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Percutaneous renal artery embolisation of non-functioning renal allografts with clinical intolerance

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Abstract The aim of the study was to evaluate the efficacy and safety of percutaneous renal artery embolisation of non-functioning renal allografts in patients with graft intolerance syndrome (GIS). Transcatheter artery embolisation was performed in 30 kidney transplant recipients with GIS. The duration of graft function had been 60 ± 45 months. Infectious disease was ruled out in all patients. Embolisation consisted of the injection of polyvinyl alcohol microspheres followed by the insertion of a stainless steel coil in the renal artery branches. Symptoms of GIS included: fever-graft pain (44%, $n = 13$), fever-hematuria-pain (20%, $n = 6$), fever-hematuria (13%, $n = 4$) and fever alone (23%, $n = 7$). Latency time between graft failure and embolisation was 184 ± 227 (17–1181) days. Embolisation was clinically successful with the prolonged disappearance of GIS in 24 patients (80%). Six patients showed initial clinical improvement, but GIS reappeared at 40 ± 18 days, and graft nephrectomy was required. There were no major complications associated with embolisation and no deaths. Perirenal collateral supply was a risk factor for the reappearance of GIS. Renal vascular embolisation is a simple, safe and effective technique for treating renal allograft intolerance syndrome and could be a feasible alternative for the first-line treatment.

Keywords Kidney transplantation · Embolisation · Vascular graft occlusion

Introduction

The most frequent cause of long-term graft loss is chronic allograft nephropathy. Patients with a non-functioning renal allograft can show clinical manifestations of intolerance after immunosuppressive therapy has been stopped. Graft intolerance syndrome (GIS) is characterised by varying degrees of fever, hematuria and graft pain [3, 10, 14]. The conventional treatment for this syndrome is surgical allograft nephrectomy. However, this procedure is associated with a substantial rate of potentially severe complications, such as haemorrhage, haematoma, abscess, wound infection or vascular injury, and even mortality [3, 19, 24].

Renal artery embolisation has been proposed as an alternative to the surgical removal of failed grafts [13]. However, the safety and long-term efficacy of this technique has not been established. The aim of the present study was to investigate the efficacy and safety of allograft embolisation for treating patients with a non-functioning renal allograft and graft intolerance syndrome.

Material and methods

Patients

From July 1995 to July 1999, transcatheter renal artery embolisation was performed in 30 kidney transplant recipients with non-

functioning renal allograft and GIS. All patients were on hemodialysis. Demographic characteristics are shown in Table 1. The causes of graft failure were chronic allograft nephropathy in 24 patients (80%), refractory acute renal rejection in 5 patients (17%) and primary non-function in 1 patient. The duration of renal allograft function and immunosuppression therapy are summarised in Table 2. At the moment when GIS was diagnosed, immunosuppressor treatment was withdrawn in all patients except the one with primary non-function. Immunosuppressor therapy was discontinued according to a standard protocol with slow, gradual tapering of the prednisone dose until the drug was stopped.

Graft-intolerance syndrome was diagnosed by the presence of fever, hematuria or graft pain in the absence of simultaneous infectious disease. Abdominal ultrasonography was performed to rule out hydronephrosis or renal allograft abscess. A 111In-labeled platelet scintigraphy study was used to confirm the diagnosis [22]. All patients were symptomatic at the time of the embolisation. Informed consent was obtained from each patient prior to the examination, and the study was approved by the local ethics committee.

