“I would rather be understandable than seem eloquent”—Johannes Joseph Fux in his introduction to musical counterpoint, *Gradus ad Parnassum*, 1725

In 1980, we reported the clinical and pathologic findings in a 49-yr-old man who had an eleven-year history of proteinuria and the nephrotic syndrome (1). We coined the diagnostic term immunotactoid glomerulopathy (ITG) to emphasize the morphology and the composition of the glomerular deposits that appeared as parallel microtubules by electron microscopy (Figure 1 and see below) and that contained IgG and complement (C3) demonstrated by fluorescence microscopy. By analogy to the linear crystallization of hemoglobin S that forms elongated tactoids in red blood cells during sickle cell crisis, we postulated that the physicochemical environment of the glomerulus favored the formation and localization of Ig tactoids in this condition (1–3). In contrast to AL amyloid, the deposits were Congo red and thioflavin T–negative. Similarly appearing glomerular deposits were known to occur in patients with systemic diseases and hematologic malignancies particularly associated with circulating cryoglobulins (4–6). We, however, believed that our patient was unique and represented a new form of primary glomerular disease. He was repeatedly evaluated over a 15-yr course of nephrotic syndrome and progressive renal insufficiency that ended in end-stage renal disease (ESRD) and never developed any of the systemic diseases that previously had been associated with organized Ig deposits. There had been reports of patients with similar clinical presentations, Congo red negativity, immunopathology, and absence of systemic disease, but when examined by electron microscopy, the glomerular deposits were organized into randomly arranged fibrils without an apparent lumen (Figure 2 and see below) (7–9).

We reported our experience with (2), and reviewed the literature on, both the fibrillar and microtubular forms of the lesion (3), excluding patients with known systemic diseases that are associated with organized glomerular immune deposits from the analysis. Because there was no consistent difference in the clinical presentation, the immunopathology, the prognosis, or the response to therapy, we decided that biopsies showing either the ultrastructural form of the organized deposits could be included under the single diagnostic rubric of ITG. By defining ITG in this fashion, we expected that clinical, pathologic, prognostic, and therapeutic information would accumulate, and when the etiology and the pathogenesis were determined, ITG would take its place as a primary renal disease. However, it must be acknowledged that this definition is not universally accepted, and controversies have arisen concerning the definition and classification of ITG.

The first alternative point of view concerns diagnostic criteria. An alternate viewpoint broadens the definition of ITG to include all diseases with non–amyloid organized immune deposits within the glomerulus. A second alternate viewpoint holds that diseases with non–amyloid organized glomerular-immune deposits should be stratified on the basis of ultrastructural differences into two categories: (1) microtubular deposits and (2) microfibrillar deposits. Before reviewing the current status of ITG, we will discuss these two viewpoints separately.

**Exclusion Criteria for the Diagnosis of ITG**

The question may be simply stated: Should the diagnosis of ITG be broadened to include all diseases that have non-amyloid organized glomerular immune deposits, or should ITG indicate only a primary glomerular disease? In constructing an answer to this question, one must consider how the definition of ITG confers relevance on the lesion in either a clinical or pathophysiological sense. It is almost certain that by including patients with systemic diseases and lymphoproliferative disorders the clinical characteristics of patients with ITG, which was described to call attention to an idiopathic primary process, would lose specific clinical relevance and become a function of a known underlying disease state. Therefore, this is both a nosological problem and a clinical concern.

Many examples of renal pathology are associated with both primary and secondary disease processes, but because the prognostic and therapeutic implications may be quite different, it is critical to separate the idiopathic forms of the disease from the secondary causes of a similar glomerular pathology. For example, membranous glomerulonephritis caused by penicillamine therapy is a drug-induced lesion, and focal segmental glomerulosclerosis secondary to HIV infection has a viral etiology. These glomerular lesions have different prognoses and therapeutic implications from the respective idiopathic forms of the glomerular diseases, which may appear histologically identical. Hence, it is important to cite whether a given histologic pattern is primary or related to a known secondary cause. Similarly, it is of no apparent clinical...
value that patients with plasma cell dyscrasias, lymphoproliferative diseases, monoclonal gammopathies, and other cryoglobulinemic states with well-defined characteristics and specific therapies should be included under the rubric of ITG just because the glomerular immune deposits happen to have discrete ultrastructural characteristics (10–13). By the same criteria of clinical utility, glomerular diseases with non-Ig organized deposits related to injury (14), as occur in some cases of diabetes mellitus (15), and inherited disorders of fibronectin biosynthesis (16) should be classified along with other examples of the fibrillary glomerulopathies (16,17), but they should not be confused with ITG.

