Significance of Delayed ^{67}Ga Localization in the Kidneys

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One hundred and seventy-five ^{67}Ga-citrate scans were performed to detect suspected occult inflammatory processes. None of the patients had a known malignancy. Renal activity was noted in 12 patients (6.8%) on the 48-hr image. These patients had either pyelonephritis, acute tubular necrosis, vasculitis, or a renal abscess. Since delayed ^{67}Ga uptake in the kidneys may be the first evidence of renal disease, further investigation, including either arteriography or biopsy, is necessary. In patients with a known malignancy, tumor involvement must be considered.

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Gallium-67-citrate has been widely investigated as a tumor-scanning agent (*1–4*). Although its lack of specificity and the superiority of other techniques have limited its use as a tumor seeker in many centers, ^{67}Ga-citrate is now being used as an abscess-seeking agent (*5–14*) in patients with fever of unknown origin or with a strong suspicion of an abscess.

Under normal circumstances, approximately 12% of the administered ^{67}Ga activity is excreted by the kidneys during the first 24 hr after injection (*15,16*). After the first day, the liver and gut become the major routes of excretion. Renal accumulation of ^{67}Ga-citrate after 24 hr may be the first indication of renal involvement with either tumor or inflammation (*17*), since renal activity should not be seen on scans obtained 48 or 72 hr after administration (*16*).

MATERIALS AND METHODS

Between June 1974 and September 1975, 175 ^{67}Ga whole-body scans were performed. The scans were usually requested for detection of a suspected occult abscess. None of the scans in this series was requested to detect malignancy or to determine the extent of a known primary tumor. Prior to ^{67}Ga scintigraphy, these patients had had routine clinical and radiologic studies that had not localized an infectious process. The ^{67}Ga was administered intravenously (50 μCi/kg), and the images were taken 48 and 72 hr later on a dual 12.7-cm-crystal rectilinear scanner with 1:2 image reduction. Activity in the kidneys at 48 hr was considered abnormal.

RESULTS

Bilateral or unilateral renal activity on the 48-hr image was noted in 12 patients (6.8%; Table 1). The abnormal activity was found in both kidneys in six patients: two with pyelonephritis; one with pyelonephritis and vasculitis; one with left renal vein thrombosis and acute tubular necrosis secondary to antibiotic therapy; one with Wegener's granulomatosis and severe glomerulitis, confirmed by renal biopsy; and one with polyarteritis nodosa and extensive cortical necrosis of both kidneys, identified at autopsy. The other six patients had increased activity in one kidney: two patients with renal transplants and acute tubular necrosis; one with acute renal failure secondary to staphylococcal septicemia; two with pyelonephritis; and one with hydronephrosis, pyelonephritis, and an upper pole renal abscess. We present two representative cases with delayed ^{67}Ga accumulation in the kidneys.

Case 1. A 63-year-old woman had been admitted to another hospital 8 weeks before this admission,

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TABLE 1. ABNORMAL ACCUMULATION OF ^{67}Ga

Patient No.	Scan finding	Diagnosis	Supporting data
1	Bilateral accumulation	Pyelonephritis	IVP, blood culture, urine culture
2	Bilateral accumulation	Pyelonephritis	Urine culture
3	Bilateral accumulation	Pyelonephritis, vasculitis	Urine culture
4	Bilateral accumulation	Wegener's granulomatosis	Biopsy
5	Bilateral accumulation	Acute tubular necrosis, left renal vein thrombosis	Renal arteriogram
6	Bilateral accumulation	Polyarteritis nodosa	Autopsy
7	Unilateral accumulation	Pyelonephritis	Nephrectomy showed pyelonephritis
8	Transplanted kidney	Acute tubular necrosis	Renogram, clinical course
9	Unilateral accumulation	Acute renal failure	Septicemia
10	Unilateral accumulation	Pyelonephritis	IVP, pyelolithotomy
11	Unilateral (upper pole) accumulation	Renal abscess	Angiography
12	Transplanted kidney	Acute tubular necrosis	Renogram, clinical course

with the chief complaints of a gradual onset of fever, night sweats, shaking chills, progressive weakness, and pleuritic chest pain. Physical examination at that time was unremarkable except for decreased breath sounds over the right lung base with scattered rales. A chest film revealed atelectasis in the right middle lobe; this remained unchanged during the 8 weeks. Bronchoscopy revealed nonspecific inflammatory changes in the right middle lobe. Laboratory tests during that hospitalization revealed a white blood count increasing from 7,100/mm^3 to 13,000/mm^3 and a hematocrit decreasing from 38% to 28% over 3 weeks. Urinalysis revealed 35 red blood cells and 15 white blood cells per high-power field and fine granular casts. Blood and sputum cultures were negative. An intravenous pyelogram (IVP) revealed nephrocalcinosis.

