Renography Before Heart Transplantation in Patients with Cardiomyopathy

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In patients with ischemic cardiomyopathy (CM), abnormal renograms may result not only from circulatory failure (which should reverse after transplantation) but also from intrinsic renal disease (which contraindicates heart transplantation). Here, the outcome of heart transplantation was related to preoperative renograms, and the differentiating and prognostic value of renography was analyzed. **Methods:** The study population consisted of 50 patients with ischemic CM expecting heart transplantation. Anatomical renal pathology was excluded in all patients. Dynamic renal scintigraphy was performed with ^{99m}Tc-mercaptoacetyltriglycine. Background-subtracted renograms were inspected visually and characterized numerically. Mean parenchymal transit time (mPTT), renal tracer content at 15 min (RTC15) and retention index (RI) were determined. The parametric renogram values were related to a normal reference group of 64 patients. The preoperative renograms were matched with the postoperative outcome. **Results:** Three characteristic types of symmetrical findings in the kidneys were found: no pathological findings, mildly delayed peak and excretion phase and severely delayed peak and excretion phase. Pathological renograms were observed in 36 of 50 (72%) patients. The mean parametric renogram values in ischemic CM were as follows: Group A (normal kidney function), mPTT = 142 ± 26.6 sec, RTC15 = 22.3% ± 4.6% and RI = 24.7 ± 11.9; Group B (mild dysfunction), mPTT = 210 ± 44.0 sec, RTC15 = 42.6% ± 10.3% and RI = 101.4 ± 50.5; Group C (severe dysfunction), mPTT = 320 ± 94.2 sec, RTC15 = 79.6% ± 15.9% and RI = 347.7 ± 194.7; and reference patients (normal kidney function), mPTT = 137 ± 31.1 sec, RTC15 = 22.8% ± 3.8% and RI = 24.6 ± 7.9. Postoperative serum creatinine levels were < 1.5 mg/dl in all Group A patients, between 1.5 and 2.5 mg/dl in 78% of Group B patients and > 2.5 mg/dl in 75% of Group C patients. **Conclusion:** Renography revealed abnormal kidney function when structural pathology was excluded. The renographic abnormalities in ischemic CM did not reflect simply the circulatory failure. The numerical grading of renograms allowed patient stratification, suggestive of possible renal insufficiency after cardiac transplantation and immunosuppressive therapy. With further experience, renography may become a useful tool for predicting postoperative outcome in ischemic CM.

Key Words: ischemic cardiomyopathy; heart transplantation; dynamic renal scintigraphy; prognosis; renal dysfunction

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The treatment of choice for advanced ischemic cardiomyopathy (CM) is heart transplantation. CM is frequently accompanied by renal dysfunction, which may be the consequence of anatomic renal pathology (cysts, scars and tumors) or renal dysfunction that is secondary to circulatory failure or hypoxemic damage to the kidneys. Renal disease is a relative contraindication to heart transplantation and presents an increased risk for immunosuppressive therapy (*1,2*). Therefore, it is important to exclude pre-existing anatomical renal disease, to recognize reversible renal dysfunction associated with congestive failure from CM and to differentiate severe and possibly irreversible renal impairment due to chronic hypoxia or metabolic or toxic damage.

The aim of this study was to analyze the prognostic value of renography before heart transplantation in patients with CM who have a proven lack of structural anatomic pathology of the kidneys.

MATERIALS AND METHODS

Study Population

The study population consisted of 50 patients (29 women, 21 men; age range 27–49 yr). They were selected from a consecutive series of patients who were evaluated for possible heart transplantation between 1989 and 1995. All patients were expecting heart transplantation and were waiting for a suitable donor organ. All patients studied suffered from CM as a consequence to end stage coronary disease. These 50 patients were selected and studied after CT or ultrasound excluded renal structural abnormalities. Patients with unilateral anatomic renal changes, such as cysts or arteriosclerotic or pyelonephritic scarring, were excluded from further analysis within this study. Patients who underwent heart transplantation began immunosuppressive therapy on the day of transplantation as follows: induction therapy with antithymocyte globulin (mg/kg of body weight) given for 7 days. Basic triple therapy was also administered, with the target levels of cyclosporin (250 mg, tapering down), azathioprine (white blood cell count of 4000–6000) and prednisone (15 mg, tapering down to 7 mg). The follow-up time of the transplanted patients considered in this study ranged between 8 mo and 6 yr.

