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CASE REPORT

CT DEMONSTRATION OF THE SPONTANEOUS REGRESSION OF A HYPERVASCULAR LESION IN CIRRHOTIC LIVER

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ABSTRACT

In patients with liver cirrhosis, arterial phase enhancement of nodular lesions on helical-CT is currently considered to be highly predictive of malignancy. We report the spontaneous regression of a hypervascular hepatic nodule in a patient with liver cirrhosis within 7 months demonstrated by helical-CT follow-up. This suggests that tumor angiogenesis known to be an obligatory step for acquisition of malignant properties could regress, and can be demonstrated by helical CT. Radiologists should be aware that CT detection of a hypervascular nodule in a cirrhotic liver is not always predictive of a malignant outcome.

CASE HISTORY

A 77 year-old male patient with a 10 year history of chronic hepatitis B disease and liver cirrhosis underwent ultrasound examination, carried out as part of a routine examination, which showed the presence of a hypoechoic lesion of the right liver lobe. A triphasic helical CT scan (CT Twin; Elscint Medical; Haifa; Israel) was performed using 5-mm collimation, and 0·7 table pitch, covering the entire liver in a single breath-hold. A first axial scanning was performed without contrast injection. A bolus of 150 ml of lobitridol (Xenetix®; Laboratoires Guerbet; Roissy; France) was power injected at a rate of 3 cc/second. Arterial phase imaging was initiated 20 seconds after the start of the injection followed by a portal phase with 45 seconds delay and caudo-cranial acquisition. A delayed phase was obtained 2 minutes after injection. CT displayed a 30 mm diameter low attenuating nodule of the right liver lobe before injection with homogeneous arterial enhancement, and with lasting hyperdensity compared to normal hepatic tissue during portal phase (Fig. 1). The nodule could not be identified on the delayed phase images. No other parenchymal, portal or arterial abnormality was detected. This large hypervascular nodule was consistent with hepatocellular carcinoma (HCC). However, the patient refused any further investigation but agreed to undergo regular CT follow-up.

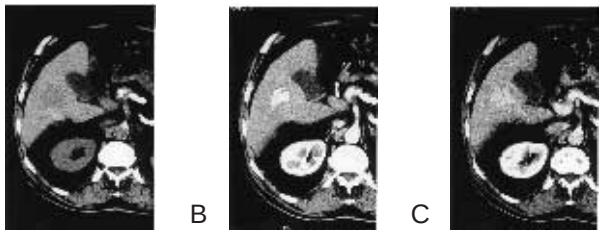

Figure 1. Initial CT scan in a 77-year-old man with hepatitis B and cirrhosis. A, Unenhanced CT scan shows a 30 mm diameter low attenuating mass of the right liver lobe. B, Arterial phase CT scan obtained at the same level as A shows homogeneous strong enhancement. C, Portal phase CT scan shows that the mass remains hyperdense.

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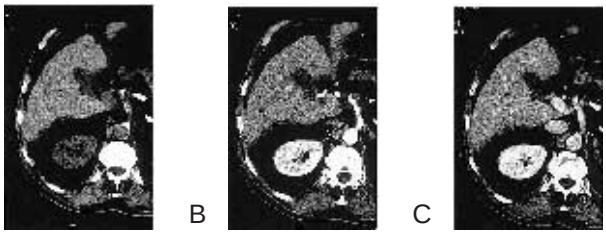

Figure 2. Follow-up CT scan achieved 7 months after initial CT. A, Unenhanced CT scan shows a 5 mm low attenuating lesion. B and C, Respectively arterial and portal phase CT scan obtained at the same level as A shows absence of parenchymal enhancement.

A second abdominal CT scan was thus performed 7 months later following the initial protocol detailed above. No hypervascular nodule could be detected. A 5-mm non-enhancing lesion was present at the location of the initial nodule (Fig. 2). After 30-months follow-up the patient is well and recent US examinations have been unable to detect initial liver lesion recurrence.

DISCUSSION

Several authors have demonstrated that arterial phase CT scan was highly sensitive in detecting HCC in cirrhotic liver. For instance, arterial phase helical CT has proved to be better in unveiling hypervascular liver lesions than either CT during arterial portography or iodized-oil CT, reaching up to 92% sensitivity[1,3]. Moreover, recent studies have tried to estimate the accurate specificity of arterial nodule enhancement in cirrhotic liver. Several authors have indeed established a link between arterial supply and malignancy, whether based on microscopic analysis[4], or on radiological data[5]. Furthermore, for Lee et al., positive predictive value of hypervascularity concerning malignancy in hepatic lesions is 100%[2]. In another series of 500 patients with cirrhosis, only 2% of arterial phase enhancing masses was not HCCs but rather benign lesions including transient hepatic attenuation difference (THAD), hemangioma, hepatic peliosis, fibrosis, splenic lobule, and cryptogenic causes[6]. In our patient, the CT appearance of the nodule was not suggestive of these causes but could be consistent either with HCC or high-grade dysplastic nodule. As a matter of fact, Krinsky *et al*. recently reported the case of a patient presenting with 11 dysplastic liver nodules which all displayed arterial phase enhancement on CT exam[7]. However, their size was smaller than in our patient ranging from 10 to 23 mm. Furthermore, these dysplastic nodules are also considered to be precursor lesions to HCC[8].

Reported cases of spontaneous HCC regression are rare. Initial histological diagnosis is usually obtained from core biopsy performed under ultrasound or CT guidance[9,10]. Regression is established on decrease in size or disappearance of the lesion within the resected liver. The exact mechanisms for such an evolution have never been established. However, the capacity of core biopsy to modify the evolution of the tumor was never considered although well-known vascular complications[11] include needle tracks, hematomas, arterio-portal fistulae and aneurysms. In our patient, as biopsy in itself could have affected the evolution and CT aspect of the tumor, the lack of pathological proof was not redhibitory. Moreover, the diagnostic sensitivity of percutaneous biopsy is low, merely reaching 69%, and tumor recurrence can occur in the needle biopsy track, so many authors believe that the presence of nodular lesion consistent with HCC in a cirrhotic liver should lead to surgical excision without biopsy[12,14].

Due to the absence of biopsy, our case provides a new point of view on nodule regression in cirrhotic liver. In our patient, disappearance of the large arterial phase enhancing nodule was clearly demonstrated by helical-CT follow-up. No invasive diagnostic procedure was performed, so the regression was truly spontaneous. Three main hypotheses may explain this regression: 1. The thrombosis of a feeding artery; 2. Recently, Misawa et al. reported the first case of spontaneous regression of a cirrhotic liver tumor demonstrated by CT[15]. However, the initial CT appearance of this liver lesion was different. This lesion had partial and peripheral enhancement, and was associated with high attenuation of adjacent parenchyma interpreted as an arterio-portal shunt.

Such vascular abnormality was not identified in our patient; 3. The necrotic involution of this lesion, as suggested by Stoelben *et al*.[9]. It is widely accepted that the histological grade of malignancy in primary liver tumors is well correlated with the presence of arterial hypervascularity on CT[5].

LESSON

Our case shows that this hypervascularity can only be transient possibly leading to tumor necrosis. This suggests that tumor angiogenesis known to be an obligatory step for the acquisition of malignant properties, or angiogenesis associated with a dysplastic nodule could regress. Moreover, our case shows that such regression can be clearly identified on helical-CT. In conclusion, radiologists should be aware that CT detection of a hypervascular nodule in a cirrhotic liver, consistent either with hepatocellular carcinoma or dysplastic nodule, is not always predictive of a malignant outcome.

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