HEPATOCELLULAR CARCINOMA WITH CENTRAL SEVERE ARTERIOPORTAL SHUNT-ENDOVASCULAR APPROACH

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HEPATOCELLULAR CARCINOMA WITH CENTRAL SEVERE ARTERIOPORTAL SHUNT-ENDOVASCULAR APPROACH (Abstract): The prognosis of hepatocellular carcinoma is related to the extension of the lesion, as well as to the liver function at the time of diagnosis and associated arteriportal shunts. Case report: We report a 70-year-old woman with C virus - related cirrhosis, hepatocellular carcinoma and central severe arteriportal shunt, presenting with upper digestive tract hemorrhage and refractory ascites, treated by embolization of the shunt and trans catheter arterial embolization of the tumor. Results: After treatment, both ascites and esophageal varices improved, with good general status for three months. Afterwards, a massive esophageal bleeding uncontrolled by emergent endoscopic hemostatic techniques caused rapid deterioration and death. Conclusions: Hepatocellular carcinoma with central severe arteriportal shunt is a challenging clinical situation, whose prognosis is influenced mainly by portal hypertension. Embolization of the shunt is the first option to reduce the pressure in the portal system and to ensure the endovascular treatment of the tumor. Keywords: HEPATOCELLULAR CARCINOMA, ARTERIOPORTAL SHUNT, EMBOLIZATION

Liver cancer is the ninth most common cancer in women, the second most common cause of death from cancer worldwide, and accounts for 7% of all cancers (1). Hepatocellular carcinoma (HCC) represents more than 90% of primary liver cancers and its prognosis is related to the extension of the lesion, as well as to the liver function at the time of diagnosis (1). Furthermore, HCC frequently involves portal vein (PV) and creates arteriportal shunts (APS), worsening portal hypertension (PHT) and increasing the risk of gastrointestinal bleeding and hepatic encephalopathy (2).

The present report describes a patient with HCC and central severe APS treated by embolization of the shunt followed by trans catheter arterial embolization of the tumor.

CASE REPORT
A 70-year-old woman, who has been diagnosed with C virus - related cirrhosis for 8
years, was referred to our hospital for rapid deterioration of liver function with refractory ascites and upper digestive tract bleeding.

The patient’s laboratory data at admission are presented in table I. Child’s classification status was B (Child-Pugh score 8).

**TABLE I**

<table>
<thead>
<tr>
<th>Laboratory data</th>
<th>Admission</th>
<th>2 days after TAE1</th>
<th>9 days after TAE 1</th>
<th>3 days after TAE 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total bilirubin level mg/dL (N: 0.2-1.2)</td>
<td>1.50</td>
<td>1.08</td>
<td>1.02</td>
<td>0.96</td>
</tr>
<tr>
<td>AST IU/L (N: 5-31)</td>
<td>73</td>
<td>43</td>
<td>35</td>
<td>27</td>
</tr>
<tr>
<td>ALT IU/L (N: 5-32)</td>
<td>112</td>
<td>72</td>
<td>52</td>
<td>46</td>
</tr>
<tr>
<td>GGTP IU/L (N: 8〜59)</td>
<td>119</td>
<td>203</td>
<td></td>
<td></td>
</tr>
<tr>
<td>WBC count /μL,</td>
<td>4010</td>
<td>5910</td>
<td></td>
<td></td>
</tr>
<tr>
<td>RBC count × 10^6 /μL</td>
<td>362</td>
<td>346</td>
<td>377</td>
<td>325</td>
</tr>
<tr>
<td>HGB level g/dL</td>
<td>11.3</td>
<td>10.7</td>
<td>11.7</td>
<td>11</td>
</tr>
<tr>
<td>hematocrit %</td>
<td>33.1</td>
<td>31.3</td>
<td>33.4</td>
<td>33</td>
</tr>
<tr>
<td>platelet count × 10^3 /μL</td>
<td>90</td>
<td>82</td>
<td>132</td>
<td>85</td>
</tr>
</tbody>
</table>

AST - aspartate aminotransferase level; ALT - alanine aminotransferase level; GGTP - γ-glutamyl trans peptidase; WBC - white blood cell; HGB - hemoglobin; RBC - red blood cell;

Upper gastrointestinal endoscopy revealed severe esophageal varices and emergent sclerotherapy was performed to control bleeding. Abdominal ultrasound (US) identified a hyper vascular lesion in the 4th segment of the liver, and hepatofugal high velocity flow in PV trunk and branches (up to 190 cm/s) with arterial waveform (fig.1). No thrombus was found in PV. Massive ascites was noticed in all peritoneal spaces. Computed tomography (CT) of the abdomen, with tri-phase acquisition after injection of contrast medium, identified a low-density lesion in the 4th segment only on non-enhanced and late scan. An APS located in porta hepatitis with early and strong enhancement of enlarged PV trunk at early hepatic arterial phase was seen. An arterial variant was also disclosed, with replaced common hepatic artery (HA) originating from superior mesenteric artery (SMA).

