</td>
</tr>
<tr>
<td>11 tortuous feeder</td>
<td>Sheated needle 19,5G</td>
<td>no</td>
<td>glue</td>
<td>Glubran/Lipiodol 1:1, 8ml</td>
<td>Glue spill minimal</td>
<td>occlusion</td>
<td>After 3 days on ultrasound occlusion, during 20 days hospital stay stable</td>
</tr>
</tbody>
</table>
Fig. 3

<table>
<thead>
<tr>
<th>Age</th>
<th>M/F</th>
<th>Artery</th>
<th>Pseudo/re</th>
<th>bleeding</th>
<th>Size (mm)</th>
<th>Etiology</th>
<th>Clinics</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>M</td>
<td>Pancreatico-dorsal</td>
<td>pseudo</td>
<td>no</td>
<td>15</td>
<td>alcoholic pancreatitis history</td>
<td>MRI follow-up imaging showed pseudoaneurysm, confirmed on angio, no symptoms</td>
</tr>
<tr>
<td>2</td>
<td>M</td>
<td>Splenic</td>
<td>pseudo</td>
<td>no</td>
<td>16</td>
<td>pancreatitis</td>
<td>pancreatitis pseudocystic bleeding treated surgically, followup CT showed small pseudo-aneur</td>
</tr>
<tr>
<td>3</td>
<td>M</td>
<td>Gastro-duodenal</td>
<td>pseudo</td>
<td>yes</td>
<td>15</td>
<td>complicated pancreatitis history</td>
<td>blood loss along abdominal drain, CT reveals arterial leak a from splenic artery and pseudoaneur gastro-duodenal artery</td>
</tr>
<tr>
<td>4</td>
<td>F</td>
<td>Splenic</td>
<td>real</td>
<td>yes</td>
<td>17</td>
<td>Ehlers-Danlos type 4</td>
<td>2 days abdominal pain, hemoglobin decreased, CT shows hematoma alcoholic, 1 week of vague abdominal pain, anorexia</td>
</tr>
<tr>
<td>5</td>
<td>M</td>
<td>Hepatic</td>
<td>pseudo</td>
<td>no</td>
<td>30</td>
<td>unknown</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>M</td>
<td>Pancreatic</td>
<td>pseudo</td>
<td>no</td>
<td>10</td>
<td>chronic alcoholic pancreatitis</td>
<td>vague abdominal pain</td>
</tr>
<tr>
<td>7</td>
<td>M</td>
<td>Splenic</td>
<td>pseudo</td>
<td>no</td>
<td>14</td>
<td>trauma</td>
<td>bar fight, spleen laceration cholecystectomy with choledochal drain, bleeding after drain removal</td>
</tr>
<tr>
<td>8</td>
<td>M</td>
<td>Cystic</td>
<td>pseudo</td>
<td>yes</td>
<td>18</td>
<td>post-surgery</td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>M</td>
<td>Right hepatic artery with artero-biliary fistula</td>
<td>pseudo</td>
<td>yes</td>
<td>19</td>
<td>unknown</td>
<td>2 years after liver transplant, 1 month after biliary Anastomosis dilatation, cholangitis, bleeding, biliary obstruction left liver lobe</td>
</tr>
<tr>
<td>10</td>
<td>M</td>
<td>Pancreatico-duodenal</td>
<td>pseudo</td>
<td>yes</td>
<td>7</td>
<td>unknown</td>
<td>sudden abdominal pain, bland history, bleeding</td>
</tr>
<tr>
<td>11</td>
<td>M</td>
<td>Right hepatic</td>
<td>pseudo</td>
<td>yes</td>
<td>25</td>
<td>post-PTC</td>
<td>hemobilia, diagnosed 3.5 weeks after rendezvous procedure</td>
</tr>
</tbody>
</table>

Fig. 2

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Conclusion

In this poster, we presented a single-center case series of visceral aneurysms treated by direct puncture approach. We evaluated the indication and effectiveness of direct puncture embolization and its safety.

Direct puncture embolization was chosen in 10 cases to solve anatomical and technical insuperabilities encountered during endovascular approach. In one case of Ehlers-Danlos type 4 (vascular type), an endovascular approach was contra-indicated because of the risk of arterial complications. Embolization was successful in all cases, with only one recurrence.

The direct puncture approach was performed with variations in operator, puncture needle, guiding imaging modality, embolization material, aneurysm location and etiology. No complications occurred after spill of glue or after puncture through the stomach.

The versatility, safety and effectiveness of this method are illustrated by the fact that despite these variations, no complications occurred, and in all 11 instances adequate occlusion was achieved.

We conclude that embolization by a direct puncture approach is an efficacious and safe alternative if endovascular embolization of visceral aneurysms is to be avoided or is not technically feasible.
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