

USE OF ^{99m}Tc -SULFUR COLLOID IN EVALUATION OF RENAL TRANSPLANT COMPLICATIONS

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Technetium-99m-sulfur colloid (TSC) was administered to 45 renal transplant patients. No TSC accumulation was seen in normally functioning transplant kidneys. Accumulation of TSC was found in 89% of transplants with rejection, in 30% of transplants exhibiting acute tubular necrosis, and in 30% of transplants of patients with sepsis. Renal accumulation of TSC in various transplant complications diminishes the value of this technique for monitoring transplant rejection.

Scintigraphic visualization of renal transplants with ^{99m}Tc -sulfur colloid (TSC) during rejection has been reported recently (1,2). The purpose of this prospective study was to evaluate the ability of TSC to distinguish among various complications of renal transplantation.

MATERIALS AND METHODS

During December 1974 and January 1975, renal transplants of 45 patients were examined with TSC. Sixteen of these patients had received transplants from related donors and 29 from cadavers. A total of 58 examinations were carried out.

The TSC (Tesuloid, E. R. Squibb and Sons, Inc., Princeton, N.J.) was withdrawn from batches prepared for routine liver imaging. The expected TSC distribution was observed in the liver-spleen studies of the test group and in controls. Two millicuries of TSC were administered by intravenous injection, together with 20 μCi of ^{131}I -ortho-iodohippurate (OIH). The OIH is used routinely for renography in the management of renal transplant patients at the University of Minnesota Hospitals (3-5).

Thirty minutes after TSC administration, a Polaroid picture was obtained in anterior projection with a scintillation camera. The field of view included the pelvis and vertebral column. Studies were considered positive if transplanted kidneys were visualized on the

scintiscan after a 4-min exposure. The degree of TSC accumulation was graded subjectively by the authors as absent, slight, or marked.

RESULTS

Twenty-four studies were performed during the immediate postoperative period, defined as the first 2 weeks after transplantation. No TSC accumulation in the transplanted kidney was seen in 14 examinations of eight patients with normal kidney function and uncomplicated postoperative course.

Acute tubular necrosis was diagnosed by exclusion, i.e., all other possible early complications were first ruled out (5). Three out of ten patients (ten studies) with acute tubular necrosis showed slight or marked TSC accumulation. Clinical and laboratory examinations during the following 2-6 weeks revealed no evidence of superimposed rejection or any other complication and no additional TSC studies were carried out. Acute tubular necrosis gradually resolved in all three instances. This was characterized by a progressive increase in urine output, improved renal function as manifested by lower serum creatinine and blood urea nitrogen values, and return of OIH renogram patterns to normal.

Thirty-four TSC examinations were performed at the time of rehospitalization of 27 renal transplant patients. Five of these patients were readmitted for routine followup and had normally functioning transplants, as assessed from clinical and laboratory data. Their OIH renograms were normal and no TSC accumulation was found in the transplanted kidney.

Five patients were readmitted for acute rejection, evidenced by elevated serum creatinine and blood urea nitrogen values, abnormal OIH renograms, and, in selected instances, abnormal arteriograms and

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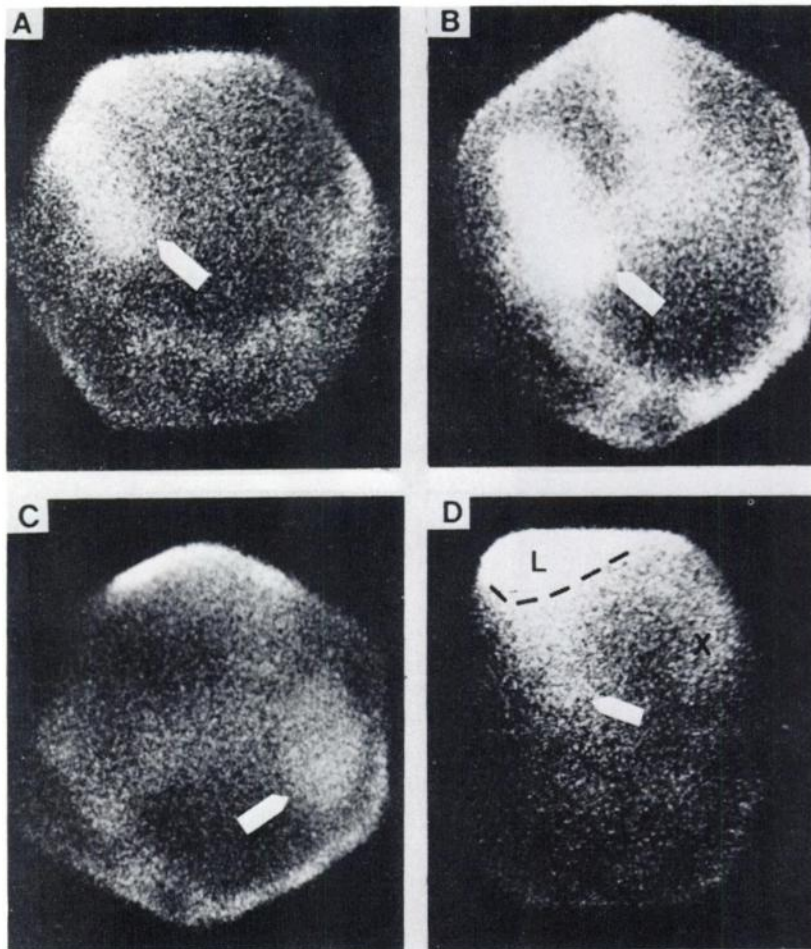


