SUPPLEMENTARY APPENDIX:

KIDNEY TRANSPLANTATION IN PATIENTS WITH FIBRILLARY GLOMERULONEPHRITIS ASSOCIATED WITH A MONOCLONAL IMMUNOGLOBULIN.

Fibrillary glomerulonephritis (FGN) is characterized by the presence of fibrillary deposits in the glomerulus, which are typically Congo-red negative. Approximately 50% of patients with FGN develop renal failure within 2–4 years after presentation. FGN typically is characterized by deposition of polyclonal IgG, but, in few cases, the fibrillary deposits consisted of a monoclonal immunoglobulin. The percentage of patients with FGN and light-chain restriction in 6 studies ranged from 3-16%.[1-6] Histology (light chain staining) and serology (serum FLC) are often discordant: evaluation of serum and/or urine in patients with a ''polyclonal'' FGN often leads to the detection of a monoclonal component. For example, Rosenstock et al observed a monoclonal immunoglobulin in serum and/or urine in 7/46 patients (15%) whereas light chain restriction in IF of the kidney biopsy was noted in only 2 patients.[1] On the other hand, in many patients with a monoclonal FGN (by IF) no hematological abnormalities in serum or urine were observed.[3]

In 2018, glomerular expression of DNAJB9 was introduced as biomarker of FGN.[7-9] Although positive staining identified FGN with high sensitivity and specificity, the initial studies showed that biopsies of patients with monotypic FGN also stained for DNAJB9 with the exception of 2 patients with FGN due to heavy chain only deposits.[4] Combining IgG subclass staining, immunohistochemistry using pronase-digested tissue, and staining for DNAJB9 contributed to better defining suspected monotypic FGN.[10] This study included 29 biopsies with FGN and apparent light chain restriction. Of these 21 biopsies were positive for DNAJB9, and 8 were negative. IgG subclass staining showed that 6 of the latter 8 cases were indeed monotypic, and in 5 of these patients a hematological malignancy was found (CLL in 4). IgG subclass staining revealed that only 7/21 DNAJB9-positive biopsies were monotypic. A monoclonal protein was detected in only one patient, and no hematological malignancies were found.[10]

Older literature data on recurrent disease in patients with FGN reported widely varying recurrence rates, ranging from 8% (1/13) to 60% (3/5). Most recurrences occurred relatively late (>5 years) and did not affect 5 and 10 year survival.[11,12] It is likely that these data are applicable to patients with a polyclonal FGN. The notion that monotypic FGN may be associated with a dismal outcome is mainly based on a cohort study of Czarnecki et al.[13] These authors described the outcome of kidney transplantation in five patients with "polyclonal" fibrillary GN and in 7 patients with monoclonal immunoglobulin-associated fibrillary glomerulonephritis (eg, patients with FGN and a monoclonal gammopathy, termed MGFGN). In this study, no recurrence occurred in the patients with polyclonal FGN, whereas recurrent disease was observed in 5/7 patients with MGFGN.[13] Although this manuscript is often cited, the study had many caveats. First, many patients with MGFGN had an underlying hematological malignancy. Second, the kidney biopsies of the native kidney demonstrated light chain restriction in only 1 patient. Also, the kidney biopsy of only 1 patient with recurrent disease showed evidence of light chain restriction. Thus these patients do not fulfil the definition of monoclonal FGN.[13] In a recent report, it was acknowledged that the patients included in this MGFGN cohort should not have been defined as FGN, rather these patients should be considered ''FGN mimics.''[14] Of interest, it was acknowledged that most patients with MGFGN did not show the typical IgG staining, in fact most patients had TMA with fluffy subendothelial deposits with vaguely fibrillar substructure.[14] El Ters et al reported outcome of kidney transplantation in 14 patients with FGN, all polyclonal (totaling 17 transplants).[14] In 3 of these patients a monoclonal protein was present in the serum. Recurrent disease was observed in only 3 patients after 5, 10 and 10 years, respectively. The patients with a known monoclonal Ig did not develop recurrence.[14] There is 1 case report of recurrent disease in a patient with heavy chain-only fibrillary glomerulonephritis. In this patient, DNAJB9 staining was negative, recurrences occurred after 2 transplantations with graft loss occurring after 2 and 3 years, respectively.[14]

Pretransplant evaluation of patients with "monotypic" FGN requires thorough evaluation of the native kidney diagnosis. Both DNAJB9 staining and IgG subclass staining of the native kidney biopsy should be performed. If DNAJB9 staining is negative, a detailed search for a hematological malignancy is warranted. In patients with DNAJB9 positive, monotypic FGN confirmed by IgG subclass staining, we suggest M-protein evaluation. Still, a kidney transplantation can be performed in patients with monotypic FGN and no documented hematological malignancy without pretransplant therapy.