The final diagnosis was HCC with central severe APS and refractory PHT. The patient was then referred to the interventional radiology unit. A common HA arteriography showed a major APS centrally located (between right HA and PV trunk with multiple feeders), but a tumor stain was not visible because of the shunt (fig. 2a). A super selective catheterization of the right HA was performed with microcatheter (3F, Renegade) followed by angiography. Due to the numerous feeders and enlarged right HA and PV, we decided to perform coils trans catheter arterial embolization (TAE) for the major APS. Three coils (Azur Pure, 4 mm in diameter, length 4 and 6 mm) were introduced into the right HA with partial occlusion of the APS (approximately 80%) (fig. 2b).

The follow-up Doppler US performed 1 week after revealed lower velocity in PV but with persistent APS. Clinical status slowly improved with diminishing rate of ascites recurrence.
Consequently, we have performed a second TAE within 3 weeks, completing the embolization with Gelfoam and occluding the shunt (fig. 3). The left HA arteriography showed HCC feeders, which were super selective embolized with Embosphere microspheres 300-500 µm. The celiac trunk (splenic, left gastric and left phrenic arteries) was catheterized, but no tumor feeders were depicted.

After treatment, ascites and esophageal varices, as well as laboratory findings improved (tab. I). Doppler US revealed reduced sized PV (14 mm), without thrombosis (fig. 4). The patient was discharged one week after with a small amount of ascites.

Three months later the patient presented a massive esophageal varices bleeding. Emergent CT showed revascularization of the HCC, with arterial supply from left HA, right internal mammary and right gastroepiploic arteries, and an APS in the lower part of the lesion. The right HA was still embolized. Unfortunately, the massive esophageal bleeding was uncontrolled by emergent endoscopic hemostatic techniques, with rapid deterioration and death.
Hepatocellular carcinoma with central severe arterioportal shunt – endovascular approach

**Fig. 3.** a) Hepatic arteriography after 3 weeks: persistence of APS; b) completion of the embolization with occlusion of the shunt.

**Fig. 4.** Liver Doppler US - follow-up after embolization: reduced sized PV (14 mm), without thrombosis and normal hepatopetal flow in the left portal branch.

**DISCUSSION**

Several clinical and imaging classifications of APS are available (3). A central APS is in *porta hepatis* and a severe APS consists in opacification of PV trunk and/or the first-order branches with enhancement at the early hepatic arterial phase, without or with early enhancement of HCC foci (3).

APS occurs in 60% of patients with HCC, marked APS of the main, right or left PV accounting for half of these patients (2, 4). Lesions associated with severe APS shows no enhancement at the arterial phase in dynamic CT due to reduced arterial flow to the tumor, as presented in our case (3).

TAE is the standard treatment for intermediate stage HCC, but there is no consensus about optimal treatment of HCC associated with central severe APS (1). EASL-EORTC Clinical Practice Guidelines have no reference related to the treatment of this complication of HCC, even if microscopic vascular invasion involves 20% of tumors of 2 cm in diameter and APS occurs in more than half of patients with HCC (1).

Moreover, HCC associated with central severe APS is a challenge for interventional radiologist since embolic material can pass through the shunt reducing the effectiveness of the treatment and producing damage of PV branches with higher risk of liver ischemia. Therefore, temporary or permanent occlusion of the supplying arteries of the APS is required before endovascular treatment of the lesion. For severe APS, the procedure is also mandatory to ameliorate PHT (5-11).

TAE of severe APS can be achieved by several embolic materials used alone or combined: gelatin sponge, ethanol, PVA
particles, N-butyl cyanoacrylate, coils (5-11). The choice of embolic agent is based on shunt size, velocity, location, angi-architecture and specific actions of each embolic agent. Coils are generally recommended only for simple APS owing to the difficulty to reach each feeder in complex shunts, but we choose them because of severity of the APS and multitude of feeders which render the selective embolization impossible.

The prognosis of HCC patients with severe APS is related to severe PHT and subsequently, to variceal bleeding, refractory ascites and portal encephalopathy (1). Surgical treatment is reported only for some severe resistant or recanalized APS with recurrent uncontrolled esophageal variceal hemorrhage (12). For centrally located APS the procedure of choice should be a major hepatectomy, unsafe in patients with poor liver function.

CONCLUSIONS
Our case outlines the challenging situation of HCC with central severe APS. Embolization of the shunt is the first option to reduce the pressure in the portal system, as well to render the endovascular treatment of the tumor more efficient and to favor patient survival.

REFERENCES