Original Article

Histopathology of Allograft Nephrectomies – A Ten Year Observational Study

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Abstract

Background: Though infrequent, allograft nephrectomies are performed for early and late graft loss. The study aims to analyze the histopathologic characteristics of allograft nephrectomy specimens. **Materials and Methods:** We conducted an observational study of 103 cases of allograft nephrectomies from 21 centers from 2013 to 2023. All the pathology slides, including hematoxylin and eosin-stained sections, masson trichrome, jones methenamine silver, PAS, GMS, AFB, and immunohistochemistry (C4d, SV40) were reviewed. Pathologic findings were analyzed based on the transplant to nephrectomy interval (0–3 months, > 3 months) and type of donor (deceased, live donor). **Results:** Of the total 103 cases, 77 were male. The mean age at the time of nephrectomy was 36.4 (range 5–64) years. The allografts were obtained from deceased (57) donors and live related (46) donors. Graft tenderness, oliguria/anuria, and fever were common clinical presentations. The majority (71.8%) of the nephrectomies were performed within the first 3 months of renal transplant. Renal vessel thrombosis (32.03%) was the most common pathologic finding. Infections were more common in the first 3 months after the transplant. Fungal infection had a significant association with deceased donor transplantation (p = 0.029). **Conclusion:** Histopathological study of allograft nephrectomy specimens aids understanding of graft loss causes. The study also provides opportunities to prevent complications and implement measures to prolong graft survival in a subsequent transplant.

Keywords: *Allograft nephrectomy, Graft infection, Renal vessel thrombosis, Histopathology, Graft loss*

Introduction

According to the transplant data submitted to Global Observatory on Donation and Transplantation from 2013 to 2018, renal transplantations were performed on 32,584 live donors and 5748 deceased donors in India.[1] Renal transplants are the most common among all solid organ transplants. The advent of immunosuppressive drugs paved the way for improved graft outcomes. Though infrequent, allograft nephrectomies are being performed for early and late graft loss.

The recognition of the etiology of graft loss is an important aspect of preventing unfavorable complications in transplant recipients. The publications from various timelines have demonstrated the gradual shift in the cause of graft loss over the decades.[2–4] The results comprehended from these studies steered us into the initiative of obtaining pathologic information from renal allografts explanted from the recipients. The current study aims to document histopathologic characteristics of allograft nephrectomy specimens analyzed and reported by us over a 10-year period.

Materials and Methods

We retrospectively analyzed all the consecutive allograft nephrectomies reported from 2013 to 2023 at a single pathology center. The specimens were received from 21 different hospitals in South India. The study enrolled a total of 103 cases. This study does not include any identifiable information of the patients. It also does not have any interaction or intervention with the patients. Hence, ethical approval is not required.

The nephrectomy specimens were formalin-fixed. Sections were taken from the ureter, the renal vessels, the renal hilum, and the renal parenchyma. The sections of 2-µm thickness taken from the paraffin blocks of all the cases were stained with hematoxylin

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and eosin, Masson trichrome, PAS, Jones methenamine silver, and immunohistochemistry (C4d). Special stains (Gomori's methenamine silver, Ziehl-Neelsen stain) were employed to identify the cause of graft failure. SV 40 immunostaining was performed in all the cases of more than 3 months post-transplant. Immunofluorescence study was not done. All the pathology slides were reviewed by two renal pathologists. Pathologic findings were analyzed based on the renal transplant to allograft nephrectomy time interval (0–3 months and >3 months) and type of donor (deceased and live donor). Statistical analysis was done using the software SPSS v21. A p value of <0.05 was considered statistically significant. We also compared our data with studies from other institutions.

Results

The study included 77 males and 26 females. The mean age of the patients at the time of nephrectomy was 36.4 (range 5–64) years and the mean age at the time of renal transplant was 36.2 years. The allografts were procured from deceased donors in 57 cases and live related donors in 46 cases, of which one was an ABO-incompatible donor. 74 patients were nephrectomized within the first 3 months of renal transplantation. 29 patients had their graft removed after 3 months, with a mean-time interval of 43.6 months (SD 42.34). The mean serum creatinine of the patients at the time of allograft nephrectomy was 5.36 mg/dL (range 2.0–8.7 mg/dL). The clinical presentations at nephrectomy included graft tenderness/pain (16.5%), oliguria/anuria (13.6%), fever (12.6%), hyperacute rejection (4.8%), absent renal blood flow (2.9%), discharge at the surgical site (0.9%) and deep vein thrombosis with necrotizing fasciitis (0.9%). Clinical presentation details were unknown in 44.6%. The indications of the allograft nephrectomy included vascular complications (34.95%), unexplained graft dysfunction (34.94%), graft rejection (17.47%), graft necrosis (7.76%) and graft rupture (4.85%).