KIDNEY TRANSPLANTATION IN CRYOGLOBULINEMIC VASCULITIS

There are very few case reports of kidney transplantation in patients with cryoglobulinemic vasculitis and most concerned patients with HCV-associated cryoglobulinemia. In the majority of patients with cryoglobulinemia type I an underlying hematological disease is present, and most patients respond to therapy. Therefore, few patients with kidney disease due to cryoglobulinemia type I develop kidney failure and information on kidney transplantation in patients with cryoglobulinemia type I is therefore not available. In the rare patient with cryoglobulinemia type I and kidney failure, we suggest that kidney transplantation is acceptable if the patient is treated and cryoglobulins are absent.

Kidney outcome in patients with kidney disease due to cryoglobulinemia type II (so called mixed cryoglobulinemia) is also favorable, with renal failure developing in only 9% of patients.[15] There are only few case reports and small case series that describe the outcome of kidney transplantation in patients with mixed cryoglobulinemia. Unfortunately, some studies have included both patients with type II and type III cryoglobulinemia, and in many (older) studies hepatitis C was not excluded.[16] This is relevant since in most patients cryoglobulinemia type II is associated with a hepatitis C virus infection: HCV positivity was reported in 5/7 patients reported by Basse et al.[17] and in 9/12 tested patients with cryoglobulinemia associated renal failure reported by Tarantino et al.[18]

Early recurrence of cryoglobulinemic kidney disease was reported by Hiesse et al,[17] occurring 30 days after kidney transplantation. Unfortunately, hepatitis C virus was not excluded, and it is likely that

viral infection was the cause of the cryoglobulinemia. In another case report, cryoglobulinemic kidney disease was observed 35 days after kidney transplantation, in a patient diagnosed with primary active CMV infection.[19] Few case reports have included patients with non-HCV associated, mixed cryoglobulinemia undergoing kidney transplantation. Unfortunately, these case reports have limited detail, eg, information on the presence and concentration of free FLC and/or information on hematological assessment is often lacking. Takeda et al reported 1 patient with HCV negative, mixed cryoglobulinemia, treated with cyclophosphamide and steroids, who developed renal failure. Kidney transplantation was uneventful, until 4 years after transplantation, when recurrent disease occurred.[20] Treatment with steroids had limited effect, still graft function remained stable >6 years. In another study, no recurrence occurred in a patient who was treated with plasmapheresis, and chemotherapy to maintain cryoglobulins levels low.[21] Basse et al reported 7 patients with recurrence cryoglobulinemic kidney disease, 2 patients were HCV negative.[17] Details are not provided, however, recurrences occurred after 7–180 months, and in patients treated with rituximab kidney function improved and stabilized. We suggest that kidney transplantation can be considered in patients with a history of mixed cryoglobulinemia if there is no evidence of active clinical disease, if cryoglobulins are undetectable or level is low, complement C4 level is normal, and there is no evidence of a hematological malignancy.

Table S1: kidney transplantation in patients with AL-amyloidosis: patient characteristics, and outcome[22-28]

Author (time period)	n	Age (yr)	Gender (m/f)	Interval from diagnosis to RRT and/or Tx (months)	Hematological therapy before Tx			Hematological response					Follow up (yr)	Survival (yr) Patient/graft	Amyloid recurrence in kidney (n), time to recurrence(y) graft loss due to recurrence
					CT only	ASCT[e]	None	CR	VGPR	PR	NR[h]	No Rx			
Angel-Korman (1987-2017)	49	60[a] (31-73)	40/9	D – Tx: 48 (5.7 - 144)	7	33	4[i]	21	9	7	8	4	7.2	10.5/6.9[c]	14 3.7 (1.1-11.9) 4 graft losses
Cohen (2004-2019)	40	53[b] (38-69)	24/16	D—RRT: 15 (0-115) RRT-Tx: 28 (3-83)	30	10	0	24	6	6	3		8.9	9/12.4[d]	3 Unknown 0 graft losses
Heybeli (1997-2018)	60	60[a] (35-74)	40/20	D – Tx: 37 (2-352)	15	36	9	37	6	5	3	9	5.1	10.2/96%[e]	13 10.1 y[f] 0 graft losses

