Gout secondary to chronic renal disease: studies on urate metabolism

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SUMMARY A report of 20 cases of gout considered to be secondary to chronic renal disease is presented. Studies of renal function and of uric acid metabolism were carried out in 16 patients. The daily production of urate remained within normal limits in the face of progressive renal dysfunction. Renal excretion of uric acid was decreased to a mean of 35.5% of the turnover. The cumulative urinary recovery of intravenously injected 14C-urate averaged 32.0%. In 3 patients 14C was successively retrieved in urinary allantoin and urea, in carbon dioxide of expired air, and in faeces. As in normal man, carbon dioxide and ammonia were the principal uricolytic products. The extrarenal excretion of uric acid assumes a greater role in chronic renal disease and eventually becomes the major route of elimination of uric acid. The possibility that gout may be secondary to intrinsic renal disease should be entertained when azotaemia is present.

Moderate hyperuricaemia frequently occurs early in the course of chronic renal disease. In such patients a positive correlation exists between the serum urate and the serum creatinine concentrations. However, as renal function further deteriorates, plasma urate level is likely to remain between 0.42 and 0.54 mmol/l until the glomerular filtration rate has fallen to 15 ml per minute. Despite the persistent mild hyperuricaemia in intrinsic chronic renal disease, acute gouty arthritis is generally thought to be a rare complication. Among 496 patients with chronic renal insufficiency Sarre found only 2 in whom gout was clearly a complication of chronic nephropathy. Richet et al. reported 17 cases of secondary gout among 1600 patients with chronic renal disease. This complication was observed during the course of renal diseases of longer duration. Certain varieties of renal disease appear to be associated with an increased incidence of gout, e.g., chronic lead nephropathy, polycystic kidney disease, and renal amyloidosis.

This communication reports 20 cases of gout considered to be secondary to primary renal disease. Studies of uric acid kinetics were performed in 16 of these patients in order to assess changes in urate metabolism due to chronic renal disease.

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Patients and methods

Sixteen patients participating in metabolic studies were maintained on a diet essentially free of purines for 5 days before as well as during the study. All drugs were discontinued 2 weeks prior to the study. Plasma and urinary uric acid was determined by an enzymatic spectrophotometric method. On the sixth day 14C-urate was injected intravenously in a dose of 20 to 40 μCi. Uric acid was isolated from the urine and purified as described elsewhere. Radioassay of 14C was done either in a vibrating reed electrometer after wet oxidation of uric acid in a vacuum combustion line or in a liquid scintillation spectrophotometer by the following technique: Prewashed amounts of uric acid were dissolved in 2 ml hyamine hydroxide, then mixed with 18 ml of a mixture of PPO (2,5-diphenyloxazole), 3 g, POPOP (1,4-bis-2-(5-phenylloxazolyl)-benzene), 100 mg, and toluene, 1000 ml. In this system 14C-toluene counted with an efficiency of approximately 70%. All samples were corrected for a modest degree of quenching, either by including internal standards or by applying a factor derived from a curve relating quenching to sample weight. Urate pool and turnover data were calculated by the isotope dilution principle.

The extent to which 14C could be recovered in
degradation products of uric acid was studied in 3 patients. The methods for determination of $^{14}$C in urinary allantoin and urea, in carbon dioxide of expired air, and in faeces have been reported in detail. No attempt was made to isolate allantoin acid, since the pH of the urine of these 3 patients was less than 6, and allantoin acid is known to degrade spontaneously to urea and glyoxylic acid at pH below 7.3.

