Oxalate deposits in biopsies from native and transplanted kidneys, and impact on graft function

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Abstract
Background. The purpose of this study was to examine the incidence of oxalate deposits in native and renal allograft biopsies, and its impact on graft function.

Methods. The renal biopsy files at The Johns Hopkins University between 2000 and 2006 were searched to identify biopsies with oxalate deposits, determine the density of oxalate deposits in renal graft biopsies, compare graft histology and function between allograft recipients with oxalate in the graft biopsies, and a control group of recipients without oxalate in the graft.

Results. Oxalate crystal deposits were observed in 61 of 5160 biopsies of native kidneys, and in 76 of 1621 renal allograft biopsies, with a frequency of 1 and 4%, respectively. Sixty-three (9%) of 680 transplant recipients showed oxalate in graft biopsies obtained within the first year from transplantation, with 1.3 ± 1.2 average number of oxalate deposits per mm² of biopsy tissue. The high oxalate density and decreased renal function were correlated in the first 2 years post-transplant (P = 0.037–0.05). Compared with a control group of 70 kidney graft recipients, the renal function was significantly lower in the oxalate group at 1 year, but not at 2 years post-transplant. High tubulo-interstitial scarring (P < 0.0001) was noted in repeated biopsies in the oxalate group, and was significantly greater than that in the control group (P = 0.027). No significant difference in graft loss was observed between oxalate and control groups, and although mortality was higher in the oxalate group, the difference was not significant.

Conclusions. In summary, this study defines the frequency of oxalate deposition in native and allograft kidney biopsies, and suggests its possible negative impact on graft function beyond the early post-transplant period.

Keywords: allograft function; kidney transplant; oxalate; rejection

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Oxalate in native and transplanted kidneys 1319
**Introduction**

Oxalate is produced by several metabolic pathways, and its plasma concentration results from a combination of different factors, including metabolic production, dietary load by ingestion of oxalate-containing food, intestinal absorption and renal excretion. The metabolic pathways of oxalate synthesis are not fully characterized, but appear to involve alanine:glyoxylate aminotransferase (AGT) and glyoxylate reductase. These enzymes are deficient in primary hyperoxalurias. Type I hyperoxaluria is due to reduced activity of AGT in the liver, and type II hyperoxaluria is due to deficiency of D-glycerate dehydrogenase or glyoxylate reductase. Glyoxylate reductase reduces glyoxylate to glycolate, and synthesis by glycolate oxidase in liver peroxisomes is considered to be the major source of oxalate. Hydroxyproline, derived from endogenous collagen turnover or from ingestion of meat and dietary gelatin, can be metabolized to glyoxylate, and a gelatin load has been shown to result in increased urinary excretion of oxalate [1,2]. The intestinal handling of oxalate is complex, with intestinal absorption estimated to range between 3 and 55% of an administered load. This involves both carrier (Slc26a)- and non-carrier-mediated transport. About 6% of oxalate is excreted through the intestinal route [3]. In addition, oxalate absorption may be influenced by the activity of intestinal bacteria such as Oxalobacter formigenes, which reduces its intestinal absorption by degrading oxalate [3]. The anion oxalate is eliminated by the kidney, but it is also reabsorbed in the proximal tubule, where it is coupled with Na transport on the cellular apical side, mediated by the anion exchanger Slc26a, and on the basolateral side, mediated by Slc26a1 [4]. Dietary hyperoxaluria may occur when food with high oxalate content is consumed in excess, and enteric hyperoxaluria can be observed with small bowel malabsorption, or injury to colonic mucosa, which non-selectively increases oxalate reabsorption.

High plasma oxalate levels are present in end-stage renal disease and in dialyzed subjects. After successful transplantation, excess plasma oxalate is cleared, which results in transient hyperoxaluria, usually lasting from 3 days to 3 weeks [5,6]. Hyperoxaluria may lead to the formation of oxalate deposits in the allograft tissue, particularly if tubular damage is present in the early post-transplant period [7–10], which may have a negative impact as an injurious element on early graft function and long-term graft outcome.

