

Clinical Image

An Unusual Parasite: Extra-hepatic Arterial Collateralization by Hepatocellular Carcinoma

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Description

A 78-year-old woman with past medical history significant for hepatitis B virus-related cirrhosis and Hepatocellular Carcinoma (HCC) was initially treated with left lateral hepatic segment surgical resection. She was subsequently referred to our Interventional Radiology clinic for liver directed therapy for large, local tumor recurrence. She initially underwent successful Transarterial Chemoembolization (TACE), although follow-up Magnetic Resonance Imaging (MRI) demonstrated persistent tumor viability, as evidenced by persistent nodular enhancement within the embolization cavity, predominantly located peripherally and along the bare surface of the liver (Figures 1 and 2).

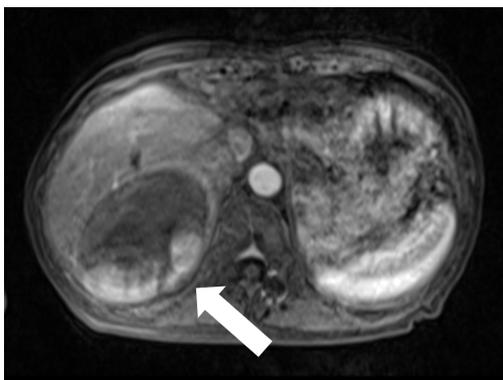


Figure 1: Persistent nodular enhancement within the embolization cavity (Marked).

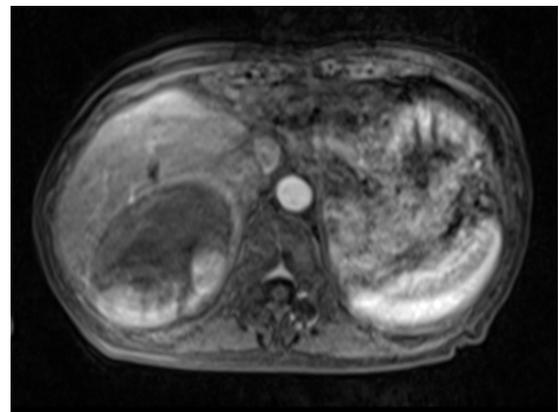


Figure 2: Persistent nodular enhancement within the embolization cavity (Unmarked).

She underwent a second TACE and the follow-up MRI once again demonstrated viable tumor in the embolization cavity, in a similar distribution. Serum alpha-fetoprotein levels continued to rise despite treatment. At this point, a Computed Tomographic Angiogram (CTA) of the abdomen was obtained to further delineate the vascular supply to the tumor. This CTA demonstrated enlargement of internal, arterially enhancing, nodular components that occupied approximately 50% of volume of embolization cavity (Figures 3 and 4) and parasitized extrahepatic collateral arterial supply arising from a hypertrophied right inferior phrenic artery (Figures 5 and 6). Once this vessel was identified, the patient underwent subsequent TACE via the nutrient vessel (Figures 7 and 8), obtaining complete response.



Figure 3: Enlargement of internal, arterially enhancing, nodular components (Marked).



Figure 4: Enlargement of internal, arterially enhancing, nodular components (Unmarked).

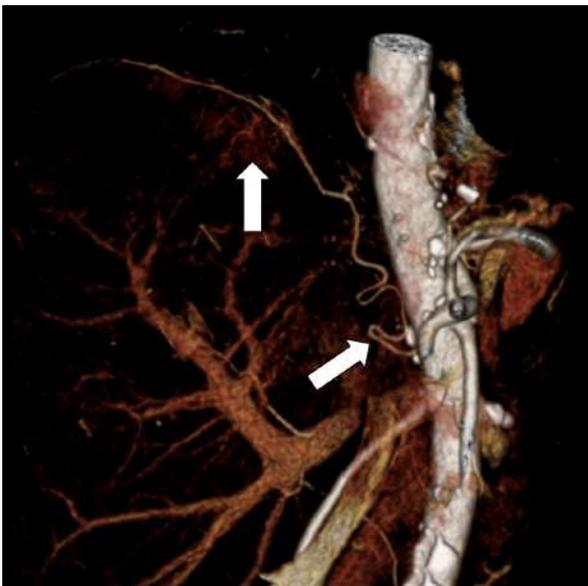


Figure 5: Parasitized extrahepatic collateral arterial (Marked).



Figure 6: Parasitized extrahepatic collateral arterial (Unmarked).

