Discrepant Sulfur Colloid and Radioparticle Liver Uptake in Superior Vena Cava Obstruction: Case Report

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The presence of collateral venous channels connecting the upper extremity veins and portal vein via the paraumbilical veins is considered the probable explanation for the observed scintigraphic hepatic "hot spot". This is seen in [^{99m}Tc]sulfur colloid liver imaging and perfusion lung imaging with ^{99m}Tc radiolabeled particles injected into an antecubital vein in the presence of superior vena caval (SVC) obstruction. The typical distribution is one of focal uptake centrally, anteriorly, and inferiorly. An unusual pattern is described in this report and mechanisms proposed for the "diffuse homogeneous" hepatic uptake also observed in a patient with SVC obstruction undergoing a perfusion lung scan.

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Focal areas of increased uptake (hot spot) in the liver have been described with sulfur colloid liver imaging in several reports (*1–14*). A selected list of differential diagnostic considerations include superior (SVC) and inferior vena caval (IVC) obstruction with caval-portal shunting, Budd-Chiari syndrome, hepatic hemangioma, hamartoma, adenoma, focal nodular hyperplasia and hepatic veno-occlusive disease (*1–3,5–12,19–22*). The most common reason appears to be SVC or IVC obstruction of varied etiologies (*2,12,15*). Thrombosis of the innominate veins and SVC can occasionally occur due to central venous catheters for hyperalimentation (*14,16*). Mediastinal diseases can extrinsically obstruct the SVC, with some notable examples being metastatic bronchogenic carcinoma, lymphoma, fibrosing mediastinitis due to histoplasmosis and tuberculosis and other causes (*5,15*). If the SVC obstruction is of relatively rapid onset, signs of increased cerebral venous pressure can dominate the clinical presentation (*15*). With gradually developing chronic SVC obstruction, various collateral channels can serve to bypass the SVC obstruction and return upper extremity, head and neck venous blood to the right heart via the IVC or portal vein (*17,18,22*). We have observed both *focal* and *diffuse* uptake in a patient with SVC obstruction undergoing a [^{99m}Tc]MAA (macroaggregated albumen) radioparticle perfusion lung scan.

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CASE REPORT

A 76-yr-old black woman presented with acute abdominal pain, anorexia, nausea, and vomiting of 4 days duration. She had a past medical history of insulin treated diabetes mellitus. Physical examination revealed a thin, elderly female in moderate distress, complaining of abdominal pain. The pulse rate was 132/min, accompanied by a systolic blood pressure of 60 mmHg. No jugular venous distension was present. The lungs were clear to auscultation. The abdomen was distended, diffusely tender, but without rebound tenderness. Her bowel sounds were hypoactive with audible tinkles. Laboratory data showed normal liver function tests. The admission chest radiograph was normal. Abdominal radiography demonstrated multiple dilated loops of small bowel with air fluid levels. The clinical impression of small bowel obstruction led to laparotomy, which revealed strangulated small bowel obstruction. Due to poor nutritional status, a central venous line was inserted for hyperalimentation. An episode of dyspnea and chest tightness and a clinical suspicion of pulmonary emboli led to a perfusion lung scan (Fig. 1). SVC disease was not clinically suspected prior to lung scanning. Chest x-ray taken after hyperalimentation was discontinued showed a widened superior mediastinum, may be due to hemorrhage or edema.

This case illustrates the presence of collateral vessels connecting upper extremity venous drainage to the portal vein. The [^{99m}Tc]MAA uptake distribution is in the right hepatic lobe, junction of the right and left hepatic lobes anteriorly and inferiorly, and part of the left lobe superiorly. The obvious abnormality is the "hot spot"; right lobe uptake is homoge-

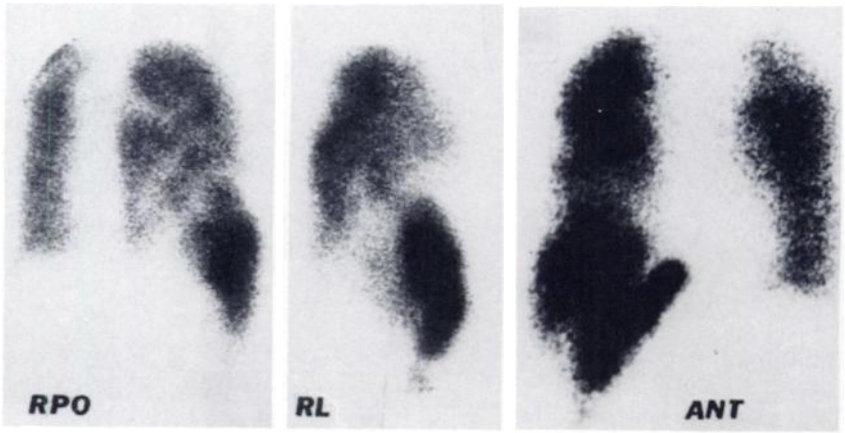

FIGURE 1
Perfusion lung scan—right posterior oblique (RPO), right lateral (RL), anterior (Ant.) views: Radioparticle (MAA) deposition in right hepatic lobe and vicinity of porta hepatis.