It is our opinion that if it is to be useful to the clinician as an organizing concept, ITG should be diagnosed only after diseases known to be associated with organized glomerular immunoglobulin deposits have been excluded. ITG, defined in this fashion, can then be used to indicate a primary glomerular disease, and data concerning the etiology, pathogenesis, clinical features, and response to therapy can accumulate and allow both the pathologist and the clinician to gain insight to this fascinating entity (18).

One Entity or Two?

It has been suggested that glomerular diseases with non-amyloid, ultrastructurally organized immune deposits be separated into two categories on the basis of fibril size and organization. In this scheme the diagnosis of ITG is reserved for cases with larger, parallel microtubules, and cases with smaller, randomly arranged fibrils are called fibrillary glomerulonephropathy/glomerulonephritis (FGN). The rationale for this morphologic stratification is that it provides a neat and reproducible division and that the different morphologic categories have clinical implications (11,19–21). Unfortunately, this terminology is infelicitous and confusing; we have used the diagnosis of ITG to describe patients with both types of deposits, and the term FGN (Figure 3) more properly denotes the general category of diseases that are characterized morphologically by fibrils seen by electron microscopy without regard to their biochemical composition (14,17,22). We will, however, use this terminology to consider the validity and the utility of separating biopsies containing glomerular non-amyloid, organized immune deposits into two categories on the sole basis of the appearance of the deposits.

The discriminating morphologic features of the microtu-

Figure 1. Electron micrograph of glomerular microtubular deposits showing a parallel, packed arrangement and measuring 35 nM in diameter. Note the thick walls and prominent lumens. These deposits colocalized with IgG3κ and the third component of complement (C3) demonstrated by fluorescence microscopy. Uranyl acetate and lead citrate. Magnification, ×32,000

Figure 2. Electron micrograph of glomerular fibrillar deposits showing a random arrangement and measuring 20 nM in diameter. The fibrils appear as elongated filaments, and in cross-section they appear as a solid point. These deposits colocalized with IgG, κ, λ, and the third component of complement (C3) demonstrated by fluorescence microscopy. Uranyl acetate and lead citrate. Magnification, ×32,000.

---

value that patients with plasma cell dyscrasias, lymphoproliferative diseases, monoclonal gammopathies, and other cryoglobulinemic states with well-defined characteristics and specific therapies should be included under the rubric of ITG just because the glomerular immune deposits happen to have discrete ultrastructural characteristics (10–13). By the same criteria of clinical utility, glomerular diseases with non-Ig organized deposits related to injury (14), as occur in some cases of diabetes mellitus (15), and inherited disorders of fibronectin biosynthesis (16) should be classified along with other examples of the fibrillary glomerulopathies (16,17), but they should not be confused with ITG.

It is our opinion that if it is to be useful to the clinician as an organizing concept, ITG should be diagnosed only after dis-

Figure 3. Algorithm for the evaluation of a patient presenting with a fibrillary glomerulopathy (17).
bules in ITG and the fibrils in FGN that have been reported include cross-sectional diameter, presence of a lumen, and random versus parallel arrangement. However, the fibrils in what has been called FGN and ITG range with considerable overlap from 10 to 49 nM (Figure 4) (3). Generally, the fibrils in reports of FGN have an average diameter between 18 to 22 nM, approximately twice the diameter of amyloid fibrils, and virtually all cases measure less than 30 nM. When Pronovost et al. (23) defined ITG by fibril size larger than 30 nM, it comprised only 6.5% of the 186 cases they reviewed. In contrast, when they defined ITG by focal parallel arrangement of the fibrils, it almost doubled its prevalence (12%). Some have argued that the morphologic definition of ITG also includes a “hollow,” electron-lucent lumen in the fibrils, but morphologic studies have suggested that the fibrils in many if not all cases of what has been called fibrillary GN (FGN) also have an electron-lucent lumen (2,17). Using arbitrary definitions of the fibril morphology, one can separate some cases (FGN) from a much smaller number of cases (ITG), but there is considerable overlap between the two diagnostic categories, and the distinction becomes artificial. In our opinion, there is no compelling reason to separately diagnose ITG and FGN on the basis of morphology alone unless it can be demonstrated that the ultrastructural features have significant pathogenetic or clinical implications.