In 12 patients renal function was determined by the clearance of inulin, in 3 by the clearance of creatinine and in 1 by the level of serum creatinine. Inulin clearance studies were carried out in the morning after a 12-hour fast. Each subject underwent aggressive oral hydration with tap water before and during the study. After administration of a priming dose, inulin was infused intravenously at a constant rate with a Harvard pump in a dose calculated to equal the estimated clearance rate. Urethral catheterisation was avoided in 6 patients. In the remaining 6 urine was collected through an indwelling Foley catheter with air washout to ensure complete collection. All catheterisations were performed with stringent aseptic technique utilising Neosporin ointment as a lubricant. Urine cultures 24 hours and 1 week after each study were sterile. Clearance periods varied from 10 to 30 minutes, and a minimum of 3 clearance periods were employed for each study. Inulin analyses were carried out according to the method of Roe et al. Informed consent was obtained from each patient.

A summary of the clinical features of patients studied is presented in Table 1. All 16 patients had experienced attacks of acute gouty arthritis. Two patients, both women, had tophaceous deposits. Crystals of monosodium urate were demonstrated by compensated polarised light microscopy in 13 cases. Nephropathy preceded the onset of gouty arthritis in all but 1 patient (case 13).

The aetiology of the primary renal disease was verified by post-mortem examination (cases 5, 8, 9, 12, 14, and 15); raised urinary lead levels, with or without stimulation by calcium disodium edetate (cases 1, 2, and 3); radiographically (cases 6 and 7); and by haemoglobin electrophoresis (case 10). Case 16 developed haemorrhagic nephritis after ingestion of turpentine oil; renal biopsy several years later showed focal chronic interstitial nephritis. Case 4 had a history of scarlatina, chorea, and kidney disease with oliguria in early childhood. Case 11 had been known to have accelerated essential hypertension for 6 years before developing his first attack of acute gouty arthritis. He died after an arterial graft replacement. Permission for necropsy was not granted. Case 13 had been studied by Dr Bartter at the Hypertension-Endocrine Branch, National Heart and Lung Institute, NIH, 7 years previously, when his creatinine clearance was 61 ml/min. This patient is case 2 in a series of 9 patients with Bartter's syndrome reported by that laboratory. Detailed accounts of the clinical histories of patients with lead nephropathy and sickle cell anaemia have been published elsewhere.

Cases 17 to 20 were not studied in detail. They have been included in this series because they serve as illuminating examples of gouty arthritis in chronic renal failure.

**CASE 17**
A 66-year-old woman presented with acute arthritis of the left first metatarsophalangeal joint, extending to the dorsum of the foot. Aspiration yielded 0·5 ml of milky fluid which under polarised light contained negatively birefringent crystals. Plasma urate concentration was 0·56 mmol/l and serum urica nitrogen was 14·6 mmol/l. Sixteen years previously, the patient underwent removal of 3 hyperplastic parathyroid adenomas, one of them weighing close to 30 g. At the time of surgery multiple bilateral interstitial renal calcifications were present. Prior to surgery serum calcium had ranged between 3·52 and 3·90 mmol/l and serum phosphorus between 0·61 and 0·90 mmol/l.

**CASE 18**
A 56-year-old woman was seen in the outpatient clinic for acute arthritis of the right ankle. Swelling and redness were present. 3 ml of inflammatory fluid was aspirated from the subtalar joint. Intracellular negatively birefringent crystals were present. The patient had experienced 5 similar episodes during the preceding 3 years. Mild hypertension had been noted off and on for 5 years, and during the past year she had taken hydrochlorothiazide. Laboratory tests revealed a plasma urate of 0·85 mmol/l while on the diuretic and 0·81 mmol/l off medication. Serum calcium was 3·15 and 3·10 mmol/l and serum inorganic phosphorous 0·87 and 0·81 mmol/l. Parathyroid hormone level was elevated at 1·9 ng/ml compared to an upper normal value of 1·2 ng/ml. Serum urea nitrogen was 17·1 mmol/l. The urine contained 3·27 mmol of uric acid in a 24-hour period, and urate clearance was 2·8 ml/min. The patient refused surgical exploration of the neck for primary hyperparathyroidism.