FIG. 1. Accumulation of ^{99m}Tc -sulfur colloid in various renal-transplant complications. (A) Acute rejection (arrow); (B) chronic rejection (arrow); (C) acute tubular necrosis (arrow); (D) normally functioning second transplant during sepsis (arrow). In D, first transplant after chronic rejection (X) and tip of right hepatic lobe (L) are visible.

renal biopsies (5). All five showed TSC accumulation in the transplanted kidney. This accumulation was slight in one and marked in the other four. Anti-rejection therapy (5) resulted in improved renal function in all instances. Six TSC followup studies of these patients were performed in the course of therapy. Accumulation of TSC vanished in one, diminished to slight in three, and remained marked in the remaining patient (two studies).

Eight patients were readmitted with presumed chronic rejection. Seven showed TSC accumulation, recorded as slight in four patients and marked in three. No significant clinical improvement of these patients was afforded by therapy; the choice was either chronic dialysis or a second transplant. One patient showed no transplant accumulation of TSC and, likewise, no concentration of OIH in the kidney. The pathologic diagnosis of his excised transplant was severe chronic rejection and infarction secondary to thrombosis of a branch of the renal artery.

Nine patients were readmitted because of clinical signs of rejection and were subsequently found to be septic. Six of these patients (three with systemic

viral infections and three with infections of the urinary tract) showed no transplant accumulation of TSC at the time of readmission. Two patients with systemic viral infections and hepatitis showed slight TSC accumulation in the transplant. In a patient with pneumonia, initial marked TSC accumulation in the transplanted kidney disappeared after appropriate treatment. None of these nine patients received anti-rejection therapy.

DISCUSSION

Following its intravenous injection, ^{99m}Tc -sulfur colloid is rapidly cleared by the reticuloendothelial system. Its tissue distribution is dependent upon its particle size and the blood flow and phagocytic activity of various body organs. Normally the Kupfer cells of the liver take up 80–90% of the radiocolloid. The remainder is usually seen within the reticuloendothelial system of spleen and bone marrow (6). Less than 2% of the radiocolloid is excreted through the kidneys (7). Scintigraphic visualization of kidneys with TSC is considered abnormal and has been reported in cases of congestive heart failure (8) and in transplanted kidneys during rejection and acute

tubular necrosis (1,2). The mechanism of TSC accumulation in transplanted kidneys during acute or chronic rejection is not well understood (1,2).

In our study there was no TSC accumulation in normally functioning transplants either during the immediate postoperative period or during subsequent followup examinations. In 19 of 21 studies of patients with acute or chronic rejection there was significant TSC accumulation in the transplant (Fig. 1A and 1B). That no TSC accumulation was noted in a partially infarcted transplant with chronic rejection indicates the need for assessing patency of the vascular system before interpreting a TSC study (9). Lack of TSC accumulation was also noted during antirejection treatment in one transplant with acute rejection. Absent accumulation of TSC preceded the return of serum creatinine, blood urea nitrogen, and OIH renogram to normal.

Accumulation of TSC was shown in three out of ten transplants with acute tubular necrosis without evidence of superimposed rejection or cardiac failure (Fig. 1C) (1,2,8). Three of ten patients with sepsis also showed TSC accumulation without evidence of a rejection triggered by the inflammatory disease (Fig. 1D) (10). The signs of sepsis during long-term immunosuppressive therapy often resemble clinically those of rejection (5). Thus, the differentiation of rejection from sepsis, as well as from acute tubular necrosis, is necessary before appropriate therapy can be instituted (5). The TSC accumulation study does not appear to provide such a differentiation.

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