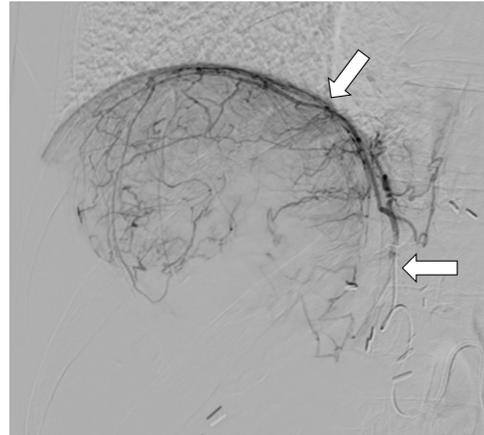


Figure 7: TACE via the nutrient vessel (Marked).

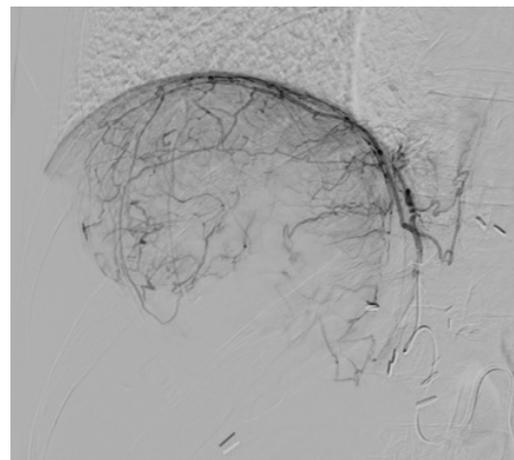


Figure 8: TACE via the nutrient vessel (Unmarked).

Discussion

Hepatocellular Carcinoma (HCC) is a hypervascular tumor that derives the entirety of its vascular supply from arterial blood, most often from the hepatic artery. The blood supply of the remainder of the healthy liver is mostly portal venous with some arterial supply as well. This physiology allows for the performance of TACE in a relatively safe and efficacious fashion as both treatment and bridging therapy to liver transplantation. However extrahepatic collateral arterial supply (EHC) to HCC may occur in a variety of scenarios [1,2]. Timely detection of this phenomenon is of fundamental importance to achieve optimal outcomes and response to treatment, as seen in the case presented above. EHC can arise at any time during the lifespan of a tumor, although certain conditions predispose to their formation and these should be kept in consideration when analyzing an image or planning treatment. Large tumors (>5 cm), exophytic with extrahepatic infiltration, peripherally growing tumor or at the bare area of the liver, prior surgery or multiple transarterial interventions can all predispose or favor EHC formation [2,3]. Additionally, EHC should be suspected when peripheral nodular enhancement persists opposite to the main hepatic arterial supply inflow and despite multiple TACEs (as in this case), when there is continued elevation of serum alpha fetoprotein levels despite interventions or when only part of the lesion demonstrates lipiodol accumulation, if used as part of the TACE [2,3]. Furthermore, conventional contrast enhanced CTA has greater utility than contrast enhanced MRI if EHC is suspected and one should be aware of this difference in imaging modalities when obtaining follow up imaging.

Of all possible EHCs, branches arising from the inferior phrenic artery (right more frequent than left) and omental branches are the most common [1-3]. These parasitized vessels are followed, in frequency, by right intercostal, right adrenal, cystic and right internal mammary arteries. In the case presented, EHC arterial supply arose from a hypertrophied right Inferior Phrenic

Artery (IPA) and several first divisional branches. Typically, the IPA may arise from the abdominal aorta or the celiac arterial trunk (less frequently from direct branches of the aorta such as the renal arteries) and courses superiorly along the bare surface of the liver to supply the ipsilateral hemi diaphragm, anastomosing with terminal branches of the internal mammary and/or intercostal arteries. Diagnostic and interventional radiologists should suspect EHC supply when a tumor arises in hepatic segment VII or is in contact with the right hemi diaphragm, which may mandate performance of selective angiography of the right IPA at time of treatment [3]. Similarly, tumor abutting the left hemi diaphragm or arising in the left lobe (segments II and III) warrants vascular interrogation of the left IPA. Parasitization of omental arteries is not bound by territory due to the inherent, high mobility of the omentum.

Ultimately, TACE procedures are increasingly performed for intermediate stage HCC, both as treatment and bridging therapy to hepatic transplant. Due to this rise, the probability of encountering an EHC supply to HCC is consequently elevated and radiologists, both diagnostic and interventional alike, and referring clinicians must be aware of this phenomenon.

References

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