The causes of native kidney disease in the study population include hypertension (14.1%), diabetes (6.7%), chronic interstitial nephritis (4.8%), IgA nephropathy (3.8%), reflux nephropathy (1.9%), focal segmental glomerulosclerosis (FSGS) (0.97%), proliferative glomerulonephritis (0.97%), polycystic kidney disease (0.97%), lupus nephritis (0.97%), congenital ectopic kidney (0.97%), renal hypoplasia with contralateral obstructive uropathy (0.97%), and bilateral renal calculus disease (0.97%). The cause was unknown in 61.1% of the patients.

The mean weight of the nephrectomy specimens was 196.84 g (range 101–480). The spectrum of pathologic findings in all the cases was grouped under six major categories: Renal vessel pathology, Infection, Rejection, Thrombotic microangiopathy, Subcapsular hematoma and others. Renal vessel pathology (34.9%) was observed to be the most common cause of graft loss in the cohort [Table 1]. Infections were more common in deceased donor transplantation than in live donor transplantation [Table 2]. Acute rejection was reported using Banff classification system in 13 cases that included active antibody mediated rejection or AMR (2/13), C4d negative AMR (2/13), AMR with thrombotic microangiopathy (5/13), AMR with acute T-cell mediated rejection or TCMR (3/13) and combined AMR, thrombotic microangiopathy and acute TCMR (1/13). The features of chronic active TCMR were noted in 4 cases. The features of rejection and C4d immunostaining were negative in all the cases mentioned under the thrombotic microangiopathy category. The non-rejection cases with pathologic features of chronicity were grouped as Others. SV 40 immunostaining was performed in all the cases more than 3 months post-transplant and found to be negative.

The majority of the nephrectomies (71.8%) were performed within the first 3 months post-transplant. Twenty-five (24.2%) allografts were explanted within the first 7 days of renal transplantation as a result of hyperacute rejection (7/25), renal vessel pathology (13/25), subcapsular hematoma (2/25), thrombotic microangiopathy (2/25) and candidal infection (1/25).

Table 1: The diverse spectrum of pathologic findings in 103 allograft nephrectomies

Spectrum of pathologic findings in the current study (n = 103)	
Renal vessel pathology (36)	Renal vessel thrombosis (33)
	Renal artery thrombosis (14)
	Renal vein thrombosis (13)
	Renal artery and renal vein thrombosis (6)
	Renal artery aneurysm (3)
Rejection (24)	Hyperacute rejection (7)
	Acute / chronic rejection (17)
Thrombotic microangiopathy (17)	
Infection (15)	Acute pyelonephritis (2)
	Pyonephrosis (1)
	Fungal infection (12)
	Aspergillosis (7)
	Cryptococcus (2)
	Mucormycosis (1)
	Candida (1)
	Mixed fungal infection (1)
	(Aspergillus + Mucormycosis)
Subcapsular hematoma (5)	
Others (6)	Advanced renal damage (3)
	Chronic pyelonephritis (1)
	Granulomatous interstitial nephritis (1)
	Dystrophic calcification of kidney (1)

Table 2: Analysis of pathologic categories in allograft nephrectomies vs. transplant to nephrectomy time interval and donor type

Pathologic category	Transplant to allograft nephrectomy time interval					Donor type				
	0–3 months		>3 months		p value	Deceased donor		Live donor		p value
	n	%	n	%		n	%	n	%	
Renal vessel pathology	29	39.18	7	24.13	0.149	18	31.57	18	39.13	0.424
Rejection	14	18.91	10	34.48	0.092	11	19.29	13	28.26	0.284
TMA	11	14.86	6	20.68	0.473	11	19.29	6	13.04	0.395
Infection	15	20.27	0	0	**0.004**	13	22.8	2	4.34	**0.006**
Subcapsular hematoma	5	6.75	0	0	0.184	4	7.01	1	2.17	0.207
Others	0	0	6	20.68	0.001	0	0	6	13.04	0.006
Total	74		29			57		46		

TMA: thrombotic microangiopathy; the bold values indicate statistically significant findings

Table 3: Analysis of pathologic findings in allograft nephrectomies vs. transplant to nephrectomy time interval and donor type