Reference Population

The reference population consisted of 64 patients with 128 renograms (left and right kidneys). The cases were drawn from the collection of routine renographies of inpatients. Inclusion was based on review of their charts after discharge. Criteria for selection were: age range 25–45 yr, no hypertension, no cardiac disease, no obstructive uropathy and no evidence of renal disease. The renographies of the reference patients and the numerical characteristics were obtained like those of the study population (vide infra). The distribution of the parametric renogram values of the reference patients was considered normal.

Dynamic Renal Scintigraphy

Dynamic renal scintigraphy was performed with ^{99m}Tc-mercaptoacetyltriglycine. The patients were examined while in a sitting position with a wide-field-of-view scintillation camera (Picker Dynacamera 415/61; Picker International, Cleveland, OH). The field of view was from the posterior and included the heart, both kidneys, ureters and the upper crest of the urinary bladder. The patients were hydrated with 500 ml of normal saline given intravenously before the study. The radiotracer was injected

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intravenously in a dose of 66.6 MBq. Diuresis was stimulated by intravenous injection of 40 mg of furosemide 15 min after the injection of the tracer.

Analog Imaging

Analog imaging by sequential scintiphotos was accomplished as follows: 30 frames of 10 sec each and 10 frames of 1 min each, followed by 10 frames of 1 min each after furosemide injection. Simultaneous computer acquisition consisted of 30 frames of 10 sec each and 10 frames of 1 min each, all in 64 × 64 matrix. After these 15 min, the acquisition was prolonged for 10 more min if renal activity had not yet reached its maximum; this was possible because of the simultaneous derivation of the renogram-like curve during acquisition. After furosemide injection, a further 10 images of 1-min duration each were acquired.

Renogram Derivation

Time-activity curves were derived from regions of interest (ROIs) placed over the kidneys, from representative renal parenchymal ROIs and from a so-called plasma region ROI including the left ventricle. The delineation between the renal parenchymal and the collecting system was performed on the rise functional image described by Oppenheim and Appledorn (*3*). This image highlights all parts with high activity concentration, independent of the time of appearance. Background subtraction was based on a sickle-shaped region after the lateral convexity of each of the kidneys. All data processing was performed using the programs developed in our department (*4*). As a matter of course, relative renal clearance values were calculated from the 2-min integral ascending section of the renogram obtained between 80 and 180 sec postinjection. The integral of each side was expressed as a percentage of the sum of the integrals.

Renogram Parameters

The following parameters were considered, and their values were calculated from the background-subtracted, computer-generated renograms (renogram values):

1. Relative renal clearance;
2. Mean parenchymal transit time (mPTT);
3. Renal tracer content at 15 min (RTC15);
4. Renal tracer content at 25 min (RTC25);
5. Washout (WO) from 15 min to 25 min (furosemide-associated WO); and
6. Retention index (RI).

Mean Parenchymal Transit

Mean parenchymal transit times were calculated from parenchymal (i.e., cortical) ROIs using discrete deconvolution of the parenchymal curves with the time-activity curve over the plasma region and were expressed in seconds. Before deconvolution, all curves were data-bounded and nine-point-smoothed (*4–6*).

Renal Tracer Content

Renal tracer content was derived from net counts within kidney ROI and expressed as percentage of peak activity: RTC15 is the ratio (× 100) of the net counts per minute (cpm) in kidney ROI at 15 min divided by the net cpm at peak; and RTC25 is the ratio (× 100) of net cpm at 25 min by net cpm at peak. The RTC15 is the prefurosemide renal tracer content, whereas the RTC25 equals the postfurosemide renal tracer content.

