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Case Report

A rare case of hepatocellular carcinoma arising from gadoxetate-retaining hepatic adenoma

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ABSTRACT

Hepatocellular adenomas (HCAs) are benign lesions of the liver which can rarely undergo malignant transformation. We report a 26-year-old woman with no underlying liver disease found to have an incidental liver lesion on noncontrast CT during workup for gastric reflux. Follow up MRI revealed a 10 cm gadoxetate-retaining lesion within the right hepatic lobe with imaging features suggestive of HCA vs focal nodular hyperplasia. Within this lesion was a focus of arterial enhancement with venous washout suggestive of hepatocellular carcinoma (HCC) within HCA, later confirmed at surgical resection. Understanding the imaging characteristics of HCAs as well as their rare ability to undergo malignant transformation is useful in differentiating HCAs from focal nodular hyperplasia.

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Introduction

Hepatocellular adenomas (HCAs) are uncommon tumors accounting for 2% of all liver neoplasms. HCAs have variable appearance on imaging; some retain gadoxetate, which is typically seen in the setting of focal nodular hyperplasia (FNH), a benign entity. This can be problematic, as HCAs can rarely undergo malignant transformation to hepatocellular carcinoma (HCC). Understanding the imaging characteristics of this transformation may be useful for early detection and management. We present a rare case of HCC arising from a gadoxetate-retaining HCA.

Case

26-year-old female with no underlying liver illness and a 6 year history of oral contraceptive use was found to have an incidental liver lesion on noncontrast CT while undergoing workup for gastric reflux. She had no complaints of abdominal pain and laboratory values including liver function tests were within normal limits. She then underwent a gadoxetate-enhanced MRI revealing a 10 cm lesion arising from the majority of the right hepatic lobe with associated hemorrhage. This lesion was heterogenous on both T1 and T2 and iso- to hyper-intense on 20 minute hepatobiliary phase, for which the differential included focal nodular hyperplasia or atypical HCAs (Fig. 1). Within this lesion was a 3 cm arterial hyperenhancing lesion with portal venous washout and hypointense on 20 minute hepatobiliary phase, suspicious for HCC. However, this finding was not diagnostic, as the patient had no

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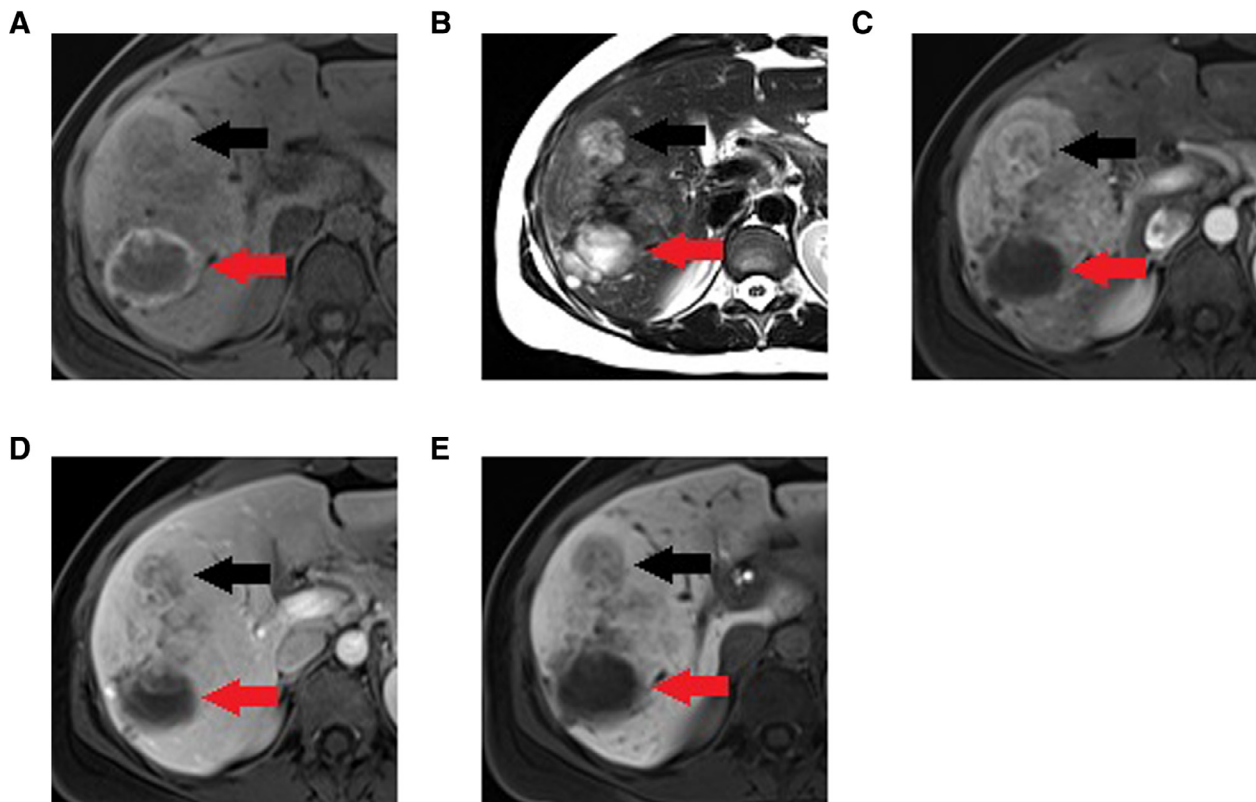