The mechanism of fibrillogenesis in Ig deposits in general and particularly in ITG and FGN is unknown. In cell biology, microtubules are cytoplasmic organelles composed of a protein tubulin, and they are contrasted morphologically with intermediate filaments composed of different proteins, cytokeratin, vimentin, and neurofilament protein, characteristic of various mature tissues. Microtubules and intermediate filaments are morphologically discrete organelles; therefore, it may be that organization into a specific organelle is an intrinsic property of the component proteins. By analogy, the dichotomous appearance of the deposits in ITG and FGN may signify different biochemical components. All 13 patients studied for IgG subgroups by Iskandar et al. (21) had a predominance of IgG4, but because membranous glomerulonephritis has the same subgroup predominance in its immune deposits and does not show ultrastructural organization of the deposits, the role of IgG4 in fibrillogenesis, despite its unique immunologic features, remains speculative. Although most cases of FGN contain both κ and λ light chains in the immune deposits, approximately 25% show κ light chain restriction (21,24) and appear to be monoclonal proteins. There is a single case of ITG that showed deposits of monoclonal IgGκ (1). Although the composition of the deposits deserves further study, the present data do not support biochemical specificity for the definition of either the microtubules of ITG or the fibrils of FGN.

The dichotomy of ITG and FGN could conceivably be justified if the morphologic distinction provided useful clinical information. In a review of 186 cases published with the diagnosis of ITG or FGN, Pronovost et al. (25) considered the relationship between the morphologic distinction between ITG and FGN and the demographics and clinical features of the patients. There was a slight female predominance in patients with ITG, but there were no age differences. They found no difference in the prevalence of hypertension, hematuria, nephrotic syndrome, and renal insufficiency at presentation whether the diagnosis was established by fibril size (FGN, ≤30 nM; ITG, >30 nM) or arrangement (FGN, random; ITG, focally parallel). They also evaluated the association between FGN and ITG and lymphoproliferative disease. As expected, patients with a serum or urine paraprotein and a lymphoproliferative disorder frequently (44%) had organized glomerular deposits of a tubular nature, as is seen in ITG. However, when patients with a paraprotein were excluded, the prevalence of neoplasia in both ITG and FGN was similarly low.

We conclude from these considerations that the morphologic differences, which some believe to support a distinction between ITG and FGN, are of unknown significance, and there is too much overlap in fibril morphology to dichotomize the biopsies in a nonarbitrary manner. There is insufficient data to suggest that either the IgG subgroup composition or the monoclonality of the deposits is critical for the pathogenesis of ITG or FGN. There are no consistent clinical differences related to fibril morphology or organization at presentation, and when secondary causes of ITG are excluded, the prevalence of hematologic malignancy is low and similar. Therefore, we consider ITG as a primary glomerular disease characterized by deposits of Ig and complement that have variable appearances when examined by electron microscopy, and the diagnosis is established only after diseases that have known associations with organized glomerular immune deposits have been excluded. In the remainder of this review, we will refer to the entity as ITG regardless of the ultrastructural appearance of the deposits.

**Clinical Manifestations and Laboratory Findings**

ITG is an uncommon condition seen in fewer than 5% of renal biopsies from nephrotic adults with primary glomerular diseases (26). The clinical presentation of ITG does not dis-
tistinguish it from the other primary causes of the nephrotic syndrome (3), and this point was confirmed in a recent review of 186 patients with ITG (23) (Table 1). The age of patients with ITG ranges from 10 to 80 yr, but the average patient is 44-yr-old at presentation (3,17,21–23). Ninety percent of the patients are white, and the distribution between men and women is approximately equal (23). All the patients have proteinuria, and over 60% have the nephrotic syndrome. Microscopic hematuria and hypertension are present in more than 60% and 70% of patients, respectively, and more than 50% of patients have some degree of renal insufficiency at the time of diagnosis, indicating the chronic and progressive nature of ITG (2,21,23). Patients with ITG only rarely have systemic symptoms unrelated to renal involvement (see below—extrarenal pathology), and serologic studies are negative. By definition, they do not have a cryoglobulinemia, a paraproteinemia, a lymphoproliferative disorder, or a plasma cell dyscrasia. Even though up to 19% of ITG patients have a positive antinuclear antibody, it is usually in low titer and/or in a speckled pattern (2,21,21). Those patients fulfilling the clinical criteria for systemic lupus erythematosus should be excluded from the diagnosis of ITG.

