ARTERIOGRAPHICALLY CONFIRMED FOCAL DEFECT IN COLLOID SPLEEN SCAN WITH NO GROSS PATHOLOGIC LESION: CASE REPORT

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A focal avascular defect in an enlarged spleen, clearly shown both by ^{99m}Tc-sulfur colloid scanning and by celiac angiography, could not be associated with any gross pathologic abnormality at splenectomy. Vascular lesions can probably produce focal defects on the scan in the absence of infarction.

Dickerman and Clements recently described a case in which a focal splenic defect, shown on two ^{99m}Tc-sulfur colloid scans, could not be confirmed at splenectomy (*1*). No mechanism was proposed and the possibility of an artifactual defect resulting from overlap of spleen and liver was raised.

We present here a similar case in which the existence of the defect and the mechanism for it, focal ischemia, were clearly shown by preoperative angiography.

CASE REPORT

A 62-year-old woman was seen by her private physician for various somatic complaints. Routine screening tests revealed a markedly elevated lactate dehydrogenase. After exhausting the usual diagnostic tests for suspected occult neoplasm, including a reportedly normal liver scan, the patient was referred for further investigation. Splenomegaly was observed on admission. Serum lactate dehydrogenase was again markedly elevated, with an abnormal isozyme detected on electrophoresis. There was mild peripheral thrombocytosis and mild lymphocytosis in both peripheral blood and marrow. A liver–spleen scan with ^{99m}Tc-sulfur colloid showed a focal defect in the upper anterior portion of the spleen (Fig. 1). (There is some suggestion of another defect toward the lower pole.) This could perhaps be attributed to splenic notching, but subsequent celiac angiography confirmed the existence of a 3.5-cm round avascular region in that location (Fig. 2). The defect was seen in an otherwise uniformly opacified splenic bed, and other films in the angiographic sequence excluded the possibility that this represented bowel gas. An exploratory laparotomy with splenectomy was consequently performed 8 weeks later. The spleen

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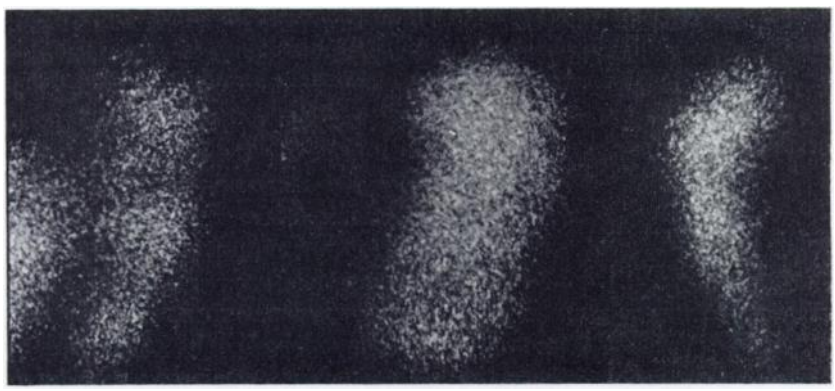

FIG. 1. Technetium-99m-sulfur colloid scans. Left: Anterior view of spleen showing focal defect at junction of upper and middle thirds. Middle: Left lateral view showing same defect. Right: Posterior view.

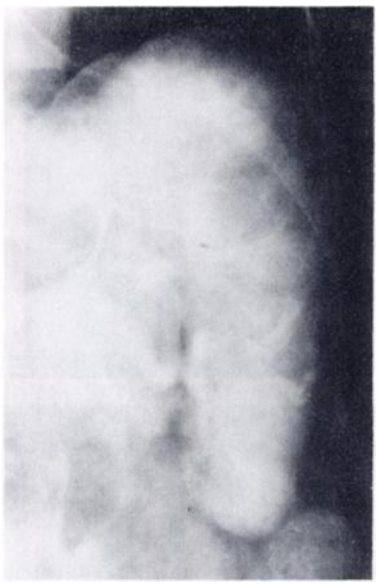

FIG. 2. Celiac arteriogram: Anterior view of spleen showing focal avascular defect at junction of upper and middle thirds.

weighed 500 gm. Multiple sections revealed diffuse lymphoid hyperplasia but no gross focal defects. Subsequent investigation suggested binding by abnormal serum proteins as the cause of the lactate dehydrogenase elevation. Followup, now in its fifth year, has been uneventful.

DISCUSSION

It is easy to see how a minute arterial lesion, if it does not produce infarction in its region of distribution, can cause a visible defect on scan and angiogram and yet be as difficult for the pathologist to find as a "needle in a haystack." We consider this to be the most likely explanation in our case and in the case reported by Dickerman and Clements. Since focal infarctions are a frequent pathologic finding in splenomegaly from whatever cause (*2*), one might expect vascular defects without infarction also to be associated with splenomegaly. In our case the spleen was enlarged to about five times its normal weight. The spleen appears generous in size in the limited views shown by Dickerman and Clements, but the weight at splenectomy was not reported in that case.

REFERENCES

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