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Case Report

Necrotizing granulomatous inflammation of the liver

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ABSTRACT

A 73-year-old patient with necrotizing granulomatous inflammation of the liver is presented. The computed tomography demonstrated 2 hypodense tumors with progressive enhancement in the liver. They became nearly isodense to the normal hepatic parenchyma on the delayed phase.

Introduction

Necrotizing granulomatous inflammation (NGI) is usually caused by *Mycobacterium tuberculosis*. It usually occurs in the lung. The extrapulmonary sites commonly include lymph node, pleura, and joints, although any organ may be involved [1]. However, its causes still remained unexplained in nearly 20%-40% of cases [2,3]. We present the computed tomography (CT) findings of a case of NGI of the liver with negative results of Ziehl–Neelsen stain for acid-fast bacilli and sputum culture for *M tuberculosis*.

Case report

A 73-year-old woman consulted the medical department with the complaint of intermittent upper abdominal pain for several weeks. She had a history of cervical squamous cell carcinoma; stage Ib, 7 years ago. She received radical hysterectomy and was regularly followed up in the gynecologic department in this 7-year period. For the present complaint, an ultrasound examination was performed and 2 mixed echoic tumors, about 2.5-4.5 cm in size, were found in right lobe of the liver. The patient was not a hepatitis B or C carrier.

Competing Interests: The authors have declared that no competing interests exist.

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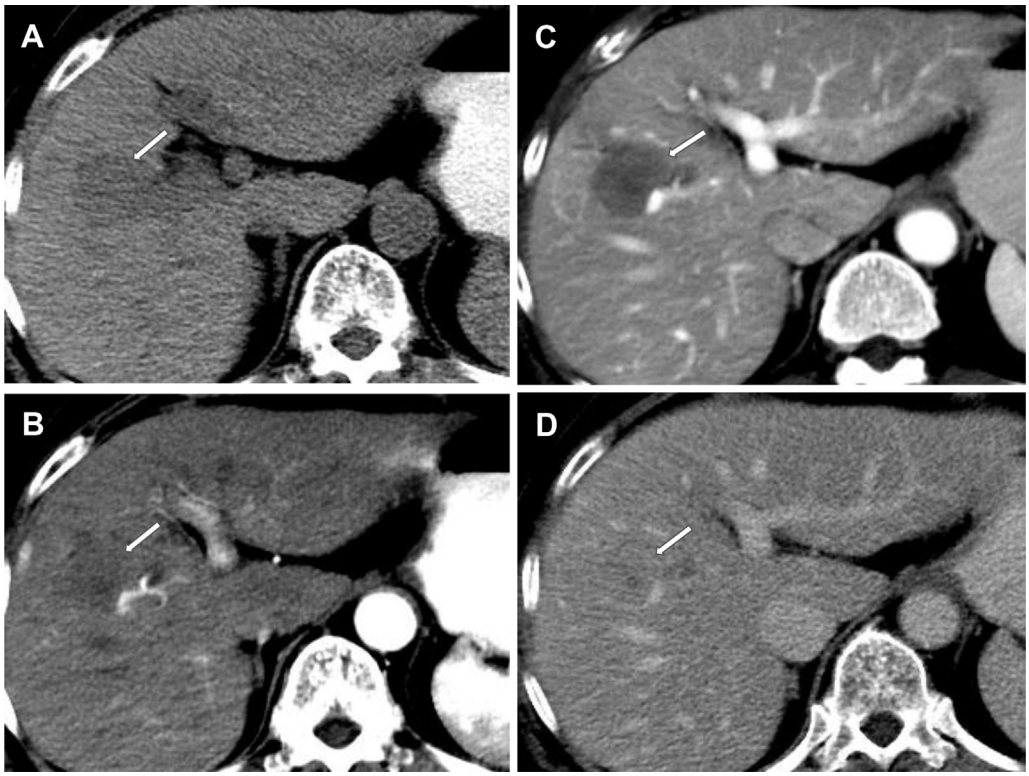

Fig. 1 – (A) Precontrast axial image. There is a hypodense tumor (arrow) in S8 of the liver. The density of the tumor was about 50 Hounsfield units (HUs). (B) Arterial phase of postcontrast scan. The hypodense tumor (arrow) did not show obvious enhancement. Its density was about 60 HUs. (C) Postcontrast image 30 seconds after Fig. 1B. The hypodense tumor (arrow) showed a moderate enhancement with a density of 90 HUs. (D) Postcontrast image 2 minute and 20 seconds after Fig. 1B. The whole tumor (arrow) became nearly isodense, about 100-105 HUs, to the normal liver parenchyma.

The serum carbohydrate antigen-199, carcinoembryonic antigen, and alpha-fetoprotein levels were all within normal limits. The other biochemical data were nonspecific. The chest plain film showed linear fibrotic scars and a soft-tissue nodule, about 26 × 12mm in size, in left upper lobe. A subsequent CT examination demonstrated 2 hypodense tumors, about 44 × 42 × 26 mm and 30 × 24 × 22 mm in sizes, in S4-S8 and S8 of the liver on precontrast scans. After intravenous contrast medium administration, both tumors showed a poor enhancement on the arterial phase. They were progressively enhanced and became nearly isodense to the normal parenchyma on the delayed phase, about 2 minutes and 20 seconds later to that of arterial phase (Fig. 1). The adjacent vessels were not involved. Under a presumptive CT diagnosis of cholangiocarcinom, she was admitted to receive a partial right lobectomy of the liver. The gross specimen showed 2 white, slightly yellowish, solid tumors in S4-S8 and S8 of the liver without obvious cavitation (Fig. 2A). The microscopic examinations described epitheliod granulomas, chronic inflammatory cell infiltrations, granulation tissue, and marked fibrosis (Fig. 2B). Langhans giant cells and caseous necrosis were present (Fig. 2C and D). Neither acid-fast bacilli nor fungi could be identified in the acid-fast or periodic acid-Schiff stains. Two lymph nodes removed from the hepatoduodenal ligament also showed granulomatous inflammation without acid-fast bacilli or fungi. Because of the negative stain results, a pathologic diagnosis of NGI of the liver was made. The patient recovered uneventfully and was discharged. A total of 7 times of sputum culture and acid-fast stains all revealed negative results of *M tuberculosis* in the after 8-month period. The later chest plain films showed no significant change of linear fibrotic scars and a soft-tissue nodule in left upper lobe. Even so, she still received anti-tuberculosis (TB) treatment and was followed up in the infectious diseases department.

Discussion

A granuloma is a focal compact collection of inflammatory cells. It is usually the end result between the invading organisms or antigens, which are failed to be removed by the host, and the persistent active cell-mediated hypersensitivity. The local inflammation attracts monocyte macrophages. These macrophages may fuse to form multinucleated giant cells or transform to epithelioid cells [4]. Necrotizing granuloma indicates the presence of tissue necrosis in the granuloma. Its causes were found to be infectious in 71% and remained unexplained in 26% after thorough study [2]. A similar result as infectious in 42% and unexplained in 39% has also been reported [3].

Granuloma formation in the liver includes a variety of conditions, most commonly tuberculosis, sarcoidosis, and hisplasmotosis [5]. They are usually discrete, sharply defined nodular infiltrates composed of epitheliod granulomas