Fig. 1 – (A) Axial T1 sequence MRI demonstrates hypointense HCC lesion (black arrow) and peripherally intense focus of hemorrhage (red arrow) within a large HCA in the right hepatic lobe. (B) Axial T2 sequence demonstrates heterogeneous hyperintense HCC (black arrow) and an even more hyperintense region of hemorrhage (red arrow). (C) Axial arterial phase (gadoxetate enhanced) sequence demonstrates arterial enhancing HCC (black arrow) and hypointense region of hemorrhage (red arrow). (D) Axial venous phase sequence demonstrates venous washout within HCC (black arrow) and hypointense region of hemorrhage (red arrow). (E) Hepatobiliary phase demonstrates large gadoxetate - retaining HCA which is iso- to hyper- intense relative to liver parenchyma and containing hypointense HCC (black arrow) and hypointense region of hemorrhage (red arrow)(Color version of figure is available online.)

underlying liver disease, precluding Liver Imaging Reporting and Data System (LI-RADS) assessment.

Core biopsy of the lesion confirmed the diagnosis of HCC within HCA. Patient subsequently underwent a right hepatectomy with resected specimen measuring 17.0 × 15.0 × 7.0-cm and containing the entirety of HCA. The gross specimen revealed a tan-brown and smooth HCA containing a 3 cm tan-grey nodular focus of HCC with central hemorrhage (Fig. 2). Histologic pathology demonstrated a sharp border between well differentiated HCA cells and moderately to poorly differentiated HCC (Fig. 3). The HCC cells contained pleomorphic nuclei and prominent nucleoli with pseudoglandular and telangiactetic architecture. Patient did well postoperatively and was discharged home on postoperative day 2. Follow-up contrast-enhanced MRI at 6 month follow up revealed no evidence of residual or recurrent disease.

Discussion

HCAs are uncommon, solid, benign tumors [1]. Incidence of these tumors is 3 per 1,000,000 of the general population and

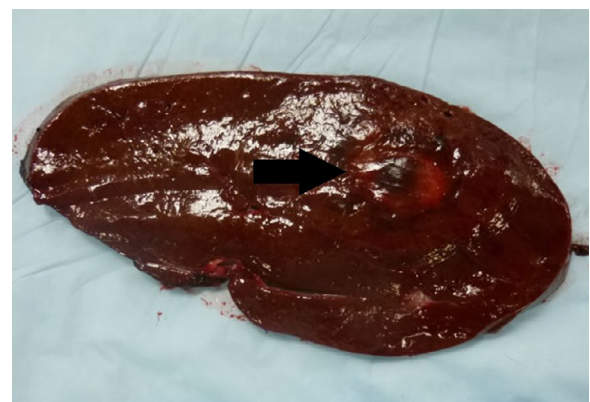


Fig. 2 – Gross specimen of right hepatic lobectomy containing entirety of tan brown smooth HCA and a 3 cm tan nodular focus of HCC containing central hemorrhage (black arrow)(Color version of figure is available online.)

