

Case Report

Hepatic hemangioma in cirrhosis : Two case reports

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Abstract

Hepatic hemangioma is the most common primary liver tumor with the reported prevalence of 0.4-20%. However, the prevalence of hepatic hemangioma in cirrhosis is considered to be very low ranging from 1.2-1.7%. Also, the morphology and hemodynamics of the hemangioma are different from those found in a non-cirrhotic liver. We reported two cases of hepatic of hemangioma in cirrhosis and their natural progression on a follow-up

Keywords: Hepatic hemangioma, Cirrhosis, Progression.

Introduction

Hepatic hemangioma is the most common primary liver tumor with the prevalence of 0.4-20%[1-3]. It is a benign tumor composed of vascular spaces lined by endothelial cells. In contrast, the prevalence of hepatic hemangioma in cirrhotic patients is considered to be very low ranging from 1.2-1.7 %[4, 5]. Cirrhosis is the end stage of the chronic liver injury characterized by the development of regenerative nodules and surrounding fibrosis. In addition to lower prevalence, the morphology and hemodynamics of hemangioma are different from those found in a non-cirrhotic liver. This is believed to be a result of alteration of blood flow and intra-hepatic environment due to fibrosis in cirrhosis. We reported two cases of hepatic hemangioma in cirrhotic liver and their natural progression.

Case Summary

Case 1

A 56-year-old woman, who is a known case of hypertension and chronic hepatitis C on interferon and ribavirin for 24 weeks, presented with relapse (high viral load) despite good compliance. She was asymptomatic. Her blood pressure was under control and physical examinations were unremarkable. The laboratory result showed mildly elevated liver enzymes. The initial ultrasound scan(USS) showed early features of cirrhosis with an ill-defined hyperechoic nodule in segment VII which could have been either hemangioma or hepatocellular carcinoma(HCC) (Figure 1).

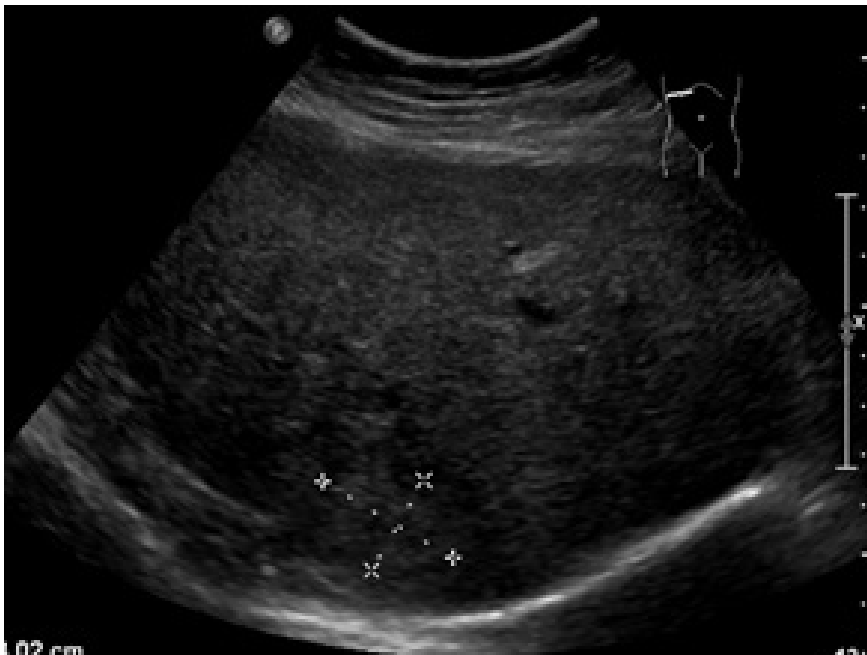


Figure 1: USS showing ill-defined hyperechoic nodule (marked) in hepatic segment VII.

Therefore, computed tomography (CT) and magnetic resonance imaging (MRI) of the abdomen were performed which confirmed the diagnosis of hemangioma at segment VII (Figure 2 and 3). No HCC was discovered.

