Unruptured Giant Aneurysm of The Inferior Pancreaticoduodenal Artery with Superior Mesenteric Artery Stenosis Obstructed by Spontaneous Thrombosis After Superior Mesenteric Artery Angioplasty: A Case Report

Yuki Wada¹, Satoshi Takahashi¹, Makoto Koga¹, Katsuhito Seki², Manabu Hashimoto¹

Abstract

Most pancreaticoduodenal artery aneurysms involve celiac trunk stenosis or occlusion. Few cases have been related to superior mesenteric artery (SMA) stenosis, and none of these was treated with SMA angioplasty before transcatheter arterial embolization (TAE) or operative resection of the aneurysm. We treated a 79-year-old woman with incidentally detected inferior pancreaticoduodenal artery aneurysms, presumably secondary to SMA stenosis. Abdominal angiography indicated that TAE of the aneurysms would disturb collateral flow and cause SMA ischemia, so SMA angioplasty was performed before TAE of the aneurysms. Angiography of the SMA six days after angioplasty revealed partial thrombosis in the giant aneurysm. The smaller aneurysm was then embolized to occlude collateral flow, which facilitated further thrombosis of the giant aneurysm without recurrence.

Key words: inferior pancreaticoduodenal artery aneurysm, superior mesenteric artery stenosis, angioplasty

(Interventional Radiology 2016; 1: 53-58)

Introduction

Pancreaticoduodenal artery aneurysms are susceptible to rupture, necessitating early treatment with either transcatheter arterial embolization (TAE) or surgery [1]. Most aneurysms in this area are caused by celiac trunk stenosis; aneurysms related to superior mesenteric artery (SMA) stenosis are very rare [2-5]. Of the previously described inferior pancreaticoduodenal artery (IPDA) aneurysms, all were treated with TAE or operative resection. We describe the case of a patient with an unruptured giant IPDA aneurysm caused by SMA stenosis, which we treated with SMA angioplasty using a bare stent.

Case Report

A 79-year-old woman was referred to our hospital with a giant IPDA aneurysm. She had complained of abdominal pulsations at another institution, where a slight abdominal aortic aneurysm was suspected on ultrasonography. An IPDA aneurysm measuring 31 mm in diameter and slight abdominal aortic dilatation were detected incidentally by contrast-enhanced computed tomography (CT). She had a history of an old cerebral infarction and left carotid artery occlusion, but no history of trauma or pancreatitis. Antihypertensive, gastrointestinal, and three types of antiplatelet drugs were prescribed. On admission to our hospital, vital signs were within normal limits. There were no abnormalities on clinical examination, complete blood count, or bio-
was neither calcification nor thrombosis in either aneurysm. An aneurysm, the IPDA, and finally into the SMA. There flowed through the dorsal pancreatic artery, gastroduodenal artery, posterior pancreaticoduodenal artery, then into the giant aneurysm, the IPDA, and finally into the SMA. There was neither calcification nor thrombosis in either aneurysm. All of the collateral arteries from the celiac trunk converged into the smaller aneurysm. Blood flow through the dorsal pancreatic artery, gastroduodenal artery, posterior pancreaticoduodenal artery, then into the giant aneurysm, the IPDA, and finally into the SMA. The giant aneurysm was saccular, had a diameter of 31 mm, and was located on the distal side of the IPDA. The smaller aneurysm was fusiform, had a diameter of 10 mm, and was located on the proximal portion of the IPDA. The SMA orifice was markedly stenotic from arteriosclerosis, which was considered the cause of the aneurysms. The celiac trunk was also dilated, possibly due to hemodynamic stress from SMA stenosis. Because the smaller aneurysm was fusiform, we were concerned that TAE of the aneurysms with preservation of the IPDA would be difficult. We predicted that TAE of the IPDA aneurysms would disturb collateral flow into the SMA and cause SMA ischemia. We therefore planned angioplasty of the proximal SMA before TAE.

A blood access sheath (Medikit Supersheath®, 6Fr, 25 cm, Medikit, Miyazaki, Japan) was inserted through the right femoral artery. The SMA orifice was accessed using a guiding catheter (Britetip®, 6Fr, 55 cm, Cordis, East Bridgewater, NJ). A microballoon catheter (Aviator Plus®, 4 mm x 15 mm, Cordis) was placed in the stenotic segment using a mi-
A metallic stent (4 mm × 18 mm PALMAZ Genesis®, Cordis) was then inserted using a microguidewire (Chevalier®, 0.014 inch, Cordis) and the balloon inflated to 10 atm for 30 s. Expansion of the SMA stenosis without complication was confirmed. Celiac arteriography immediately after angioplasty showed decreased flow and stagnation in the giant aneurysm. Superior mesenteric arteriography showed mild antegrade flow into the IPDA with faint opacification of the giant aneurysm (Fig. 2). The three kinds of antiplatelet drugs were continued after angioplasty.

We planned TAE of the giant aneurysm six days after angioplasty of the SMA. We inserted blood access sheaths (Medikit Supersheath®, 6Fr, 25 cm, Medikit) in the femoral arteries bilaterally; one for intervention and the other for controlled angiography. Angiography of the celiac trunk was performed first and showed partial thrombosis of the giant IPDA aneurysm (Fig. 3). We feared coil embolization of the giant aneurysm, and instead attempted to embolize the smaller aneurysm abutting the giant aneurysm to occlude collateral flow from the celiac trunk into the SMA and accelerate thrombosis in the giant aneurysm. A balloon catheter (Aviator Plus® 4 mm × 15 mm, Cordis) was placed in preparation for repeat dilatation of the SMA stent, but was not used. A guiding catheter (RH®, 4.2 Fr, Hanaco Medical, Saitama, Japan) was positioned in the celiac trunk. A microcatheter (Excelsior 1018®, Stryker Japan, Tokyo, Japan) was navigated into the smaller aneurysm with a microguidewire (d’Azur Wire®, 0.016 in, 180 cm, CREATE MEDIC, Kanagawa, Japan) by way of the celiac trunk-common hepatic artery-gastroduodenal artery-posterior pancreaticoduodenal artery. The smaller aneurysm was embolized using nine detachable microcoils (in order: two Precidio18®, Johnson and Johnson, Tokyo, Japan; one Cashemere®, Johnson and Johnson; two Galaxy®, Johnson and Johnson; one Cashemere®; three GDC18® soft detachable coils, Stryker Japan) while evaluating blood flow into the smaller aneurysm by angiography with a catheter (SHK-KANAZAWA®, Hanaco Medical) in the celiac trunk. Imme-

