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Preparing for a kidney transplant: Medical nephrectomy in children with nephrotic syndrome

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Abstract

Nephrotic syndrome is characterized by proteinuria, hypoalbuminemia, and general edema. These symptoms may persist in children who reach ESRD, which is unfavorable for the patient's allograft outcome. In addition, this may hamper early diagnosis of a relapse after transplantation. Surgical bilateral nephrectomy is often considered for that reason, but medical nephrectomy may be a less invasive alternative. In this retrospective single-center case series, we identified all children on dialysis with ESRD due to nephrotic syndrome in which a medical nephrectomy was attempted before kidney transplantation between 2013 and 2018. Outcome was measured by urine output and serum albumin levels. Eight patients with either congenital nephrotic syndrome or focal segmental glomerular sclerosis were included in the study. All patients received an ACE inhibitor as drug of first choice for medical nephrectomy, to which 5 patients responded with oligoanuria and a significant rise in serum albumin, and 3 patients responded insufficiently. In 1 of these 3 patients, diclofenac was added to the ACE inhibitor, with good result. In the other 2 patients, indomethacin was initiated without success, and surgical bilateral nephrectomy was performed. Overall, 6/8 patients had a successful medical nephrectomy and did not need surgical nephrectomy. No recurrence of nephrotic syndrome was found after kidney transplantation in all but one. Medical nephrectomy with ACE inhibitors and/or non-steroidal anti-inflammatory drugs is a safe and non-invasive therapy to minimize proteinuria in children with ESRD due to nephrotic syndrome before kidney transplantation. We suggest that this strategy should be considered as therapy before proceeding with surgical nephrectomy.

KEYWORDS

ACE inhibitors, kidney transplantation, medical nephrectomy, nephrotic syndrome, NSAIDs

Abbreviations: ACE, angiotensin-converting enzyme; CNS, congenital nephrotic syndrome; ESRD, end-stage renal disease; FSGS, focal segmental glomerular sclerosis; NSAIDs, non-steroidal anti-inflammatory drugs.

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1 | INTRODUCTION

Nephrotic syndrome is a glomerular disorder that is defined by massive proteinuria, hypoalbuminemia, and general edema. Children with nephrotic syndrome mostly respond well to steroids and have a favorable prognosis. In contrast, therapy-resistant nephrotic syndrome generally results in ESRD within a few years.^{1,2}

Kidney transplantation in patients with active nephrotic syndrome poses 2 major risks. First of all, hypoalbuminemia leads to an unfavorable circulatory status and an increased risk of thromboembolic events, both resulting in major risks for the renal transplant.^{3,4} In fact, optimal hydration is the most important perioperative intervention to ensure graft function.⁵ Second, nephrotic syndrome may relapse after transplantation. Early detection of proteinuria after transplantation is vital to start treatment for such relapses in order to improve prognosis. "Contamination" of the urine with proteinuria from the native kidneys may hinder such early detection. Furthermore, proteinuria may also indicate rejection of the transplant kidney, for which prompt diagnosis and treatment are also necessary.⁶ Ponticelli et al have shown that renal survival rates of patients with proteinuria are worse in comparison with non-proteinuric patients.⁷ In fact, the quantity of proteinuria is a reliable predictor of the allograft outcome.⁷

In order to improve the circulatory status during and after renal transplantation and allow for early detection of proteinuria of the transplant kidney, treatment to stop the native proteinuria is often needed. Currently, the treatment of choice in such patients is surgical nephrectomy.^{2,8} This leads to at least one additional operation in these patients, which is known to have high rates of morbidity and even mortality (up to 2.0%).^{9,10} A less invasive but successful therapy is bilateral renal artery embolization, which may have severe side effects, such as severe flank pain and fever, and requires specific interventional skills that may not be readily available.¹¹⁻¹³

