
Cat-Scratch Disease: Report of a Case with Liver Lesions and No Lymphadenopathy

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The usual presentation of Cat-scratch disease (CSD) is a subacute regional lymphadenitis following cutaneous inoculation. We present the case of a 10-yr-old white female with a 4-wk history of abdominal pain and fever, without associated lymphadenopathy. A ^{67}Ga scintigram showed inhomogeneous uptake by the liver. An abdominal computed tomographic (CT) scan revealed multiple low density lesions in the liver and the spleen, that were confirmed at laparotomy. Stellate microabscesses were seen on a wedge biopsy of the liver and a CSD antigen skin test was positive. CSD should be considered in the differential diagnosis of liver lesions, even in the absence of lymphadenopathy. This case emphasizes the importance of inhomogeneous ^{67}Ga uptake by the liver.

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Cat-scratch disease (CSD) most frequently presents as a regional chronic lymphadenitis, associated with a primary dermal lesion, a cat scratch in 62% of the cases (1). The incubation period ranges from 3-30 days, but is usually 7-12 days. Lymph node enlargement usually last 4 to 6 wk, but can persist for 12 mo. Fever above 102°F is present in 30% of cases. Another manifestation of the disease is the oculoglandular syndrome if the primary lesion affects the eye. In most of cases, the course of the disease is benign. Rare complications include encephalopathy, encephalitis, thrombocytopenic purpura, and osteolytic bone lesions. Mesenteric lymphadenitis, pneumonia, arthralgia, subacute iriditis, lymphedema and thyroiditis, have been described (1). Liver involvement has been described in only a few cases (2-5), and focal liver lesions have been demonstrated only in a single case (5). All these cases presented with associated lymphadenopathy.

The etiologic agent of CSD has only recently been identified. It is believed to be a small gram negative bacillus, best seen with the Warthin-Starry silver stain or the tissue gram stain in the biopsy sections of the lesions. However this organism has not yet been isolated in culture (6). Lymph node biopsy shows follicular hyperplasia in the early stage, granulomatous changes in the intermediate stage, and macro- and microscopic

abscesses with stellate necrosis and neutrophils surrounded by palisading histiocytes in the late stage.

We present an unusual case of CSD without lymphadenopathy but with diffuse liver and spleen microabscesses seen on ^{67}Ga scintigram, CT and laparotomy. A CSD antigen skin test was positive and a liver biopsy confirmed the diagnosis.

CASE REPORT

A 10-yr-old white female presented with a 4 wk history of abdominal pain and daily spikes of fever up to 105°F. One week into her illness, she was treated in a peripheral hospital with sulfamethoxazol and trimethoprim (Bactrim) per os for a suspected urinary tract infection, followed with ampicillin intravenously, and finally vibramycin per os for suspected Rocky Mountain spotted fever. However, her symptoms did not improve, and she was transferred to Vanderbilt University Medical Center on July 19, 1987. At the time of admission, the abdominal pain was localized in the left upper quadrant. Social history revealed that several cats were living at home, and the patient had been scratched prior to the onset of her illness. Physical examination was notable for a temperature of 103°F, slight hepatosplenomegaly, and tender abdomen with slight guarding but no rebound. There was no lymphadenopathy detected in the cervical, supraclavicular, axillary, or inguinal regions. Abnormal laboratory findings included a white blood cell count of 16,600/mm³, with a differential of 79% segmented neutrophils, 3% band forms, 14% lymphocytes, and 4% monocytes. The red blood cell count was 3.76 million/mm³, hemoglobin 9.9 gm/dl and the platelet count 710,000/mm³. The sedimentation rate was 130 mm/hr. Liver

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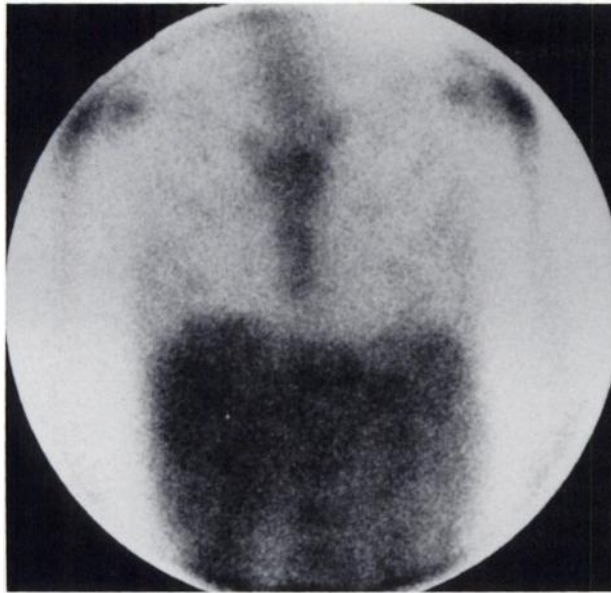


FIGURE 1
Anterior view of the abdomen obtained 48 hr following intravenous injection of 3.5 mCi of [⁶⁷Ga]citrate, showing inhomogeneous uptake in the liver, the areas of increased uptake correspond to microabscesses.

function tests were slightly abnormal at admission and reached their peak 1 wk later: Serum glutamic oxaloacetic transaminases 293 U/ml (normal = 4–40), serum glutamic aspartic transaminases 341 U/ml (normal = 4–40), alkaline phosphatases 580 U/ml (normal = 4–110), and lactic dehydrogenases 337 U/ml (normal = 150–250). A cat-scratch antigen skin test was positive. A ⁶⁷Ga scintigram revealed inhomogeneous uptake by the liver but no focal discrete lesions (Fig. 1). An abdominal CT scan with and without contrast showed multiple low density lesions scattered throughout the liver and the spleen (Fig. 2). Numerous polymorphonuclear neutrophils were seen in a needle biopsy of the liver, but the cultures of the specimen were negative. A laparotomy and open wedge

