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## CT of Hepatic Sarcoidosis: Small Nodular Lesions Simulating Metastatic Disease

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Summary	
<b>Background:</b>	Sarcoidosis is a multisystemic inflammatory disease of unknown origin. The lymphoid system and the lungs are the most commonly involved organs. The frequency of signs or symptoms of hepatic involvement is very low.
<b>Case Report:</b>	We present a case of symptomatic granulomatous liver disease secondary to sarcoidosis, mimicking a metastatic disease on ultrasonography and CT.
<b>Conclusions:</b>	Hepatic involvement in sarcoidosis might be a perplexing diagnostic problem. The decisive CT finding with respect to the differential diagnosis was the absence of a mass effect and intact vascular architecture around the lesions.
<b>MeSH Keywords:</b>	<b>Cholestasis • Liver • Multidetector Computed Tomography • Neoplasm Metastasis • Sarcoidosis</b>
<b>PDF file:</b>	<a href="http://www.polradiol.com/abstract/index/idArt/893330">http://www.polradiol.com/abstract/index/idArt/893330</a>

### Background

Sarcoidosis is a multisystemic non-caseous granulomatous disease of unknown origin. It emerges as hilar adenopathy and interstitial lung disease in most of the cases. The central nervous system, bones, skin, eyes, liver and spleen can also be involved [1]. Fifty to 65% of patients with sarcoidosis have histologically proven liver involvement. Therefore, patients are usually asymptomatic for hepatic involvement [2]. Sarcoidosis presents as focal nodular, hypodense lesions in the liver and spleen that can be seen on computed tomography (CT) [3].

### Case Report

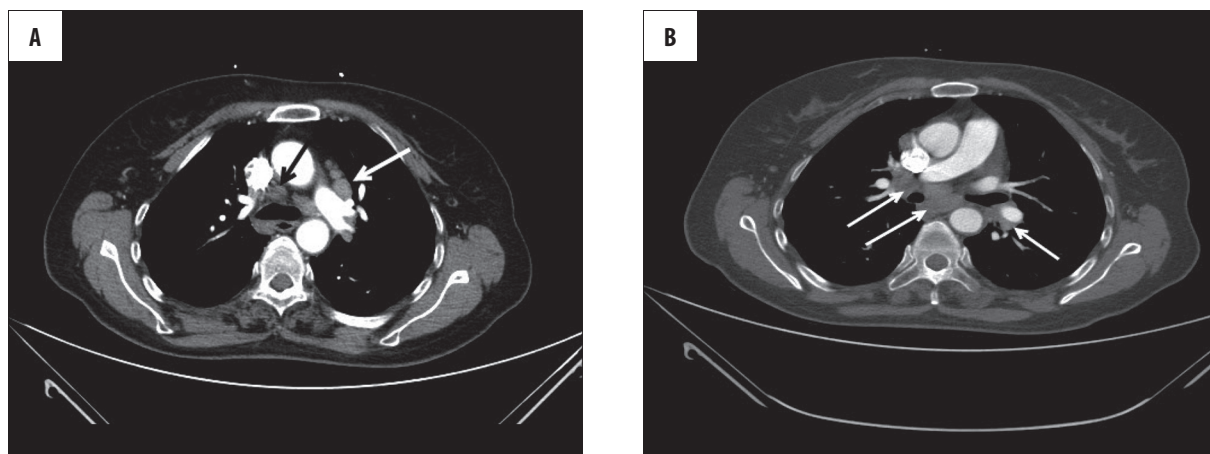
A 67-year-old female with elevated liver function tests and increased serum tumor markers (CA 125, CA 19-9 and CA 15-3) was referred from the hepatology department for a CT scan of the chest and abdomen to assess the characteristics of nodular liver lesions that had recently been identified on ultrasonography. According to the patient's history, she had been complaining for approximately 2 months of generalized weakness, fever and mild scleral jaundice. Her past medical history was unremarkable. Family history was not significant for the presence of any malignancy or gastrointestinal disorder. Laboratory

investigations revealed elevated liver enzyme levels such as;  $\gamma$ -glutamyl transpeptidase (1668 U/L, reference value 6–42 U/L), alanine transaminase (201 IU/L, reference value 4–33 IU/L), aspartate transaminase (163 IU/L, reference value 4–32 IU/L), alkaline phosphatase (2098 IU/L, reference value 35–105 IU/L), total bilirubin (1.83 mg/dL, reference value <1.2 mg/dL), direct bilirubin (1.62 mg/dL, reference value <0.2 mg/dL). Serum CA 125 (86 U/mL, reference value 0–35 U/mL), CA 19-9 (50 U/mL, reference value 0–27 U/mL), and CA 15-3 (38 U/mL, reference value 0–28 U/mL) levels were also increased, as mentioned before.

