Retrograde access using the pancreaticoduodenal arcade to embolise aneurysms in Sutton Kadir Syndrome

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Introduction

Association between coeliac axis (CA) stenosis or occlusion and aneurysm formation in the collateral vasculature of the CA territory first described in 1973.  

125 cases reported so far. Pancreaticoduodenal artery (PDA) affected with aneurysms in 84% of these cases.  

30% of cases presented with aneurysm rupture. Size not been found to correlate with risk of rupture.  

Pathophysiology of this syndrome still unknown but strong association with median arcuate ligament (MAL) compression of the CA seen.

Close proximity of inferior PDA aneurysms to the superior mesenteric artery (SMA) makes this treatment challenging.

Discussion

Aneurysms affects the inferior PDA at the point it branches off the SMA or close to it in the majority of cases.  

Current hypothesis suggests CA stenosis or occlusion results in increased retrograde flow from the SMA into collateral arcades; this increased abnormal flow in these small diameter vessels then leads to aneurysms forming.

MAL compression of the CA has been found to be a recognised risk factor. Compression occurs from a young age whereby likely predisposing the collateral vasculature to abnormal high flows for a long duration. The prevalence of this association is likely under recognised. MAL compression was present in all our cases.

Case 1: 71-year-old male with a symptomatic inferior PDA aneurysm associated with CA stenosis from median arcuate ligament compression; treated with Histoacryl-Lipiodol embolization.

Presented acutely with sharp abdominal pain, nausea and anorexia. CTA and DSA confirmed a ruptured bi-lobed aneurysm measuring 6mm in diameter.

Case 2: 55-year-old male with a symptomatic inferior PDA aneurysm associated with CA stenosis from median arcuate ligament compression; treated with coil embolization.

Presented with a three-day history of abdominal cramps, diarrhoea and vomiting. CTA and DSA confirmed a retroperitoneal bleed from a ruptured aneurysm.

Case 3: 64-year-old male with a symptomatic inferior PDA aneurysm associated with CA stenosis from median arcuate ligament compression; treated with Histoacryl-Lipiodol embolization.

CTA confirmed a ruptured aneurysm measuring 11mm in diameter.

Case 4: 71-year-old male with a symptomatic aneurysm of the inferior PDA and common hepatic associated with CA occlusion from median arcuate ligament compression; treated with coil embolization.

Presented with sharp left flank pain. CTA and DSA confirmed two aneurysms; an inferior PDA saccular aneurysm measuring 26mm in diameter and a fusiform aneurysm of the common hepatic measuring 8mm in diameter.

Case 5: 55-year-old female with a symptomatic inferior PDA aneurysm associated with CA stenosis from median arcuate ligament compression; treated with coil embolization.

Presented with severe central abdominal pain associated with two episodes of bilious vomiting. CTA and DSA confirmed a contained retroperitoneal haematoma with evidence of an aneurysmal segment of the inferior PDA.

Table 1: This case series describes the treatment of five patients with Sutton-Kadir Syndrome by the Interventional Radiology Department at the Sir Charles Gairdner Hospital in Perth, Western Australia. Aneurysms were approached using retrograde access via the superior mesenteric artery (SMA) to select the PDA in all cases.

<table>
<thead>
<tr>
<th>Age</th>
<th>Gender</th>
<th>Presentation</th>
<th>MAL compression of CA</th>
<th>Aneurysm location</th>
<th>Size</th>
<th>Rupture</th>
<th>Embolic agent</th>
<th>Outcome</th>
<th>Complications</th>
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</table>

Figure 1: Pre-embolization (A) and post-embolization (B). Embolized with 1mL of 1:3 Histoacryl:Lipiodol mixture.

Figure 2: Contrast extravasation suggestive of aneurysm rupture (A) proximal CA stenosis (B).

Figure 3: Pre-embolization (A) and post-embolization (B). Embolized with 1.5mL of 1:4 Histoacryl:Lipiodol mixture.

Figure 4: Pre-embolization (A) and post-embolization (B).

Figure 5: Pre-embolization (A) and post-embolization (B).

Conclusion

This series has shown that embolisation with glue and coils using the SMA to access the inferior PDA is a safe and feasible approach to manage Sutton-Kadir Syndrome.

Future research should consider longitudinal studies in this patient cohort and the potential role for CA revascularization as an answer or as an adjunct to treatment of Sutton-Kadir.

References