A CASE OF A RECURRENT FNH-LIKE LESION TREATED BY RADIATION THERAPY

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Abstract: A 38-year-old woman underwent liver resection for an FNH-like lesion. Regrowth of the residual nodule occurred 9 months after liver resection. The recurrent nodule was treated with 50 Gy by external conformal radiation therapy. The nodule shrank remarkably 20 months after radiation therapy without evidence of recurrence. The literature was reviewed for an ‘FNH-like lesion’ and radiation therapy for the benign liver tumors.

Key words: Benign nodular hepatocellular lesion, FNH-like lesion, Radiation therapy

INTRODUCTION
Focal nodular hyperplasia (FNH) is a benign tumor-like lesion of the liver considered to be the result of a hyperplastic rather than a neoplastic process. An ‘FNH-like lesion’ can be used if the nodule resembles FNH to some extent. In general, surgical resection is inadaptable for an FNH or FNH-like lesion. However in the case which is difficult to distinguish it from a malignant tumor or in a symptomatic case, surgery is sometimes provided. We have experienced the radiation effective recurrent FNH-like lesion after the surgical resection of a primary bulky FNH-like lesion. We report the case including a review of the literature.

CASE REPORT
A 38-year-old woman presented with the symptom of abdominal pain. CT showed a hypervascular bulky tumor, 6x7 cm in size, in the right lobe of the liver (Fig. 1a). She had not taken any oral contraceptive before. Hepatic functions were within the normal range. A malignt hepatic tumor could not be ruled out at the time. Although she was recommended to receive resection of the tumor by the attending physician, she refused surgery and chose a wait-and-see approach. Four years later, she was pointed out increasing of the tumor in size. Although the findings of the tumor including hypervascularity and a central scar-like appearance suggested a benign hepatic tumor such as FNH, the possibility of malignant tumor could not be excluded because of its increase in size (Fig. 1b). After obtaining informed consent to surgical therapy, resection of the tumor in the right lobe was performed. The removed specimen showed a poorly-demarcated bulky tumor without a capsule, which was 13x12x9 cm in size. Pathological diagnosis of the lesion was an FNH-like nodule associated with blood flow abnormality primarily in the portal system and/or in the arterial system in the broad sense of the term4) (Fig. 1c, d). Nine months after surgery, a recurrent nodule was noted in the surgical margin of the liver. Surgical treatment was difficult because of adhesion of the lesion to adjacent organs such as the small intestine and the inferior vena cava. The attending surgeon referred her to the department of radiology for the possibility of radiation therapy for the recurrent nodule. Although observation was one of the managements, the growing nature of the nodule concerned her and us. She was explained that non-invasive external radiation therapy was one of the possible therapeutic options and informed of the potential risk of hemorrhage or rupture by the recurrent increasing nodule similar to FNH,5) the potentiality of shrinkage of the nodule by external radiation therapy in view of the radiation effect on the hepatic benign tumor, hemangioma, in spite of no previous report on radiation therapy for the particular FNH-like lesion and the risks of radiation hepatitis and later secondary cancer due to radiation therapy. After receiving the above information, She consented to external radiation therapy for the lesion. Thus, external conformal irradiation was performed with 6-MV X rays from a linear accelerator (Mitsubishi Medical Systems, Tokyo, Japan). The planning dose was 50 Gy in 25 fractions (Fig. 2). Although there was no significant change in the nodule when the radiation therapy was completed, there was a remarkable reduction in nodule size 20 months later (Fig. 3) and were no significant changes in the subjective symptoms, liver functions and irradiated normal liver on CT.

DISCUSSION
The frequency of detection of hepatic nodular lesions has increased because of recent advances in diagnostic imaging techniques. Sometimes benign hepatocellular lesions are also detected today such as focal nodular hyperplasia (FNH), nodular regenerative hyperplasia (NRH), nodular lesions associated with idiopathic portal hypertension (IPH), non-cirrhotic large nodular regenerative nodules (LRN),
Fig. 1  (a) Enhanced CT at the initial visit on March 2000 shows a bulky hypervascular tumor (6×7 cm in size) in the right lobe of the liver.(b) Preoperative gadolinium enhanced MRI shows the increased tumor in the right lobe of the liver.  (c) Cut section of the gross specimen of the lesion shows a huge nodular lesion (*) and ambiguous margin (arrows). (d) Microscopically, the specimen shows fibrous septum, and fibrous expansion and bridging of the portal area with proliferation of cholangiocytes and chronic inflammatory cell infiltration (arrows). The portal area shows vascular channels with irregular thickened walls or obstruction (arrowheads). The hepatocytes show no cellular atypia. (Victoria bleu-Hematoxylin eosin (VB-HE) ×25)