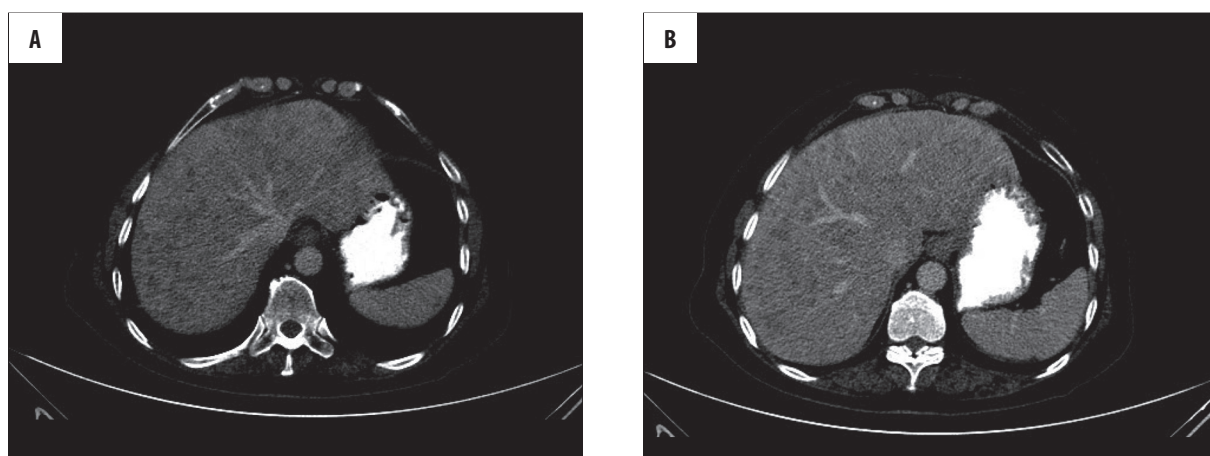
### Results

Contrast-enhanced CT of the chest showed lymph nodes in the bilateral hilar region and mediastinum, with a maximum short axis diameter of 14 mm (Figure 1A, 1B). The lung parenchyma was normal. Abdominal CT revealed multiple hypodense, ill-defined, variable-sized (ranging from 5 mm to 15 mm in diameter) nodular lesions in the liver. The most striking characteristic of the lesions was the absence of a mass effect. Vascular architecture in the region of the lesions around the portal and hepatic veins was intact (Figure 2A, 2B).

Ultrasound-guided tru-cut liver biopsy using an 18-gauge needle showed typical sarcoidosis with granulomatous



**Figure 1.** Contrast enhanced axial CT scan of the thorax. The images demonstrate (A) precarinal (black arrow), subaortic (white arrow) (B) bilateral hilar and subcarinal lymphadenopathies (white arrows).



**Figure 2.** (A, B) Enhanced axial CT scan of the abdomen. The images demonstrate multiple hypodense ill-defined, variable-sized, nodular lesions. Note the intact vascular architecture around the lesions.

inflammation without caseating granuloma or acid-fast bacilli.

## Discussion

Sarcoidosis is a multisystem disease of unknown etiology characterized by proliferation of noncaseating granulomas. The mediastinal and hilar lymph nodes and lung parenchyma are the most commonly affected sites, being involved in up to 90% of patients [1]. Liver biopsy demonstrates granulomas histologically in 50–65% of the patients. Therefore, sarcoidosis of the liver is often histologically present in patients but usually patients have no symptoms related to liver disease [2]. Symptomatic liver disease occurs in less than 5% of patients with sarcoidosis. Systemic symptoms are represented by fever, physical exhaustion and jaundice (consistent with cholestasis) which were also present in our patient because of the hepatic involvement. Chronic intrahepatic cholestasis due to sarcoidosis can mimic sclerosing cholangitis or primary biliary cirrhosis [3,4].

The most common abnormal presentation of liver function tests in hepatic sarcoidosis is an elevation in serum alkaline phosphatase level. However, this finding is only present in as few as 15% of patients with histological evidence

of the disease. Hyperbilirubinemia may also rarely occur with chronic progressive disease [5]. Both of these manifestations were present in our patient.

Hepatomegaly is the most common liver abnormality seen on CT but was not present in our case [6,7]. Multiple focal hepatic lesions, most of which are small (mean diameter of 1.0 cm) are found in less than 5% of patients with sarcoidosis. Hepatic nodules are seen less frequently than splenic nodules. Although nodular lesions are reported to be seen mostly in patients presenting with hepatomegaly, our patient had normal liver size with multiple nodular lesions [7]. Differences in immunological response to the disease may be a possible reason for the difference.

The differential diagnosis of hypodense multiple hepatic nodules includes tuberculosis, metastatic disease, fascioliasis, candidiasis, Langerhans' cell histiocytosis (LCH), and lymphoma. Hepatic tuberculosis is divided into two groups: miliary form and local form. Miliary pattern is seen as multiple hypodense nodules mostly less than 3 mm in diameter on CT. The local form manifests as multiple hypodense hepatic lesions with hepatomegaly or a single tumorlike mass. The definitive diagnosis of hepatic tuberculosis can only be made by histopathological

examination [8]. After intravenous contrast medium injection, liver metastases usually enhance less than the surrounding liver. The enhancement pattern is typically peripheral and a washout of contrast medium from the lesions is seen in the delayed phase [9]. Hepatic fascioliasis includes multiple, small, hypodense lesions, with peripheral contrast enhancement on CT. Focal thickening of the liver capsule and subcapsular, low-attenuation branching tracts can be demonstrated on CT [10]. Hepatic candidiasis is usually observed in immunocompromised hosts. On contrast-enhanced CT, fungal microabscesses usually appear as multiple, discrete, low-density, nodular lesions of similar size with peripheral contrast enhancement [11]. Hepatic Langerhans' cell histiocytosis (LCH) presents as a focal nodular disease. Accompanying lytic bone lesions are important for the differential diagnosis [12]. Solid, hypodense,

nodular lesions are also seen in hepatic lymphoma but existence of lymphadenopathies with periportal location of the lesions and disruption of the vascular architecture are helpful in making the differential diagnosis. Hepatic involvement in lymphoma is often seen as a secondary form in patients with known disease [13].

## Conclusions

Hepatic involvement in sarcoidosis might be a perplexing diagnostic problem. The decisive CT finding with respect to the differential diagnosis was the absence of a mass effect and intact vascular architecture around the lesions. In addition, bilateral hilar and mediastinal lymphadenopathy is most commonly identified by a chest x-ray or CT.

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