Pathology

The pathology of ITG has been recently reviewed in depth, and we will present only the highlights (16,23). The glomeruli show various patterns that largely reflect the distribution of the deposits. Mesangial expansion by eosinophilic, periodic acid-Schiff–positive deposits and mild mesangial hypercellularity is the most frequent finding. Variable glomerular basement membrane (GBM) thickening is also frequent, and although the GBM pathology may be subtle, irregular thickening, subepithelial projections, and silver-negative defects may be seen with special stains. Occasionally, there are large subendothelial and subepithelial GBM deposits and thrombi. However, it must be emphasized that the glomeruli and all the deposits seen by fluorescence microscopy, implying that they contain immunoglobulins and complement as principal components. Other ultrastructural findings are nonspecific. Deposits are described throughout the glomerulus, but only isolated mesangial involvement is seen in approximately 25% of the cases. Deposits associated with the basal lamina have a predilection for the lamina densa and the lamina rara externa, appear to diffusely infiltrate and replace the basal lamina in some instances, and may form prominent subepithelial and subendothelial deposits.

Significant glomerular inflammatory lesions have been reported in ITG with diffuse proliferation and crescents in 40% and 19% of biopsies, respectively (22). In several reports, ITG was associated with a rapidly progressive course and crescents and/or necrosis in more than 50% of the glomeruli (16). In our own experience, we have not seen crescents, endocapillary proliferation, or necrosis when cryoglobulinemia, paraproteinemias, and systemic disease were excluded. With advancing disease, epithelial proliferation may accompany segmental scarring, and this, in addition to ischemic crescents in obsolescent glomeruli, should not be confused with active glomerular inflammation.

All cases of ITG have glomerular Ig and complement deposits in the mesangium alone or in combination with capillary wall deposits. The capillary wall deposits are either diffuse and coarsely granular or discontinuous and pseudo-linear. Tubular basement membrane and interstitial and vascular deposits determined by fluorescence microscopy have not been observed. The Ig class is most frequently IgG, and the deposits usually contain both κ and λ light chains (Table 2). Monoclonal Ig deposits were seen in approximately 20% of the 65 cases of ITG studied with light chain antisera, and κ light chain restriction was present in all cases, usually in combination with γ heavy chain (16). As previously noted, a study of IgG subgroups restricted to cases with smaller, randomly arranged fibrils found IgG4 as the dominant subclass with weak staining for IgG1 and absent IgG2 and IgG3. Monoclonal IgGκ was reported in one case with 35-nm microtubular deposits (1).

ITG is characterized by the ultrastructural appearance of the glomerular deposits that consist of extracellular, elongated, nonbranching microfibrils/microtubules, which show neither periodicity nor substructure. They are seen at the same sites as immune deposits seen by fluorescence microscopy, implying that they contain immunoglobulins and complement as principal components. Other ultrastructural findings are nonspecific. Deposits are described throughout the glomerulus, but only isolated mesangial involvement is seen in approximately 25% of the cases. Deposits associated with the basal lamina have a predilection for the lamina densa and the lamina rara externa, appear to diffusely infiltrate and replace the basal lamina in some instances, and may form prominent subepithelial and subendothelial deposits.

Table 1. Presenting clinical features

<table>
<thead>
<tr>
<th>Patients (n)</th>
<th>186</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (range in yr)</td>
<td>10 to 81</td>
</tr>
<tr>
<td>Proteinuria</td>
<td>100% (186/186)a</td>
</tr>
<tr>
<td>Nephrotic</td>
<td>72% (105/146)</td>
</tr>
<tr>
<td>Hematuria</td>
<td>71% (110/166)</td>
</tr>
<tr>
<td>Hypertension</td>
<td>70% (92/131)</td>
</tr>
<tr>
<td>Renal insufficiency</td>
<td>53% (83/158)</td>
</tr>
</tbody>
</table>

a Percent with feature (proportion with feature). Data from reference (23).
Pathogenesis

