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Hepatic Kaposi Sarcoma in AIDS: US and CT Findings[1]

Abdominal ultrasonography (US) and computed tomography (CT) were performed in two patients with acquired immunodeficiency syndrome (AIDS) and necropsy-proved hepatic Kaposi sarcoma. At US, small (5–12-mm) hyperechoic nodules and dense periportal bands were seen in the liver. These lesions appeared hypoattenuated on baseline and dynamic CT scans and enhanced on delayed scans after a bolus injection of contrast material. Although nonspecific, these features strongly suggest tumor involvement in the liver in patients with AIDS and Kaposi sarcoma.

Index terms: Acquired immunodeficiency syndrome (AIDS) • Kaposi sarcoma, 761.349 • Liver neoplasms, CT, 76.1211 • Liver neoplasms, diagnosis, 761.349 • Liver neoplasms, US studies, 761.1298

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KAPOSI sarcoma in patients with acquired immunodeficiency syndrome (AIDS) is an aggressive, multicentric neoplasm frequently involving the skin and visceral organs and occurs mainly in homosexual men. Hepatic Kaposi sarcoma is rarely diagnosed during life, although autopsy studies have demonstrated tumor involvement in the liver in 34% of cases (1).

The computed tomographic (CT) and ultrasonographic (US) findings in two patients with AIDS and necropsy-proved hepatic Kaposi sarcoma are described.

CASE REPORTS

Case 1.—In May 1986, a 38-year-old bisexual man had a diagnosis of cutaneous and ganglionar biopsy-proved Kaposi sarcoma and AIDS. Liver function tests disclosed the following elevated values: alkaline phosphatase, 573 IU/L; aspartate aminotransferase, 147 IU/L; and alanine aminotransferase, 180 IU/L. Abdominal US revealed hepatomegaly and thickened periportal bands around the porta hepatis and peripheral branches. Multiple, small, peripheral, hyperechoic nodules were also seen and apparently did not involve the portal veins (Fig 1a). CT was performed on a Somatom DR3 scanner (Siemens Medical Systems, Erlangen, Federal Republic of Germany). Baseline hepatic CT showed enlarged hilar and peripheral vascular areas. A dynamic CT study (8-mm-thick sections at 10-mm intervals, 3-second scanning time, 4-second interscanning delay time) was rapidly per-