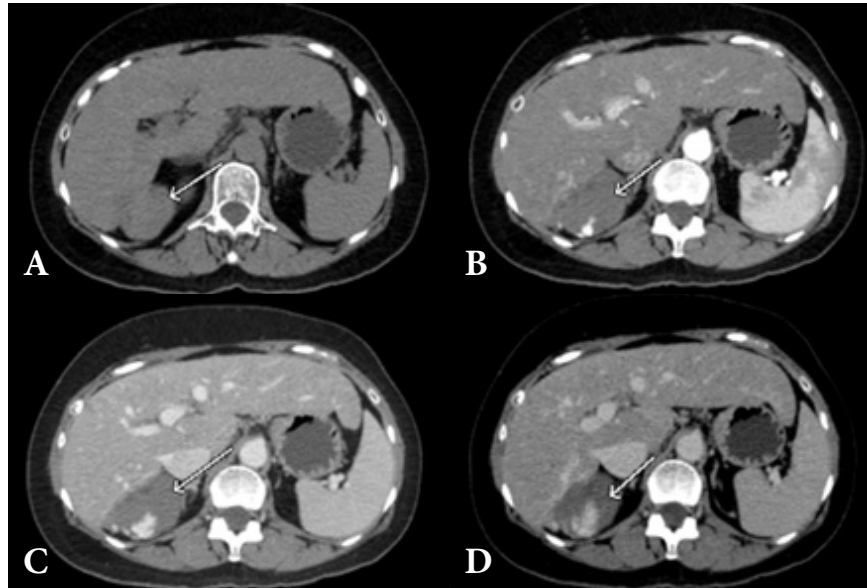


Figure 2: CT upper abdomen in A) plain, B) arterial, C) venous and D) delayed phase showing cirrhotic liver with lobulated hypodense mass and peripheral nodular progressive enhancement typical for hemangioma (arrow) in segment VII. Two small hypodense lesions with homogenous enhancement pattern matching blood pool (flash-filling) was seen at segment II and III, typical of small hemangioma (not shown).

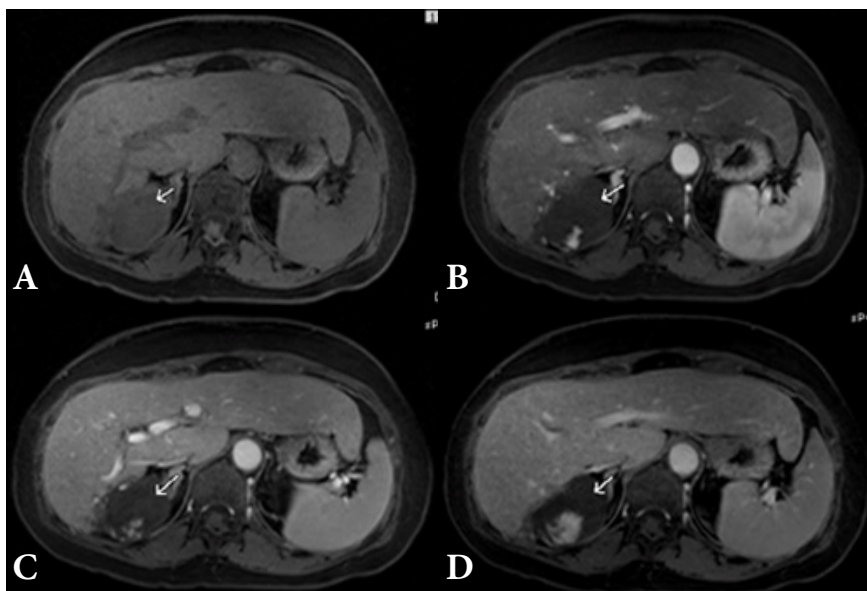


Figure 3: MRI upper abdomen A) pre-gadolinium B) 30 seconds(s), C) 60s and D) 120s showing early features of cirrhosis with typical enhancing pattern of hemangioma (arrow) in segment VII.

She was restarted interferon and ribavirin which was continued for 43 weeks without any complications. At the end of an anti-viral therapy, rapid and sustained virological response was achieved. She continued to undergo a routine USS liver with occasional MRI/CT scan. The follow-up images showed progressive cirrhosis of the liver with decreased size of hepatic hemangioma in segment VII while the hemangioma at segments II and III disappeared. On top of that, the peripheral nodular and progressive centripetal enhancement was not observed. Instead, irregular progressive enhancement seen in only some parts of the hemangioma was noted and the lesion was diagnosed as atypical hemangioma. Eventually, the hemangioma became avascular.

Later on, the patient developed small HCC, about 1.0 cm, in segment IVA/VIII, about 7 years later which was treated successfully with two sessions of selective transarterial chemoembolization (sTACE) (Figure 4).