The pathogenetic mechanism of ITG must account for three features: the Ig found in the deposits must be produced by lymphocytes and/or plasma cells; the Ig precursors must reach the kidney via the circulation; and the exclusive glomerular localization of the deposits implies a role for local factors in fibrillogenesis. The first two features suggest that the mechanism of formation of these structures is somehow similar to that of cryoglobulinemia or monoclonal gammopathy. Cryoglobulins and paraproteins constitute exclusions from the diagnosis of ITG; therefore, the Ig must circulate in quantities that escape routine testing. In view of the fact that the morphologic structures seen in many cases of cryoglobulinemia are similar, it is conceivable that production of immunoglobulins or immune complexes that are not cold precipitable but are otherwise capable of forming tactoids in the glomerulus explains this phenomenon. The glomerular deposits are polyclonal in most cases, and this makes it unlikely that the immunoglobulins are produced by neoplastic plasma cells or lymphocytes. Rastagno et al. (30) reported a unique case in which biochemically identical fibrils were seen in the glomeruli and in a serum precipitate that formed after 4 mo at 4°C. In this case, the presence of fibronectin in fibrils seen in the glomeruli and in the serum precipitate led the authors to suggest that fibronectin plays a role in fibrillogenesis. Although that remains a possibility in their unique case (30), a previous study from the same group found no fibronectin in the glomerular deposits in a series of patients with ITG (27). In fact, the extreme conditions required to form the serum precipitate (4 mo at 5°C) and its insolubility upon rewarming suggest that it is not a classic cryoglobulin. Furthermore, the appearance of the fibrils were quite different in the precipitate and in the glomerulus with the serum fibrils showing a large size (diameter, 90 nM) and periodicity in contrast to the glomerular fibrils that were 15 to 20 nm, typical immunotactoids. The role of the physical conditions and the specific glomerular environment exemplified by this last point supports our original conjecture that the filtration process creates an environment that favors fibrillogenesis and precipitation (1–3). The fact that fibrils composed of the same serum protein components have different morphology under different physical circumstances shows that, at least in this instance, morphologic differences in fibril appearance do not imply biochemical divergence. Although ITG has a clinical course similar to primary glomerular diseases, such as membranous glomerulonephritis, pathogenetic speculation has favored a search for systemic factors such as a normal or pathologic Ig, the properties of which favor glomerular precipitation and tactoid formation. However, the absence of both a demonstrable paraprotein and a population of neoplastic plasma cells/lymphocytes suggest that some unidentified defect in glomerular function plays a central role in the localization and formation of immunotactoids.

Recent studies in CD2-associated protein (CD2ap) knockout mice provide support for a defect in glomerular function in ITG that is responsible for tactoid formation and suggest that the defect is localized to the podocyte (31,32). CD2ap is an 80-kD protein found in the specialized junction that forms between mouse T lymphocytes and antigen-presenting cells (31,33), and it is widely distributed in mature and developing kidney (31). Within the glomerulus, the molecule binds to the cytoplasmic domain of nephrin, a component of the podocyte slit diaphragm (31). CD2ap-deficient mice have congenital nephrotic syndrome and compromised immune function, and they die of renal failure 6 to 7 wk after birth (34). The glomeruli show podocyte pathology and mesangial hypercellularity (34). In addition, they have mesangial and subendothelial deposits that comprise parallel arrays of microtubules seen by electron microscopy (35). Thus, congenital absence of CD2ap leads to glomerular ultrastructural pathology similar to that seen in ITG in humans. The podocyte is known to lose mature differentiation antigens and functional proteins in acquired disease states such as FSGS and HIVAN (36); therefore, one may postulate that glomerular deposits in ITG are secondary to acquired defects in critical podocyte cellular functions involved in the clearance of filtered and retained Ig. However, the rigid, crystalline nature of the deposits suggest that physicochemical homogeneity of the component molecules remains an important distinguishing pathogenetic feature between ITG and other primary Ig-mediated glomerular diseases in which the deposits are not organized.

Therapy

ITG is an Ig-mediated lesion, and the apparent role of Ig in the pathogenesis of this lesion has led to a variety of immunotherapies with the expectation of a favorable response. The reported experience is limited and anecdotal, but therapeutic trials with steroids alone, steroids with cytotoxic agents, and steroids with plasmapheresis have been associated with clinical remission of proteinuria in fewer than 10% of the cases (Table 3).

Patients with ITG frequently present with renal insuffi-
iciency, and they have a progressive course to ESRD that is not affected by current therapy. Because the patients generally do not have systemic involvement related to ITG, their prognosis, independent of renal survival, is quite good. Therefore, renal transplantation has been performed in ITG patients with ESRD, and renal transplantation has been reported in eight patients who were followed from 2 to 11 yr after transplantation (Table 4) (37). ITG recurred in four of these grafts. In one patient, recurrence occurred 21 mo after transplantation and led to loss of the graft 3 yr later (38). In the remaining three patients with recurrent disease, renal function continued to be adequate after 5 to 11 yr of follow-up (23,24,38). In patients with recurrent disease, the ultrastructural morphology in the transplants is similar to that seen in the native kidneys (24,38). Thus, renal transplantation is a viable option for patients with ITG.

