CASE REPORTS

Local Colloid Trapping in the Liver in the Inferior Vena Cava Syndrome

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Local radioactive areas in the liver were observed as a result of superficial cavoportal shunting of radiocolloids in two patients with the inferior vena cava syndrome. In one patient a paraumbilical and/or a recanalized umbilical vein was apparently involved in the hepatopetal shunting. In the other patient a superficial anastomosis other than the paraumbilical vein shunted colloid to the liver. Relatively discrete areas of increased radioactivity, single or multiple, were seen in the left lobe. Virtually all reported instances of hepatopetal shunting of radioparticles in a superior or an inferior vena cava syndrome have demonstrated similar findings. Various hepatopetal collateral pathways in infrarenal caval obstruction are considered, and factors that could affect liver scan findings in the infrarenal obstruction are discussed.

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In the superior vena cava (SVC) syndrome, radiocolloid liver scans may show an area of increased activity in the left lobe of the organ. At least 26 cases of focal colloid liver concentration have been reported in the literature through 1979 (*1–14*). The accumulated data suggest that the area is produced by collaterally shunted colloids. A paraumbilical vein or a dilated umbilical vein has been mentioned as a last leg in the hepatopetal collateral journey following administration of radioparticles to an upper extremity. Analogous areas of increased uptake should occur in an inferior vena cava (IVC) syndrome if the colloid is given through a leg vein. In this communication, we report two such cases and review related observations in the literature.

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Case 1. A 47-year-old man presented with edema of both lower extremities and a long history of right-leg swelling and right-groin varicosity. Two weeks before admission, he noted aggravation of the leg swelling and for the first time developed swelling in the left leg. Inspection of the abdomen showed prominent veins over the epigastrium and both lower quadrants, with a midline scar from a prior umbilical herniorrhaphy. Urinalysis, SMA-12, and chest radiographs were all normal. A right-leg contrast venogram showed extensive pelvic collaterals with nonfilling of the right common ilica vein and retrograde filling of the left external iliac vein. He was treated conservatively with anticoagulants.

One month later, he was readmitted because of a gross hematuria. Cystoscopy showed blood coming from the right ureter. An intravenous pyelogram and an abdominal sonogram were normal, although the latter failed to visualize the IVC. A Tc-99m sulfur colloid study was obtained following a deliberate administration in the right leg. The colloid arrived in the left lobe through what appeared to be a paraumbilical vein and produced multiple focal increased concentrations in the lobe (Fig. 1). The hematuria was ascribed to the anticoagulant and did not recur after the drug was discontinued.

Case 2. A 58-year-old man was admitted for evaluation of lower abdominal pain. Past history included resection of a colonic adenocarcinoma 2 yr before admission, and a brief episode of severe edema of both lower extremities and groins 1.5 yr before admission. Physical examination on admission showed a scaphoid abdomen with prominent veins and surgical scars, but with no palpable mass. Edema was absent in all extremities. Urinalysis was normal. Figure 2 shows a Tc-99m SC study obtained after admission. With no suitable veins in the arms, the colloid was administered through a leg vein. A hepatopetal shunting occurred, producing an area of increased uptake in the quadrate area of the left lobe. The observation indicated an IVC syndrome, which had escaped recognition until then. Subsequently we learned that he had had a sulfur-colloid scintigram 11 mo before the admission; difficulty was encountered with intravenous administration, and the colloid had

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FIG. 1. Superficial hepatopetal collaterals and focal areas of activity in liver (Case 1). Tc-99m sulfur colloid (3 mCi) was administered through a right leg vein. Shown are eight consecutive dynamic frames (8 sec/frame, beginning 17th second after injection) and anterior and right lateral static images. Note cephalad flow in visualized right inferior epigastric vein, subsequent visualization of paraumbilical vein, nonuniform uptake in left lobe, and nonvisualization of inferior vena cava.

been injected into a femoral artery. Although initially interpreted as normal, upon review the scintigram indicated an interruption of the IVC at a high infrarenal level without visible collateral or localized liver uptake.

DISCUSSION

In both cases, the IVC syndrome appeared to be due to an infrarenal IVC obstruction. There was no nephrotic syndrome or other renal functional impairment to suggest involvement of a renal vein. In Case 1, the chronicity of the course and the history of asymmetric involvement of lower extremities indicated a benign thrombosis rather than malignant obstruction of the infrarenal IVC (*15*). Contrast venography in this case showed apparent bilateral iliac venous occlusion. Obstruction of the IVC itself was not directly demonstrated, but could have been present since no segment of the IVC was visualized in the dynamic colloid study (Fig. 1). Bilateral occlusion of the common iliac veins, without an obstruction of the IVC, produces almost the same syndrome as that of an infrarenal IVC obstruction (*16*).