Retention Index

The RI was calculated from the RTC15 and RTC25 (percentages) according to the following equation: $RI = (RTC15 \times RTC25)/10$.

Washout

The normal response to furosemide is an increase in urine flow resulting in a forced diminution of the amount of labeled urine in the renal collecting system (*7*). This WO effect of furosemide was expressed as the percentage change of kidney net counts from the RTC15 (prefurosemide) to the RTC25 (postfurosemide): $[(RTC15 - RTC25)/RTC15] \times 100$.

Statistical Analysis

The individual parametric values of the patient renograms were compared to those of 128 renograms of 64 reference patients verified to be free of kidney disease or hypertension. They were related further by regression analysis to the values of serum creatinine and left ventricular ejection fraction (EF). On the basis of the values of RTC15 and RI, the patients were classified as normal, with RTC15 < 30 and RI < 50, or pathological, having RTC15 > 30 and RI > 50. There were no patients with RTC15 < 30 and RI > 50 or RTC15 > 30 and RI < 50. A further categorization of the pathological renograms was based on the RI values: patients with a RI of 50–200 were classified as Group II, and patients with a RI of >200 were classified as Group III. Later, the postoperative follow-up allowed classification of the patients into three categories of renal dysfunction, as follows: Group A, no postoperative renal dysfunction; Group B, episodic and mild renal dysfunction; and Group C, severe and persisting renal dysfunction.

Statistical analyses were performed using the statistical software package STATGRAPHICS Plus (Windows Version 2.1). Descriptive statistical values (mean, s.d., coefficient of variation, minimum and maximum) were calculated for mPTT, RTC15, RI and WO of all left-sided renograms. We avoided using parametric renogram values of the right side because of possible artifacts from liver congestion. The renogram parameters of Groups I, II and III were compared to each other and to those of the reference group. The statistical significance of the Groups II and III patients were compared to the reference group and to the Group I patients using nonparametric multiple-range tests. A p value of ≤ 0.05 was considered to be significant. The regression analysis between the renogram values and the creatinine or EF values (measured on the day of scintigraphy) was performed using the mathematical model of the hyperbolic function $(A + B/x)$.

Ancillary Studies

Serum creatinine was measured by the JAFFE method on Hitachi 747 using Boehringer Mannheim reagents (normal range 0.6–1.1 mg/dl). Echocardiography was used to obtain left ventricular EFs.

RESULTS

Visual Inspection

Three characteristic types of renograms, i.e., three patient groups, were found:

1. No pathological findings (Group I);
2. Mildly delayed peak and excretion phase (splayed) (Group II); and
3. Severely delayed peak and excretion phase (obstructed) (Group III).

Symmetry between the two kidneys was present in each patient. Overall, pathological renograms were observed in 36 of 50 (72%) patients.

Numerical Analysis

Mean values for the parameters of the normal reference group and the study groups are presented in Table 1, the ranges are given in Table 2 and the statistical significance of the differences in the three patient groups in Table 3. The visually determined symmetry between the right and left kidney was confirmed numerically for the entire collection of patients. In individual patients, a slightly prolonged mPTT was found on

TABLE 1
Numerical Characteristics of Renograms in Ischemic Cardiomyopathy

Group	n	mPTT (mean ± s.d.)	RTC15 (mean ± s.d.)	RI (mean ± s.d.)	WO (mean ± s.d.)
Reference	128	137 ± 31.1	22.8 ± 3.8	24.6 ± 7.9	52.3 ± 13.6
I	14	142 ± 26.6	22.3 ± 4.6	24.7 ± 11.9	47.4 ± 10.9
II	26	210 ± 44.0	42.6 ± 10.3	101.4 ± 50.5	43.7 ± 10.9
III	10	320 ± 94.2	79.6 ± 15.9	347.7 ± 194.7	47.7 ± 15.4
All CM patients	50	220 ± 79.7	44.3 ± 22.4	129.1 ± 146.8	45.5 ± 11.8

mPTT = mean parenchymal transit time; RTC15 = renal tracer content at 15 min; RI = retention index; WO = washout; cpm = counts per minute.