Pathologic finding	Transplant to allograft nephrectomy time interval					Donor type				
	0–3 months		>3 months		p value	Deceased donor		Live donor		p value
	n	%	n	%		n	%	n	%	
Renal vessel thrombus	27	36.48	6	20.68	0.122	17	29.82	16	34.78	0.591
Renal artery aneurysm	2	2.70	1	3.44	0.442	1	1.75	2	4.34	0.333
Hyperacute rejection	7	9.45	0	0	0.091	5	8.77	2	4.34	0.218
Acute/ chronic rejection	7	9.45	10	34.48	**0.002**	6	10.52	11	23.91	0.042
TMA	11	14.86	6	20.68	0.473	11	19.29	6	13.04	0.395
Fungal infection	12	16.21	0	0	**0.014**	10	17.54	2	4.34	**0.029**
Acute pyelonephritis	2	2.70	0	0	0.514	2	3.50	0	0	0.303
Pyonephrosis	1	1.35	0	0	0.718	1	1.75	0	0	0.553
Subcapsular hematoma	5	6.75	0	0	0.184	4	7.01	1	2.17	0.207
Others	0	0	6	20.68	0.001	0	0	6	13.04	0.006
Total	74		29			57		46		

TMA: thrombotic microangiopathy; the bold values indicate statistically significant findings

When comparing the pathological findings of the graft nephrectomies done within and after 3 months, all pathological findings were comparable in incidence except for fungal infection, which was exclusively seen in the first 3 months, and rejection which was significantly more common after 3 months of transplant [Table 3]. Fungal infection was also found to be significantly associated with deceased donor transplant. Features of acute/chronic rejection were more common in live donor transplant. The rest of the pathologic findings had no association with the donor type.

The pathologic findings correlated with the previous graft biopsy reports in 19 patients. Fungal infection (4/17), thrombotic microangiopathy (7/17), renal vessel thrombus (2/17), acute rejection (3/17) and subcapsular hematoma (1/17) were the new pathologic findings unveiled from nephrectomy specimens in 17 non-correlated cases. The graft biopsy prior to nephrectomy was either not done or unavailable in 67 patients.

Discussion

This is a large case series from India that describes pathologic findings in allograft nephrectomies. Allograft nephrectomies are performed to confiscate the failed graft resulting from either early complications (<3 months post-transplant) or late complications (>3 months post-transplant). The procedure becomes unavoidable in life-threatening early complications. The patients planned for subsequent transplantation following late complications also underwent graft removal. Most patients developed early complications that culminated in graft nephrectomy. This is in concordance with the study by Ariyarathenam *et al*. where 60% of the total (42) nephrectomies were performed in the first month post-transplant.[5] The graft intolerance syndrome, characterized by fever and graft tenderness, was the common clinical presentation of graft failure observed by Bonilla *et al*.[6] Our patients had a similar clinical picture at the time of nephrectomy. Primary non-function of the allograft is defined as the permanent absence of kidney

function since implantation. Among the nephrectomies done within the first 3 months post-transplant, 13 patients had primary non-function of the allograft. The pathologic findings in these cases were hyperacute rejection (7), renal artery thrombosis (3), subcapsular hematoma (2) and thrombotic microangiopathy (1).

Renal vessel thrombosis remained as the most common pathologic finding in our cohort. This category featured fibrin thrombus in the lumen of the renal artery and/or renal vein [Table 1]. Transplant renal vein thrombosis is a devastating complication with an incidence of 0.5%–4%.[7] Renal artery thrombosis has a low incidence of 0.1%–0.2% of cadaveric transplants.[8] Mazdak *et al.* stated that renal vein thrombosis was the most common pathologic event within the first 6 months of transplant, in their study of 39 allograft nephrectomies.[9] Similarly, Toth *et al.* and Muramatsu *et al.* found renal vein thrombosis in 37% of 49 cases and 22.6% of 124 allograft nephrectomies, respectively.[10,11] Bunthof *et al.* had recorded renal vein thrombosis in 2.5% of the cases.[12] The study, however, included 197 explanted grafts following 3 months of renal transplantation. The thrombus formation in the allograft vessels is often due to mechanical and technical factors like external compression of the vessel, disparities in the vessel sizes of the donor and recipient. Other etiologies include drugs, donor or recipient related factors, late hemolytic uremic syndrome, antiphospholipid antibody syndrome, and the factor V Leiden mutation.[7,13–15]

