CASE REPORT

Metastatic rectal adenocarcinoma to the liver associated with focal nodular hyperplasia

P J Nisar, A M Zaitoun, A Damera, Z Hodi, G M Tierney, I J Beckingham

A 45 year old female patient underwent right hemihepatectomy for metastatic rectal adenocarcinoma. Preoperative imaging demonstrated an area of focal nodular hyperplasia (FNH) in segment VIII and metastatic carcinoma in segment VI of the liver. Gross and microscopic examination of the former lesion showed features typical of FNH with an intralosomal metastatic adenocarcinoma. To the best of our knowledge, this is the first reported case of metastatic adenocarcinoma located within a lesion of FNH. The possibility of a pathogenetic association behind this occurrence is discussed.

A previously well 43 year old female patient underwent anterior resection for Dukes's stage C1 adenocarcinoma of the rectum. Postoperatively, she received adjuvant chemotherapy for six months. Liver ultrasonography at one year follow up demonstrated an irregular hypoechoic lesion measuring 25 mm in diameter and extensive fatty change.

After referral to a liver surgeon, contrast enhanced magnetic resonance imaging (MRI) was performed. This demonstrated a 34 mm circular hypervascular mass in segment VIII of the liver (lesion A) with decreased signal on T1 and T2 weighted images, showing early and persistent enhancement. The mass contained an eccentric low attenuation area on dynamic imaging, which was suggestive of a scar within an area of focal nodular hyperplasia (FNH). A 15 mm non-enhancing spherical lesion in segment VI was demonstrated (lesion B), which did not conclusively represent a metastatic focus.

After an interval of three months MRI was repeated. Lesion B had grown to 35 mm in diameter and showed irregular rim enhancement with reduced signal on T1 and T2 weighted images; features that are typical of neoplastic disease. The patient was admitted for elective right hemihepatectomy. The patient recovered without complications and was discharged.

PATHOLOGY

Macroscopy

The resected specimen weighed a total of 1056 g and measured 21 × 17 × 9 cm. A well circumscribed unencapsulated tumour (lesion B), which measured 2.7 × 2.2 × 2 cm, was present at 2 cm from the resection margin in liver segment VI. Lesion A measured 5 × 3 × 4 cm, and was situated in segment VIII, 6.5 cm from lesion B, so that it extended closely (0.3 cm) to the margin of resection.

Lesion A had an ill defined, lobulated, flesh coloured, nodular appearance when sectioned, and contained a central scar, surrounded by irradiating fibrous septa—a picture typical of FNH. A solid white tumour measuring 1 × 1 × 0.7 cm was seen arising within the main mass of FNH (fig 1A). More than 80% of the metastatic tumour nodule was surrounded by the FNH and the remaining outline was composed of fibrous capsule.

Microscopy

Microscopic examination of lesion A revealed nodular hyperplastic parenchyma showing thickened hepatic plates and prominent tortuous vessels of arteriolar calibre (fig 1B), consistent with FNH. Located within the FNH was a focus of metastatic adenocarcinoma (fig 1A).

Abbreviations: FNH, focal nodular hyperplasia; MRI, magnetic resonance imaging

Figure 1  (A) Section of liver showing lesion A. Focal nodular hyperplasia almost completely surrounding a solid white nodule of metastatic carcinoma. (B) Focal nodular hyperplasia showing steatosis; haematoxylin and eosin stain; original magnification, x4.
metastatic adenocarcinoma with features indicating a colorectal origin (fig 2A). Sections from lesion B had the typical appearance of metastatic adenocarcinoma, with extensive necrosis.

The margins of resection were noted to be tumour free, and there was no evidence of vascular or bile duct invasion. Generalised macrovesicular steatosis in zones 2 and 3, and multiple foci of biliary microhamartoma (fig 2B), with no evidence of cystic changes, were present throughout the resected liver.

DISCUSSION

The term focal nodular hyperplasia was instituted by Edmondson in 1958 and is accepted to describe benign liver lesions with a pathognomonic gross appearance, as seen in our case. The microscopic architecture of hyperplastic liver parenchyma and the central scarred fibrous area is characteristic. Several atypical solitary morphological variants have been reported; telangiectatic, mixed hyperplastic, adenomatous, and large cell type FNH have been reported.1 It has been generalised that FNH with a macroscopic scar corresponds to the microscopic classic architecture.

FNH is considered to be the result of a hyperplastic process, secondary to increased blood flow. FNH has been associated with liver haemangiomas, arterial recanalisation, systemic arterial dysplasia, and cerebral vascular malformations.2

“Focal nodular hyperplasia is thought to be a non-neoplastic entity, although it may have behaved as a host lesion in our case because it is well vascularised and would therefore provide an appropriate environment for tumour deposition and growth.”

Radiological appearances of FNH have been described for MRI; FNH is generally hypo-intense on T1 weighted images and exhibits intense post contrast enhancement. A central scar, as seen in our case, can be seen in 10–49% of lesions.3 Metastases can have the same signal characteristics as FNH on precontrast scans and may enhance in a similar fashion to FNH. We conclude that the intrallesional metastasis was not demonstrated by MRI in our case because it was obscured by the pronounced enhancement of the FNH.

Metastatic disease associated with a benign lesion is also seen in the phenomenon of “tumour to tumour” metastasis. FNH is thought to be a non-neoplastic entity, although it may have behaved as a host lesion in our case because it is well vascularised and would therefore provide an appropriate environment for tumour deposition and growth.

FNH contiguous with fibrolamellar variant hepatocellular carcinoma has been well described.5 Convincing histological evidence of the transformation of FNH to hepatocellular carcinoma does not exist, and studies of clonal analysis have rejected the potential for malignant transformation within FNH to hepatocellular carcinoma. Ewing’s sarcoma metastasising to FNH has also been described.6 It is interesting to note that in our case intrallesional metastasis in the FNH coincided with diffuse hepatic steatosis. A similar finding has been reported by other authors.7

Our case compounds the evidence for hyperaemia playing a role in the pathogenesis of FNH and shows that FNH may act as a host lesion to metastatic carcinoma. Malignant lesions located within FNH may not be demonstrated radiologically.

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REFERENCES


Figure 2  [A] Metastatic carcinoma in the right lower corner surrounded by focal nodular hyperplasia. [B] Multiple hamartomas and background liver showing steatosis. Haematoxylin and eosin stain; original magnification, ×4.


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