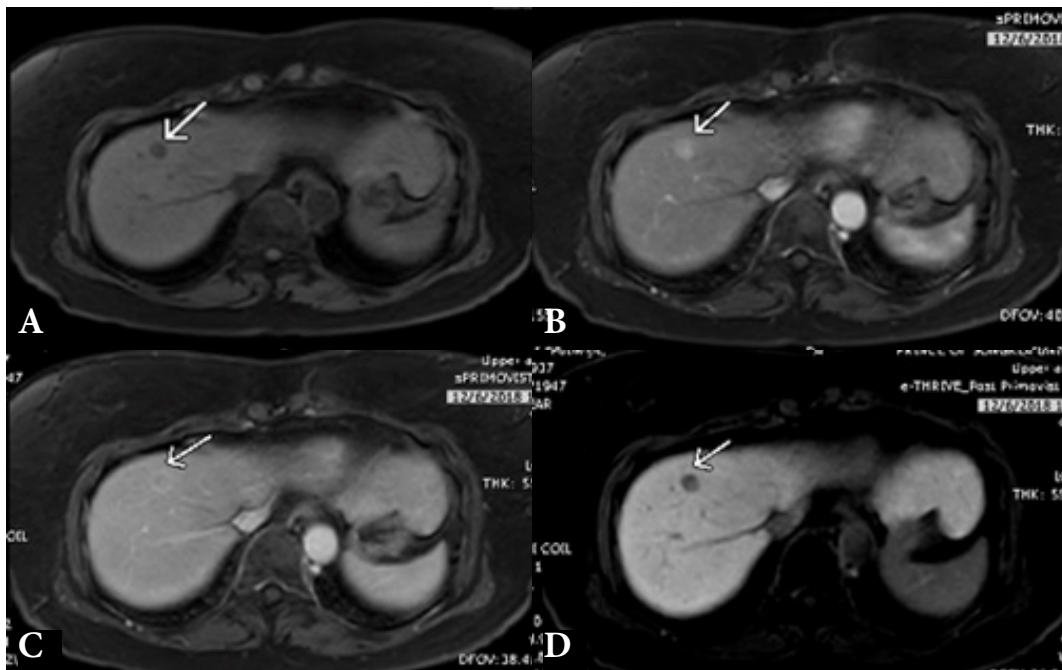


Figure 4: MRI liver A) pre-gadolinium, B) 30s, C) 60s and D) primovist 20 minutes showing a small nodule (arrow) in segment 4A/8 with early arterial hyperenhancement, rapid venous washout and no primovist uptake at 20 minutes, highly suggestive of HCC.

The latest MRI scan showed previously seen hemangioma as a T2 hyperintense lesion at segment VII without enhancement (avascular). No recurrent or new HCC was noted (Figure 5).