ACE inhibitors and NSAIDs have antiproteinuric effects and are used as such in patients with (congenital) nephrotic syndrome.¹⁴⁻¹⁶ Case reports show that non-invasive nephrectomy by using NSAID and/or ACE inhibitors may have an immediate effect on urine output and proteinuria, resulting in a sharp rise of serum protein levels.^{14,17,18} Unfortunately, little effect was seen in some other reports.^{13,19}

In our center, we have used medical nephrectomy as a first-line option in children with therapy-resistant nephrotic syndrome on dialysis during the workup for renal transplantation. Our retrospective case study investigated the feasibility and safety of the use of ACE inhibitors and/or NSAIDs instead of surgical nephrectomy or renal artery embolization in order to achieve medical nephrectomy in a safe non-invasive way in patients with persistent severe proteinuria in the workup toward renal transplantation.

2 | PATIENTS AND METHODS

In this single-center retrospective case study, we did a data search of all pediatric renal transplant candidates with ESRD due to nephrotic syndrome in the period 2013-2018. This period was chosen for

reasons of data availability in the digital hospital data system. Of these patients, we identified those who were treated with medication in order to induce a medical nephrectomy prior to renal transplantation. Such patients received an ACE inhibitor and/or NSAID after starting dialysis. The dosage of the medication was based on body weight and blood pressure, and adjusted based on the anti-diuretic effect, blood pressure, and side effects such as liver function tests. In all cases, the medication was stopped at least 1 day before kidney transplantation.

Patient characteristics extracted from electronic patient records were date of birth and diagnosis, gender, weight, date of medical nephrectomy, date of dosage changes of ACE inhibitor and/or NSAIDs, and date of kidney transplantation. Furthermore, we extracted clinical and genetic diagnosis, medication type and dosages pre- and post-surgery, and complications like thrombosis. Clinical and laboratory results extracted were blood pressure, urine output, amount of proteinuria if measured, and serum albumin levels before and after the start of the medical nephrectomy.

2.1 | Statistical analysis

Descriptive analyses were performed using SPSS, version 22.0 (IBM). Results are expressed as median with range and counts with percentages.

