Colloid Shift Following Blunt Trauma

Russell C. Briggs and Steven M. Amberson

Division of Nuclear Medicine, Department of Radiology, Maine Medical Center, Portland, Maine

Thirty-three patients who underwent spleen imaging with [^{99m}Tc]sulfur colloid following blunt trauma to the left chest and/or left upper abdominal quadrant were evaluated. Nineteen of these patients were found to have reversed liver-spleen activity ratios (colloid shift). The incidence of this finding was surprising in view of the absence of any predisposing factors in these patients. Colloid shift was seen as early as 2 hr after trauma and was found to persist as long as 188 days after the traumatic event. The explanation for colloid shift in this group of patients is unknown, but the results suggest that it is secondary to reticuloendothelial system stimulation.

J Nucl Med 28:188–190, 1987

Reversal of liver-spleen activity ratio (colloid shift) is frequently associated with cirrhosis, malignant neoplasms, and diabetes (*1–3*). We have noted colloid shift in several patients undergoing spleen scanning following trauma and therefore conducted a retrospective review of the records of 54 patients who underwent spleen scanning after trauma performed during a period of 43 mo.

MATERIALS AND METHODS

Patients

The records of 54 patients referred for spleen scans during the period from January 1, 1981 through July 31, 1984 for the question of traumatic splenic injury were examined. Ten of these cases had known underlying problems which might alter the liver-spleen ratio (heavy ethanol intake—three; infectious mononucleosis—two; ethanol intake and substance abuse, serum hepatitis, myeloid metasplasia. Factor IX deficiency, reactive splenitis,—one each) and were excluded from this study. One stab wound was excluded because our interest was limited to blunt trauma and ten cases with blunt trauma were excluded because the posterior view was inadequate for accurate assessment of liver to spleen ratio. The remaining 33 cases were analyzed to determine (a) date and time of trauma, (b) mechanism of trauma, (c) body areas traumatized, (d) laboratory values prior to spleen imaging, (e) other factors which might alter the liver-spleen ratio such as hyperalimentation, anesthesia, or blood transfusions, and (f) the imaging results including spleen size, evidence of traumatic injury, and liver-spleen activity ratio.

Received Nov. 15, 1985; revision accepted July 17, 1986.

For reprints contact: Russell C. Briggs, MD, Div. of Nuclear Medicine, Dept. of Radiology, Maine Medical Center, 22 Bramhall St., Portland, ME 04102.

Imaging Procedure

Spleen imaging was performed with intravenously administered technetium-99m sulfur colloid. The same commercial source for the pharmaceutical was used throughout the study period. The colloid particle size with this preparation is 400–700 mμ. Adults received 5 mCi doses, while pediatric doses were calculated by the modified Young's rule (*4*). All patients were imaged within 10 to 20 min postinjection with anterior, posterior, LAO 45°, LAO 70°, left lateral and left posterior oblique views. All images were obtained on a large-field gamma camera with 37 photomultiplier tubes using a low-energy, all-purpose collimator, and 600,000 counts per image were recorded. Data were displayed in digital format in a 128 × 128 matrix. Film recording did not utilize background subtraction, but contrast enhancement was used to display splenic detail, a very important maneuver when searching for subtle splenic abnormalities. These factors may lead to a patchy-appearing liver in any instance in which the liver-spleen ratio is reversed and should not be interpreted as an absolute indicator of underlying liver pathology. Six of the 33 cases had two or more scans.

Liver-spleen ratios were established by visual evaluation of anterior and posterior views which included both liver and spleen. Ratios in which liver activity equaled or exceeded spleen activity by visual inspection were considered normal. Those in which spleen activity exceeded liver activity on the posterior view only were graded as 1+ reversal, while those which revealed liver-spleen ratio reversal on both the anterior and posterior views were graded as 2+ reversal.

RESULTS

A total of 44 imaging procedures were carried out on the study group of 33 patients. Table 1 indicates that 19 patients (58%) demonstrated colloid shift, while 14 patients had normal liver-spleen ratios. Each of these

TABLE 1
Correlation of L/S Ratio with Other Splenic Abnormalities

L/S ratio	No. of patients	No. with splenic enlargement	No. with focal splenic lesion
Normal	14	3	5
Reversed	19	5	9

14 with normal ratios was studied only once. Thirty imaging procedures were performed on the 19 patients who demonstrated colloid shift at some point. One patient was scanned on five separate occasions; two patients were scanned three times; and three patients were scanned twice.