---

**Figure 2.** A: Percutaneous transluminal angioplasty is performed for stenosis of the superior mesenteric artery (SMA). The black arrow shows the metallic stent positioned in the stenotic lesion. Stenosis is markedly improved and antegrade flow into the inferior pancreaticoduodenal artery (IPDA) from the SMA is demonstrated (arrowhead). B, C: Arterial phase (B) and portal phase (C) of the celiac angiograms obtained immediately after SMA angioplasty show decreased flow and stagnation of the giant aneurysm (arrow).
Immediately after TAE, celiac arteriography revealed occlusion of all collateral flow from the celiac trunk into the SMA and no opacification of the aneurysms (Fig. 3). Superior mesenteric arteriography showed sufficient patency of the SMA orifice with the stent, and antegrade flow into the IPDA with faint opacification of the giant aneurysm (Fig. 4). After TAE of the smaller aneurysm, one antiplatelet drug was discontinued to prevent recanalization of the thrombosed aneurysm.

Finally, the giant IPDA aneurysm was completely thrombosed four days after TAE (Fig. 5). The patient was discharged 16 days after TAE without complications and is now followed up with CT studies at another institution. The aneurysm size has remained stable for two years.

Discussion

True pancreaticoduodenal artery aneurysms are rare, but a high mortality rate has been reported for ruptured cases, thus necessitating rapid treatment [1]. Approximately 80% of these aneurysms are related to celiac trunk stenosis [6] that induces hemodynamic change of the pancreaticoduodenal arcade, the speculated cause of these aneurysms [2]. Although SMA stenosis or occlusion also increases flow into the pancreaticoduodenal arcade, pancreaticoduodenal aneurysms with SMA stenosis or occlusion are very rare.

A PubMed search for these aneurysms with the keywords “pancreaticoduodenal artery” and “aneurysm,” retrieved only four cases thought to be caused by SMA stenosis [2-5]. Table 1 compares the characteristics of past patients and the current patient. SMA angioplasty was not performed before TAE of the aneurysm in any case except for ours. Two of the seven aneurysms were surgically resected, two were treated with TAE, two were observed carefully, and the present aneurysm needed no further treatment because thrombosis occurred in the aneurysm after SMA angioplasty. Ichiyokawa et al. selected resection because the size of the aneurysm was too large for TAE with metallic coils [3], and Kitaoka et al. selected resection considering that TAE could lead to SMA ischemia due to occlusion of collateral flow [2]. Ikoma et al. treated the aneurysm with TAE and performed SMA angioplasty immediately after TAE [4].

In the present case, we thought resection of the aneu-
Interventional Radiology 2016; 1: 53-58

Figure 5. Contrast-enhanced computed tomogram obtained 4 days after embolization. The giant aneurysm is completely thrombosed (arrow).

Table 1. Pancreaticoduodenal arterial aneurysms related to stenosis or occlusion of superior mesenteric artery

<table>
<thead>
<tr>
<th>Age</th>
<th>Sex</th>
<th>Size of SMA</th>
<th>Celiac trunk condition</th>
<th>Treatment</th>
<th>Angioplasty of SMA</th>
</tr>
</thead>
<tbody>
<tr>
<td>76</td>
<td>F</td>
<td>30</td>
<td>Occlusion</td>
<td>Resection</td>
<td>No</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>n.a.</td>
<td>Observation</td>
<td>No</td>
</tr>
<tr>
<td>80</td>
<td>M</td>
<td>11</td>
<td>Occlusion</td>
<td>Observation</td>
<td>No</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>n.a.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>71</td>
<td>M</td>
<td>20</td>
<td>Stenosis</td>
<td>TAE</td>
<td>Done</td>
</tr>
<tr>
<td>73</td>
<td>F</td>
<td>20</td>
<td>Stenosis</td>
<td>Resection</td>
<td>No</td>
</tr>
<tr>
<td>79</td>
<td>F</td>
<td>31</td>
<td>Stenosis</td>
<td>Spontaneous</td>
<td>Done occlusion</td>
</tr>
</tbody>
</table>

F: female, M: Male, n.a.: no abnormality, TAE: transcatheter arterial embolization
ficient pathways between the celiac artery and the SMA other than the aneurysm-related artery—TAE can precede angioplasty; otherwise, preceding angioplasty is recommended. Complete thrombosis occurred in the giant IPDA aneurysm after SMA angioplasty in this case; however, it does not always occur in treated aneurysms. Additional embolization of the aneurysm may be necessary when thrombosis is incomplete.

In conclusion, precise evaluation of the angioarchitecture of the aneurysm and collateral pathways between the celiac artery and SMA is vital in case of pancreaticoduodenal arcade aneurysm related to SMA stenosis. The strategy for TAE of the aneurysm and SMA angioplasty should be carefully designed, as well as the order in which these procedures should be performed.

Conflict of interest: The authors declare that they have no conflicts of interest to report.

This study has been presented at JSIR, ISIR & APICO 2015.

References