Scan Findings in a Case of Splenic Infarction Due to Amyloidosis: Case Report

Euishin Kim and Adel G. Mattar

Mallinckrodt Institute of Radiology,
Washington University School of Medicine, St. Louis, Missouri

Spleen images in a patient with monoclonal gammopathy showed numerous areas of decreased radiocolloid accumulation. Microscopic examination revealed areas of coagulation necrosis with diffuse amyloid infiltration in the spleen. In this patient, amyloidosis is considered to have caused the infarct.

J Nucl Med 17: 902–903, 1976

Abnormal findings on a spleen scan led to the complete workup of a patient with splenic infarction secondary to amyloidosis. The definitive diagnosis was established after exploratory laparotomy and histologic examination of the spleen.

CASE REPORT

A 61-year-old woman was referred for clinical evaluation of monoclonal gammopathy. Her condition had been discovered through routine laboratory tests 4 years previously when she underwent a cholecystectomy. Tests at that time, as well as annual bone-marrow examinations, failed to indicate the presence of multiple myeloma.

Physical examination on admission was unremarkable. Laboratory tests revealed mildly abnormal levels of liver enzymes, blood urea nitrogen, serum creatinine, and uric acid. The peripheral blood count was normal, but the erythrocyte sedimentation rate was elevated. The serum was positive for antinuclear antibodies, with increased levels of IgG and IgM. Proteinuria was also present. A liver–spleen scan was ordered for further workup. The ^{99m}Tc-sulfur colloid scintigram revealed mild hepatosplenomegaly and numerous areas of absent radioactivity in the spleen, consistent with either space-occupying lesions or infarcts (Fig. 1).

In view of the scan and laboratory abnormalities, the most likely diagnosis was malignant lymphoma. At exploratory laparotomy, the spleen was found to be firm and slightly enlarged, and numerous yellow geographic areas were seen on its surface (Fig. 2). The spleen was removed, and biopsies of

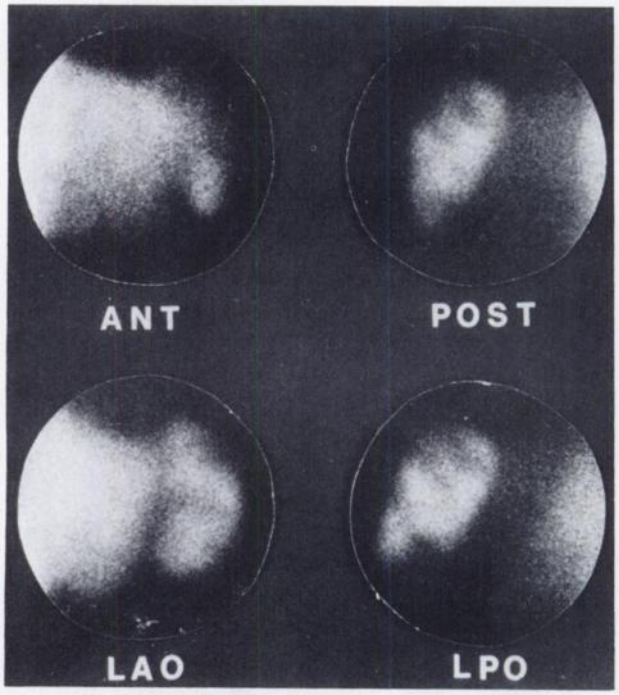

FIG. 1. Scintigrams of spleen taken with ^{99m}Tc-sulfur colloid show numerous areas of absent radioactivity, consistent with either space-occupying lesions or infarcts.

Received Feb. 3, 1976; revision accepted March 16, 1976.

For reprints contact: E. Kim, Div. of Nuclear Medicine, Edward Mallinckrodt Institute of Radiology, 510 S. Kingshighway Boulevard, St. Louis, MO 63110.

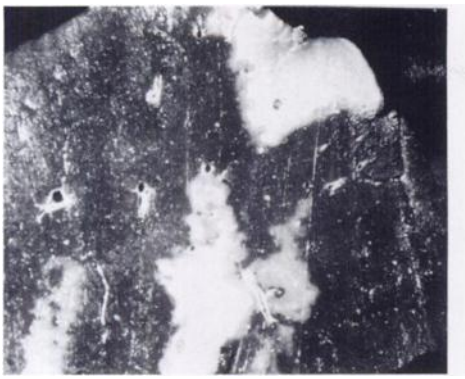

FIG. 2. Cross section of spleen shows numerous yellow geographic areas.

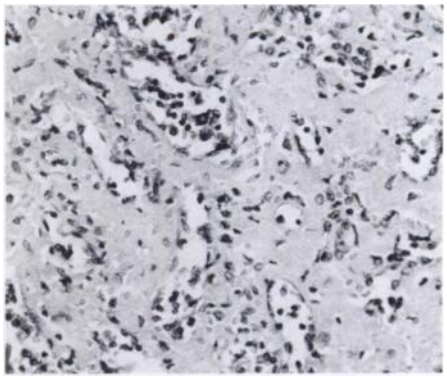

FIG. 3. Microphotographs of spleen reveal diffuse amyloid infiltration of pulp and media of arteries and arterioles. (×175).

the liver, kidney, and a celiac lymph node were obtained. Microscopic sections of the spleen revealed coagulation necrosis in the yellow areas, with diffuse infiltration by an eosinophilic homogeneous material involving the cords of the red pulp and the media of the arteries and arterioles (Fig. 3). The material was identified as amyloid by its congo-red and thioflavine-T staining characteristics. Amyloid was also found in the perisinusoidal spaces of the liver, the glomerular lobules of the kidney, the interstitium of the celiac lymph node, and the media of the arterioles of each of these tissues. No evidence of lymphomatous involvement was found. Due to the absence of factors predisposing toward amyloidosis, the condition was considered idiopathic in origin.

DISCUSSION

Since the splenic arteries do not communicate with each other, occlusion of these arteries leads directly to the death of splenic tissue. Such occlusion is commonly due to emboli derived from mural thrombi in the heart or aorta. Local thrombosis may also occur in sickle cell anemia, malaria, and leukemia. Multiple infarcts can be produced by polyarteritis nodosa and in situ thrombosis, due to the acute infections that initiate arteritis and arteriolitis (*1*). We assume that in our case the extensive amyloid deposition in the spleen led to ischemia and necrosis.

The scan features of hepatic and splenic involvement with amyloidosis are nonspecific and include hepatosplenomegaly, inhomogeneous radiocolloid distribution, or focal areas of decreased radioactivity. Such abnormalities are thought to be due to impairment of the reticuloendothelial cell function (*2*) and to focal or diffuse amyloid deposition.

REFERENCES

1. MacPherson AIS, Richmond J, Stuart AE: *The Spleen*. Springfield, Ill., C. C. Thomas, 1973, p 101

2. Suzuki K, Okuda K, Yoshida T, et al.: False-positive liver scan in a patient with hepatic amyloidosis: Case report. *J Nucl Med* 17: 31–32, 1976