As can be seen in Table 2, in 20 studies performed between 2 and 24 hr after blunt trauma, ten showed evidence of colloid shift. Twelve procedures were carried out between 1 and 7 days after injury of which seven demonstrated colloid shift; and of 12 studies performed between 8 and 201 days, colloid shift was seen in nine instances.

Table 1 also notes that in ten of 19 cases with reversal of the liver-spleen ratio, no scintigraphic evidence of focal splenic injury was demonstrated, while the remaining nine were positive for traumatic splenic abnormality. Five of the 14 patients with normal liver-spleen ratios had focal splenic lesions by scan evaluation. The presence of splenomegaly did not correlate with the presence of colloid shift.

The six cases in this series in which multiple scintigraphies were performed all demonstrated colloid shift and focal splenic lesions. It is noteworthy that in three of the six cases, no evidence of colloid shift was seen on the initial study. In one case the final scan at 201 days post-trauma showed a normal liver-spleen ratio. However, this same patient had evidence of 2+ ratio reversal at 144 days. In this group of six patients, the longest interval between injury and spleen imaging in which colloid shift could be seen, was 188 days.

The 14 patients with normal liver-spleen ratios and the 19 patients with ratio reversal were compared for age, sex, hyperalimentation, blood transfusions, and anesthesia prior to imaging and these factors bore no relationship to whether or not colloid shift was observed. Hematocrit, white blood cell count, and serum amylase values also failed to show any correlation with the colloid shift. Both groups contained patients with decreased hematocrits and elevated white blood cell counts. Transient elevation of serum amylase was found in only two of 19 patients with reversed liver-spleen ratios. Liver function tests including total protein, serum albumin, total bilirubin, and alkaline phosphatase were all normal in two patients with reversed liver-spleen ratios for whom these studies were performed. Similar studies were normal in one patient with a normal ratio, while one case with a normal liver-spleen ratio demonstrated a mildly elevated alkaline phosphatase and one case had an elevated lactic dehydrogenase level on the day of the scan.

TABLE 2
Colloid Shift Correlated with Time After Blunt Trauma

Time after trauma scan performed	No. of patients	No. of patients with splenic lesion	No. of splenic scans	No. of scans with colloid shift
2–24 hr	20	9	20	10
1–7 days	11	5	12	7
8–201 days	8	6	12	9

The severity of trauma had no bearing on whether or not colloid shift occurred. Twenty-two of the 33 total cases were involved in motor vehicle accidents. Both groups, i.e., "reversed" and "normal," contained some individuals subjected to minor trauma and some to major multi-organ trauma.

Patients who demonstrated colloid shift in this study all survived with no sequelae, so that the fact that colloid shift was demonstrated as long as 188 days after trauma, did not correlate with any lack of well being.

DISCUSSION

Our observation of colloid shift on splenic images following trauma is not unique. Examples of this finding may be seen on a study at 6 days postinjury in a report by Morayati et al. and for 11 mo in the publication of Fischer and co-workers (*5,6*). The incidence of colloid shift on liver-spleen scans for reasons other than trauma in our laboratory is 45% and is similar to that of Wilson and Keyes (*2*). Therefore, the observed incidence of 58% found in otherwise healthy individuals subjected to acute blunt trauma is significant. Lutzker has provided a review of the current status of imaging the injured spleen (*7*). She refers to a study of experimental traumatic splenic injury by Washburn et al. who demonstrated transient increase in spleen to bone marrow activity ratios in dogs following the trauma (*8*). The literature otherwise has been silent on the subject of colloid shift following trauma.

Trauma is associated with stimulation of the extrahepatic portions of the RES and this may account, at least in part, for the colloid shift following blunt trauma found in the cases reported here (*9,10*). Factors associated with the traumatic event such as alteration in mesenteric and portal venous flow, changes in splanchnic arterial flow, changes in circulating epinephrine levels, and adrenal steroid release, might well be involved with the colloid shift seen in a matter of hours postinjury. The liver-spleen ratio would be expected to return to normal as these immediate physiological re-

sponses dissipate. We have demonstrated that such is not necessarily the case.

Our results suggest that, in addition to immediate post-trauma physiologic responses, at least two other mechanisms may be postulated to account for the observations of colloid shift appearing within the first few days or weeks after injury and persisting for periods exceeding 6 mo.

First, traumatic injury is associated with an immediate fall in plasma fibronectin levels followed at 24 to 48 hr by a rise to supranormal levels which then return to normal over a period of days. Plasma fibronectin is an alpha-2 globulin and a major opsonic protein required for the phagocytosis of foreign particulate matter and tissue debris (*10*). Perhaps these elevated fibronectin levels permit enhanced phagocytic capacity in the extrahepatic RES regardless of whether or not the spleen has been directly damaged.