3-4 per 100,000 women [1,2]. The high incidence of HCAs in women is attributed to the hormone-induced nature of these

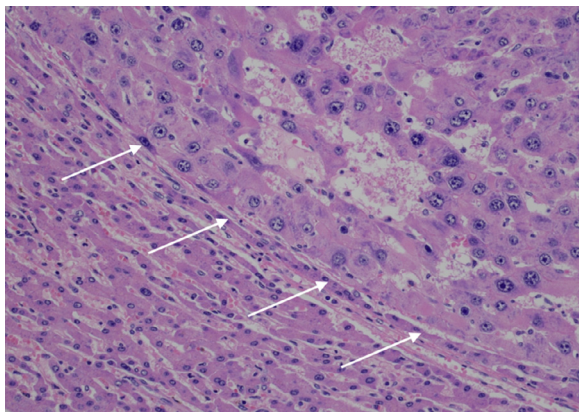


Fig. 3 – Histologic pathology using H&E (Hematoxylin and eosin) stain at 40x power demonstrates a sharp border (white arrows) between well differentiated HCA cells (bottom left) and moderately to poorly differentiated HCC (top right). The HCC cells contained pleomorphic nuclei and prominent nucleoli with pseudoglandular and telangiectatic architecture

tumors and use of oral contraceptive pills and other estrogens in women of childbearing age [3]. Hepatic adenomas are usually solitary, have a tendency for hemorrhage, and usually arise in the right hepatic lobe [4]. Patients with larger tumors over 5 cm are more likely to report symptoms such as vague abdominal pain. Elevated levels of serum alpha-fetoprotein should raise suspicion for malignant transformation to HCC, the most severe complication of HCAs. Although the overall risk of malignant transformation has not been well defined in the literature, previous reports demonstrate 4%-8% of resected HCAs have undergone malignant transformation [5]. Surgical resection is recommended for HCAs larger than 5 cm, intralesional HCC, increasing size, rising alpha fetoprotein, and adenomas in glycogen storage disease [6]. Surgery is not recommended for HCAs smaller than 5 cm, anatomically challenging locations, and those that undergo regression on steroid withdrawal [7]. For these patients, conservative management is recommended which includes discontinuation of hormone therapy and follow up imaging surveillance. Most HCAs are heterogeneous on both T1 and T2 weighted sequences and hypointense on hepatobiliary phase, an important distinguishing factor with focal nodular hyperplasia (FNH) which is often iso- to hyper- intense on hepatocyte specific contrast [8]. It should be noted, however, that up to 25% of HCAs can also retain hepatocyte specific contrast such as gadoxetate, limiting the diagnostic differentiation from FNH [9].

Our patient had no complaints of abdominal pain and laboratory values including liver enzymes were normal. Interestingly, the liver lesion was hyperintense on hepatobiliary phase, making it difficult to discern between HCA and FNH. Presence of hemorrhage and intralesional hypervascular lesion, however, favored HCC arising from HCA, which was later confirmed.

Conclusion

Hepatic adenomas are rare benign liver lesions typically affecting women of childbearing age. While most adenomas demonstrate hepatobiliary hypointensity, some adenomas are hyperintense on hepatobiliary phase, making it difficult to discern from FNH. Presence of hemorrhage, heterogeneity, T2 hyperintensity, and intralesional arterial enhancing lesion provided useful diagnostic clues to favor hepatic adenoma over FNH.

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