neous and unremarkable on [^{99m}Tc]sulfur colloid scan (Fig. 2). When interpreted in light of the radioparticle (MAA) study, it becomes apparent that there exists a component leading to a "homogeneous" distribution due to collateral flow to the liver which is obscured because it is inseparable from the normally occurring homogeneous uptake on sulfur colloid imaging. Using "hot spots" as criteria for inferring distribution of collateral flow on sulfur colloid scan underestimates the full extent of the collateral flow, which is more accurately depicted on the MAA study, as seen in this case. This may have no clinical significance, but presents an interesting imaging observation and is thought provoking. The distribution observed in this report is unusual, depicting a *diffuse* as well as *focal* pattern of uptake.

DISCUSSION

The principle mechanism underlying localization of macroaggregated albumin (MAA, 5–100μ particle size) or albumin microspheres (HAM 20–40μ particle size) is capillary blockade. Hepatic uptake of radioparticles (MAA,HAM) has been previously reported and is attributed to caval-portal collaterals seen in SVC and IVC obstruction (*1–3,11,12,21,22*). In the presence of SVC obstruction, collateral pathways develop and venous flow is directed via these collateral channels to bypass the SVC block. These venous collaterals can include the internal mammary and lateral thoracic veins which may communicate with the superficial epigastric and paraumbilical veins, which communicate with the portal venous system (*11*). By this route, the MAA particles injected antecubitally would impact in the first capillary bed encountered, which of course is in the liver. Other collateral channels exist as well and can lead to "downhill" varices, diverting flow via the hemiazygous system in retrograde fashion; lumbar and retroperitoneal collaterals can also result which can allow blood to bypass the obstructed SVC (*17,18*) and follow a tortuous collateral route eventually reaching the right heart via the IVC, or to portal vein via mesenteric or periesophageal venous collaterals (*21,22*).

The distribution within the liver reflects the combined effects of streaming, preferential flow and the intrinsic anatomic peculiarities of the particular collateral channel (*12,21*). If part of the bypassed upper extremity venous blood flows via the superficial epigastric veins to connect with the IVC via the iliac veins (*11*), the particles taking this lengthy route would eventually of course end up in the lungs. In the case described, caval-portal collateral mechanisms seem to be the logical explanation for the hepatic uptake of radiolabeled MAA (*21,22*). This mechanism of course also accounts for the sulfur colloid hot spot in SVC obstruction following an antecubital injection (*3,11,12*).

The distribution in the current case is unusual, since there is extensive right hepatic lobe deposition, a finding not ordinarily expected with the umbilical vein to left portal vein flow route in caval-portal shunting. Left lobe uptake would be the expected finding and is described in the literature. Weissman and Lin point out, however, that there may be variations in the level and degree of umbilical and portal venous communications that might explain unusual distributions, such as encountered in this case (*12,21*). It is interesting to note that the apparent "hot spot" distribution is disparate between the MAA and sulfur colloid scan. One might speculate that if the umbilical vein were to join the portal vein at the junction of the right and left portal vein branches, uptake in both lobes may be present. A mixing effect in the longer right portal vein may lead to a homogeneous distribution, whereas streaming effect experienced by part of the blood flow entering the left portal vein would give rise to the hot spots seen in the vicinity of the umbilical and portal venous area. This is of course speculative, but seems to explain the observed findings; other mechanisms may exist as well, such as branching of the umbilical vein prior to joining the portal branch and differential flow rates in the two vessels that may lead to streaming or mixing effects. A similar phenomenon may explain the sulfur colloid hot spot distribution (*12,21,22*). If mixing with blood oc-