mPTT is expressed in seconds. RTC15 is calculated as the net cpm in the kidney ROI at 15 min divided by the net cpm at the peak × 100 and is expressed as a percentage. RTC25 is the same value at 25 min. RI is calculated as: RI = (RTC15 × RTC 25)/10. Division by 10 serves to simplify the numbers. WO denotes the furosemide-induced diuretic effect; it is expressed as the percentage change of renal net cpm at 15 min (RTC15, prefurosemide tracer renal content) to 25-min renal net cpm (RTC25, postfurosemide renal tracer content). WO = [(RTC15 − RTC25)/(RTC15)] × 100.

If tracer is not removed from kidney speedily, RTC15 and RTC25 remain high and RI becomes very high. If tracer removal is protracted, RTC25 is usually distinctly lower than RTC15, and RI becomes smaller but still higher than normal.

the right kidney. When numerical characterization was used, the renograms remained in the same groupings that were determined visually.

Group I consisted of 14 patients. Renograms were normal on visual inspection (Fig. 1). The Group I values of mPTT, RTC15, RI and WO fell within the range of the reference group, and the mean values were very close to each other (Tables 1–3). Heart transplantation was performed in 11 of 14 patients. Preoperatively, serum creatinine was normal. The postoperative course was uncomplicated and, in particular, there was no renal functional impairment seen during either the immediate postoperative period or later follow-up in each of these patients. Group II consisted of 26 patients with a mild disorder in renal function (Fig. 1). Their renogram values differed significantly from Group I patients for the parameters mPTT, RTC15 and RI (Table 3). For WO, there was no significant difference between Group II and Group I patients. Implicitly, Group II patients also differed from the reference group because the latter was statistically the same as Group I. Heart transplantation was performed in 14 of 26 of these patients. Soon after surgery, 11 of the 14 patients developed an episode of distinctly increased serum levels of creatinine (>1.5 and <2.5 mg/dl). The three other patients had no increased creatinine postoperatively. None of the transplanted patients in this group suffered from an episode of late postoperative or long-term renal dysfunction, and they showed rapid return to normal and steady cyclosporin levels. Group III consisted of 10 patients whose renogram curves showed continuously increasing renal activity and restricted tubulo-epithelial secretion (Fig. 1). However, the ability to empty the renal collecting system after Lasix administration was unimpaired. The renogram values of Group III patients differed significantly in mPTT, RTC15 and RI from Group II and Group I patients (also from reference patients) but not in WO (Table 3). Heart transplantation was performed in 8 of 10 patients. Postoperatively, all patients developed episodes of distinctly increased serum creatinine levels with severe increase in 6 of these 8 patients (>2.5 mg/dl). In the first year follow-up after transplantation, continued renal functional impairment of a low degree was observed in 4 patients and of a high degree, requiring hemodialysis, in 2 patients. One of the transplanted patients died postoperatively from acute pancreatitis.

Matching the preoperative renal studies with postoperative outcome revealed a correlation (Table 4) between Group I and Group A, meaning that the Group I/Group A patients had only transient and slightly increased (>1.5 mg/dl) serum creatinine levels. A good match was also seen in Group II/Group B patients: 11 of 14 (78%) surgical patients with Group II renographic abnormality had increased levels (>1.5 to <2.5 mg/dl) of serum creatinine postoperatively, whereas 3 (22%) surgical patients had no postoperative creatinine elevation. None of the patients suffered from long-term renal dysfunction. In Group III/Group C patients, 6 of 8 (75%) surgical patients developed postoperative episodes of creatinine levels of >2.5 mg/dl (requiring hemodialysis in two patients). In addition, renal dysfunction was seen in 6 of 8 patients for at least 1 yr after transplantation, of a persistent low degree in 4 patients and of a more severe degree requiring hemodialysis in 2 patients.