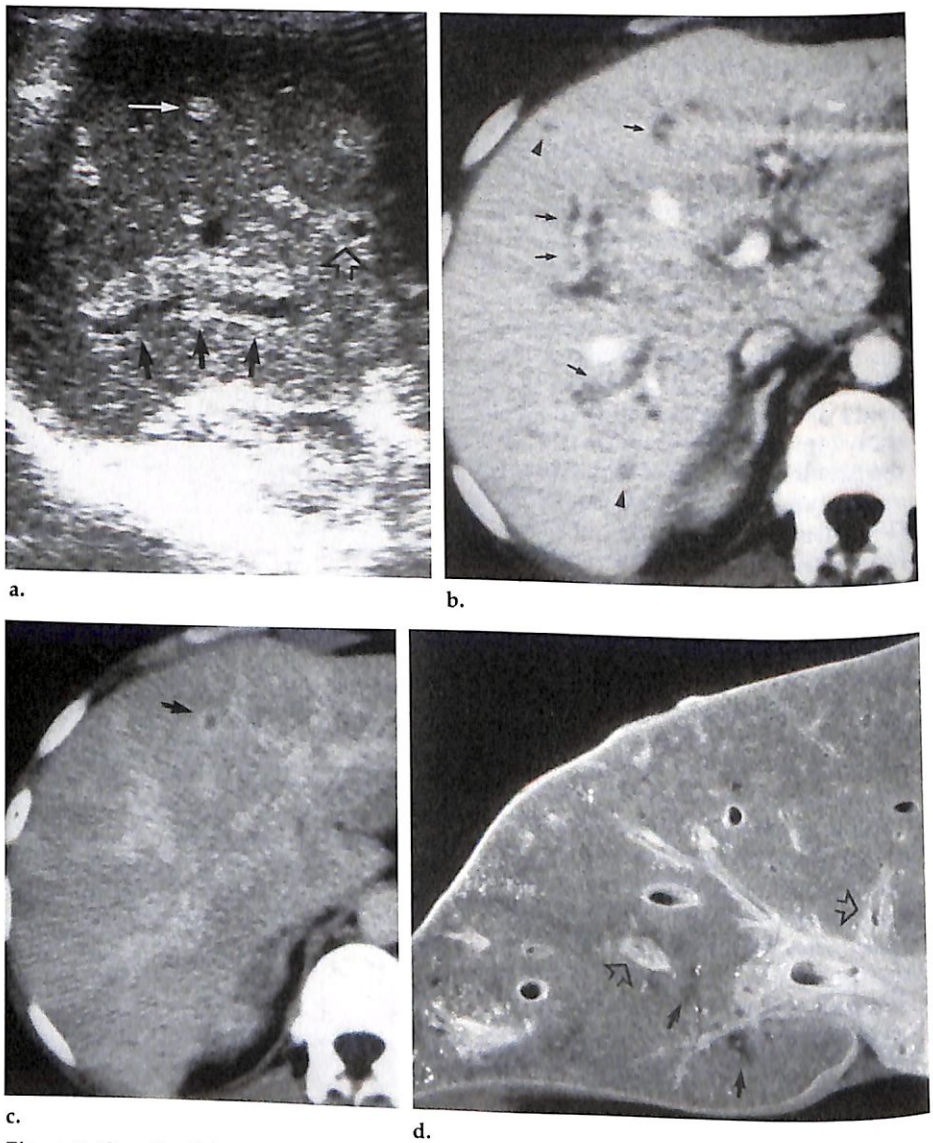

Figure 1. Case 1. **(a)** Oblique US scan of the right lobe of the liver shows prominent hyperechoic bands encasing portal veins in both longitudinal (black arrows) and cross-sectional (open arrow) views. Small hyperechoic nodules (white arrow) are seen. **(b)** Dynamic CT scan shows low-attenuation periportal tissue (arrows) throughout the liver and small peripheral hypoattenuated nodules (arrowheads). **(c)** Delayed CT scan obtained at same level as that in **b** shows marked enhancement of the low-attenuated lesions with a linear and branching pattern. Some lesions have only a rim of enhancement (arrow). **(d)** Photograph of gross specimen demonstrates fibrous thickening of portal tracts (open arrows) and dark red nodules in the periportal areas and diffusely infiltrating the liver parenchyma (arrows).

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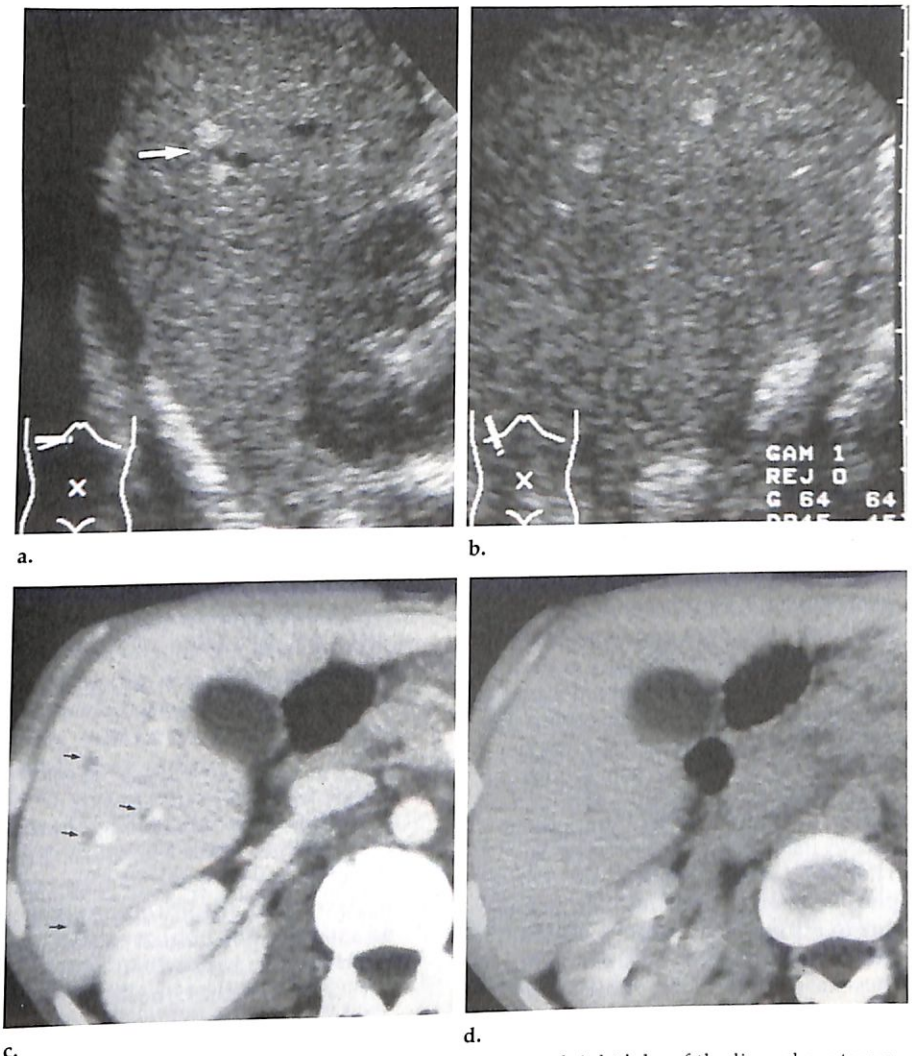