Although oxalate deposits in kidney biopsies are generally considered to be a relatively infrequent finding, a specific estimate of their occurrence in biopsies from native kidneys has not been defined. Widely different incidences of oxalate deposits in transplanted kidney have been described, ranging from 4 to 52% in biopsies, and up to 87% in transplant nephrectomies [8,9,11,12]. Very few reports deal with the impact of oxalate deposition on the renal function and graft survival of kidney transplant recipients.

The purpose of this study is to define the incidence of oxalate deposition in allograft and native kidney in a large number of patients, and to examine whether oxalate deposition could affect negatively the clinical course and renal graft function of graft recipients beyond the early post-transplant period, as was indicated in two previous studies [8,9].

**Materials and methods**

**Patients**

The records of renal biopsies received from 2000 to 2006 in the Department of Pathology at Johns Hopkins University were searched to identify biopsies where oxalate deposits were reported. A total of 5160 biopsies from native kidneys were recorded. A diagnostic kidney allograft biopsy database was also searched, and included 1621 kidney allograft biopsies from 680 recipients of renal graft from living or deceased donors from the same period. The transplanted kidney database was searched to identify those cases in which deposition of oxalate crystals in the kidney tissue in the first year post-transplantation was described, and the slides of those biopsies were examined to evaluate the abundance of oxalate deposits. Most of the biopsies were performed for clinical evidence of decreased graft function; few protocol biopsies were also available. For comparison, as a control group, 70 recipients of renal graft from living or deceased donors, transplanted from 2000 to 2006, were selected from the same list of patients, following the alphabetical order of their last names.

The demographic and clinical information for subjects with native kidney biopsies and for those with renal graft biopsies were recorded, when available, and included: age at biopsy, age at transplant, gender, race, serum creatinine (mg/dL) at 1 week, 2 weeks, 1 month, 12 months, 2 years, 3 years and 4 years after transplant, if available, and graft failure. The last serum creatinine values at follow-up were recorded for failed grafts if available. The estimated GFR was calculated by the Modified Diet in Renal Disease (MDRD) formula [13–15].

Baseline immunosuppression treatment included prednisone, calcineurin inhibitors cyclosporine or tacrolimus and mycophenolate. The study was approved by the Institutional Review Board at The Johns Hopkins Medical Institutions.

**Biopsies and histological evaluation**

The tissue of kidney biopsies was processed as previously described [16]. About 3 mm of tissue was frozen in OCT matrix (Tissue-Tek, Sakura Finetek, Torrance, CA, USA) for immunofluorescence studies. Most or all of the remaining tissue was fixed in 10% buffered formalin and embedded in paraﬁn sections, which were stained with haematoxylin-eosin (H&E), periodic acid-Schiff (PAS) methenamine silver, and Masson's trichrome for light microscopic examination. Frozen sections were stained by indirect immunofluorescence with an anti-human-C4d mouse monoclonal antibody (Quidel, San Diego, CA, USA) diluted at 1:40, followed by fluorescein-isothiocyanate (FITC) conjugated goat anti-mouse IgG antibody (Jackson Research Laboratories, West Grove, PA, USA).

Biopsies were evaluated using the Banff classification [17–19] for grading acute cellular rejection type I, II and
Oxalate in native and transplanted kidneys

III, as well as antibody-mediated rejection, and other pathological findings were also recorded. The intensity and distribution of staining for C4d in the peritubular capillaries (PTCs) was graded as follows: 0 (negative stain), 1 (1 < 10% focal stain), 2 (10–50% focal stain), 3 (> 50%, diffuse stain). The degree of tubular atrophy and interstitial fibrosis was expressed as a sum of the Ci and Ct grading in the Banff classification scheme.