Second, to account for the persistance of colloid shift for periods exceeding 6 mo in patients with splenic injury, we should consider the powerful phagocytic stimulator, tuftsin, reported by Najjar (*11*). This is a tetrapeptide (L-threonyl-L-lysl-prolyl-L-arginine) which is derived from a subclass of gamma globulin, called leukokinin. Tuftsin becomes active after excision from carrier leukokinin by two enzymes, one of which is present in the spleen (tuftsin endocarboxypeptidase) and the other on the surface of the phagocyte (leukokininase) (*12*). If splenic tissue is absent the phagocytic stimulatory effect of tuftsin does not occur. Spirer et al. have demonstrated that patients subjected to splenectomy for splenic trauma continue to have normal or only partially decreased tuftsin levels in the serum for periods of up to 20 yr post-splenectomy, in contrast to those individuals with nontraumatized spleens subjected to splenectomy on an elective basis (*13*). These authors note that the incidence of overwhelming sepsis is low in the post-trauma splenectomy group compared with the elective splenectomy group. Pearson and coworkers have similarly noted a low incidence of sepsis following splenectomy for splenic injury which they believe is related, at least in part, to splenosis (*14*). Perhaps splenic implants occur outside the spleen more often than hitherto recognized in patients with traumatic splenic injury even though the spleen is not removed, and these implants might represent a source of excess tuftsin leading to a supranormal RES stimulatory effect and prolonged, demonstrable colloid shift.

As has been noted, there appeared to be no difference in clinical outcome between those patients who demonstrated colloid shift and those who did not. Saba has indicated that tissue injury alone does not lead to progressive multiorgan failure and low survival rates after trauma (*10*). Superimposed invasive wound sepsis seems to be the crucial factor. It would appear quite possible that the colloid shift seen in the majority of the patients reported here might represent an indicator of the stimulated reticuloendothelial system armed to respond to the forces of infection and tissue destruction subsequent to blunt traumatic injury.

REFERENCES

1. Bekerman C, Gottschalk A. Diagnostic significance of relative uptake of liver compared with spleen in ^{99m}Tc-sulfur colloid scintiphotography. *J Nucl Med* 1971;12:237–240.
2. Wilson GA, Keyes JW. The significance of the liver-spleen uptake ratio in liver scanning. *J Nucl Med* 1974;15:593–597.
3. Harvey WC, Kopp DT, Podoloff DA, et al. Reversed liver-spleen ratio and the normal liver-spleen scintigram. *J Reticuloendothel Soc* 1976;19:19–27.
4. Webster EW, Alpert NM, Brownell GL. Radiation doses in pediatric nuclear medicine and diagnostic x-ray procedures. In: James AE, Wagner HN, Cooke RE, eds. Pediatric nuclear medicine. Philadelphia: WB Saunders, 1974:38.
5. Morayati S, Sagar VV, Warsal NF, et al. Rapid resolution of spleen scan changes after trauma. *Clin Nucl Med* 1985;10:440.
6. Fischer KC, Eraklis A, Rossello P, et al. Scintigraphy in the followup of pediatric splenic trauma treated without surgery. *J Nucl Med* 1978;19:3–9.
7. Lutzker LG. Radionuclide imaging of the injured spleen and liver. *Semin Nucl Med* 1983;13:184–198.
8. Washburn ME, Balk MW, Mazat BA, et al. Experimental subcapsular hematoma of the spleen. *Ann Surg* 1978;187:407–410.
9. Saba TM, Scovill WA. Effect of surgical trauma on host defense. *Surg Ann* 1975;7:71–102.
10. Saba TA. Plasma fibronectin and hepatic Kupffer cell function. *Prog Liver Dis* 1982;7:109–131.
11. Najjar VA, Nishioka K. "Tuftsin": a natural phagocytosis stimulating peptide. *Nature* 1970;228:672–673.
12. Najjar VA. Biochemical aspects of tuftsin deficiency syndrome. *Med Biol* 1981;59:134–138.
13. Spirer Z, Zakuth V, Diamant S, et al. Decreased tuftsin concentrations in patients who have undergone splenectomy. *Br Med J* 1977;2:1574–1576.
14. Pearson HA, Johnston D, Smith KA, et al. The born-again spleen: return of splenic function after splenectomy for trauma. *N Engl J Med* 1978;298:1389–1392.