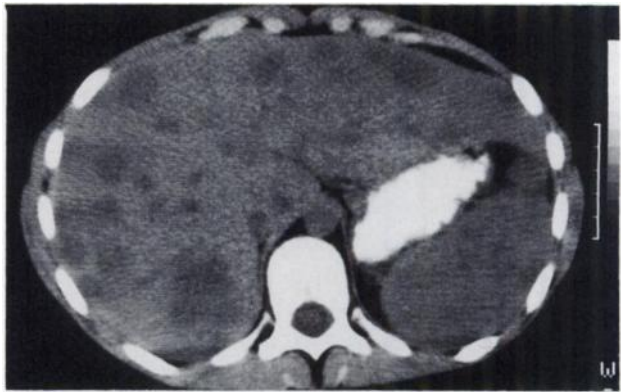


FIGURE 2
Abdominal CT scan showing multiple small low density lesions diffusely in the liver and the spleen.

biopsy of the liver was then performed. Palpation of the liver revealed multiple 0.5 to 1.0 cm nodules containing white creamy material. A touch preparation of this material showed inflammatory cells, but no organisms could be identified by special stains and the cultures were negative. Microscopic examination of the tissue showed stellate microabscesses characteristic of CSD (Fig. 3), and compatible organisms were demonstrated by special stains. Serology for Epstein-Barr virus, cytomegalovirus and brucella were negative. Antinuclear antibodies were negative. A PPD was negative. Blood, urine, throat, stool, and bone marrow cultures were negative. A bone marrow biopsy, chest x-ray, abdominal, and pelvic ultrasound were normal. The child was diagnosed as having CSD, and improved after treatment with gentamycin intravenously.

DISCUSSION

Multiple atypical cases of CSD have been described in the literature. However, CSD remains a diagnosis of exclusion due to the fact that the etiologic agent has

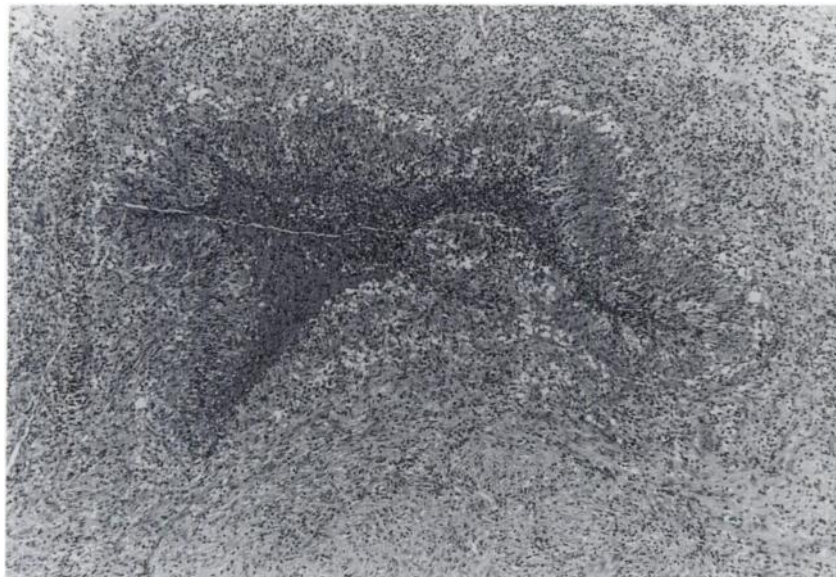


FIGURE 3
Photomicrograph (100x) of a wedge biopsy of the liver showing a typical stellate microabscess, consisting of a central zone of neutrophils and cellular debris with a surrounding rim of palisading histiocytes.

not been isolated in culture, although it has been recently identified only by staining of tissue specimens (6).

The case reported meets the criteria for the diagnosis of CSD: (a) history of cat scratch, (b) positive cat-scratch antigen skin test, (c) negative laboratory findings for another disease and, (d) characteristic histopathologic findings in a biopsy.

This case is unusual in that the patient presented without lymphadenopathy. Liver involvement has been described in rare cases (2-5), but none of them presented with diffuse multiple small abscesses in the liver and spleen, and all of them presented with associated lymphadenopathy. The case reported by Rocco et al. (5) presented with lymphadenopathy and two large defects (2-3 cm) seen on CT scan and liver-spleen scintigram.

In addition, this case emphasizes the importance of careful examination of the liver on a ^{67}Ga scintigram. The ^{67}Ga scintigram was performed prior to the CT scan, to rule out an abdominal abscess because at admission the major problem was fever of unknown origin with no clear localization of the symptoms. At that time there was only slight hepatomegaly and the liver function tests were only slightly elevated. Careful examination of the liver showed inhomogeneous uptake of ^{67}Ga , which raised the suspicion for microabscesses or diffuse involvement of the liver by a neoplastic

process. However, by the time the ^{67}Ga scintigram was completed, the cat-scratch antigen skin test became positive and an infectious etiology was more likely than a neoplastic process. In this instance, it was not necessary to perform a [$^{99\text{m}}\text{Tc}$]sulfur colloid scan. The CT performed the following day clearly showed the microabscesses suspected on ^{67}Ga scintigram, a biopsy was obtained and confirmed the diagnosis of CSD.

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