3 | RESULTS

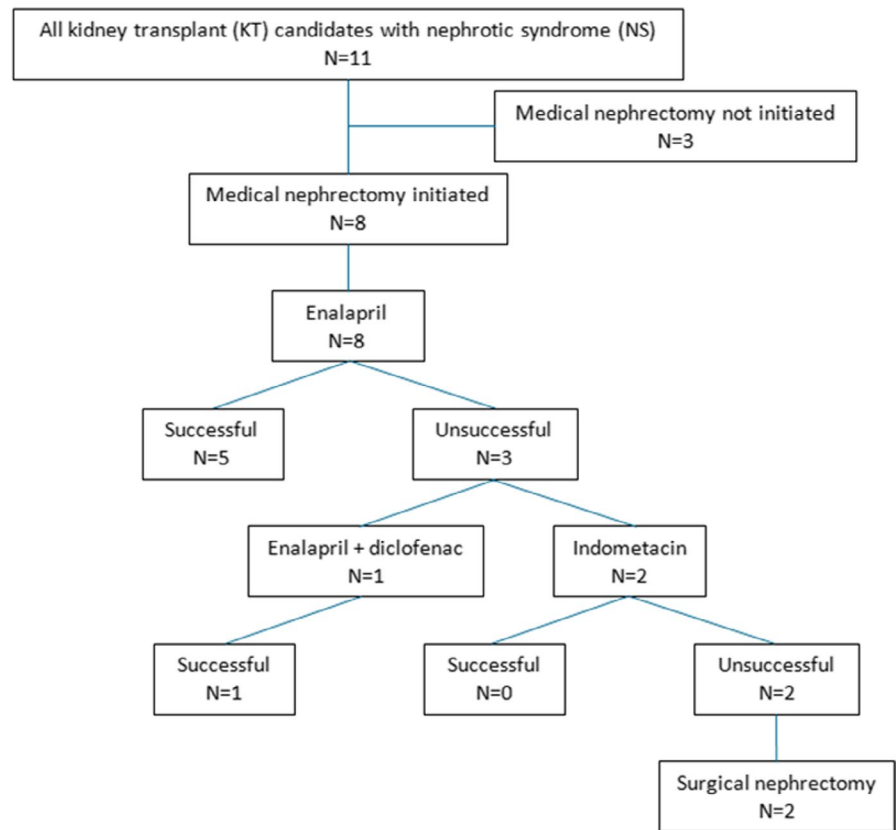
3.1 | Demographics

Eleven pediatric patients with nephrotic syndrome who were prepared for renal transplantation were identified in this study. Three patients were not started on ACE inhibitors for medical nephrectomy, because of severe illness ($N = 1$, surgical nephrectomy was performed to stabilize the patient due to extensive fluid overload) and therapy-resistant malignant hypertension ($N = 2$). Eight patients did receive medication to induce a medical nephrectomy prior to renal transplantation (Figure 1 and Table 1). The diagnosis leading to ESRD was CNS due to a mutation in the NPHS1 gene ($n = 2$) or NPHS2 gene ($n = 3$), and FSGS without a proven genetic basis ($n = 3$). All patients were on dialysis prior to the kidney transplantation, whereof 4 patients on hemodialysis and 4 on peritoneal dialysis.

Table 1 shows the demographics, preoperative characteristics, and post-operative outcome of these patients. The median age at transplantation was 5.7 (range 3.4-10.0) years with a median age of 4.2 (range 2.3-9.4) years at the start of medical nephrectomy, 2.1 (range 0.04-3.67) years after diagnosis.

Currently, 7 of the 8 patients had a successful kidney transplantation (6 living and 1 post-mortem heart beating), whereof 6 patients without recurrence of nephrotic syndrome despite keeping their native kidneys. One patient was still on hemodialysis waiting for kidney transplantation, and 1 patient had a therapy-resistant recurrence of the disease after transplantation and had to restart dialysis.

FIGURE 1 Flow diagram of medical nephrectomy in pediatric patients with ESRD due to nephrotic syndrome in a single-center study (2013-2018)



3.2 | Medical nephrectomy

Figure 1 shows a flow diagram of the medication used for medical nephrectomy in our patients. The medication dosages are presented in Table 1. All eight patients received enalapril with a median daily starting dose of 0.09 mg/kg (range 0.07-0.31 mg/kg). Adjustments in dosage were made based on blood pressure and urine output in the individual patients, with a median maximum daily dose of 0.30 mg/kg (range 0.03-0.76 mg/kg). Reasons to lower or stop enalapril were hypotension or persistent significant urine output despite increases in the dose. Two patients received indomethacin (1.0 and 2.0 mg/kg/day, respectively) after unsuccessful medical nephrectomy with enalapril: Patient 3 was taken off enalapril after 2.5 months for hypotension, after which indomethacin was started; patient 8 was treated with enalapril for 14 months when indomethacin was added. In both patients, medical nephrectomy with indomethacin had to be stopped due to elevated transaminases after 1 and 6 months, respectively. In 1 patient, diclofenac was added to enalapril because of persistent proteinuria and significant urine output despite an acceptable serum albumin level of 34 g/L. It was started at a dose of 50 mg 3 times a week after the hemodialysis session and increased to 100 mg 3 times a week.

Albumin levels increased or stayed in within normal range in all patients (Table 2). After starting enalapril albumin levels increased from a median of 18 (range 11-28) g/L to a median of 31 (range 21-36) g/L. With indomethacin albumin levels increased from a median of 25 (range 21-29) g/L to a median of 29 (27-31) g/L. The patient who received diclofenac in addition to enalapril showed no change

in serum albumin levels, as the albumin level before start was already 34 g/L.

In all patients, the medication for medical nephrectomy was stopped at least 1 day before kidney transplantation.