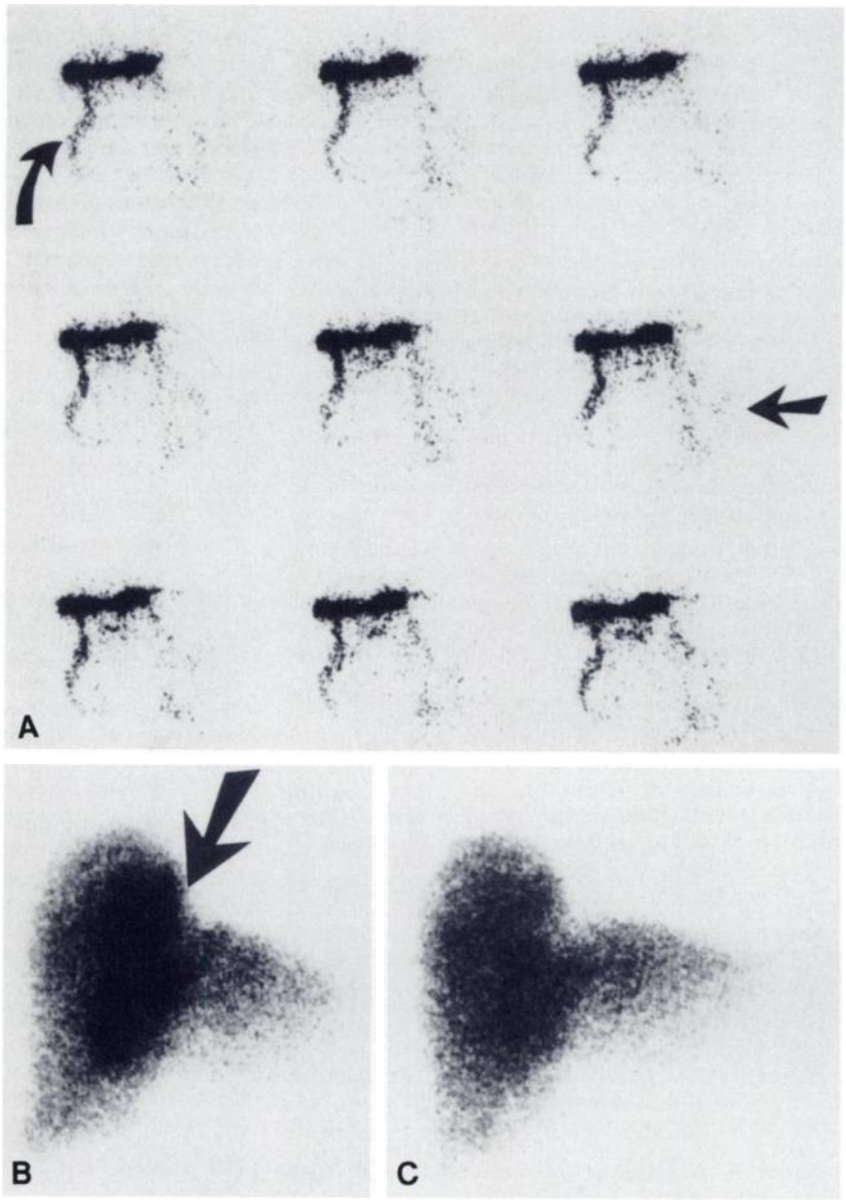

FIGURE 2
Radiocolloid study via right antecubital injection. A: Superior vena cava flow study: Note collaterals bypassing the completely obstructed superior vena cava (lateral thoracic vein: curved arrow; transthoracic collateral veins: straight arrow). B: Radiocolloid hyperconcentration is present around the porta hepatis and in the right hepatic lobe anteriorly, medially and superiorly (arrow). C: Radiocolloid study following left pedal vein injection. Hepatic "hot spots" are no longer visualized.

curs in the right portal branch, a focal hot spot, per se, may not be visualized in the right hepatic lobe and would in fact appear homogeneous on the static images. The hot spots in the vicinity of the porta hepatis region anteriorly and inferiorly are due to the high concentration of radiopharmacuetical in blood flowing in this region (*12,22*).

In general, sulfur colloid hot spots appear to be due to local increase in perfusion and/or number of phagocytic cells (*13*). In SVC obstruction, a relatively undiluted, high specific activity bolus is delivered to the liver via the portal vein; additionally, the inherently high first pass extraction of sulfur colloid by the liver probably accounts for the observed hot spot (*22*). The phagocytic cells perfused with a high local concentration of radiocolloid are able to achieve higher cellular concentration due to the collateral blood flow mechanisms (*1,12*). The failure of the hot spot to visualize following an IVC injection of sulfur colloid confirms this theory.

A hot spot is easily perceived visually, being focal. A faint diffuse increase in sulfur colloid concentration may not be readily evident and would appear normal such that one may not suspect caval-portal shunting to the right hepatic lobe on sulfur colloid imaging. With radioparticles (MAA, HAM), however, due to the difference in mechanism of localization, the distribution portrays the collateral network participating in caval-portal shunting more accurately. The sulfur colloid distribution underestimates the full extent of caval-portal shunting and collateral flow, particularly to the right hepatic lobe.

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