The OTPN/UNOS renal transplant registry analysis from 1988 to 2010 recorded marked improvement in short-term graft survival as a consequence of a decline in acute rejection episodes in the early post-transplant period.[16] This progress is due to the advent of immunosuppressive agents. The tapering of immunosuppressive agents could lead to immunological activity and inflammation in the allograft. Bonilla *et al*. and Toth *et al*. had witnessed chronic rejection and chronic allograft nephropathy as the most common pathologic diagnosis in allograft nephrectomies.[6,10] Bunthof *et al.* included 197 allograft nephrectomies of more than 3 months post-transplant and featured rejection in 93% of the cases.[12] Mazdak *et al.* reported that chronic T-cell mediated rejection (41%) were significantly found more than 6 months post-transplant.[9]

In our study, rejection (23.3%) was comparatively lower than the previous studies. Endarteritis or vasculitis was noted in all the cases of hyperacute rejection. The acute/chronic rejection findings were significantly higher after 3 months of transplantation. The Banff classification and details of C4d immunostaining for all the cases in the category (n = 17) are summarized in Table 4.[17,18] Acute T-cell mediated rejection Banff III was noticed in three cases of more than 3 months post-transplant. Four cases showed chronic active T-cell mediated rejection, out of which one had Banff III as well as features of chronic TMA. We had limited clinical information on the withdrawal of immunosuppressive therapy, and therefore its correlation with the rejection features could not be ascertained.

Banff TMA working group consensus has recently framed diagnostic criteria for transplant thrombotic microangiopathy (TMA).[19] TMA in kidney transplants and native kidneys share

Table 4: Banff classification and scoring of acute/chronic rejection cases in the study

TX-AN months	AMR	TCMR	C4d	TMA	Activity scores							Chronicity scores			
					g	ptc	i	i-IFTA	t	t-IFTA	v	cg	ci	ct	cv
>3	-	Chronic active/Banff IB	Neg	-	0	0	1	3	0	3	0	0	3	3	3
0–3	+	-	Neg	-	2	2	3	-	0	-	3	0	0	0	0
0–3	+	-	+	+	1	2	2	3	0	1	2	0	3	3	0
>3	+	Chronic active/Banff IA	+	-	2	1	1	3	0	2	0	2	3	3	3
>3	+	-	+	+	2	2	1	0	0	0	2	0	0	0	0
>3	+	Chronic active/Banff IA	+	-	2	1	1	2	0	2	0	3	3	3	3
>3	+	Acute/Banff III	+	-	1	2	3	-	3	-	3	0	0	0	0
>3	+	Acute/Banff III	+	+	2	2	2	1	2	0	3	0	1	1	0
0–3	+	-	+	+	2	1	1	-	0	-	3	0	0	0	0
0–3	+	Acute/Banff IIB	+	-	2	2	3	-	2	-	2	0	0	0	0
>3	+	Chronic active/Banff III	+	+[#]	1	1	1	3	0	2	3	0	3	3	2
0–3	+	-	+	-	2	3	2	-	0	-	0	0	0	0	0
0–3	+	-	+	+	2	2	1	-	0	-	0	0	0	0	0
>3	+	-	Neg	-	1	2	2	-	0	-	0	0	0	0	0
>3	+	-	+	-	2	2	2	-	0	-	0	0	0	0	0
0–3	+	-	+	+	3	3	1	-	0	-	2	0	0	0	0
>3	+	Acute/Banff III	+	-	2	2	2	-	2	-	3	0	0	0	0

TX: transplantation; AN: allograft nephrectomy; AMR: active antibody mediated rejection: TCMR: T-cell mediated rejection, TMA: thrombotic microangiopathy, g: glomerulitis, ptc: peritubular capillaritis, i-interstitial inflammation of non-scarred cortex, i-IFTA: inflammation in scarred cortex, t: tubulitis within tubules of non-scarred cortex, t-IFTA: tubulitis within tubules of scarred cortex, v-vasculitis, cg: chronic glomerulopathy, ci: interstitial fibrosis in cortex, ct: tubular atrophy in cortex, cv: arterial intimal fibrosis, Neg: negative; [#] Chronic TMA.

similar histomorphology. Unlike in native kidneys, transplant TMA is renal limited and has multiple confounding factors like antibody mediated rejection, drug toxicity, and disease recurrence. The rejection-associated TMA in our study are set apart by categorizing them under Rejection category. TMA category of our study includes only non-rejection cases (17). The precise cause of TMA in each case couldn't be determined further.