**CASE 19**
A 64-year-old woman presented with acute inflammation of the metatarsophalangeal and interphalangeal joints of the left big toe. Synovial analysis showed numerous negatively birefringent crystals mainly inside polymorphonuclear leucocytes.
### Table 1  Summary of clinical features and data on uric acid metabolism in 16 patients with chronic renal disease studied while on a purine-free diet

<table>
<thead>
<tr>
<th>Case</th>
<th>Sex</th>
<th>Age</th>
<th>Renal diagnosis</th>
<th>Gouty arthritis</th>
<th>Tophi</th>
<th>Inulin clearance</th>
<th>Urate clearance</th>
<th>Plasma urate</th>
<th>Miscible pool of uric acid</th>
<th>Turnover of uric acid</th>
<th>Urinary uric acid</th>
<th>Urinary uric acid</th>
<th>Recovery of $^{14}$C-uric acid</th>
<th>Per cent of turnover</th>
<th>Per cent of dose</th>
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<td>50</td>
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<td>30*</td>
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<td>49*</td>
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<td>0.302</td>
<td>9.10</td>
<td>4.40</td>
<td>1.010</td>
<td>23.0</td>
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<td>0.768</td>
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<td>707**</td>
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<td>0.491</td>
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<td>16</td>
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<td>Focal chronic interstitial nephritis</td>
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<td>0.709</td>
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<td>3.45</td>
<td>1.180</td>
<td>34.2</td>
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</table>

*Creatinine clearance.  **Serum creatinine, μmol/l.
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During the preceding 2 years she had experienced 3 similar attacks localised to the first metatarsophalangeal joints. The history was significant for several congenital abnormalities, including complete absence of the left kidney, and recurrent urinary tract infections. On a purine-free diet plasma urate concentration was 0.46 mmol/l, urinary uric acid 2·12 mmol/24 h, urate clearance 3·21 ml/min, and creatinine clearance 42 ml/min with a serum creatinine of 141 μmol/l.

Case 20
A 55-year-old woman was admitted to hospital for fatigue, malaise, weight loss, and intermittent left flank pain of 10 months’ duration. During the preceding 12 years she had had multiple episodes of dysuria and urinary frequency for which she had been treated with sulphonamides. A diagnosis of gouty arthritis based on 2 attacks of acute inflammation of the left foot, promptly responding to colchicine, and associated with hyperuricaemia, was made 2 years earlier. Serum urea nitrogen was 15·4 mmol/l and plasma urate 0·613 mmol/l. Urine analysis revealed 15 to 18 leucocytes per high power field. Retrograde pyelograms showed bilateral hydronephrosis with narrowing of the left ureter. An inferior vena cavaogram showed displacement and encroachment upon the inferior vena cava by a retroperitoneal process. Since the BUN continued to rise it was felt that the indwelling ureteral catheters did not provide adequate drainage. Surgical exploration showed the ureters to be embedded in a thick fibrous tissue, which was partly excised. To ensure adequate drainage bilateral nephrostomies were performed. However, the BUN continued to rise and the patient succumbed to anaemia and superimposed infection. The anatomical diagnosis was retroperitoneal fibrosis.

Results
A summary of data on the miscible pool, daily turnover, and urinary excretion of uric acid in 16 patients with chronic renal disease is presented in Table 1. The clearance rate of inulin ranged from 3 to 68 ml/min. Despite significant reduction in glomerular filtration the miscible pool of uric acid was only modestly raised, far less than that of other nitrogenous waste products.

Three patients with chronic lead nephropathy had plasma urate levels below 0·42 mmol/l while on a purine-free diet. On a regular diet plasma urate levels rose by 0·12 mmol/l. A similar rise in plasma urate with the addition of exogenous purines has been noted in patients with saturnine gout.14

The daily turnover of uric acid was normal in all patients except for the patient with sickle cell anaemia, who had a uric acid production of 5·46 mmol/day, a value which is about 40% higher than expected on the basis of his weight. An increased production of uric acid is not unexpected in a patient with chronic haemolytic anaemia.

