

CLINICAL PROFILE AND OUTCOMES IN POST TRANSPLANT COLLAPSING GLOMERULOPATHY

Dr. A. R. A. Changanidi, Dr. Natarajan Gopalakrishnan, Dr. Jayachandran Dhanapriya, Dr. Sheik Sulthan Alavudeen, Dr. Shivakumar Dakshinamoorthy, Dr. Solomon Dolphin, Dr. Ravindran Krishna, Dr. Paulpandi Vathsalyan, Dr. Srinivasan Kannan Badri, Dr. Sajmi Shaji
Madras Medical College, Chennai.

INTRODUCTION

Collapsing glomerulopathy(CG) is a podocytopathy with diverse etiologies that confers a detrimental prognosis and often causes ESRD. Post transplant CG likewise has a poor patient and graft outcome.

AIM:

To study the clinical profile and outcome in post transplant CG.

METHODS:

A **retrospective analysis** of records of kidney transplant recipients (KTR) was done. KTRs with allograft biopsy revealing collapsing glomerulopathy during **2011 to 2021** were studied. Donor type, immunosuppressive regimen, serum creatinine and viral profile (HIV, HCV, CMV) were analysed.

RESULTS

All the six KTRs with collapsing glomerulopathy were **males**. Of them, two had received kidney from living donors and four from deceased donors. Two patients were on cyclosporine and four were on tacrolimus. Viral profile was negative in all patients except one in whom **CMV** was positive.

Patient	A	B	C	D	E	F
Age(years)	28	29	36	19	32	31
Induction	ATG	ATG	basiliximab	basilixima b	Basilixima b	-
Period post transplant(days)	990	24	1620	31	390	300
Serum creat at diagnosis of CG(mg/dl)	3.8	1.8	11.3	2	2.2	3.9
Number of Collapsed glomeruli	6	3	2	2	2	2
IFTA (%)	-	-	25-30	-	5-10	35-40
Associated rejection	-	-	ACMR Banff IB	-	ACMR Banff IA	Chronic ACMR Banff IA
Outcome	Dialysis dependant(DD)	Recovered	DD	CGDF	DD	DD
Present status	Died	Died due to unrelated causes	Died	Alive	Alive	Died
Current serum Creat(mg/dl)				2.5	9.6	

DISCUSSION:

CG was first reported by Weiss et al in 1986. Although it was initially reported among PLHA, now varied other causes have been identified. Etiology can be infectious, autoimmune, malignancies, genetic, drug induced and post transplant.

PATHOLOGY

- podocytes get **transdifferentiated** into macrophage like cells
- podocyte hypertrophy and hyperplasia forming **"pseudocrescents"** in atleast one glomerulus.
- Podocyte- effacement, swelling or disappearance of primary foot processes, loss of actin based skeleton
- GBM wrinkling and folding
- Extensive tubulointerstitial disease with occasional microcystic transformation
- Dense cellular infiltrate predominantly mononuclear phagocytes.

CONCLUSION:

Aetiology of CG was uncertain in most KTRs. **Outcome was poor** in all patients.