Fig. 2  Irradiation of the tumor with conformal radiotherapy. Field configuration and isodose lines for the tumor by 6-MV X rays.
hepatocellular adenoma (HA)-like hyperplastic nodules and partial nodular transformation (PNT). However, there are some nodular hepatocellular lesions that are difficult to be diagnosed definitively. They partially resemble some typical lesions but are not entirely consistent with any of the typical lesions and are classified as non-typical cases such as FNH-like lesions.

Our case is a non-capsulated nodular lesion. The specimen showed fibrous septum, fibrous expansion and bridging of portal areas with proliferation of cholangioles and chronic inflammatory cell infiltration. The portal region showed wall thickening and obstruction of the vessels due to wall thickening. Macroscopic examination of the classical FNH shows a nodular architecture, a central or eccentric scar containing various types of vessels with vascular changes that resembles arteriovenous malformations, abnormal architecture devoid of portal tracts, and bile ductular proliferation of variable degree. In our case, the absent of a central scar and the presence of the portal region are the findings different from FNH. Although diagnostic images suggested FNH, final pathological diagnosis was an FNH-like lesion in the broad sense of the term.

The etiology of an FNH and an FNH-like lesion is explained by a common causation, that is, an anomaly of the portal tract (Glisson’s sheath) or an anomaly of hepatic vessels including hepatic veins. Both the arterial and portal blood flows may be impaired by the vascular anomaly and this disturbance of the blood flow causes hyperplasia and consequent nodule formation in areas with a richer blood flow.

The natural course of FNH is variable. In particular, the lesion regression is not rare. Thus there is no indication of aggressive medical treatment in principle. However the therapy is indicated for the large tumor size and/or symptomatic case. The natural course of an FNH-like nodule is not known, but considered stable similar to FNH. However progressive or recurrent FNH cases were also reported. Our case showed a rapid growth during 4 years and recurrence after surgery. Radiation therapy for the benign hepatic tumor is few with rare exceptions of unresectable cavernous hemangiomas. We could not find an FNH or FNH-like

Fig. 3 Serial recurrent tumor changes before and after radiation therapy on CT.
(a) CT at the time of recurrence before radiation therapy shows a recurrent hypervascular tumor (arrow) in the surgical edge of the liver.
(b) CT at the end of radiation therapy shows no remarkable change (arrow).
(c) CT at 20 months after radiation therapy shows a significant reduction of the lesion (arrow) in size.

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lesion which was treated by radiation therapy in the range of our literature search. Tolerance dose of the whole liver is considered to be 30 Gy.10), 12) Cheng et al. presented that patients receiving a mean hepatic dose of more than 28 Gy developed radiation induced liver damage more frequently than those with a mean dose of 28 Gy or less after three-dimensional conformal radiotherapy for patients with hepatocellular carcinoma.13) Although the report of radiation therapy for the benign hepatic tumor is limited, radiation therapy with a total dose of 30 Gy (2 Gy per fraction) has been reported to be effective for giant hemangioma.10)

Our case was treated with radiation therapy of a total dose of 50 Gy (2 Gy per fraction), because the patient had no underlying hepatic disorders such as chronic hepatitis or liver cirrhosis in the hepatic tissue surrounding the lesion and the tumor grew rapidly. We used conformal radiation therapy not to exceed over 20 Gy to the surrounding hepatic tissue. It is unknown whether a total dose less than 50 Gy had been effective on our particular tumor. The shrinkage of the recurrent lesion may be due to the radiation damage of hepatocytes and reduced blood flow by radiation damage of abnormal vessels, which is similar to hemangiomas.11)

Although treatment for a benign hyperplastic hepatic nodule is a contentious subject, radiation therapy would be applicable to a symptomatic or rapid growing unrespectable benign hepatic nodule.

References