The urinary excretion of uric acid ranged from 22·9 to 64·2% of the uric acid produced, with a mean value of 35·5%. The cumulative urinary excretion of injected 14C-urate acid is shown in Fig. 1 for the first 13 subjects and in Figs. 2, 3, and 4 for the remaining 3 patients. The urinary recovery of 14C-urate acid varied from 22·0 to 53·6% of the administered dose, the average value being 32·0%. This figure corresponds closely to the urinary uric acid as a percentage of the daily turnover, validating the calculation of the turnover rate of uric acid. In 9 healthy subjects urinary recovery of 14C-urate acid ranged from 63·0 to 75·5% of the injected dose, the mean value being 67·8%.15 With the 2 tailed, nonpaired Student’s t test the difference in urinary recovery of 14C-urate acid between the 2 groups is significant at a P value of <0·001.

In all but 2 of the 16 patients extrarenal excretion of uric acid was the major route of elimination. Figs. 2, 3, and 4 illustrate the recovery of 14C in uric acid and in various breakdown products for 3 of the patients. The amount of 14C recovered in the degradation products was 1·45, 2·06, and 3·24 times greater than that recovered in urinary uric

![Fig. 1 The recovery from urine of intravenously injected 14C-urate acid in 13 patients with chronic renal disease. The index number refers to case number in Table 1.](http://ard.bmj.com/)

*Fig. 1 The recovery from urine of intravenously injected 14C-urate acid in 13 patients with chronic renal disease. The index number refers to case number in Table 1.*
Fig. 2 Recovery of $^{14}$C-uric acid administered intravenously to a patient with chronic pyelonephritis (case 14 in Table 1) (Reproduced by permission of Springer-Verlag, Berlin-Heidelberg).

Fig. 3 Recovery of $^{14}$C-uric acid administered intravenously to a patient with diabetic glomerulosclerosis (case 15 in Table 1).

Fig. 4 Recovery of $^{14}$C-uric acid administered intravenously to a patient with chronic interstitial nephritis (case 16 in Table 1).

Discussion

Although acute gouty arthritis has been reported infrequently and even questioned as a complication of chronic renal disease, personal experience suggests that intrinsic renal disease is the underlying cause of gout more frequently than is generally accepted. The true incidence of this sequel is difficult to assess with certainty. It will obviously depend on how thoroughly it is looked for, but frequently it is not possible to dissociate the role of such factors as hypertension and drugs that can contribute to hyperuricaemia by affecting uric acid excretion. In years past, before the era of efficacious treatment of hyperuricaemia, primary gout was not infrequently complicated by gouty nephropathy. There are, however, no firm data to suggest that primary hyperuricaemia will induce renal damage by urate crystal deposition within the renal parenchyma before it causes acute gouty arthritis.

The 20 reported cases represent the most clear-cut examples of gout secondary to a primary renal disease among some 400 gout patients who have
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come to our attention during the past 20 years. It has been possible to define the nature of the renal
disease with reasonable certainty, and the renal
disease preceded the onset of gouty arthritis in all
but 2 patients. Although the diagnosis of a well-
defined nephropathy antedating gout strengthens
the diagnosis of secondary renal gout, this is not an
absolute requirement. Case 18 is an example in point.
This patient developed acute gouty arthritis as the first
manifestation of primary hyperparathyroidism
with renal involvement.

Yü and Berger16 found that 253 of their 1700
patients with primary gout had evidence of varying
degrees of renal involvement. Of these, 84 were
considered to have primary intrinsic renal disease,
such as chronic glomerulonephritis, chronic pye-
lonephritis, congenital renal disease, and proteinuria
of undetermined aetiology preceding gout by a mean
of 15 years. Three patients had polycystic kidney
disease and 2 women had onset of nephropathy
during pregnancy. Although Yü and Berger con-
sidered these 84 cases to have primary gout with
independently occurring intrinsic renal disease, it
can be argued that at least some of their cases represent
gout that is secondary to a nephropathy, especially
since the onset of renal disease antedated gouty
arthritis by many years in a good number of the
patients.