Renal transplant recipients face the serious risk of acquiring community acquired and opportunistic infections owing to their immunosuppressive status. Graft removal in infections is due to recurrent episodes of pyelonephritis resistant to therapy or clinical deterioration in septicemia. In our study, infection in allograft nephrectomies was significantly detected in the first three months post-transplant. Angio-invasive fungal infection (11.6%) was the chief cause of graft loss among our cases in the infection (14.5%) category. The most common fungal infection among the spectrum was aspergillosis [Table 1]. The fungal infections occurred significantly in the first 3 months post-transplant and following deceased donor transplantation [Table 3]. The fungal infection in our study population was not anticipated prior to surgery. The prompt pathologic diagnosis facilitated the initiation of anti-fungal therapy in the patients. The acquisition of fungal infection in the recipients could occur during organ procurement, preservation, transport, or transplantation.[20–22] The prolonged cold ischemia time, HLA (human leukocyte antigen) mismatch may increase the risk of invasive fungal infection in deceased donor transplantation.[23] The rupture of an abdominal viscus during multi-organ procurement could be a potential source of candida. The fungal arteritis and aneurysmal rupture pose serious threat to the recipient and the graft.

Anupama *et al.* had described acute on chronic pyelonephritis in five of the total 18 allograft nephrectomies.[24] No fungal infection was observed in the cohort. Muramatsu *et al.* classified 124 nephrectomized allografts according to the surgical indication. In the infection (19.4%) category of their study, urinary tract infection (13.7%) was the common cause of nephrectomy, followed by viral infection.[11] One case of fungal infection (candida) was incidentally revealed by the pathologic study. Bunthof *et al.* investigated the pathologic reports of 197 allograft nephrectomies. Two cases of fungal infection (candida) were recorded in the study.[12] Both were anticipated prior to the surgery. The pathologic evaluation by Mazdak *et al.* found no cases of infection among 39 allograft nephrectomies.[9]

Hot and humid climate, late presentation, unhygienic conditions and endemic infections are some of the factors linked to the incidence of infections among transplant recipients in tropical countries.[25] For instance, previous studies from India describe a diverse spectrum of infections in renal allograft biopsies. Gupta *et al.* reported 10.2% of invasive fungal infections among 550 renal transplant recipients.[26] Mucormycosis, followed by aspergillosis, were the common findings. Bhargava *et al.* had described 7.1% graft infection in allograft kidney biopsies of 1684 recipients.[27] Most of them acquired viral infection. Fungal infection contributed the most to allograft nephrectomy. Mucormycosis, followed by aspergillosis, were the most common fungal infections.

Spontaneous rupture of the renal allograft is a rare but dreadful complication with an incidence of 0.3%–9.6%.[28] The graft rupture is the tear of the renal capsule and renal parenchyma, accompanied by hemorrhage. Acute rejection, renal vein thrombosis, trauma, and septic infection could be the notable causes of graft rupture.[29,30] Our study could not attribute to a specific cause of graft rupture in five cases as they had isolated pathologic finding of subcapsular hematoma. We had to categorize them separately, as none of them exhibited other concomitant pathologic findings or features of chronicity.

Graft loss has declined in the current era of effective immunosuppressive therapy. There are undesirable circumstances where patients return to dialysis following the renal transplant. The need for re-transplantation escalates the pre-existing demand for organ donors. The identification of specific pathologic causes of graft loss would help achieve a higher graft survival rate in the future. The current study has shown that routine pathologic assessment of allograft nephrectomy specimens plays a vital role in diagnosing treatable causes like angio-invasive fungal infection. Our retrospective study has limitations with regard to the clinicopathologic correlation. We had incomplete clinical data on patients' DSA levels, immunosuppressant usage, initiation of dialysis prior to nephrectomy. This also applies to the specifics of deceased donor transplantation, like the age and serum creatinine of the donor, microbiological culture to rule out contamination during organ handling. Also, an immunofluorescence study could not be performed as the removed grafts were sent in formalin and no fresh 'unfixed' tissue was available.

Histopathological study of the allograft nephrectomies helps us better understand the causes of graft loss. Our study is distinctive in illustrating the association between the pathologic cause of graft loss in nephrectomies and the donor type. Histomorphology is the gold standard method to establish treatable causes like angio-invasive fungal infection. The study on pathologic findings in allograft nephrectomies provides opportunities to prevent complications and implement appropriate measures to attain prolonged graft survival in the subsequent transplant.

Conflicts of interest

There are no conflicts of interest.

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