In 6 patients, medical nephrectomy prior to renal transplantation was successful. In two patients, treatment was unsuccessful and surgical bilateral nephrectomy was performed as preparation for renal transplantation. Both these patients were diagnosed with NPHS1 mutations. In 1 patient, medical nephrectomy failed because of side effects (enalapril because of hypotension and indomethacin because of elevated transaminases). The other patient had no relevant change in urine output and proteinuria despite enalapril and indomethacin, and medication dose could not be increased because of a low blood pressure and a slight increase in liver function tests.

3.3 | Side effects

The only side effect of enalapril seen was hypotension. In 3 cases, ceasing other anti-hypertensive drugs was enough to normalize blood pressure, in 1 case a reduction in dosage was sufficient and in 2 cases enalapril needed to be stopped. In both patients that were started on indomethacin, liver function tests were elevated as a side effect, leading to cessation of treatment and subsequent surgical bilateral nephrectomy. In contrast, there were no side effects seen in the patient that was treated with diclofenac in combination with enalapril. After transplantation, no thrombosis, delayed graft function, hypotension, or need of diuretics was noted.

TABLE 1 Demographics and clinical characteristics of individual patients with ESRD due to nephrotic syndrome who underwent medical nephrectomy

	1	2	3	4	5	6	7	8
Disease	CNS	CNS	CNS	FSGS	CNS	FSGS	FSGS	CNS
Mutation	NPHS2	NPHS2	NPHS1	–	NPHS2	–	–	NPHS1
Gender (M/F)	M	F	M	M	M	F	M	F
Age at diagnosis	0.8	1.2	0.06	3.0	3.0	3.5	3.7	0.04
Age at start MN (y)	2.9	4.0	2.3	9.4	4.3	5.4	6.2	4.0
Age at KT (y)	3.4	4.4	4.6	10.0	5.7	6.1	–	6.5
RRT mode	PD	PD	HD	HD	HD	PD	HD	PD
Enalapril (mg/kg/d)	+	+	+	+	+	+	+	+
Initial dosage	0.07	0.09	0.09	0.08	0.31	0.11	0.10	0.08
Maximum dosage	0.15	0.40	0.09	0.76	0.37	0.22	0.10	0.08
Indomethacin (mg/kg/d)	–	–	+	–	–	–	–	+
Initial dosage	–	–	1.15	–	–	–	–	1.01
Maximum dosage	–	–	1.15	–	–	–	–	2.02
Diclofenac (mg/kg ^a)	–	–	–	+	–	–	–	–
Initial dosage	–	–	–	1.52	–	–	–	–
Maximum dosage	–	–	–	3.05	–	–	–	–
Surgical nephrectomy	–	–	+	–	–	–	–	+
UO before KT	Reduced	Reduced	Anuria	Reduced	Anuria	Reduced	Reduced	Anuria
Serum albumin before MN (g/L)	12	19	11	28	24	17	11	19
Serum albumin before KT (g/L)	30	27	39	33	32	26	–	37
Protein-creatinin ratio after KT ^b (g/10 mmol)	0.30	0.42	0.25	3.63	0.33	0.40	NA	0.70

Abbreviations: CNS, congenital nephrotic syndrome; FSGS, focal segmental glomerular sclerosis; HD, hemodialysis; KT, kidney transplantation; MN, medical nephrectomy; NA, not applicable; PD, peritoneal dialysis; RRT, renal replacement therapy; UO, urine output.

^aDosed after each hemodialysis session.

^b9–38 d after KT.

4 | DISCUSSION

Our case series showed that controlled medical nephrectomy in children with ESRD due to nephrotic syndrome is a feasible and safe non-invasive treatment in the preparation of renal transplantation. ACE inhibitors seem to be the drug of first choice because of the high success rate and mild side effects in a dosage up to 0.30 mg/kg per day. In contrast, indomethacin has a low success rate and considerable side effects, that is, hepatic impairment, which makes its use questionable for medical nephrectomy, even in low doses, in our experience. No conclusion can be drawn with regard to diclofenac since it was only given to one patient in our institution.