Pinney (1987-2011)	25	60[b] (52-63)	9/16	D-RRT: 13 (0-43) RRT-Tx: 27 (13-60)	22	?	3	5	13[f]		4	3	4.6	/5.8	7 5.9 (3.8-6.3) 0 graft lossed
Sathick (1999-2018)	16	53[a] (36-63)	7/9	RRT-Tx 21 (3-58)	0	16[e]	0	14	2		0^^	0	3.3	../ 11.3 yr[d]	3 Unknown 1 graft loss
Havasi[j] (1987-2020)	237	61[a] (31-79)	156/81	D-RRT: 46 (-5.7y – 12.8y) RRT-Tx 24 (0-140)	103	117 (as first line in 75)	17	145	40	24	11	17	8.5	8.6/7.8 (death censored graft survival)	N =69 (29%) hematologic relapse N = 50 in kidney 6.6 y 6 grafts lossed

CT chemotherapy; CR complete response; D diagnosis; NR non-response; PR partial response; RRT renal replacement therapy; Tx kidney transplantation; VGPR very good partial response; CT = chemotherapy, ADCT = autologous stem cell transplant; Rx= treatment

* [a]age at transplantation ** [b]age at diagnosis - [+c]includes death, [++d]death-censored, [+++e]death-censored 5y graft survival - ^[f]includes detection of amyloid deposits in protocol biopsies; # [e]some patients received CT before ASCT. ## [f]8 patients had VGPR, 2PR, 3 undefined. † [g]patient counts see remarks, †† [h]12 NR , but 4 NR were untreated. ^^ [i]two patients initially responded but had progressive disease at time of transplantation [## j]important overlap between this study and previously reported studies.

A recent study of Law et al which summarized outcome of 51 patients with AL-amyloidosis is not included,[17] since this report included the patients reported by Pinney et al[86] and Cohen et al.[13] We prefer presenting the 2 separate studies, since the latter study included patients transplanted in the period 2004-2018, which better reflects current practice.

Table S1 (continued)

Author (time period)	Remarks
Angel-Korman (1987-2017)	Overall 20 recurrences, includes extrarenal and hematological, treated (HDM-ASCT6, CT 14). Recurrence was cause of death in 8 patients. No differences in graft survival between patients with and without amyloid recurrence. No difference in survival between patients who underwent ASCT vs patients treated with CT only; undergoing ASCT before or after transplantation did not change survival. Note: details on pretransplant therapy are missing. 39 patients received ASCT, of these 33 were treated pretransplant. Posttransplant ASCT was done few months – 7 years after kidney transplantation. Some of these patients likely had received chemotherapy before transplantation, but are not included in the chemotherapy only group.
Cohen (2004-2019)	9 (hematological) recurrences, treated, 5 died due to extrarenal relapse
Heybeli (1997-2018)	Amyloid recurrence in kidney occurred earlier in patients with NR/PR vs VGPR/CR 4 patients with extrarenal recurrence (includes hematological disease), of these 3 died. Bortezomib used in 8 patients, CR/VGPR in 7
Pinney (1987-2011)	5 clonal relapses, treated with response in 4

Sathick (1999-2018)	Two patients with progressive disease at time of transplantation, receiving salvage therapy posttransplant, survived with stable graft function with follow-up of 40 and 125 months respectively. Four patients had hematological progression, all were successfully treated, with 1 graft loss. Four patients have died during follow-up , 1 PTLD.