TABLE 2
Range of Numerical Characteristics of Renograms in Ischemic Cardiomyopathy

	mPTT		RTC15		RI		WO	
Group	Min	Max	Min	Max	Min	Max	Min	Max
Reference	30	250	11	36	4	53	21	86
I	100	180	13	29	11	45	28	62
II	130	330	31	71	43	225	21	60
III	150	450	54	97	124	646	26	67

Min = minimum; Max = maximum; mPTT = mean parenchymal transit time; RTC15 = renal tracer content at 15 min; RI = retention index; WO = washout; cpm = counts per minute.

mPTT is expressed in seconds. RTC15 is calculated as the net cpm in the kidney ROI at 15 min divided by the net cpm at the peak × 100 and is expressed as a percentage. RTC25 is the same value at 25 min. RI is calculated as: RI = (RTC15 × RTC 25)/10. Division by 10 serves to simplify the numbers. WO denotes the furosemide-induced diuretic effect; it is expressed as the percentage change of renal net cpm at 15 min (RTC15, prefurosemide tracer renal content) to 25-min renal net cpm (RTC25, postfurosemide renal tracer content). WO = [(RTC15 − RTC25)/(RTC15)] × 100.

If tracer is not removed from kidney speedily, RTC15 and RTC25 remain high and RI becomes very high. If tracer removal is protracted, RTC25 is usually distinctly lower than RTC15, and RI becomes smaller but still higher than normal.

TABLE 3
Differences in Averages of Numerical Characteristics of Renograms

Difference	mPTT	RTC15	RI	WO
Group II–Group I	68.2*	20.4*	76.6*	−3.7
Group III–Group I	177.9*	57.3*	322.9*	0.34
Group III–Group II	109.6*	36.9*	246.3*	4.0

*Statistically significant difference (Duncan test).

mPTT = mean parenchymal transit time; RTC15 = renal tracer content at 15 min; RI = retention index; WO = washout; cpm = counts per minute.

mPTT is expressed in seconds. RTC15 is calculated as the net cpm in the kidney ROI at 15 min divided by the net cpm at the peak × 100 and is expressed as a percentage. RTC25 is the same value at 25 min. RI is calculated as: RI = (RTC15 × RTC 25)/10. Division by 10 serves to simplify the numbers. WO denotes the furosemide-induced diuretic effect; it is expressed as the percentage change of renal net cpm at 15 min (RTC15, prefurosemide tracer renal content) to 25-min renal net cpm (RTC25, postfurosemide renal tracer content). WO = [(RTC15 − RTC25)/(RTC15)] × 100.

If tracer is not removed from kidney speedily, RTC15 and RTC25 remain high and RI becomes very high. If tracer removal is protracted, RTC25 is usually distinctly lower than RTC15, and RI becomes smaller but still higher than normal.

Regression Analysis

The correlation coefficients between the renogram parameters and serum creatinine are listed in Table 5; those compared to EF are listed in Table 6. Renal tracer content at 15 min correlated significantly to creatinine in Group I patients (normal) and in Group III patients (tubular dysfunction and high serum creatinine). Renal tracer content at 15 min did not correlate to creatinine in Group II patients, suggesting that their renogram alteration was due to circulatory failure. Tubulo-epithelial secretion of the tracer and primary urine flow determine the RTC15 postpeak. Congestive failure and tubular dysfunction inhibit the elimination of creatinine from the body. The parametric renogram values correlated to EF only if all CM patients were considered together; they did not correlate when the groups were considered separately. The EF values were derived from echocardiography (not by radionuclide ventriculography).

DISCUSSION

Heart transplantation is used as the definitive therapy for patients suffering from ischemic CM (*8*). After transplantation, survival is endangered by acute rejection, infection, graft dysfunction, hypertension or renal failure (*9,10*), and renal failure was described as a predominant problem in patients presenting with complications of immunosuppression (*11*). It is important to be able to assess postoperative risk of kidney failure before transplantation.