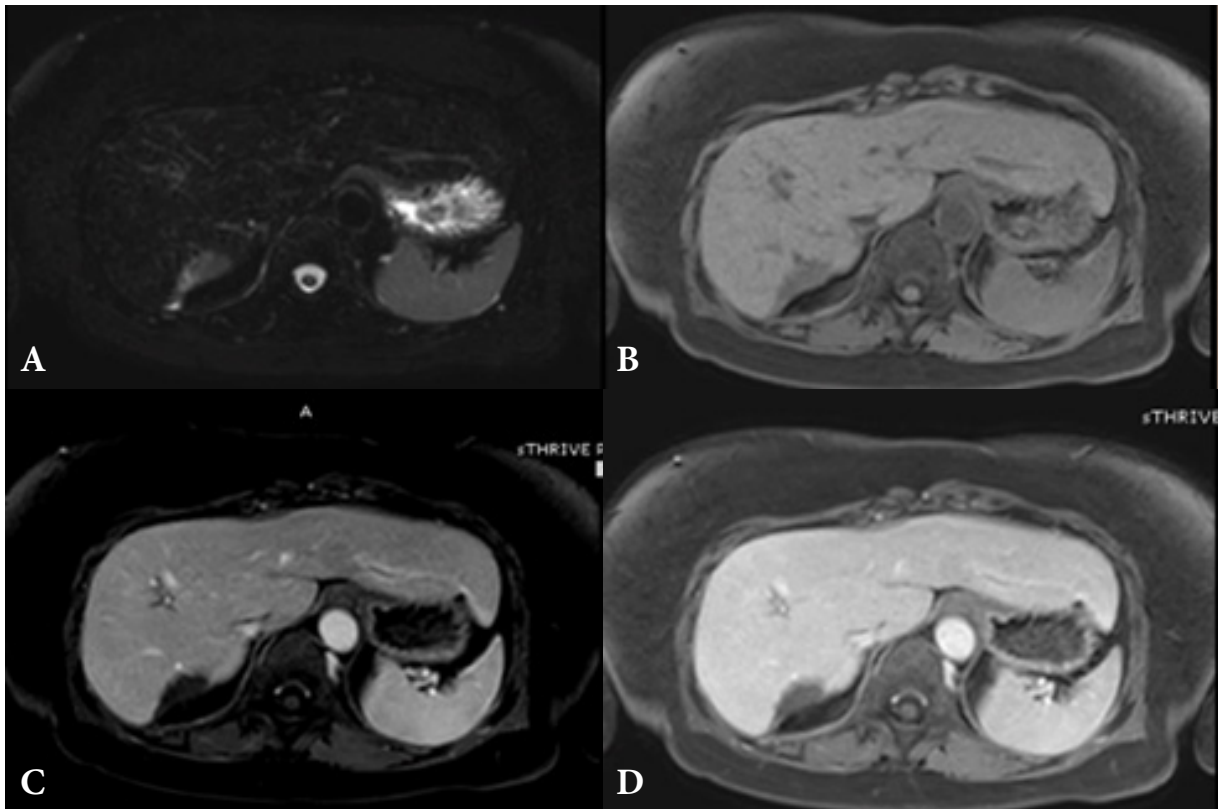


Figure 5: MRI liver A) T2*, B) pre-gadolinium, C) 60s and D) 120s showing avascular T2 hypersignal lesion in segment VII with progressive cirrhosis.

Case 2

A 52-year-old female, who is a known case of chronic hepatitis C with cirrhosis and portal hypertension, presented with a well-defined heterogeneous mass and a few small hyperechoic nodules at segment VII during routine HCC surveillance on the ultrasound scan which was likely to be hemangioma (Figure 6).

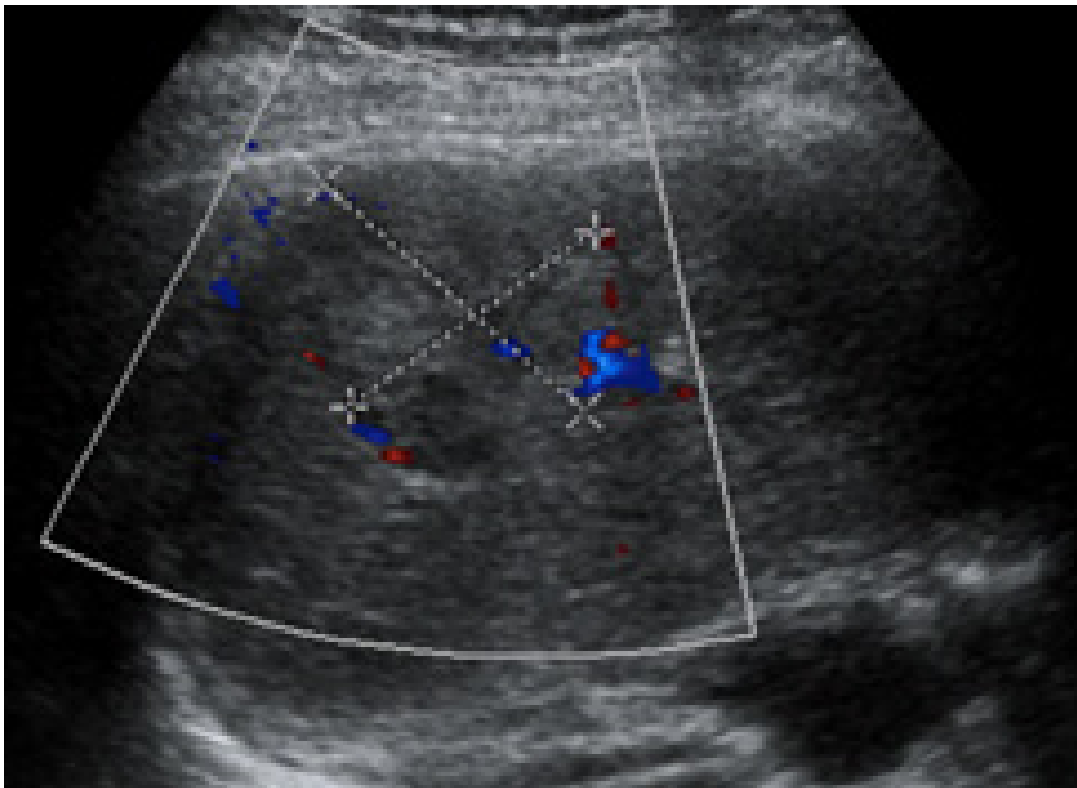


Figure 6: USS liver showing well-defined heterogeneous hyperechoic mass (marked) with internal hypoechogenicity and internal flow at segment VII, likely to be hemangioma. Small hyperechoic nodules were also seen, likely to be small hemangiomas (not shown).