Table S2: kidney transplantation in patients with MIDD, with and without pretransplant hematological treatment[29-33]

Author (time period)	Patients with pretransplant hematologic treatment				Patients without pretransplant hematologic treatment		
	n	ASCT(n)	Recurrences	Remarks	n	Recurrences	remarks
Angel-Korman (1996-2017)	4	3	1 after 114 months	Another patient developed MM	3	3 after 5, 44 and 106 months	Included 2 patients with nonresponse to melphalan and prednisone; graft loss in 2 untreated patients. One patient received HDM-ASCT with functioning graft > 11 years
Joly (1981-2015)	14	< 2[a]	4 after 38 (32-42) months	Recurrences treated with CT, graft loss in 1 after 60 months	9	9 after 32 (23-42) months	"Recurrences" were patients with MIDD diagnosed after transplantation. 2 untreated, 7 treated with CT. Graft loss in 4/9 at 9-24 months after diagnosis. No details of treatment and period. In this study patients with native kidney MIDD diagnosed after 2004 had markedly better outcome.
Sayed (2002-2015)	3	3	0		2	2 after 18 and 21 months	Untreated, both patients had graft loss
Molina (2010-2019)	6	6	1 after 99 months	Recurrence occurred 24 months after hematological relapse, which did not respond to CT	0	NA	

Heybeli (1987-2016)	10	4	3 after 46 (45-76) months	Graft loss in 2 patients; additional 2 patients with hematological relapse (MM)	9	8 after 20 (2-33) months	In 3 patients “recurrence” was a diagnosis of MIDD after kidney transplantation. This study included patients reported by Leung[21] 5 patients untreated, graft loss in 4/5 3 patients treated, graft loss in 2 patients after 22 months resp > 22 months; 1 patient with CR graft survival > 60 months

CT = chemotherapy ; HDM-ASCT high dose melphalan and autologous stem cell transplantation ; MM = multiple myeloma

[a]approximation, ASCT was mainly limited to patients with MIDD and multiple myeloma.

Table S3: kidney transplantation in patients with LCPT[33-39]

Author	Pre-Tx Therapy	Age (yr)	Gender (m/f)	Time to recurrence (month)	Hematological malignancy	Hematological therapy	Outcome	remarks
Angioi (2016)	No	66	F	12	No (SMM?)	BorDex	Improved Screat 2.6 to 1.2 mg/dl	Diagnosis of LCPT unknown at KTx Serum free kappa 500 -600 mg/l Plasma cells 5-10%
Drieux (2015)	Yes	53	M	29	No	NA	NA	Allograft biopsy because of proteinuria 1.5 g/day. Normal Screat. Kappa LC increased (306 mg/l), λLC normal (9 mg/l). No evidence of tubular dysfunction. BJP not reported. This patient had a diagnosis of LCDD. No crystals
Taneda (2009)	No	64	M	3	Yes MM	VDDex	Improved	Allograft biopsy with casts in the distal tubules, suggesting concomitant cast nephropathy.
Balamuthusamy (2009)	No	47	?	8	Yes	NA	NA	Massive BJP 1.7 gr/day
Kapur (2007)	No	47	F	48	No	CYBorDex	Stable	
Kamal (2020)	No	75	M	13	Yes MM	Unknown	NA	
Kamal (2020)	No	38	F	197	Yes MM	Unknown	Failure	No details , could be de novo disease
Heybeli (2021)	Yes, CR	?	F	No recurrence		Pre KTx	Stable	CR, follow-up 111 months

	No	?	F	2	?	No	Failure	Diagnosis of LCPT unknown at KTx; kidney biopsy for severe rejection which caused graft failure.
	No	?	F	13	?	BorDex	Improved	Stable eGFR, follow-up 78 months, persistent glucosuria. Report mentions hematological progression at time of recurrence. This case is likely the same patient as described by Angioi, however details differ eg, kappa light chain are reported 166 mg/l
	No	?	M	1	?	DEX+ASCT +PE	Stable eGFR	Death after 25 months with stable eGFR.The use of PE suggests that this patient had a high light chain load

Bor = bortezomib, Dex = dexamethasone, V = Vincristine, D = doxorubicin, PE = plasme exchange, ASCT = autologous stem cell transplant. BJP = Bence Jones proteinuria, LC = light chains, MM = multiple myeloma. KTx = kidney transplantation

Table S4: recurrent Immunotactoid Glomerulopathy after kidney transplantation[40-44]