To evaluate oxalate deposits in renal transplant biopsies, tissue dimensions were measured on the slide, and expressed as mm², and the total number of oxalate deposits (each single deposit may contain more than one oxalate crystal in aggregate) present in the tissue on the slide was counted under polarized light for each biopsy, and recorded. The count of oxalate deposits under polarized light has been used previously in several studies and found to be adequate for measurement [8–10]. Slides were examined independently by two renal pathologists in a blinded fashion. The count of oxalate deposits represents the average from determination by the two pathologists, with very high agreement between the measurements of each observer ($r^2 = 0.9914$).

Statistical analysis

Continuous variables with normal distribution were analysed by unpaired, two-tailed Student’s $t$-test, with $P \leq 0.05$ as an indicative of significant difference. Regression analysis was used for correlation of continuous variables. The chi-square test or the Mann-Whitney test was used for categorical variables. Statistical analyses were performed with STATA statistical software, version 9 (Stata Corp, College Station, TX, USA), and with GraphPad Prism software, version 4 (GraphPad Software, Inc., San Diego, CA, USA).

Results

Biopsy findings and patient’s characteristics

To obtain an estimate of the frequency of oxalate deposits in kidney biopsies, we searched the diagnostic reports on renal biopsies reviewed in the Department of Pathology of Johns Hopkins University from 2000 to 2006, and found that oxalate crystals were described in 61 of 5160 native kidney biopsies, with a frequency of 1%. These patients included 23 females and 38 males, with an average age of 49 ± 19 years at the time of biopsy. Clinical information was provided in 42 of the 61 reports, and revealed that, with the exception of one individual with normal renal function, the others had renal failure, of acute onset in 34 cases. The main diagnostic renal biopsy findings included diverse lesions such as glomerulonephritis (14), acute tubular injury (12), acute interstitial nephritis with eosinophils (10), FSGS or minimal change nephropathy (8), glomerulosclerosis or hypertensive nephrosclerosis (6), diabetic glomerulosclerosis (3), interstitial nephritis (2), lupus nephritis (2), oxalate nephropathy (1), cholesterol emboli (1), thin glomerular basement membrane disease (1) and non-specific changes (1).

Among 1621 kidney allograft biopsies from 680 recipients transplanted between 2000 and 2006, 76 biopsies from 63 recipients collected within the first year from transplantation were found to have oxalate crystal deposition. Thus, according to our observations, the frequency of oxalate deposits in renal allograft biopsies in the 12 months post-transplant can be estimated to be 4.6%, and 9.2% of renal graft recipients show the presence of oxalate deposits in at least a biopsy in the first year post-transplant.

We then analysed in more detail the pattern of oxalate crystal deposition in the transplant biopsies. The average tissue surface examined was 20 ± 11 mm²/biopsy (range 7–64 mm²), with the cortex occupying 75 ± 20% of the kidney tissue. The average oxalate density was 1.3 ± 1.2 deposits/mm² total tissue (range 0.1–4.5 deposits/mm²). Similar to a previous report [8], most oxalate deposits were identified in the cortex, almost always within proximal tubular cells or in the tubular lumen for large precipitates (Figure 1). However, some oxalate deposition also occurred in the medulla. The average total oxalate count in the cortex was 24 ± 41 deposits, compared with 5 ± 8 deposits from the medulla.

Fig. 1. Representative images of oxalate crystals in allograft biopsies are shown. (A) Multiple deposits in a single high-power cortical field, highlighted by polarized light in the bottom panel. (B) Luminal oxalate crystals and isometric vacuolization (arrows) in adjacent proximal tubules.
Table 1. Characteristics of patients with oxalate deposits in renal allograft biopsies

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of patients</td>
<td>63</td>
</tr>
<tr>
<td>Gender (F/M)</td>
<td>22/41</td>
</tr>
<tr>
<td>Race (B/W)</td>
<td>31/32</td>
</tr>
<tr>
<td>Age at transplant, years</td>
<td>49 ± 15</td>
</tr>
<tr>
<td>Donor (living/deceased)</td>
<td>20/42</td>
</tr>
<tr>
<td>ATN in first allograft biopsy</td>
<td>56/63</td>
</tr>
<tr>
<td>Biopsy, days post-transplant</td>
<td>58 ± 66</td>
</tr>
<tr>
<td>Oxalate deposits, N/mm²</td>
<td>1.3 ± 1.2</td>
</tr>
</tbody>
</table>

Age, time interval between biopsy and transplant and oxalate count are shown as mean ± SD.