Renography is one of the standard tests during the preoperative evaluation of patients considered for heart transplantation. It was abnormal in 72% of the study population, although structural kidney disease had been excluded by anatomical imaging. Abnormal renograms may result from circulatory failure because of reduced cardiac output and diminished kidney perfusion (*12,13*), but they may also indicate intrinsic renal disease, such as hypoxic damage. The differentiation is important because irreversible renal dysfunction is one of the major contraindications to heart transplantation.

This study revealed that:

1. Considerable preoperative renal dysfunction may be discovered in patients with CM without any structural pathology of the kidneys; and
2. The postoperative renal function appears to be related to the preoperative renogram.

Matching the preoperative renal studies with postoperative outcome revealed correlations suggesting a prognostic interpretation of the renographic changes. Overall, the numerical grading of renograms allowed patient stratification, suggestive of possible renal decompensation after cardiac transplantation and immunosuppressive therapy. None of the surgical patients with normal renograms had postoperative renal dysfunction. The moderate renographic abnormalities of Group II still meant good postoperative prognosis from a renal standpoint (only transient slightly increased creatinine). On the other hand, 75% of the Group III patients developed postoperative creatinine levels of >2.5 mg/dl, and two patients required hemodialysis. The parametric renogram values of Group III patients, therefore, appeared to predict markedly increased risk of prolonged postoperative renal dysfunction and poor prognosis. This secretory nephropathy of the Group III patients is well known to occur in ischemic, metabolic or toxic tubulo-epithelial damage; in acute tubular necrosis; or in acute renal transplant rejection (*14–19*). The parametric renogram values allowed a meaningful differentiation between three ranges of renographic tracer elimination (healthy, mild renal dysfunction and severe renal dysfunction). Mean parenchymal transit time, RTC15 and RI were useful parameters, whereas WO was not. RTC15, which is quite similar to the R20/3 described by Li et al. (*20*), was, despite its simplicity, quite effective in estimating the degree of inhibition of tracer elimination. The RTC15 correlated significantly in the Group I patients with the normal levels of serum creatinine and in Group III patients with the increased levels of serum creatinine. On the other hand, RTC15 did not correlate to creatinine in Group II patients. The RTC15 is, evidently, not only determined by kidney perfusion and primary urine flow but also, strongly, by tubulo-epithelial function. It was the one parameter most capable of differentiating healthy kidneys (Group I) from dysfunctional patients (Groups II and III). For all CM patients (Groups I, II and III), it had the clearest

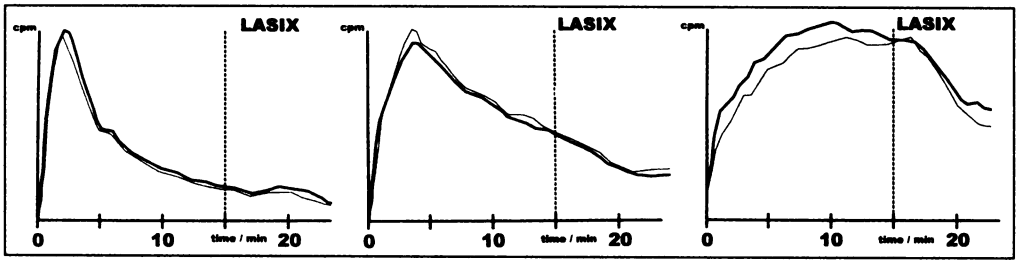

FIGURE 1. Renograms before heart transplantation may indicate adequate renal function (Group I), mild renal dysfunction (Group II) or severe renal dysfunction (Group III). Post-transplantation outcome in Group II was transient mild increase of serum creatinine and in Group III was hemodialysis in 2 patients and increase of serum creatinine above 2.5 mg % in 75% of transplanted patients.

