De-novo and recurrent focal segmental glomerulosclerosis (FSGS) in the past twenty years of kidney transplantation: a retrospective monocentric experience

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OBJECTIVES

•**FSGS** shows **high recurrence rates** in renal grafts and often portends an ominous prognosis[1].
•**De-novo FSGS are also reported**, but data about this subgroup are scarce and elusive[1,2].
•Since the histological diagnosis of the underlying disease may be lacking in many ESRD cases, **the FSGS recurrence and de-novo rates** are not well defined in Literature[2].
•**No randomized clinical trial about treatment or prevention of recurrence** is available.
•We report a **retrospective monocentric experience about both de-novo and recurrent FSGS.**

METHODS

•In the period between January 1995 and March 2013 we performed **1954 renal transplants in 1887 patients**. 1162 kidney biopsies were done.

•We found **42 cases of FSGS**, **8 recurrent** (Group 1, 19%) and **16 de-novo** (Group 2, 37%).
•Patients whose clinical data in native kidney follow-up were consistent with FSGS but had no histological diagnosis were defined as **FSGS of uncertain attribution** (n= 19, Group 3, 44%).

•All biopsies were performed for cause (proteinuria and/or increase in serum creatinine>20% from baseline).
•The **rebiopsy rates** in Group 1, Group 2 and Group 3 were **63%, 19% and 26%**, respectively.

	Group 1 n=8 (19%)	Group 2 n=16 (37%)	Group 3 n=19 (44%)	p (Gr1 vs Gr2)
Serum creatinine at discharge, mg/dl	2.55 (1.5-3.9)	2 (0.97-2.9)	2 (0.8-3.4)	ns
Proteinuria at discharge, g/day	0.79 (0.17-1.76)	0,35 (0,1-2,4)	0,6 (0,2-1,72)	ns
Time between transplant and diagnosis, months (m)	5.5 (1-74)	50.5 (2-168)	31 (1- 204)	ns
RAAs blockers at diagnosis	4/8 (50%)	6/16 (38%)	15/19 (79%)	ns
Steroids at diagnosis	8/8 (100%)	15/16 (94%)	12/19 (63%)	ns
CNI at diagnosis	7/8 (88%)	16/16 (100%)	18/19 (95%)	ns
Other immunosuppressive agents* at diagnosis	7/8 (88%)	8/16 (50%)	14/19 (74%)	ns
Serum creatinine at diagnosis, mg/dl	2.43 (1.4-3.8)	2.15 (1.14-5.2)	2,8 (1.2-5.6)	ns
Proteinuria at diagnosis, g/day	3.55 (0.84-10)	2.5 (0.25-6)	2.45 (0.21-18)	ns
No treatment	1/8 (13%)	5/16 (31%)	7/19 (37%)	ns
RAAs blockers increase/modulation	2/8 (25%)	3/16 (19%)	5/19 (26%)	
Immunosuppressive modulation**	0/8 (0%)	3/16 (19%)	3/19 (16%)	
High doses steroids	1/8 (13%)	4/16 (25%)	2/19 (11%)	
Plasma exchange	4/8 (50%)	1/16 (6%)	1/19 (5%)	
Plasma exchange + rituximab	0 (0%)	0 (0%)	1/19 (5%)	
Total follow-up, m	30.5 (1-93)	12 (1-124)	22 (2-155)	ns
Graft failure	6/8 (75%)	5/16 (31%)	8/19 (42%)	**<0.05**
Time between diagnosis and graft failure, m	25.5 (1-42)	3 (1-41)	9 (3-36)	ns

Table 1. Patients characteristics. *Azatioprine, m-TOR inhibitors or mophetil mycophenolate **Addition of others immunosuppressants or dose increase of the ongoing immunosuppresseants

RESULTS

•In our patients **recurrence of FSGS:**
  •**occurs rapidly** at a post-transplantation median time of **6 months**
  •**affects negatively** graft survival: **graft failure in 75%** in a median time of 30 months from diagnosis).
•Patients in Group 2 and 3:
  •**develop the disease lately** at a post-transplantation median time of 50 months and 31 months, respectively
  •**have better outcomes** (graft failure in 31.2% and 42.1%, respectively).
•The **odds ratio for graft failure** in Group 1 vs Group 2 is **6.6**.
•In spite of their lower failure rate, we noted a **more rapid decrease of renal function** in **Group 2 and 3 vs Group 1**: one hypothetical explanation could be **a different treatment rate.**

•As for the **therapeutic approaches**, we herein report about different policies over twenty years. So we cannot adequately compare this different regimes. With this limitation, **Group 2 seems to have the best prognosis in terms of graft failure** (31% vs 75% vs 42%) in spite of a **lower treatment rate.**

CONCLUSIONS

•Also in our experience the **post-transplantation development of FSGS represents a serious complication**.
•As for now, we are unable to suggest a therapeutic strategy to be labelled as the "golden standard".
•**De-novo FSGS** seems to have a **better prognosis** and a **lower failure rate** in comparison with the recurrent form.
In our opinion the **adoption of repeated biopsies** may help **in defining the indications for further treatments**; particularly nowadays when **newer therapies as monoclonal antibodies[3]** are suggested.

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