The asterisk refers to the first biopsy where oxalate deposits were identified for each allograft recipient.

Table 2. Pathologic findings and type of rejection in biopsies of graft recipients with oxalate deposits, and all allograft recipients with biopsies between 2000 and 2006

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Oxalate = 63</th>
<th>All = 680</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of patients</td>
<td></td>
<td></td>
</tr>
<tr>
<td>No rejection</td>
<td>18</td>
<td>196</td>
</tr>
<tr>
<td>Borderline</td>
<td>15</td>
<td>106</td>
</tr>
<tr>
<td>Acute cell-mediated rejection I</td>
<td>8</td>
<td>86</td>
</tr>
<tr>
<td>Acute cell-mediated rejection II</td>
<td>19</td>
<td>205</td>
</tr>
<tr>
<td>Acute cell-mediated rejection III</td>
<td>3</td>
<td>47</td>
</tr>
<tr>
<td>Antibody-mediated rejection</td>
<td>5</td>
<td>53</td>
</tr>
</tbody>
</table>

Banff score is reported as the highest recorded among one or more biopsies through follow-up, according to the Banff classification [18,19].

in the medulla. There was no difference in the average oxalate density among patients with no rejection or borderline findings and those with rejection (1.4 ± 1.1 versus 1.2 ± 1.3 deposits/mm², P = 0.39).

The characteristics of 63 recipients with oxalate deposits are listed in Table 1. Except for two individuals of 12 and 16 years of age all patients were adults. Of these, 88% showed acute tubular injury (ATI) in the first biopsy post-transplant.

Implantation (zero-time) biopsies were performed in 26 of these 63 recipients, and showed no evidence of oxalate deposition in the donor kidney. The average time interval of the first biopsy showing oxalate after transplantation was ~8 weeks. The time between the ‘index’ biopsy showing oxalate and transplant ranged from 5 to 330 days, with 32 patients showing oxalate in a biopsy 1 month or later after transplant, which is longer than the expected time of 3 days–3 weeks for the oxalate load to be cleared after successful kidney transplantation [5,6]. None of the patients had a history of primary hyperoxaluria, none had recent Roux and Y procedure, or severe pancreatic failure [7,20] at the time of biopsy.

Table 2 shows that the proportion of patients with and without rejection was very similar between the graft recipients with oxalate in their biopsies and all the 680 kidney allograft recipients with graft biopsies performed in the same period.

The 70 kidney transplant recipients, included as a control group to compare with recipients showing oxalate in at least one biopsy, did not show significant difference in gender, race, age at transplant and Banff score (reported as the highest recorded among one or more biopsies through follow-up), compared to the oxalate group, although the control group had a higher proportion of living donors (P < 0.05).

Allograft function and graft scarring over time in the oxalate group

The course of allograft function in patients with oxalate in their biopsies is illustrated in Figure 2A up to 48 months post-transplantation.

There was a correlation between higher oxalate count and lower renal function at 1 year (P = 0.05, n = 48), and at 2 years after transplantation, shown in Figure 2B (P = 0.037, n = 23).

There was no difference in the renal function of those patients with oxalate in their biopsy without episodes of rejection and those with episodes of rejection after 1 year (P = 0.4536) and 2 years (P = 0.9517).