By the time of admission to this hospital, the patient had lost 14 pounds since the onset of her illness. Her fever continued, with occasional night sweats. On the second hospital day, a 48-hr ^{67}Ga-citrate scan showed accumulation of ^{67}Ga only in the kidneys (Fig. 1). The culture of a catheterized urine sample produced more than 10,000 *E. coli*, and she was started on Keflex, 500 mg four times a day. The patient became afebrile and remained so for the duration of her hospital stay. Neurologic examination following the gallium scan revealed significantly decreased deep tendon reflexes on the right with a marked right foot drop. Right gastrocnemius biopsy revealed changes compatible with vasculitis and polymyositis. Her subsequent hospital course was complicated by gastrointestinal bleeding, hemoptysis, and progressive renal failure, with a blood urea nitrogen level of 74 mg/dl, creatinine of 5.0 mg/dl, potassium of 5.8 meq/liter, urine sodium of 59 meq/liter, and a urine-to-plasma creatinine ratio of 10. The creatinine clearance was 9 ml/min. For her vasculitis, the patient was started on steroids and cyclophosphamide with improvement in her condition, particularly in her renal function. She was discharged with a diagnosis of vasculitis, which was related to her gastrointestinal bleeding, hemoptysis, renal failure, anemia, and myositis.

Case 2. A 60-year-old man was transferred for treatment of fever, leukocytosis, left maxillary sinusitis, bilateral basal atelectasis, and renal failure following Keflex and gentamicin therapy. His creatinine levels had increased from 4 to 11 mg/dl over 3 weeks. By the intravenous pyelogram, the right kidney measured 15 cm and the left kidney 17.3 cm,

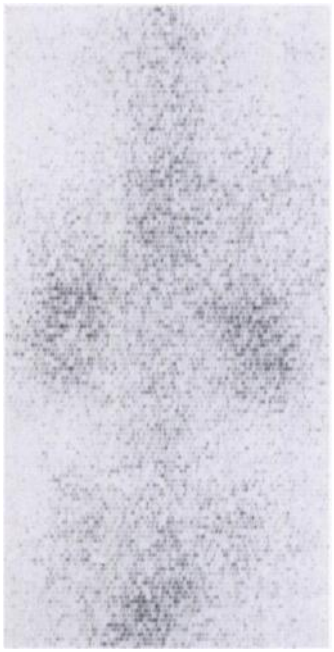

FIG. 1. Abnormal 48-hr ^{67}Ga-citrate scan showing increased renal activity in patient with pyelonephritis, renal failure, and vasculitis.

FIG. 2. Abnormal 48-hr ^{67}Ga-citrate scan showing increased renal activity in patient with Wegener's granulomatosis.

and there was nonvisualization of the collection systems. Cultures for *Mycobacterium* and fungi and routine cultures of blood and bone marrow were negative. The patient continued to be febrile, his blood urea nitrogen increased to 184 mg/dl, and his mental status began to deteriorate. He was started on hemodialysis.

Liver–spleen and brain scans were normal. Sinus films showed bilateral maxillary sinusitis with both air and fluid within the maxillary sinus. A ^{67}Ga-citrate scan showed increased activity in both kidneys on the 48-hr study (Fig. 2). Treatment with ampicillin and oxacillin was started. A bilateral antral irrigation was performed without improvement. A left renal biopsy was consistent with Wegener's granulomatosis and also revealed severe glomerulitis with large fibrin deposits throughout. The patient was started on cyclophosphamide, and 3 days later his urine output increased and his renal function improved significantly.

DISCUSSION

Gallium localization in acute pyelonephritis has been described in several previous reports. Frankel et al. (*17*) reported renal localization of ^{67}Ga in 1.7% of 2,000 patients. Thirteen of these 34 patients were autopsied: nine had tumor bilaterally, one had chronic pyelonephritis, one had acute fungal inflammatory disease, and two had no renal disease. In the remaining 21 patients, the ^{67}Ga kidney localization showed poor correlation with other studies, but several of these patients did not have a complete urologic evaluation. Frankel et al. stress the nonspecificity of ^{67}Ga localization, and renal angiography or biopsy may be required to determine the exact significance of abnormal ^{67}Ga uptake.

Our study shows a good correlation between abnormal ^{67}Ga accumulation and other evidence of renal disease. In several patients the first evidence of renal involvement was the abnormal ^{67}Ga scan. None of our patients were found to have renal tumors. This is presumably due to the patient population studied: all had suspected inflammatory lesions and no known tumors.

We had two patients with renal transplants and acute tubular necrosis; both had abnormal ^{67}Ga accumulation. These two patients subsequently recovered from the acute tubular necrosis. George et al. (*18*) reported abnormal gallium uptake in patients rejecting renal transplants. However, neither of our two transplant patients exhibited evidence for rejection. The mechanism of ^{67}Ga uptake in patients with acute renal failure is uncertain.

In patients with abnormal accumulation of ^{67}Ga in the kidneys, further investigation, including either arteriography or biopsy, is necessary since infection, acute tubular necrosis, or vasculitis may be present. In a patient with a known malignancy, renal involvement by tumor must be considered.

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