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Case Presentation

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Hepatocellular Carcinoma Arising from Ectopic Liver Tissue in the left Triangular Ligament: A Case Report

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Abstract

Accessory liver lobes represent an infrequent anatomical anomaly. The probability of hepatocellular carcinoma (HCC) developing in these accessory lobes remains exceptionally rare. Herein we report a case of a HCC arising from accessory liver lobe. A 63-year-old man came to our institution because of an abnormally elevated level of alpha-fetoprotein (AFP). There were no previous medical issues related to chronic liver disease or cirrhosis. Initially, MRI, PET-CT, and preventive interventions failed to detect tumor lesions. After conducting three follow-ups, it was concluded that the elliptical HCC was linked to the mother liver through a linear structure. This report shows when AFP levels continue to escalate without apparent etiology and routine examinations of the main liver reveal no conspicuous pathology, fastidious retrospective scrutiny of the anatomical elements encompassing the ectopic HCC or accessory liver should be undertaken.

Keywords: Case report; HCC; Hepatocellular carcinoma; Accessory liver lobe

Introduction

Accessory Liver Lobe (ALL) refers to an excessive parenchymal extension of the liver connected to the main hepatic organ by hepatic tissue or a stalk [1]. Watanabe reports that the incidence of ALL is extremely low, estimated at 0.09% when assessed via laparoscopy [2]. Ozaki et al. observed that 80% of ALL cases localize in liver segment VI or the right hepatic lobe [3]. While accessory lobes are documented in the left hepatic lobe, the occurrence of Hepatocellular Carcinoma (HCC) in these left accessory lobes remains exceptionally rare [4]. We present an exceedingly rare case of HCC originating from an ALL situated in the left subphrenic space,

adjacent to the triangular ligament. This diagnosis was confirmed through surgical resection and histopathological analysis.