Further evaluation and imaging were advised. The contrast-enhanced CT and MRI liver showed cirrhotic liver with a well-defined hypodense subcapsular mass with peripheral nodular and progressive centripetal enhancement, completely filling in, in segment VII, typical of hemangioma (Figure 7 and 8). No HCC was noted.

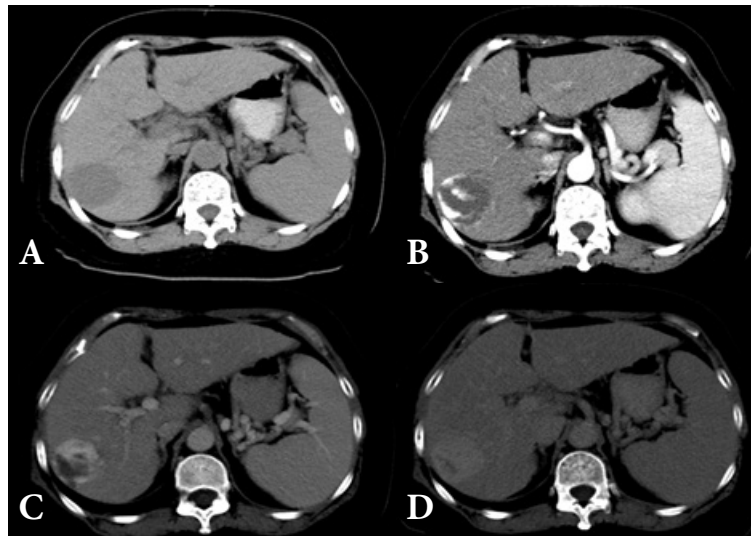


Figure 7: CT liver A) plain, B) arterial, C) venous and D) delayed phase showing peripheral nodular with progressive centripetal enhancement at segment VII, characteristic of hemangioma.

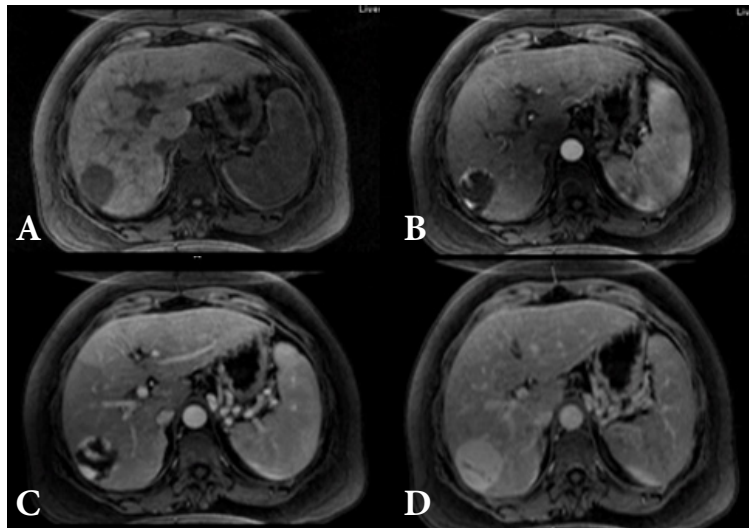


Figure 8: MRI liver A) plain, B) 30s, C) 60s and D) 120s showing peripheral nodular with progressive centripetal enhancement at segment VII, characteristic of hemangioma.

The most recent contrast enhanced CT abdomen scan showed a decrease in the size of previously seen hemangioma at segment VII with less intense and atypical enhancement pattern (Figure 9).



Figure 9: CT abdomen A) plain, B) arterial and C) venous phase showing decrease size of previously seen hemangioma (arrow) at segment VII. A less obvious typical enhancing pattern of hemangioma with enhancement seen only in some parts of the lesion.