Outcome/Prognosis

The overall course is usually slowly progressive (Table 5). The survival at 1 yr is 100%, with over 80% of patients alive at 5 yr (3). The course in patients with ITG is characterized by renal insufficiency progressing to ESRD over 2 to 4 yr in 50% of patients (2,21,23). This course is similar to other primary glomerulopathies, but it is distinct from patients with amyloidosis who experience a more rapid decline to ESRD (17). Features at presentation that appear to be associated with a poor renal prognosis are hypertension, nephrotic proteinuria, and the presence of renal insufficiency (2,3,23). Even though detailed pathologic analyses have been reported, there is little information on the prognostic significance of the various morphologic aspects of ITG. In our experience, those patients with more extensive glomerular involvement are associated with a poorer prognosis (2,3).

Summary

The clinical and pathologic expressions of ITG are almost always solely confined to the kidney, and at the present time, the patient with ITG is best served by considering ITG as a primary glomerular disease. Its presentation is similar to the other primary diseases causing the nephrotic syndrome, and although it is unresponsive to current therapy and frequently progresses, renal transplantation is a viable option for the patient with ESRD. ITG is one of the many conditions that are appropriately categorized as fibrillary glomerulopathies, but the diagnosis should only be applied after systemic diseases that may be associated with organized glomerular immune deposits have been excluded. Restricting the diagnosis of ITG to the primary form of the disease focuses research efforts by avoiding confusion with well-defined diseases that may have organized glomerular immune deposits as part of the pathologic process. Most importantly, it will allow us to develop lesion-specific information for this group of patients in the future (39–42).

---

**Table 3. Therapeutic response of ITG**

<table>
<thead>
<tr>
<th>Treatment</th>
<th>n</th>
<th>Partial Remission</th>
<th>Complete Remission</th>
<th>No Response</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prednisone</td>
<td>4</td>
<td>1</td>
<td>0</td>
<td>3</td>
</tr>
<tr>
<td>Cytotoxics</td>
<td>2</td>
<td>0</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>Prednisone and cytotoxics</td>
<td>22</td>
<td>2</td>
<td>0</td>
<td>20</td>
</tr>
<tr>
<td>Plasmapheresis</td>
<td>3</td>
<td>0</td>
<td>0</td>
<td>3</td>
</tr>
<tr>
<td>Nonsteroidals</td>
<td>2</td>
<td>0</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>Total</td>
<td>33</td>
<td>9%</td>
<td>0</td>
<td>91%</td>
</tr>
</tbody>
</table>

*Data from references (3, 17, 20, 24, 29, and 39 to 42).

---

**Table 4. Posttransplant course of ITG**

<table>
<thead>
<tr>
<th>Study</th>
<th>Patients (n)</th>
<th>Recurrence</th>
<th>Follow-up (yr)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alpers <em>et al.</em> ((24))</td>
<td>1</td>
<td>1</td>
<td>5</td>
</tr>
<tr>
<td>Sturgill <em>et al.</em> ((37))</td>
<td>1</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>Korbet <em>et al.</em> ((38))</td>
<td>2</td>
<td>1 (ESRD)</td>
<td>5.5</td>
</tr>
<tr>
<td>Pronovost <em>et al.</em> ((23))</td>
<td>4</td>
<td>2</td>
<td>4–11</td>
</tr>
<tr>
<td>Total</td>
<td>8</td>
<td>4</td>
<td>2–11</td>
</tr>
</tbody>
</table>

---

**Table 5. Renal and patient survival with ITG**

<table>
<thead>
<tr>
<th>Years</th>
<th>Renal Survival</th>
<th>Patient Survival</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>100%</td>
<td>100%</td>
</tr>
<tr>
<td>1</td>
<td>96%</td>
<td>95%</td>
</tr>
<tr>
<td>2</td>
<td>81%</td>
<td>92%</td>
</tr>
<tr>
<td>5</td>
<td>56%</td>
<td>83%</td>
</tr>
<tr>
<td>10</td>
<td>24%</td>
<td>83%</td>
</tr>
</tbody>
</table>

*Data from references in Korbet *et al.* (3) and Korbet *et al.* (17).
References


31. Li C, Ruotsalainen V, Tryggvason K, Shaw AS, Miner JH: CD2AP is expressed with nphrin in developing podocytes and is found widely in mature kidney and elsewhere [In Process Citation], Am J Physiol Renal Physiol 279: F785–F792, 2000


