Poor Technetium-99m-DMSA Renal Uptake with Near Normal Technetium-99m-DTPA Uptake Caused by Tubulointerstitial Renal Disease

Richard J. Quinn and Grahame J. Elder

Department of Nuclear Medicine and Ultrasound and Department of Renal Medicine, Lidcombe Hospital, Sydney, Australia

We present a patient with tubulointerstitial renal disease and poor renal ^{99m}Tc-DMSA uptake. A ^{99m}Tc-DTPA scan was normal and the creatinine clearance only minimally decreased. In this case, ^{99m}Tc-DMSA uptake did not correlate with "global renal function," but rather with the functioning tubular mass.

J Nucl Med 1991; 32:2273–2274

In general, the renal uptake of ^{99m}Tc-dimercaptosuccinic acid (DMSA) is a good measure of global renal function and correlates with the effective renal plasma flow (ERPF), glomerular filtration rate (GFR) as measured by creatinine clearance and renal scanning with ^{99m}Tc-diethylenetriaminepentaacetic acid (DTPA) (*1,2*). In this report, we describe a patient who had minimal uptake of ^{99m}Tc-DMSA on two occasions with only moderate GFR impairment as measured by creatinine clearance and a near-normal renal scan with ^{99m}Tc-DTPA.

Biochemical and biopsy evidence of renal tubular damage was present. The lack of uptake of ^{99m}Tc-DMSA is most likely due to a discrete tubular defect and does not reflect overall renal impairment.

CASE REPORT

A 23-yr-old male presented for review of abnormal renal function. During a febrile illness at age 14 mo, proteinuria had been noted. At age 14, he was investigated for growth retardation, as his height was below the third centile and his bone age was measured at 10 yr. Examination at that time revealed bilateral loin tenderness and proteinuria. A renal biopsy was performed, which showed changes of interstitial nephritis with tubular atrophy, interstitial fibrosis and foci of chronic inflammation. There were secondary glomerular changes with one-third of them showing partial or complete sclerosis. He remained well with no treatment.

At this presentation, his height was 150 cm and his weight was 47 kg. Blood pressure was 105/65 lying and 85/65 standing. Urinalysis showed pH 7, 30 m*M*/liter glucose, 0.1 g/liter protein and was negative for blood. Quantitative white blood cell (WBC) excretion rate was 200,000 WBC per hour (normal less than 100,000 per hour) and there were 1,150 casts per ml (waxy, granular, hyaline and WBC). Blood sugar level and glucose tolerance tests were normal, serum electrolytes were normal, serum creatinine was 0.175 m*M*/liter (normal less than 0.11), ionized calcium was 1.16 m*M*/liter (1.08–1.3), serum phosphorous was 0.62 m*M*/liter (0.8–1.55) and alkaline phosphatase 125 InU (normal less than 95). Serum uric acid and magnesium were normal. 1,25 OH Vitamin D was normal, intact PTH was 11.7 pmol/liter (1.1–6.4). Creatinine clearance was 65 ml/min per 1.73 m (70–150) and there was slight generalized aminoaciduria. Fractional phosphate reabsorption was reduced. Maximal urinary osmolality following desmopressin was 355 mOsm/kg H2 (normal greater than 830).

Since a retrograde study showed normal collecting systems and ultrasound demonstrated some cortical thinning with no focal abnormalities, a ^{99m}Tc-DMSA study was performed to determine whether focal scarring was present. The 2-hr postinjection images demonstrated very poor renal uptake compared to background (Fig. 1).

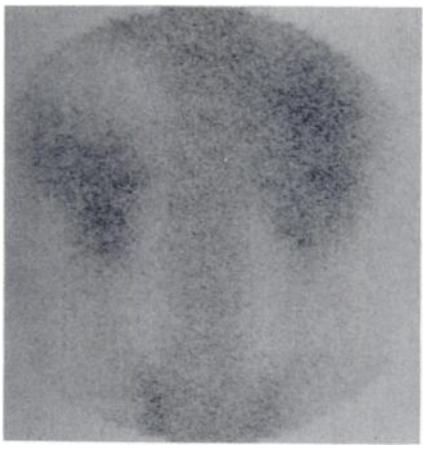

FIGURE 1. Posterior ^{99m}Tc-DMSA image, demonstrating bilaterally poor uptake.

Received Apr. 3, 1991; revision accepted Jun. 26, 1991.

For reprints contact: Dr. Richard J. Quinn, Department of Nuclear Medicine, St. George Hospital, Belgrave St., Kogarah, NSW, Australia 2141.