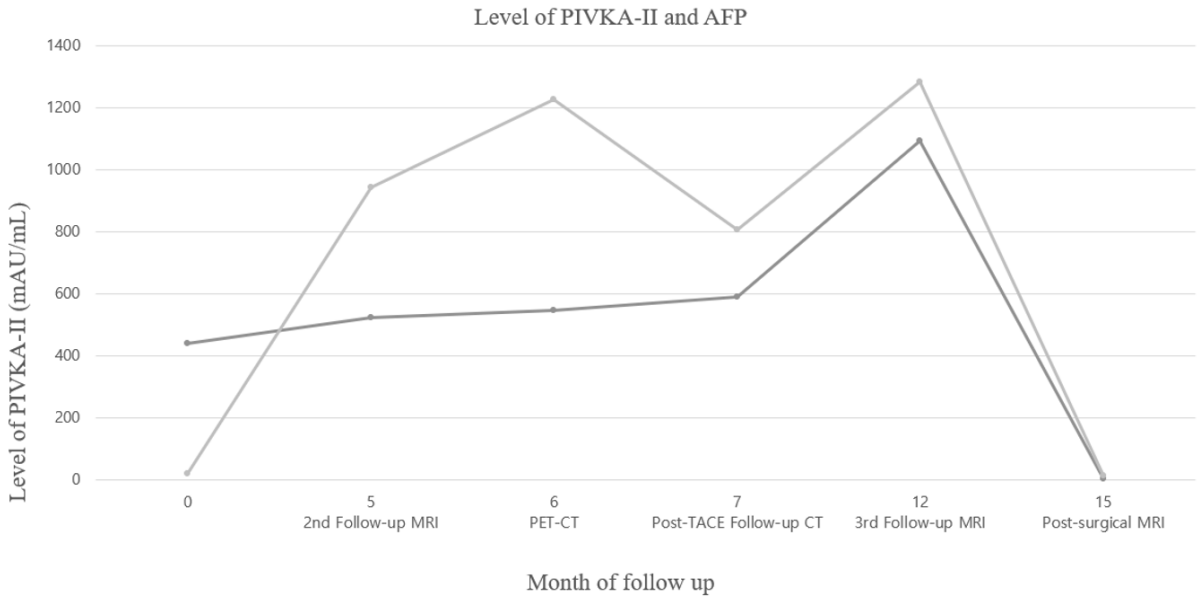
Case Presentation

A 63-year-old male was referred to our institution from a health assessment center due to elevated Alpha-Fetoprotein (AFP) levels. Previous MRI and abdominal ultrasonography at another healthcare facility showed no significant findings. The patient was asymptomatic and had an unremarkable medical history. Upon evaluation at our hospital, an MRI revealed no mass-like hepatic lesions, except for minor shunts in the right hepatic lobe. A Positron Emission Tomography-Computed Tomography (PET-CT) scan also showed no malignancy indicators. The patient opted for outpatient follow-up. However, over several monthly visits, AFP levels rose from 522 ng/mL to 590 ng/mL, and Prothrombin Induced by Vitamin K Absence-II (PIVKA) levels fluctuated between 800-900, remaining elevated **(Table 1).** To exclude HCC, hepatic angiography was performed but yielded no substantial findings. The prominent hepatic dome shunt identified via MRI prompted the decision to proceed with Trans-Catheter Arterial Chemoembolization (TACE) as a precaution.

Table 1: Laboratory findings of patient's tumor marker.

	Outside MRI (06.24.2019)	2nd Follow-up MRI (11.02. 2020)	PET-CT (12.02.2020)	Post-TACE Follow-up CT (01.13.2021)	3rd Follow-up MRI (06.17.2021)	Post-surgical MRI (11.04.2021.)
AFP (ng/ml)	438	522.24	545.77	590.28	1092.64	4.1
PIVKA-II (mAU/mL)	20	941.64	1223.83	804.43	1282.91	14.34

†Abbreviations: TACE: Transcatheter Arterial Chemoembolization; AFP: Alpha-Fetoprotein; PIVKA-II: Protein Induced by Vitamin K Absence or Antagonist



Three months later, during the patient's third outpatient visit, AFP levels showed a more significant increase compared to January, post-TACE. PIVKA levels followed the same pattern. An MRI scan at this time revealed a mass-like lesion approximately 2 cm proximal to the spleen, located in the left subphrenic space, seen during the hepatobiliary phase **(Figure 1).** T2-Weighted Imaging (T2WI) showed elevated signal intensity, while the arterial phase exhibited no appreciable difference from the spleen. Diffusion-Weighted Imaging (DWI) revealed subtle diffusion restriction or no significant change compared to the spleen. Retrospective review of the non-enhanced CT performed during TACE showed the lesion as smaller in size. Due to its location near the spleen, it was initially considered an ectopic spleen. Subsequent MRI demonstrated enhancement and morphological characteristics similar to those of the spleen. However, compared to previous images, the lesion had grown, and a coronal scan revealed a linear structure tethered to the left hepatic lobe, culminating in an oval-shaped mass **(Figure 2).** This raised the possibility of HCC arising from aberrant liver tissue.