TABLE 4
Preoperative Renogram Compared to Postoperative Renal Dysfunction

Preoperative renogram	Postoperative renal dysfunction			Heart Transplantation
	Group A	Group B	Group C	(No. of patients)
Group I	11 (100%)	0	0	11
Group II	3 (22%)	11 (78%)	0	14
Group III	0	2 (25%)	6 (75%)	8

Groups I, II and III denote degree of renogram abnormality. Groups A, B and C classify patients according to postoperative course [normal (<1.5 mg/dl serum creatinine), mild (>1.5 mg/dl serum creatinine) and severe (>2.5 mg/dl serum creatinine)]. Values are numbers of patients (%).

correlation of all renogram parameters to serum creatinine levels. Renal tracer uptake at 15 min in Group II indicated that their renogram alteration was mainly due to circulatory failure. Our results of renography evidently did not reflect only circulatory failure. Mean parenchymal transit time, an accepted parameter for the numerical characterization of renography, differentiated well between normal and impaired renal function and permitted grading of the renal dysfunction. Makoba et al. (*21*) reported 129 ± 30 sec as a normal value. This is quite similar to our reference group and Group I patients. On the other hand, the values of mPTT of Group II patients imitated those values that Makoba et al. (*21*) reported for patients with dilated renal pelvis (166 ± 45 sec), an entity that was excluded in this patient study. Finally, values of 212 ± 79 sec were reported as a sign of secretory nephropathy in that study. This latter finding was similar to the mPTT values of the Group III patients. The RI provided an index of the global renal functional deficit. It was the best parameter for differentiation of severe renal dysfunction (Group III) from mild dysfunction (Group II).

The cardio-renal interactions in congestive failure and after heart transplantation are complex. Cardiac insufficiency of CM develops gradually. One important compensatory mechanism for maintaining cardiovascular homeostasis is the increase in ventricular filling pressures that is produced by an increase in plasma volume as a result of salt and water retention by the kidneys. The precise mechanisms for the inital changes in renal function that produce water and salt retention are still unclear. The renal adaption to cardiac insufficiency may occur early or may fail completely if the circulatory dysfunction is acute (*22–24*). Renal insufficiency, i.e., deficient elimination of body wastes, is usually caused by severe intrinsic renal disease, such as glomerulonephritis or diabetic vasculopathy (which are regarded as relative contraindications to heart transplantation). It may also develop from circulatory failure (*22*). The latter, in turn, may cause renal ischemia and hypoxia with intrinsic damage to the renal tissues, particularly the tubulo-epithelial cells. Restitution of adequate blood flow to the kidneys after heart transplantation should normalize the renal effects of circulatory failure and, thus, improve elimination of body wastes and urine. Hypoxemic or other metabolic damage to the kidneys may delay or even prevent normalization of renal dysfunction despite re-establishment of adequate renal blood flow. It is of grave importance to be able to judge the likelihood of such kidney damage and, consequently, futile heart transplantation. With further experience, renography may become a useful prognostic tool.

TABLE 5
Regression Analysis: Renogram Parameters Compared to Creatinine

Group	mPTT		RTC15		RI		WO	
	r	p	r	p	r	p	r	p
Group I	0.02	0.937	0.69	0.006*	0.14	0.641	0.30	0.299
Group II	0.05	0.799	0.10	0.616	0.06	0.774	0.13	0.520
Group III	0.62	0.055*	0.73	0.017*	0.24	0.500	0.54	0.106
All CM patients	0.42	0.002*	0.50	0.001*	0.42	0.002*	0.01	0.917

*Significant correlation, $p \leq 0.05$.

r = correlation coefficient; p = probability value of r; mPTT = mean parenchymal transit time; RTC15 = renal tracer content at 15 min; RI = retention index; WO = washout; cpm = counts per minute.

Regression analysis was performed with creatinine as the independent variable, compared to each parametric renogram value of each group. Reciprocal-x model: $y = a + b/x$.

This regression analysis showed a positive correlation between the renogram parameters and creatinine of all patients were considered together and also if Group I and Group III patients were considered separately. RTC15 correlated significantly to creatinine in Group I patients with normal serum levels of creatinine and in Group III patients with tubular dysfunction and high serum creatinine levels. RTC15 did not correlate to creatinine in Group II patients, suggesting that their renogram alteration is due to circulatory failure. Tubulo-epithelial secretion of the tracer and primary urine flow should determine the RTC15 postpeak. Congestive failure and tubular dysfunction inhibit the elimination of creatinine from the body.