Discussion

Hemangioma is the most common primary tumor of the liver. It has the prevalence of 0.4-20% [1-3]. It is a benign tumor consisting of vascular spaces lined by endothelial cells. The exact etiology or pathophysiology of hemangioma is not known. However, they are thought to be congenital vascular malformation or hamartoma. It is most commonly found in adults between 30-50 years old and is more common among females with a 3:1 ratio [6]. Most hemangioma are asymptomatic and are diagnosed incidentally when investigated for other pathologies. However, some hemangioma especially giant hemangioma (>10 cm), can present with symptoms such as right upper quadrant pain, discomfort, early satiety, nausea and vomiting [7]. No malignant transformation of hemangioma has been reported so far [8].

Cirrhosis is the end stage of the chronic liver injury characterized by the development of regenerative nodules and surrounding fibrous bands. According to the Global Burden of Disease (GBD), Thailand has the highest incidence of cirrhosis in Asia[9]. The common causes of cirrhosis in Asia include chronic viral hepatitis (B and C)[10]. Other causes of cirrhosis include alcoholic liver disease, non-fatty liver disease, hemochromatosis, autoimmune, cystic fibrosis and so on. In both of our cases, the patient had been diagnosed with chronic hepatitis C infection and they developed cirrhosis.

Unlike the general hepatic hemangioma prevalence, the prevalence of hemangioma in the cirrhotic liver is considered to be very low ranging from 1.2-1.7 % [4, 5]. However, these data were older than two decades ago which was based exclusively on histopathological findings (autopsies and surgical specimens). According to the more recent study, the prevalence of the hepatic hemangioma in cirrhosis was 8.8% and there was no statistically significant difference with the prevalence in the non-cirrhotic liver[11]. Now with the advancement in both technologies, protocols, better understanding of the histopathology as well as the increasing amount of imaging (CT/MRI), the prevalence of hemangioma is expected to be much higher than previously reported.

Many believe that as the cirrhosis progresses, it alters the morphology and hemodynamics of the hemangioma[12]. The previous studies have shown that the hemangioma in cirrhosis tends to be smaller in size which could be attributable to obliteration from fibrosis seen in cirrhosis[13–15]. One case in a study even reported a complete disappearance of hemangioma in cirrhosis[14]. Dodd et al demonstrated extensive fibrosis within and surrounding hemangioma in cirrhotic liver specimens[4]. The decreased blood flow in cirrhosis is thought to alter hemodynamics of the hemangioma. Typically, the hemangioma will appear as well-defined hypodense or pre-gadolinium hypointense in the unenhanced image and after contrast injection; it will show characteristic peripheral nodular and progressive centripetal enhancement, matching with the blood pool all the time[7]. Additionally, in T2 and T2* MRI sequence, the hemangioma will appear

bright and 'light-bulb' bright respectively. However, in case of small hemangiomas (<2 cm), they might show homogenous enhancement described as flash-filling while giant hemangioma might show incomplete central filling in. All these characteristic enhancing patterns of hemangioma are either lost or not obvious in cirrhosis[16]. In both of our cases, they presented with a well-defined subcapsular lesion in segment VII showing peripheral nodular and progressive centripetal enhancement typical of hemangioma. The hemangioma in both patients was hyperintense in T2 and light bulb bright in T2*. No active biopsy or intervention was done as the imaging features were characteristic of hemangioma.

During the follow-up in both cases, the hemangioma gradually decreased in size. The intensity of the enhancement also decreased with rather incomplete centripetal filling in. In the first case, the hemangioma eventually became avascular without any enhancement while in the second case, minimal enhancement was observed in some parts of the lesion. No hemangioma associated capsular retraction was seen which is in agreement with Soyer et al which stated that capsular retraction adjacent to the hepatic tumor is a specific sign of hepatic neoplasm [17].

HCC is the fifth commonest cause of cancer and the second commonest of cancer-related deaths in the world[18]. Cirrhosis is a known risk factor for HCC with >90% HCC developing in the cirrhotic liver[19]. The risk of HCC in HCV-infected patients is increased by 15- to 20-fold, with an annual incidence of HCC being estimated at 1-4% in cirrhotic over a 30-year period[20]. The first case eventually developed HCC at segment 4A/8 and was treated successfully with two sessions of sTACE.

Conclusion

The prevalence of the hepatic hemangioma in the cirrhotic liver might be higher than previously estimated. The cirrhosis alters the morphology and hemodynamics of hepatic hemangioma resulting in a decreased size and loss of the characteristic enhancement pattern.

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