Figure 2. Case 2. Axial **(a)** and oblique **(b)** sonograms of right lobe of the liver show hyperechoic Kaposi sarcoma nodules, some clearly adjacent to portal veins (arrow). **(c)** Dynamic CT scan shows low-attenuation periportal lesions grossly mimicking dilated biliary ducts (arrows). **(d)** Delayed CT scan obtained at same level as that in **b** shows that the lesions have enhanced and are not visible.

formed after a bolus of 100 mL of contrast material (iohexol 64.7% [Omnitrast 300]; Schering, Berlin) was injected by hand. Low-attenuated tissue around hilar and distal portal branches (Fig 1b) was demonstrated during both the arterial and portal phases of the study. The vena porta was not enlarged. On delayed scans obtained 7 minutes after the bolus injection, most of the low-attenuation lesions enhanced and showed attenuation higher than that of the surrounding liver (Fig 1c). A few of the lesions had only a peripheral rim of enhancement. Two US-guided percutaneous biopsies of liver lesions did not disclose any abnormality.

The patient received chemotherapeutic treatment for 3 months. Although partial cutaneous response was obtained, his general condition worsened, and the patient died several weeks later. At autopsy, widespread cutaneous and visceral Kaposi sarcoma was found. In the liver, pathologic features corresponded well with the imaging findings (Fig 1d). Kaposi sarcoma was seen involving the portal tracts and parenchyma. The former were expanded, with dense collagenous sheaths around arteries and veins. This fibrous tissue contained small amounts of hemosiderin and had poorly formed slitlike spaces lined by slightly atypical spindle cells. In addition, groups of dilated blood-filled cavities reminiscent of cavernous hemangiomas extended from the portal triads into the hepatic lobules.

All the lesions found at US and CT, including periportal or parenchymal lesions, were assumed to be Kaposi sarcoma. However, the limitations of this study include the length of time between CT examination and autopsy, the chemotherapy administered during that time, and the lack of a direct radiologic-pathologic correlation for each lesion.

Case 2.—In July 1984, a 40-year-old homosexual man had a diagnosis of biopsy-proved cutaneous Kaposi sarcoma and AIDS. The patient refused any treatment at that time. In May 1988, he was admitted because of numerous cutaneous Kaposi sarcoma lesions and lymphadenopathy. Kaposi sarcoma was detected at biopsy of lymph nodes. Results of liver function tests were normal. At US, three small (7–12-mm) hyperechoic nodules, having the appearance of angiomas, were seen in the liver. The patient underwent treatment with combination chemotherapy, and cutaneous response was considered to be total. At follow-up US, the size of two of the hepatic lesions had decreased, and the third was not visible. The patient was readmitted 6 months later because of a severe relapse of cutaneous Kaposi sarcoma. US showed bilateral pleural effusion, mild hepatomegaly, and multiple nodules throughout the liver, some of which were close to the portal veins (Fig 2a, 2b). Baseline CT disclosed two low-attenuation nodular lesions (1 cm) in the hepatic edge. Results at dynamic CT—performed in the same way as in case 1—helped confirm the lesions and revealed multiple hypoattenuated nodules adjacent to the portal vessels (Fig 2c). On delayed CT scans obtained 4 minutes after bolus injection of contrast material, the periportal lesions became isoattenuated and were indistinguishable from the rest of the liver (Fig 2d). The two peripheral foci revealed at baseline CT remained hypoattenuated. Although chemotherapy was reinitiated, the general condition of the patient rapidly deteriorated, and he died 1 month later. Wedge-shaped samples of hepatic tissue taken postmortem showed ill-defined purple macular lesions on the surface. At histologic examination, the lesions proved to be Kaposi sarcoma, displaying periportal groups of dilated, blood-filled cavernous spaces lined by flat endothelial cells and interspersed with bundles of spindle cells. Among these, there were slitlike spaces containing extravasated erythrocytes and minimal hemosiderin deposits. No significant cellular atypism was present.