Author	Pre-Tx Therapy	Age (yr)	Gender (m/f)	ITG type	Time to recurrence (month)	Hematological malignancy	Hematological therapy	Outcome	remarks
Korbet (1990) Case 1	No	55	M	?	48	No	No	Failure	De novo membranous nephropathy was diagnosed 18 months after transplantation. At that time no abnormalities in EM. A second biopsy showed signs of ITG
Case 2	No	76	F	?	No recur	No	NA	Stable	Follow-up 84 months
Carles (2000)	No	38	F	M	19	No	Cyclophosphamide	Stable	Initial improvement of eGFR and proteinuria; thereafter stable for 24 months
Javaugue (2021) case 1	No	?	?	M	48	No	?	?	
Javaugue (2021) Case 2	No	?	?	?	No recur	No	NA	NA	
Cadnapaphornchai (1989)	No	45	M	?	18	Yes MM	Methylpredn.	Failure	
Nasr 2021 Case 1	No	56	F	M	10	Yes CLL	Fludarabine, cyclophosphamide, rituximab	Stable	53 months follow-up

Case 2.1[a]	CsA/pred	46	M	P → M^	10	No	Cyclophosphamide prednisone	Failure	36 months
Case 2.2		49	M	P → M^	36	No	Rituximab	Stable	73 months follow-up
Case 3	RTX/pred	57	M	P → M^	4	No	-	Screat 4,3mg/dl	Recurrence at 4 months detected by IF/EM of kidney biopsy which showed rejection but no glomerular abnormalities. After 18 months MPGN pattern of injury
Case 4	?	?	?	P	No recur				
Case 5	?	?	?	P	No recur				

[a]Case 2.1 received a second transplant

In these patients the recurrence was characterized by monoclonal Ig staining (IgG Lambda)

Table S5: kidney transplantation in PGNMID[45-59]

			Pretransplant		Posttransplant			
Author	Year	Patient (gender,age)	Therapy	Response	Recurrence	Time to recurrence	Therapy	Outcome; Follow-up (Mo)
Merhi	2017	M 53	-	-	Yes	12 months	RTX 2x	Stable eGFR, UPCR 12 M
		M 69	-	-	Original disease?	9 months	RTX 2x	UPCR , stable 24 M
Batal	2014	M 61	-	-	unknown	17 yrs	-	Graft function
Al Rabadi	2015	F 61	NA	NA	possibly	7 yr	Bortezomib, carfilzomib	eGFR =; UPCR =
		M 74	BorDex	?	yes	6 months	-	HD 9 months
		F 40	NA	NA	Possibly	11 yrs	-	Stable
Katsuno	2019	M 46	NA	NA	Likely	3.4 yrs	NA	NA
Sawada	2016	M 21	NA	NA	Unlikely	4 yr	Rituximab+PE	± stable
Tsuji	2016	M 33	-	-	Unlikely	4 yr (EBV related)	Rituximab	Stable
Tewari	2016	M 55	-	NA	Yes	3 months	ASCT	UPCR
Wen	2018	M 38	NA	NA	Yes	19	PE	†
		M 44	NA	NA	Likely	15	RTX (late)	renal failure

		F 30	NA	NA	?	10.5	BOR	renal failure
		M 51	NA	NA	?	10.5	BOR	Stable, UPCR low
		M 51	NA	NA	Likely	5 (HCV)	BOR	No FU
Ranghino	2012	F 66	CP	NA	Yes	18 months	PE	renal failure
Sumida	2013	M 52	NA	NA	Yes	4 months	MP	Improved
Visger	2019	M 44	-	-	Yes	14 months	Rituximab	Improved
Kawanishi	2014	M 56	CP	-	Yes	1 month	No	Failure
Albawardi	2011	M59	-	-	Yes	13 months	No	Death cardiac
		M 64	-	-	Yes	22 months	No	Failure BKV?
		F 68	-	-	NO	156 months	No	Dialysis
		M 24	-	-	No	30 months	No	??
Setoguchi	2014	M63	-	-	Yes	12 months	Rituximab	Failure after 31 months
Namba-Hamana	2020	M64	Prednisone	No	Yes	3 months	mycophenolate mofetil	Failure after 8 months

NB: Batal reported a retrospective data analysis which revealed 8 patients with posttransplant PGNMID; time to PGNMID 1.5 months – 22 years; no detailed data, 4/8 developed graft failure within 13 months. - early recurrence in Kawanishi diagnosed in biopsy with acute rejection; showed small deposits in mesangial area only; only after 2-4 months mesangial proliferation; similar findings in Katsumo: after 1 month mesangial deposits; only after 3 years full picture with MPGN, subendothelial deposits

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