As mentioned in the introduction, there are a few
recognised entities in which gout occurs as a signi-
ficant complication of chronic renal disease. 
Approximately 50% of patients with chronic lead
nephropathy develop gouty arthritis.4, 5 Other
nephropathies associated with an increased inci-
dence of gout include polycystic kidney disease6, 7
and renal amyloidosis.5 A prevalence of hyperurica-
aemia of 50% and of gouty arthritis of 20% has
been observed in a group of patients with Bartter's
syndrome.11 It was suggested that systemic alkalosis,
a prominent feature of Bartter's syndrome, may
contribute to hyperuricaemia by decreasing the
clearance of uric acid. Our study, together with the
experience of Richet et al.,9 suggests that any type
of renal disease associated with prolonged hyper-
uricaemia may result in gouty arthritis.

Studies on uric acid metabolism showed a modest
increase in the miscible pool of uric acid, averaging
about 50%. The production of uric acid was within
the normal range, and thus renal failure does not
affect extrarenal aspects of urate kinetics. All the
patients studied had a decreased excretion of uric
acid in the urine. The mean recovery of 14C-uric
acid in the urine was 32-0% whereas in healthy
subjects we have found an average recovery of
67-8% of injected 14C-uric acid in the urine.15
The difference between the daily turnover of uric
acid and the uric acid excreted in the urine in a 24-
hour period represents that uric acid which has
entered the gut in digestive juices. The recovery of
14C in uricolytic breakdown products emphasises the
enhanced enteral uricolyis that occurs as a com-
ensatory measure in chronic renal disease with
compromised renal excretion of uric acid.

The distribution of 14C in various breakdown
products indicates that carbon dioxide and ammonia
are the principal degradation products of uric acid.
Three patients in this study excreted 44, 54, and 71%
of injected 14C as carbon dioxide in expired air or in
faeces. Almost all 14C in faeces has been shown to be
incorporated into bacteria.9 In other words, the
bacterial flora utilises carbon dioxide derived from
uric acid as a result of uricolyis. Carbon dioxide
and ammonia are likewise the major breakdown
products in individuals who have no overt renal
disease.17 Small amounts of 14C-uric acid was
recovered in urinary allantoin (from 0-4 to 4-7%) and
urinary urea (from 0-4 to 2-5%). Allantoic
acid, another intermediary product in the breakdown
of uric acid is inconsistently present in the urine.
This compound is unstable, particularly at an acid
pH, and spontaneously degrades to glyoxylic acid
and urea.

Although increased extrarenal excretion of uric
acid assumes the major role in urate homeostasis
as renal function deteriorates, some patients with
azotaemia show a remarkable adaptation in their
renal handling of uric acid. Renal functional studies
revealed that 2 of our patients (cases 8 and 12) had
urate/inulin clearance ratios of 1:3. The findings
conform with the experience of Steele and Riesel-
bach18 that, with a reduction in glomerular filtration
rate, there is a progressive increase in uric acid
excretion per nephron. Their data show that residual
nephrons of the chronically diseased kidney retain
integrity of urate reabsorptive and secretory trans-
port until inulin clearance has fallen to 15 ml/min.
Further impairment of renal function is associated
with a marked increase in the fractional excretion of
filtered urate. On the other hand not all our
patients had an enhanced excretion of uric acid by
residual nephrons. Patients with chronic lead
nephropathy, for example, had very low urate
clearances in relation to their corresponding glom-
nerular filtration rates. A similar finding has been
reported by Emmerson.14

With the elucidation of the mechanism of the
acute attack of gout it is to be expected that this type
of complication may occur during the course of
renal disease of long duration. At present, when
primary gout rarely leads to gouty nephropathy and
primary renal disease is likely to be diagnosed at an
early stage, the concomitant presentation of gout
and azotaemia should lead the physician to consider that intrinsic renal disease may represent the underlying cause for hyperuricaemia.

References

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