DISCUSSION

Although Kaposi sarcoma is the most common intrahepatic neoplasm in AIDS, it remains undetected antemortem in most cases. Hepatic Kaposi sarcoma is usually seen in a perivascular location at autopsy (1–4). The tumor is multifocal; tends to originate in the capsular, hilar, and portal areas; and invades the parenchyma from these sites. Focal areas of involvement may have the gross appearance of purple-brown nodules, "hemorrhage" within expanded portal tracts, or even angiomas of the liver. At histologic examination, Kaposi sarcoma is characterized by proliferation of endothelial cells, extravasated erythrocytes, fibroblastic proliferation with spindle cells, and vascular lakes involving predominantly periportal areas. Other vascular hepatic lesions have been reported in patients with AIDS: sinusoidal dilatation (5), peliosis hepatis (blood-filled cystic spaces), and angiosarcomatous changes. It has been suggested that they may represent transitional phases in the development of Kaposi sarcoma (6).

When present, clinical findings in hepatic Kaposi sarcoma, are nonspecific. Hepatomegaly and elevation of serum alkaline phosphatase levels—related to the infiltration of the hepatic hilum and portal branches—may be seen. However, these abnormalities are encountered in nearly two-thirds of patients with AIDS and may be due to opportunistic intrahepatic infections (eg, *Mycobacterium avium-intracellulare*, *M tuberculosis*, fungi, and cytomegalovirus) or other neoplasms (such as lymphoma) (2). Cholangitis caused by cytomegalovirus or *Cryptosporidium* species may also result in anicteric cholestasis (7). Percutaneous liver biopsy has a high diagnostic yield in cases of opportunistic intrahepatic infections or lymphoma (2). However, positive findings at liver biopsy in hepatic Kaposi sarcoma have been reported in only two cases (6,8). False-negative results may occur and might be due to sampling error, since most of the biopsies were not performed with US or CT guidance. On the other hand, Kaposi sarcoma shows a number of unusual histologic patterns (1). Our first case illustrates two of them, namely, the so-called sclerotic and cavernous hemangioma variants. These are known to occur in African patients and have also been described occasionally in patients with AIDS. These unusual patterns, which bear minimal or no cytologic atypia, may be very difficult to diagnose in biopsy material.

In the study by Moon et al (9), abdominal CT revealed mild hepatomegaly, a nonspecific finding, in 19% of patients with AIDS-related Kaposi sarcoma. However, imaging evidence of hepatic Kaposi sarcoma during life has been reported in only two cases (9,10), probably because the lesions tend to be microscopic or too subtle to identify radiographically. In the case reported by Moon et al (9), multiple hypoattenuated nodular lesions were seen in the liver at CT. In the case described by Defalque et al (10), an echogenic periportal infiltration and small hyperechoic nodules were shown at US, with this abnormal tissue being hypoattenuated on nonenhanced and contrast material–enhanced CT scans. No delayed scanning was performed in either case.

Awareness of the US and CT features of hepatic Kaposi sarcoma may result in an increase in the cases detected antemortem. In our patients, abdominal US showed mild hepatomegaly and multifocal hyperechoic nodules, ranging in size from 5 to 12 mm, both adjacent to portal veins and diffusely infiltrating the liver parenchyma. Follow-up US studies showed a progressive increase in the number of Kaposi sarcoma nodules. In one patient, hyperechoic periportal bands were also noted. CT findings corresponded well with those seen at US. Baseline CT revealed small hypoattenuated nodules and an irregular enlargement of hilar and peripheral portal branches. Dynamic CT revealed multiple, discrete, low-attenuation nodules and periportal tissue. Dynamic enhanced scans depicted more lesions than baseline scans. On delayed scans performed 4–7 minutes after bolus injection of contrast material, most of the lesions enhanced and became either homogeneous with, or more attenuated than the surrounding parenchyma. To our knowledge this finding is previously unreported in hepatic Kaposi sarcoma and is related to the vascular nature of the tumor. In both cases, some lesions had atypical characteristics: They did not enhance or showed only peripheral enhancement. This may be due to intranodular thrombosis. Additional delayed scans, not obtained in these cases, might have shown complete enhancement. A direct radiologic-pathologic correlation for each lesion is not available, although we believe that all the lesions are Kaposi sarcoma. No other disease was found in the samples.

When the hepatic pattern of Kaposi sarcoma is predominantly nodular, considerations in the differential diagnosis should include angiomas, fungal microabscesses, and hepatic metastases. Focal hepatic involvement in other common AIDS-related diseases, such as mycobacterial infections or lymphoma, generally produce hypoechoic lesions at US. Prominent hyperechoic periportal bands appearing hypoattenuated at CT and showing enhancement after administration of contrast material have been described in hepatic schistosomiasis mansoni (11,12).

In summary, the described US and CT findings, although nonspecific, strongly suggest hepatic involvement in Kaposi sarcoma. US may be a useful screening technique for the identification of these lesions when hepatic involvement is suspected. Dynamic and delayed enhanced CT scans may help characterize such lesions. ■

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