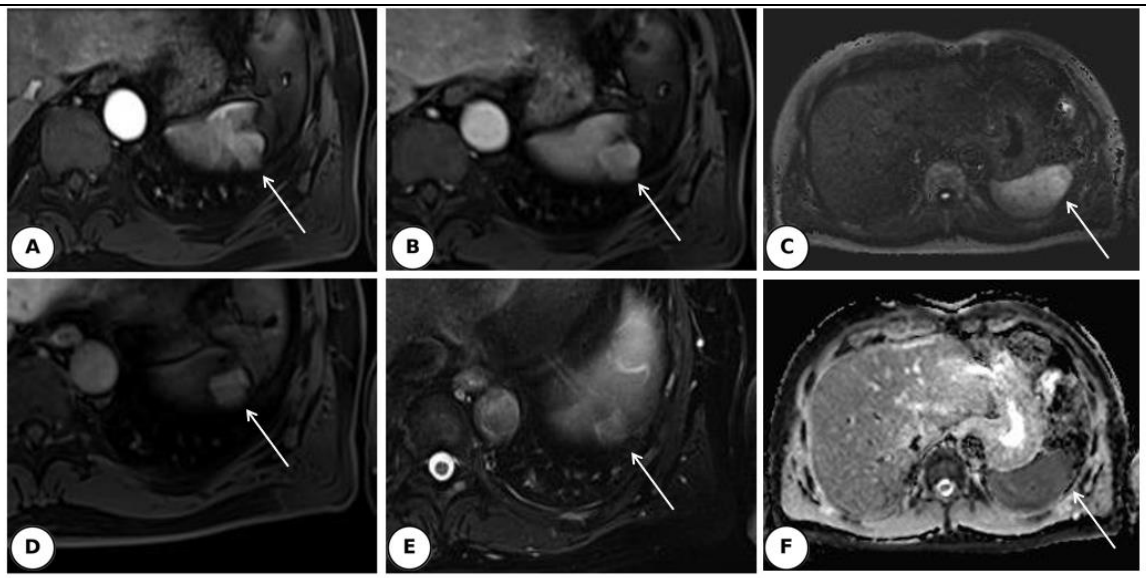


Figure 1: Follow-up MRI. Arterial phase of axial T1WI (A), portal phase of axial T1WI (B), DWI (C), hepatobiliary phase of axial T1W1(D), axial T2WI (E) and ADC (F) MRI unveiled the presence of a mass-like lesion (white arrow), approximately 2 cm proximal to the spleen, situated within the left subphrenic space.

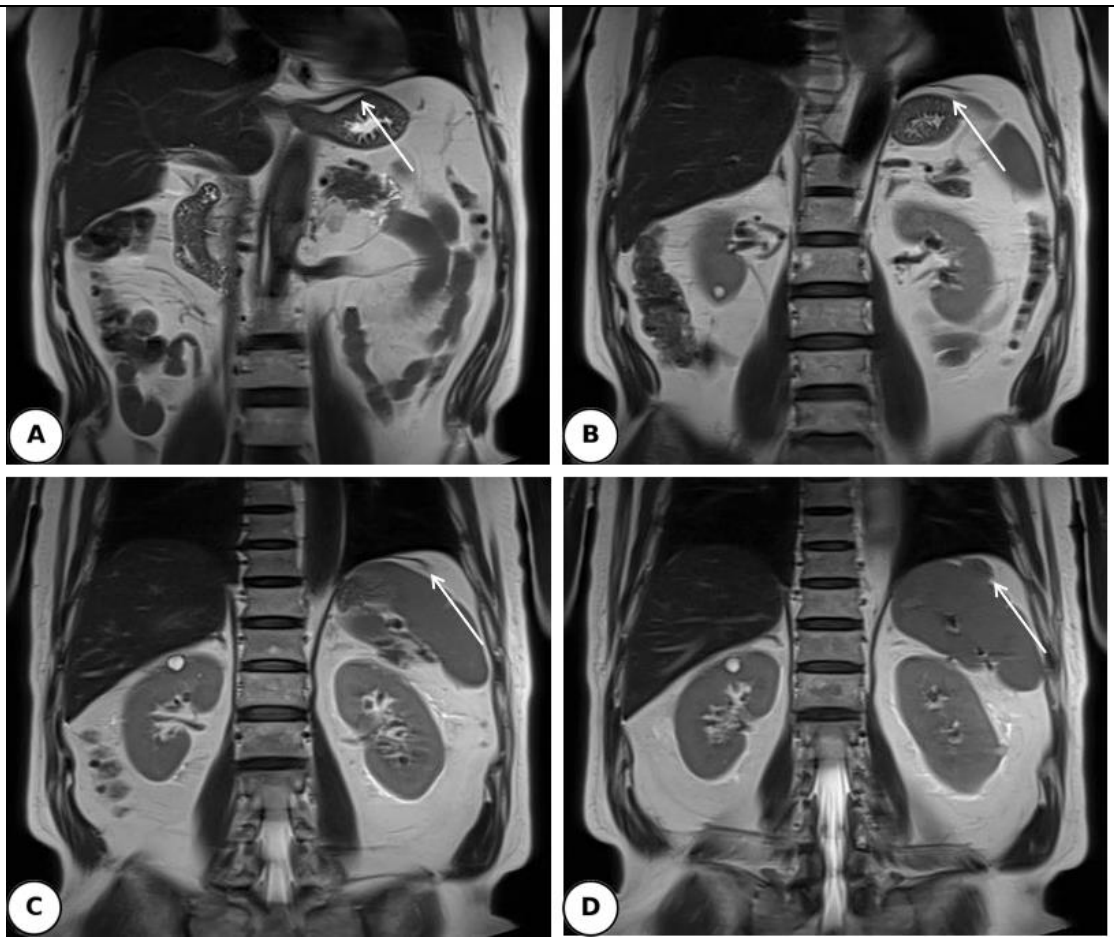


Figure 2: Follow-up MRI with coronal scan. Serial images of coronal T2WI MRI (A)~(D) show a linear structure tethered to the left hepatic lobe, culminating in an oval-shaped mass (white arrow).