Twenty-two allograft recipients in the oxalate group had biopsies in the early post-transplant period (11 ± 13 days post-transplant) and later follow-up biopsies (147 ± 150 days post-transplant), in which the change in

![Fig. 2. (A) Estimated GFR in the patients with oxalate in their graft biopsies is shown over 48-months follow-up. The number of patients for whom the serum creatinine value was known at each time point is indicated in the bottom part of the figure. (B) The relationship between oxalate count and renal function at 2 years after transplant is shown (P = 0.037, n = 23).](image-url)
A

First bx  Last bx

Fig. 3. (A) Difference in tubular atrophy and interstitial fibrosis between the first and last post-transplant biopsies in allograft recipient in the oxalate group with multiple biopsies ($P < 0.0001, n = 22$). The median of the time interval between the first and the last biopsy from transplantation is shown in days. (B) No significant correlation was found between change in tubular atrophy and interstitial fibrosis from the first to the last graft biopsy and the density of oxalate deposits ($P = 0.504, n = 22$). The degree of tubular atrophy and interstitial fibrosis as the sum of the Banff score ($C_i + C_t$) provided in each biopsy was examined. Figure 3A showed that in general a significant increase in tubulo-interstitial scarring was observed between the first and last biopsy ($P < 0.0001$) in these individuals. However, there was no significant correlation between worsening of tubular atrophy and interstitial fibrosis over time and a higher density of oxalate deposits ($P = 0.504$, Figure 3B).

Comparisons between oxalate and control groups
Compared to a control group of 70 transplant recipient without oxalate in any of their graft biopsies, the renal function of the graft recipients in the oxalate group was significantly lower at 1 year after transplantation (Figure 4A), but was not statistically different after 2 years from transplant (Figure 4B).

The degree of tubular atrophy and interstitial fibrosis over time was also compared between transplant recipients with oxalate and control recipients without oxalate. Forty-three of the 70 control patients had more than one biopsy within 1 year post-transplant. The average time at which the first biopsy was performed in this group was $34 \pm 48$ days post-transplant, and $209 \pm 93$ days post-transplant for the last biopsy. The increase in tubulo-interstitial scarring between the first and last biopsy was significantly larger in the oxalate group compared with the controls ($P = 0.027$, Figure 5), during a similar time interval elapsed between the biopsies ($135 \pm 84$ days in the oxalate group and $171 \pm 91$ days in the control group, $P = 0.13$).

Graft loss during follow-up was essentially the same in both oxalate and control group; nine patients lost the graft in each group. However, a higher number of graft recipients with oxalate positive biopsies (14) died within 3 years of transplantation, whereas five died in the control group during follow-up, although the difference did not reach significance ($P = 0.11$). Six of these patients had oxalate in more than one biopsy, and the oxalate density in the
biopsies from these individuals (2.0 ± 1.4 deposits/mm²) was significantly higher ($P = 0.047$) than the average in the whole oxalate group (1.3 ± 1.2 deposits/mm²). Due to limited data for late follow-up of several patients in the oxalate cohort, analysis of the impact of oxalate crystal deposition on graft survival could not be performed.

**Discussion**

Although the presence of oxalate has been considered to be a relatively rare finding in renal biopsies, a specific estimate of its frequency has not been determined. Based on our analysis of a large number of cases, it seems that ~1% of native kidney biopsies show oxalate deposits. The pathologic changes described in these biopsies included diverse lesions, and the majority of patients had renal failure.

It is known that oxalate crystal deposition is detected more often in transplanted kidneys, but very few previous reports that examined oxalate deposits in renal allograft biopsies [8,9], included smaller cohorts of allograft recipients, and the results vary widely. In the current study ~9% of graft recipients show oxalate deposits in the graft tissue in the first year after transplant. We found a 4.6% frequency of oxalate deposits in allograft biopsies, which is similar to a previous report by Truong et al. [8], while Pinheiro et al. describe a much higher frequency (52.8%) of this finding in a study conducted on 97 renal transplant recipients [9].

Our observations were conducted on a group of recipients with oxalate deposits in the allograft biopsy detected in the first year of transplantation, which goes beyond the initial 3-month period selected by Pinheiro et al. [9]. Another difference with the study of Pinheiro et al. is that in the present study the total number of oxalate deposits are counted on the whole tissue surface represented in the slide as oxalate deposits/mm², which seems a more direct measurement than scoring oxalate deposition based on evaluation of 5–20 high power fields used by Pinheiro et al.