Previous case reports evaluating medical nephrectomy observed variable results (for an overview, see Table 3).^{12–14,17,19} In all reports, ACE inhibitors, indomethacin, or another NSAID were used to achieve an acceptable circulatory state before transplantation. Dosages used in these case reports are 2 mg/kg/day to 14 mg/kg/day of indomethacin, a maximum of 5 mg/kg/day of captopril, and a dose of 1 mg/kg/day of quinapril. A possible reason for the difference in outcome between some of the previous case reports and our experience could be the difference in type of nephrotic syndrome. Another reason could

be the fact that in our center all patients were on dialysis, which we used as an effective method to achieve intravascular volume depletion in addition to the medical nephrectomy. The differences in dosage are not likely the cause, as we used lower dosages in our study compared to the above-mentioned case reports.

Of the 11 patients identified, 5 underwent surgical nephrectomy. The complication rate in these patients was favorable, with no mortality and only minor morbidity, mainly related to the surgery. As described, 2 patients were already treated with anti-hypertensive agents, including ACE inhibitors, and had a surgical nephrectomy for uncontrolled blood pressure. In addition, in 1 patient surgical nephrectomy was performed to stabilize the patient due to extensive fluid overload, in whom it was deemed impossible to perform a medical nephrectomy. These cases illustrate that surgical nephrectomy may be a preferred option, and an individualized consideration of the treatment options should be made.

Another option to shut down the kidneys is by bilateral renal artery embolization. The reported success rate is higher than in medical nephrectomy, but side effects can be severe.^{11–12,19} Side effects that have been previously described are fever and challenging flank pain, requiring narcotic analgesics in combination with epidural

TABLE 2 Effect of medical nephrectomy with ACE inhibitor and/or NSAID on urine output and albumin level in patients with ESRD due to nephrotic syndrome

	Enalapril	Indomethacin	Enalapril and diclofenac
Total (%)	8/8 (100)	2/8 (25.0)	1/8 (12.5)
Urine output			
No difference	1/8	0/2	0/1
Reduced	6/8	2/2	1/1
Anuria	1/8	0/2	0/1
Albumin level (g/L)			
Premedication ^a	18 (11-28)	25 (21-29)	34
Post-medication ^a	31 (21-36)	29 (27-31)	33
Difference pre- and post-medication ^a	12 (2-25)	4 (-2-10)	-1

^aMedian (range).**TABLE 3** Literature review of medical nephrectomy and renal artery embolization in patients with ESRD due to nephrotic syndrome

Study	Year	N	Disease	Method	Success rate (%)	Side effects
Medical						
Baumelou ¹⁷	1982	1 ^a	Membranous nephropathy	Indomethacin (150 mg/day)	1/1	—
Hagerty ¹⁸	1989	1 ^a	Type 1 MPGN	Naproxen (500 mg/day)	1/1	—
Pomeranz ¹⁴	1995	1	Finnish CNS	ACEi (max 5 mg/kg/day) + indomethacin (max 4 mg/kg/day)	1/1	—
Solak ¹³	2010	1 ^a	NS secondary to amyloidosis	ACEi (80 mg/day) + indomethacin (150 mg/day)	0/1	No effect
Maeda ¹⁹	2011	1 ^a	Membranous nephropathy	Indomethacin (max 1000 mg/day)	0/1	No effect
Sallam ¹²	2012	1 ^a	FSGS	NSAID (high dose)	0/1	Gastrointestinal
Vos (current)	2018	8	FSGS, CNS	ACEi, ACEi + diclofenac, indomethacin	6/8	Low blood pressure, elevated transaminases
Total		14			9/14 (64.3)	
Embolization						
Capozza ¹¹	2007	1	Massive proteinuria		1/1	Flank pain, (fever)
Solak ¹³	2010	1 ^a	NS secondary to amyloidosis		1/1	—
Maeda ¹⁹	2011	1 ^a	Membranous nephropathy		1/1	Fever, elevated LDH, WBC, CRP
Sallam ¹²	2012	1 ^a	FSGS		1/1	Fever, flank pain, leukocytosis
Total		4			4/4 (100)	