In Case 2, the obstructive level appeared to be high infrarenal. Patency of the IVC below this level, and of the common iliac veins, was left uncertain. The most common cause of infrarenal and midcaval IVC obstructions is an ascending thrombophlebitis from one or both iliofemoral veins (*16*). Thrombosis can also occur primarily in the IVC, but may subsequently propagate distally. Thrombotic occlusion of one or both common iliac veins in addition to the IVC would encourage the development of superficial abdominal collaterals and thus possibly the development of paraumbilical shunting to the liver as well (*17*).

A paraumbilical vein, and occasionally a persistent umbilical vein, it is said, join the left main branch of the portal vein (*18,19*). After entering the left main branch, shunted colloid would distribute itself evenly in the left hepatic lobe, if there is no streaming of the colloid in the left main branch (*18*). Activity in the whole lobe, rather than in a focal area, would result. However, uniform trapping in the entire left lobe did not occur in either of these two cases of IVC syndrome or in any of 26 reported cases of SVC syndrome (*1-14*). Only in a few colloid studies (*4*) does the left lobe, aside from focal areas of uptake, look generally more active than the right one. The present Case 2 is an example of such exceptions (Fig. 2). Hepatopetal shunting of Tc-99m albumin microspheres has been observed by others in two cases of IVC syndrome (*10,20*). In both cases, a single area of microsphere uptake was seen within but not over the entire region of the left lobe.

Even if shunted colloid enters through the paraumbilical vein into the left main branch of the portal vein in an SVC or IVC syndrome, the left lobe would not become uniformly radioactive, if it contains lesions such as metastases. However, disease in the left lobe as a possible basis for the nonuniformity appears to be exceptional (*10,11*). In virtually every instance of SVC syndrome, the liver scan is normal except for visualized collaterals and the area of localized activity (*1-14*). In most instances, there is just a single well-defined area of activity, which is most commonly seen in the inferior (quadrate) area of the medial segment of the left lobe (*18*). Many other patterns, however, such as that of the present Case 1, have been observed. The nonuniformity and varied patterns of the abnormal left-lobe uptake in an SVC or IVC syndrome suggest a few possibilities. First, paraumbilically shunted colloid that enters the left main branch of the portal vein could be subject to a streaming effect within the left main branch. Second, there might be places other than the left main branch where paraumbilically shunted colloid could enter the left-branch system, and these entry sites could vary from one patient to another.

Cavoportal pathways in infrarenal IVC obstruction also include visceral routes through the inferior and superior mesenteric veins (*21*). A liver-scan pattern resulting from shunting through these mesenteric pathways would depend in part on the extent of any streaming in the portal vein (vide infra) and on the formation of the portal vein from its major tributaries, namely, the splenic vein and the mesenteric veins (*18*). Whether the venous blood from the major tributaries completely mixes in the portal vein before reaching the main branches is not clearly known (*18*). At the 1973 annual meeting of the Radiological Society of North America, Henkin and others presented studies in a young woman with both IVC and SVC occlusion. Technetium-99m albumin microspheres,

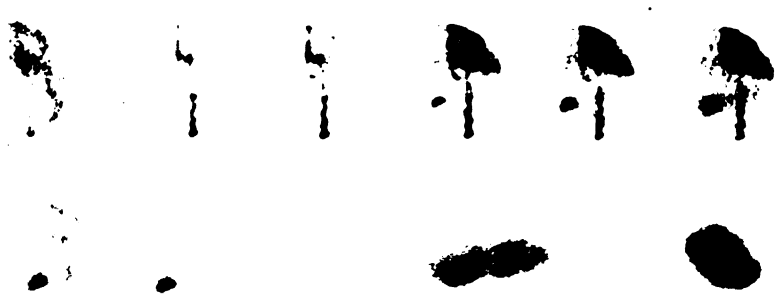

FIG. 2. Superficial hepatopetal shunting producing increased concentration in quadrate area in Case 2. Tc-99m sulfur colloid (3 mCi) was given into left leg vein. Shown are eight consecutive dynamic frames (8 sec/frame, beginning 9th second after injection) and anterior and right lateral static images. Note spiraling midline collaterals with faint offshoot to right, producing uptake in quadrate area and nonvisualization of inferior vena cava. Offshoot does not follow course of paraumbilical or umbilical vein.

admistered through a pedal vein, localized almost exclusively in her liver, producing a normally appearing liver scan. A contrast inferior cavogram and the dynamic phase of the microsphere study both showed the inferior mesenteric vein to be the dominant collateral (*21*). In this woman, the hepatic microsphere distribution was consistent with an absence of portal venous streaming and any portal vein formation (*18*).

A focal area of increased activity in the liver, striking as it is, may not necessarily be a finding in an infrarenal IVC syndrome as in the case of an SVC syndrome (*11*). Potential collateral pathways may not have developed fully at an early stage of the obstruction. A collateral venous return through mesenteric veins, though detectable on dynamic images, may not be apparent on static images in the absence of portal venous streaming. Finally, nonhepatopetal pathways may dominate the return (*21*).

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