The oxalate density in this study ranges from 0.1 to 4.5 deposits/mm². As noted by Truong et al. [8], tubulo-interstitial oxalate precipitation did not appear to incite significant inflammation per second. Our observations show a very similar frequency of rejection in patients with oxalate in their biopsy and those without, and the presence of oxalate deposits does not appear to be associated with increased occurrence of acute rejection. The higher oxalate tissue density, however, seemed to correlate with lower renal function in the 2 years post-transplant. Moreover, although the allograft biopsies examined were not performed at regular interval time for surveillance, as would be ideal for a study like this, we had a group of allograft recipient in both oxalate and control cohorts, who had repeat biopsies within 1 year from transplantation. The analysis of repeat biopsies in the allograft recipients with oxalate showed a significant increase in tubular atrophy and interstitial fibrosis over a relatively short time.

In order to evaluate the possible long-term impact of oxalate deposits on graft condition and function, we compared renal function and tubulo-interstitial scarring in subsequent biopsies within 2 years post-transplant in the oxalate group and a group of allograft recipients who underwent renal transplant and were followed in the same centre during the same period. While the graft function was significantly worse in the oxalate group in the first year, it was not different after 2 years. However, significantly greater tubular atrophy and interstitial fibrosis developed in the oxalate group in subsequent biopsies. Considering that the majority of oxalate patients had some tubular injury in the first post-transplant biopsy, it is plausible that tubular injury may facilitate oxalate deposition, and it is not clear that oxalate, by itself, is a major factor promoting scarring in the transplanted kidney.

For most cases, it is not clear what the exact cause of oxalate precipitation may have been, particularly for post-transplant time intervals longer than 1 month, when the oxalate load should be eliminated by the transplanted kidney. Also, it is not known whether or how the tubular pathways involved in oxalate handling are altered in a single transplanted kidney, compared to the normal situation with two functioning kidneys.

Based upon the clinical history, we can exclude primary hyperoxaluria in this oxalate cohort. The possible role of excessive oxalate intake or possible effects of pharmacological treatment on oxalate metabolism/excretion could not be ruled out in any of our patients. Oxalate nephropathy has been mentioned as a potential side effect of antibiotic treatment in transplant recipients in the report by Lefaucheur et al. [10], and interestingly, 7 out of 14 patients who died within 3 years post-transplant, and had high oxalate density, underwent prolonged antibiotic treatment.

In conclusions, this study provides estimates of the frequency of oxalate deposits in native and allograft kidney biopsies. Our observations suggest a possible negative impact of oxalate deposition on graft function beyond the early post-transplant period, although less severe than that indicated in previous studies [9].

More detailed clinical studies will be needed to identify possible mechanisms involved in abnormal oxalate handling by renal allografts.

**Conflict of interest statement.** None declared.

**References**

Early conservative intervention for candida contamination of preservative fluid without allograft nephrectomy

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Abstract

Background. Fungal contamination of kidney allograft preservative fluid can lead to renal arteritis and arterial wall rupture.

Methods. We have evaluated a conservative management strategy based on early antifungal therapy, rigorous morphological monitoring of the graft artery and surgical second look (SSL). Since November 2004, preservative fluid was routinely cultured on specific media for all kidney transplant recipients.

Results. In 8/474 cases, results were positive for Candida (albicans 5, glabrata 2, tropicalis 1). Two patients also had candida infection of drainage fluid leading to the diagnosis of operative site infection. Radiological and surgical exami-

nations of the renal graft artery were normal in all cases and nephrectomy was not required. At 12 months, all patients were alive with a functioning allograft.

Conclusion. Early antifungal therapy with microbiological and morphological follow-up should be recommended as soon as contamination is detected, but SSL is advised only in patients with risk factors for arterial anomalies.

Keywords: arteritis; candida; management nephrectomy; rupture

Introduction

Fungal contamination of the preservative fluid can lead to renal arteritis and a high risk of arterial wall rupture [1]. Because of this potentially life-threatening risk, some authors have favoured the possibility of removing the graft, while others have followed a more conservative approach [1,2]. While transplant nephrectomy may...