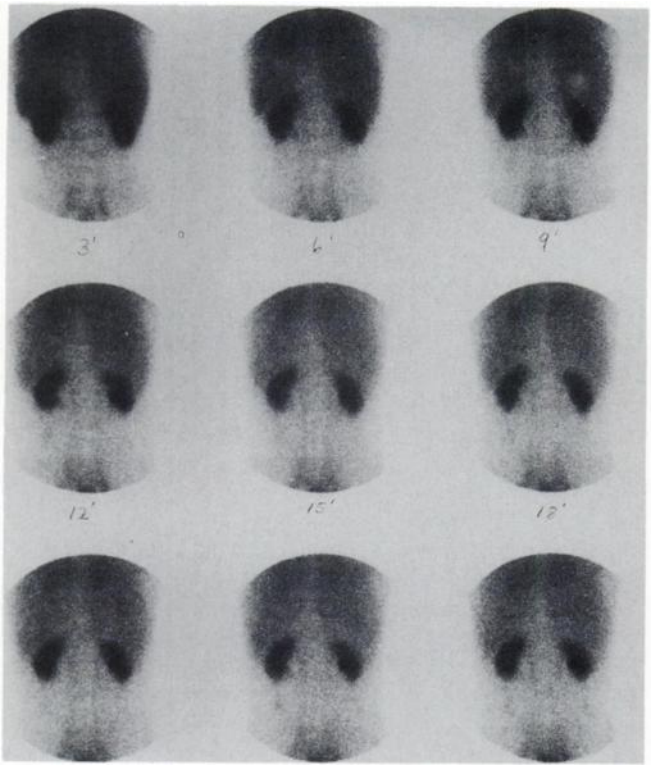

FIGURE 2. Sequential ^{99m}Tc images to 27 min postinjection demonstrating changes consistent with mild functional impairment.

Fearing a pharmaceutical problem, the study was repeated 1 mo later. This time the radiopharmaceutical was prepared and another patient undergoing a ^{99m}Tc-DMSA study at the same time was injected with a dose from the same vial. Again there was very poor renal uptake of ^{99m}Tc-DMSA, while the other patient demonstrated normal renal uptake.

A ^{99m}Tc-DTPA scan was performed and the cortical uptake and excretion phases demonstrated changes consistent with a mild functional deficit only (Fig. 2). A repeat renal biopsy demonstrated similar histology to that performed 9 yr before with interstitial fibrosis, chronic inflammation and tubular atrophy. The blood vessels were thick-walled, one-third of the glomeruli were obsolete, and the remainder were hypertrophied but otherwise normal. Immunofluorescence was negative and electron microscopy showed no immune deposits.

DISCUSSION

This patient demonstrated poor renal uptake of ^{99m}Tc-DMSA and normal handling of ^{99m}Tc-DTPA. Biochemical studies in this patient showed tubular abnormalities of both proximal and distal origin and renal biopsy showed significant tubulointerstitial damage with little abnormality of remaining glomeruli.

The exact mechanism of ^{99m}Tc-DMSA handling by the human kidney is uncertain. Technetium-99m-DMSA is highly protein-bound and in rat renal uptake it appears to be from peri-tubular capillaries with insignificant glomerular filtration (*3*). Technetium-99m-DMSA binds in the rat to cytoplasmic proteins in the proximal tubular cells and in the first part of the loop of Henle with minimal glomerular activity (*4*). This underlies its usefulness in showing focal diffuse scars, which historically are areas of fibrosis and tubular damage. Technetium-99m-DMSA uptake in the kidneys has been shown to correlate closely with ERPF in patients with serum creatinines up to and including 2 mg/dl (0.177 m*M*/liter) (*1*). Technetium-99m-DMSA uptake generally correlates well with serum creatinine. Kawamura et al. suggest that it is an index of renal function (*5*). Technetium-99m-DMSA uptake at 24 hr has been shown to correlate excellently with ^{99m}Tc-DTPA accumulation at 1–3 min postinjection in patients with serum creatinines up to 2.5 mg/dl (0.221 m*M*/liter) (*6*). Of interest, Taylor et al. have described absence of ^{99m}Tc-DMSA uptake in a patient with tubular necrosis (*7*).

We have presented a patient in whom there was very poor uptake of ^{99m}Tc-DMSA on two occasions despite a ^{99m}Tc-DTPA scan demonstrating only mild impairment and a serum creatinine of 0.175 m*M*/liter. This demonstrates the need to consider ^{99m}Tc-DMSA uptake an index of "functioning tubular mass" rather than "global renal function" and that in the face of predominant tubulointerstital disease the previously reported correlation with GFR and ERPF may be poor.

REFERENCES

1. Taylor A. Delayed scanning with DMSA: a simple index of relative renal plasma flow. *Radiology* 1980;136:449–453.
2. Kawamura J, Hosokawa S, Yoshida O. Renal function studies using Tc-99m-dimercaptosuccinic acid. *Clin Nucl Med* 1979;4:39–47.
3. Goldraich NP, Alvarenga AR, Goldraich IH, Ramos OL, Siqulem D. Renal accumulation of ^{99m}Tc-DMSA in the artificially perfused isolated rat kidney. *J Urol* 1985;134:1281–1286.
4. Treves S, Martinez DA, Dauss MA, et al. Cellular localization of Tc-99m radiopharmaceuticals by radioautography [Abstract]. *J Nucl Med* 1976;17:536.
5. Kawamura J, Hosokawa S, Yoshida O, Fujita T, Ishii Y, Torizuka K. Validity of Tc-99m-dimercaptosuccinic acid renal uptake for an assessment of individual kidney function. *J Urol* 1978;119:305–309.
6. Kipper MS, Witzum K, Taylor A. Visual determination of differential renal function. What to do until the computer comes [Abstract]. *J Nucl Med* 1982;23:64.
7. Taylor A, Akiya F, Gregory M. Failure to visualize acutely injured kidneys with technetium-99m-DMSA does not preclude recoverable function. *J Nucl Med* 1986;27:377–379.