After reviewing the MRI findings, the physician proceeded with surgical exploration, revealing mass tissue with a linear tail interlinked with the left triangular ligament **(Figure 3A).** Histopathological analysis confirmed HCC within the accessory liver, marked by positive glypican-3 staining **(Figure 3B).**

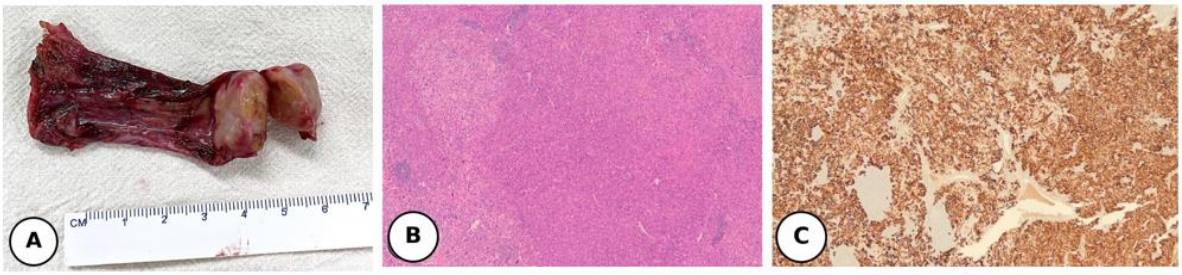


Figure 3: Mass tissue yields by surgical exploration characterized by a linear tail, interlinked with the left triangular ligament (A), Glypican-3 immunohistochemistry stain, positivity in tumor cells that support the malignant nature of hepatocellular proliferation(B) and H&E stain, zoom x 10 shows poorly differentiated area with severe cytological atypia (C).

Discussion

The definitive diagnosis was HCC originating from an ALL adherent to the left triangular ligament. ALLs are embryological anomalies often found in various anatomical regions **[1].** However, the majority of these anomalies are located in the right hepatic lobe, especially liver segment IV **[3,6].** The occurrence of an accessory lobe in the left hepatic lobe, along with the development of HCC, is exceptionally rare, as seen in our case. Literature suggests that helical dynamic CT scans can be useful in diagnosing HCC. Features include arterial-phase contrast enhancement, lower density than hepatic parenchyma in the portal phase, and an irregularly enhanced tumor with contrast pooling in the delayed phase **[4].** MRI findings typically show low signal intensity on T1-weighted images and irregularly high signal intensity on T2-weighted images, aiding in lesion characterization **[8].** However, our case highlights the variability in imaging patterns. The ectopic HCC closely resembled an ectopic spleen in morphology and contrast enhancement, making differentiation challenging. Upon retrospective review, as the lesion increased in size, axial MRI during the hepatobiliary phase revealed a contrast uptake pattern distinct from both the mother liver and the spleen. This allowed for the identification of the lesion's malignant potential. In addition, the risk factors typically associated with HCC, such as HBV infection and cirrhosis, have not been linked to ALL-derived HCC **[9].** This complicates preoperative diagnostics. Some reports recommend Digital Subtraction Angiography (DSA) to confirm HCC, particularly hypervascular lesions, and guide Trans-catheter Arterial Embolization (TAE). However, accurately identifying the feeding artery is crucial. In cases where the mass location remains unclear, random artery selection for TAE, as in our patient's case, can cause diagnostic dilemmas. Arakawa's study found that among 16 patients with HCC from ALL or ectopic liver, two received vascular supply from the left hepatic artery, two from the right hepatic artery, and twelve from other sources entirely **[9].** Given this variability, arbitrary arterial selection for intervention is not ideal. Considering these factors, we recommend maintaining a high index of suspicion in cases with persistently elevated AFP levels, similar to our patient's profile. This should prompt a thorough evaluation of the anatomical surroundings of the ectopic HCC or accessory liver, keeping in mind the potential for HCC development within these structures. Despite Cunningham's reservations about the utility of AFP **[10]**, Arakawa's observations emphasize the diagnostic value of elevated AFP levels, especially when no clear clues are present **[9].** In our case, the definitive diagnosis was achieved only after a comprehensive retrospective MRI analysis, which eventually revealed the lesion's elevated AFP level. The delay in diagnosis was due to the lesion's location within a blind spot in the subphrenic space. This underscores the crucial role of radiologists in diagnosing complex cases like this one.

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