Abbreviations: ACEi, angiotensin-converting enzyme inhibitors; CNS, congenital nephrotic syndrome; FSGS, focal segmental glomerular sclerosis; MPGN, membranoproliferative glomerulonephritis; N, number of patients; NS, nephrotic syndrome; NSAID, non-steroidal anti-inflammatory drugs.

^aAdult.

infusion of local anesthetic.^{11-12,19} This method seems to be more invasive, especially in children, frequently needing general anesthesia. In addition, an experienced physician is needed to perform this treatment and there is a risk of injuring the femoral artery, which may hamper arterial anastomosis during kidney transplantation.

Despite the small patient group and the retrospective character, our study supports the use of ACE inhibitors and/or NSAIDs as first-choice therapy in the induction of medical nephrectomy prior to kidney transplantation. Limitations of the present study are the

retrospective design, the lack of a hard definition for reduced urine output, the lack of fixed time-points to measure proteinuria and thereby missing data on pretransplant proteinuria, and the small sample size. No conclusions can be drawn regarding indomethacin and diclofenac. Surprisingly, unsuccessful medical nephrectomy seems to be associated with having a NPHS1 gene mutation. Both our patients with NPHS1 gene mutation did not respond to medical nephrectomy, while no other patient needed bilateral surgical nephrectomy. Since such information could not be obtained from other

reports, and it remains to be determined whether this was due to chance or not. Preferably, such information should come from a prospective multi-center study, which is needed to investigate the advantages of ACE inhibitors, possibly in combination with NSAIDs as first-choice therapy in the management of future kidney transplant candidates with proteinuria from the native kidneys.

Since thrombosis is an uncommon outcome, it cannot be determined whether medical nephrectomy decreases the risk of thrombosis post-transplant. Prospectively showing improvements in clotting factors such as ATIII would be useful in future studies. Another potential pitfall of medical nephrectomy could be the recurrence of proteinuria after transplantation. We did not observe any rebound proteinuria, which may be explained by the use of tacrolimus as part of the immunosuppressive regimen, as tacrolimus is well known to result in vasoconstriction of the afferent arterioles, with a subsequent reduction in glomerular permeability to protein and thereby a reduction of proteinuria.²⁰

Based on the presented data, we conclude that medical nephrectomy with ACE inhibitors and/or NSAIDs should have a place in the process prior to renal transplantation before surgical or embolization nephrectomy. Since our study was limited to 8 patients, we acknowledge that complications may still occur and patients should be on close monitoring. However, these data suggest that complications other than standard (side) effects of the medication are rare and treatment can be started safely, starting with a low dose of 0.1 mg/kg/d of enalapril.

AUTHORS' CONTRIBUTIONS

Michiel F. Schreuder, Elisabeth A. M. Cornelissen, Linda Koster-Kamphuis, Nicole C. A. J. van de Kar, and Charlotte M. H. H. T. Bootsma-Robroeks: conceived case series; Eefke Vos and Michiel F. Schreuder: designed the case series; Michiel F. Schreuder, Linda Koster-Kamphuis, Charlotte M. H. H. T. Bootsma-Robroeks, and Elisabeth A. M. Cornelissen: helped including cases; Eefke Vos: coordinated the running of the case series, the data collection, and carried out the initial statistical analysis. Eefke Vos: wrote the first draft of the manuscript. Michiel F. Schreuder: assisted Eefke Vos with further analysis and contributed to the interpretation of the analysis and the writing of the manuscript. All authors contributed in revising the manuscript. All authors read and approved the final manuscript.

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