TEXT

A SIX MONTH STEROID BASED THERAPY FOR RECURRENT OR DE NOVO IgA GLOMERULONEPHRITIS (IgAGN) IN KIDNEY TRANSPLANTATION (KT): A MONOCENTRIC EXPERIENCE.

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OBJECTIVES

IgAGN is the most common primary glomerular disease. In KT, IgAGN can be recurrent or de novo, leading to graft loss in at least 5-10% of cases. In native kidney, Pozzi et al. have proposed a therapy with steroids iv and oral for 6 months with favourable results on long term.In KT with IgAGN recurrent or de novo no therapeutic regimen has been apporved or sustained by numerous favourable results.

METHODS

- ***Aim*** : To evaluate in IgAGN de novo or recurrent in KT the results of the steroid-based protocol of treatment proposed by Pozzi (The Lancet 1999).
- ***Population***: 1948 KT in 1922 recipients over 17 years (January 1995-December 2012). 1098 biopsies were performed for cause: increase in serum creatinine >30% or increase in proteinuria >0,5 g/day.
- In 75/1098 biopsies (6.8%) the histological diagnosis was IgAGN: 23 recurrence GN, 7 de novo GN, 45 GN of an uncertain attribution (ESRD or unknown dignosis in native kidney).
- The treatment was performed in 18 patients (one patient perfomed the steroid course twice) starting in July 2007 as described in Table 1
- ***Treatment***: **methylprednisolone intravenously 500 mg/day for three consecutive days repeated at month 3 and 5; oral prednisone at a dose of 0,5 mg/Kg every other day for 6 months)**
- ***Indication:*** proteinuria (>1,5 g/day), crescents on kidney biopsy possibly associated with increasing serum creatinine.

Characteristics	Steroid treated IgAGN (N=18)	IgAGN not steroid treated (N=57)
Age (years)	44 +/- 14,8	47,4 +/- 15,4
Male/Female	17/2	47/10
Deceased/living donor	14/5	55/2
native kidney disease :		
Chronic GN (no biopsy)	5/18 (27,7%)	14/57 (24,6%)
ESRD (of unknown origin)	6/18 (33,,3%)	15/57 (26,3%)
IgAGN	4/18 (22,3 %)	18/57 (31,6%)
Other GN	2/18 (11,2%)	9/57 (15,8%)
ADPKD	1/18 (5,5%)	1/57 (1,7%)
Type of IgAGN :		
Recurrence	5/18 (27,8%)	18 /57 (31,5%)
De Novo	2/18 (11,1 %)	5 /57 (8,8%)
Unclassified	11/18 (61,1%)	34/57 (59,7%)
Steroid withdrawal pre-biopsy	8/18 (44,4%)	33/57 (57,8%)
Median post transplantation time of diagnosis	64,2 +/- 41,8	78,6 +/- 51,8
Follow up duration	117,2 +/- 45	152,1 +/- 49,2

RESULTS

- All patients completed the therapy without experiencing any major side-effects. They are all alive.
- 2/18 cases graft loss occurred 1 and 3 years after the Pozzi course.
- Data of the 16 patients who are still in follow up are reported in Table 2.

	Pre steroid course (N =16)	Post steroid course (N=16)
ACE inhitors treatment	13/16 (81,2%)	15/16(93,7%)
Urine Protein excretion (g/day)	2,2 +/- 1,9	0,75 +/- 0,5
Plasma cratinine (mg/dL)	1,92 +/- 0,53	1,94 +/- 0,76
Infections/Cardiovascular events /Fractures	0	0
Diabetes	1	5 (transient in 3/5 patients)

CONCLUSIONS

- The immunologic mechanisms involved in the development and progression of IgAGN in native kidney suggest a potential beneficial role for immunosuppressive therapies.
- Particularly, in Pozzi' s and others reports, steroid therapy reduces proteinuria and may prevent ESRD in patients with IgAGN who are proteinuric but without a severely impaired renal function. In KT the IgAGN pathogenesis and treatment is even more difficult to define, given the concomitant immunosuppressive therapy.
- In our experience the use of a six-month steroid therapy in IgAGN after KT can reduce proteinuria and progression of the disease without producing serious side-effects.
-We adopted this regimen in patients with a form of IgAGN we define "aggressive" at some extent (crescents on the renal biopsy, proteinuria >1g/day and/or increase in serum creatinine>20% of baseline.
-Our results may be considered encouraging for the treatment of IgAGN in KT, a conditionfor which encoded treatments are still lacking.

References

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