CONCLUSION

Dynamic renal scintigraphy, associated with numerical characterization of the renogram, allowed the stratification of patients with ischemic CM for possible risk of heart transplantation failure and postoperative follow-up difficulties in a functional manner in this small group of patients. Renography elucidated renal dysfunction when anatomical imaging was normal. It was not only needed to assess preoperative renal

TABLE 6
Regression Analysis: Renogram Parameters Compared to Ejection Fraction

Group	mPTT		RTC15		RI		WO	
	r	p	r	p	r	p	r	p
Group I	0.02	0.957	0.28	0.336	0.12	0.684	0.11	0.702
Group II	0.10	0.621	0.12	0.544	0.09	0.639	0.48	0.817
Group III	0.70	0.024*	0.55	0.098	0.34	0.328	0.63	0.053
All CM patients	0.65	0.001*	0.70	0.001*	0.69	0.001*	0.11	0.463

*Significant correlation, $p \leq 0.05$.

r = correlation coefficient; p = probability value of r; mPTT = mean parenchymal transit time; RTC15 = renal tracer content at 15 min; RI = retention index; WO = washout; cpm = counts per minute.

Regression analysis was performed with ejection fraction (EF) as the independent variable, compared to each parametric renogram value of each group. Reciprocal-x model: $y = a + b/x$.

The parametric renogram values correlated to EF only if all cardiomyopathy patients were considered together; it did not correlate when the groups were considered separately. The EF values were derived from echocardiography (not by radionuclidventriculography).

function, but it also appeared useful as an important predictor for postoperative outcome. Normal findings on renography meant no contraindication to heart transplantation and good conditions for immunosuppressive therapy. Pathological findings on renograms, as seen in Groups II and III, indicated a high probability of postoperative renal dysfunction, possibly a contraindication to heart transplantation. The more severe the abnormal changes on pretransplant renography, the more they represented important signs heralding risk for the heart transplantation itself, predicting a stormy postoperative course. We now consider these abnormal changes to be a relative contraindication to heart transplantation. Renal scintigraphy provided a simple and economical method for functional evaluation, giving information that can readily be combined with anatomic renal parameters, as demonstrated by CT, ultrasound or MRI, if necessary.

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Post-Therapy Iodine-131 Localization in Unsuspected Large Renal Cyst: Possible Mechanisms

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Sensitive and specific, whole-body ^{131}I scintigraphy remains an important technique for diagnosing metastases from differentiated papillary or follicular thyroid carcinoma. False-positive ^{131}I localization is well recognized and can occur in a variety of conditions. We present a case of intense ^{131}I localization in a previously unsuspected large renal cyst; the lesion was not visualized on routine preablation diagnostic ^{131}I scintigraphy but was obvious on post-therapeutic whole-body imaging, underscoring the value of post-therapy imaging in detecting abnormalities not apparent on diagnostic studies. Radioiodine within the urinary bladder or, at times, the renal collecting system is expected, because ^{131}I excretion is primarily by glomerular filtration. In the case presented here, ^{131}I activity within the renal cyst supports the concept that iodide is subject to an active secretory process by the renal tubule.

Key Words: thyroid carcinoma; iodine-131; renal cyst

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Whole-body ^{131}I scintigraphy and monitoring of thyroglobulin levels remain the mainstays for follow-up of patients with well-differentiated papillary or follicular thyroid cancer. False-positive ^{131}I localization on diagnostic scans has been well documented (*1–9*); it is important to recognize false-positive sites to avoid unnecessary ablation therapy. Radioiodine localization in a renal cyst during diagnostic ^{131}I imaging has been described (*1*). We report marked radioiodine activity in an unknown large renal cyst visualized only on the postablation ^{131}I scan.

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