Table 1. Demographic characteristics of patients

Age	44 ± 16	(15–71)
Sex		
Men	$n=18$	60%
Women	$n=12$	40%
Years on dialysis		
< 5	$n=19$	64%
5–10	$n=7$	23%
> 10	$n=4$	13%
Aetiology of CRF		
Glomerulonephritis	$n=6$	20%
Polycystic kidney	$n=3$	10%
Diabetes mellitus	$n=4$	13%
Nephrosclerosis	$n=2$	7%
Urological disease	$n=2$	7%
Unknown	$n=6$	20%
Others	$n=7$	23%

Renal embolisation

Renal embolisation was performed on an angiographic unit (Politron Plus, Siemens) with local anaesthesia and no sedation. Prophylactic antibiotic treatment, consisting of a single intravenous dose of 1,000 mg ceftriaxone, was administered just before the procedure. Retrograde catheterisation of the femoral artery ipsilateral to the renal allograft and digital subtraction angiography of the aorto-iliac sector were carried out in all patients. Before starting embolisation, we assessed the renal allograft anatomy, renal artery patency and the presence of perirenal collateral supply. We performed selective retrograde catheterisation of the allograft renal artery with a standard angiographic catheter (Radiofocus Glidecath, Terumo, Japan). Additionally, the renal artery branches were selectively catheterised with a microcatheter to achieve occlusion at the most distal point and avoid the entry of embolisation material into the iliac artery.

We began by embolisation of the distal vessels with 100–500 micras polyvinyl alcohol microparticles (Contour, Boston Scientific) mixed with iodine contrast until slow flow was achieved in the intrarenal arteries. Embolisation was completed with the insertion of a 6–8 mm Tungsten steel coil (Free P Tungsten Spirale, Balt, France) into the renal artery or its branches. After completing renal embolisation, intra-arterial digital subtraction angiography of the aorto-iliac sector was performed to confirm occlusion of the renal artery and to check for collateral renal supply. With the detection of cortical supply, we attempted superselective catheterisation of the artery with a coaxial microcatheter and embolisation with 100 micras polyvinyl alcohol particles.

Table 2. Clinical features of renal transplantation. *Aza* azathioprine, *CsA* cyclosporine, *Pdn* prednisone, *MMF* mofetil-mycophenolate, *Rapa* rapamycin

Duration of renal function		
Overall	60 ± 45 months	
Primary non-function	$n=1$	3%
< 24 months	$n=8$	27%
24–60 months	$n=7$	23%
60–120 months	$n=10$	34%
> 120 months	$n=4$	13%
Immunosuppression therapy		
Aza-Pdn	$n=3$	10%
CsA-Pdn	$n=17$	57%
CsA-Aza-Pdn	$n=5$	17%
CsA-MMF-Pdn	$n=4$	13%
Rapa-MMF-Pdn	$n=1$	3%

Evaluated parameters

The following variables were recorded: clinical features of renal transplantation and graft intolerance, characteristics of the embolisation technique, time transpired between graft loss and embolisation, immediate angiography results, existence of collateral supply, duration of ospitalisation and short- and long-term clinical outcome.

Complications including haemorrhage, haematoma, infection, pain, allergy to the contrast, vascular dissection or limb ischemia and post-embolisation syndrome were recorded. The characteristics of the group in which embolisation was ineffective were analysed independently.

Statistical analysis

Results are expressed as mean and standard deviation (SD). Statistical analysis was carried out according to the Student's t test and ANOVA for the analysis of quantitative variables and Chi-squared and Fisher's exact probability test for the analysis of qualitative variables. We considered a P value of less than 0.05 to be statistically significant.

Results

Three patients (10%) had received a simultaneous kidney-pancreas transplantation and 27 patients (90%) a kidney transplantation alone. This was the first renal transplantation in 21 patients (70%), the second in 8 (27%) and the third in 1 (3%). The renal allograft was located in the right iliac region in 13 patients (43%), in the left iliac in 13 patients (43%) and in the lumbar region in 4 patients (14%). All the patients showed fever. The common clinical presentations were fever-graft pain (44%, $n=13$), fever-hematuria-pain (20%, $n=6$), fever-hematuria (13%, $n=4$) and fever alone (23%, $n=7$). A 111In-labeled platelet scintigraphy study was performed in 24 patients, and 87% of them ($n=21$) showed abnormal uptake (index > 1.5). In three patients, radionuclide imaging was negative (index 1) or inconclusive

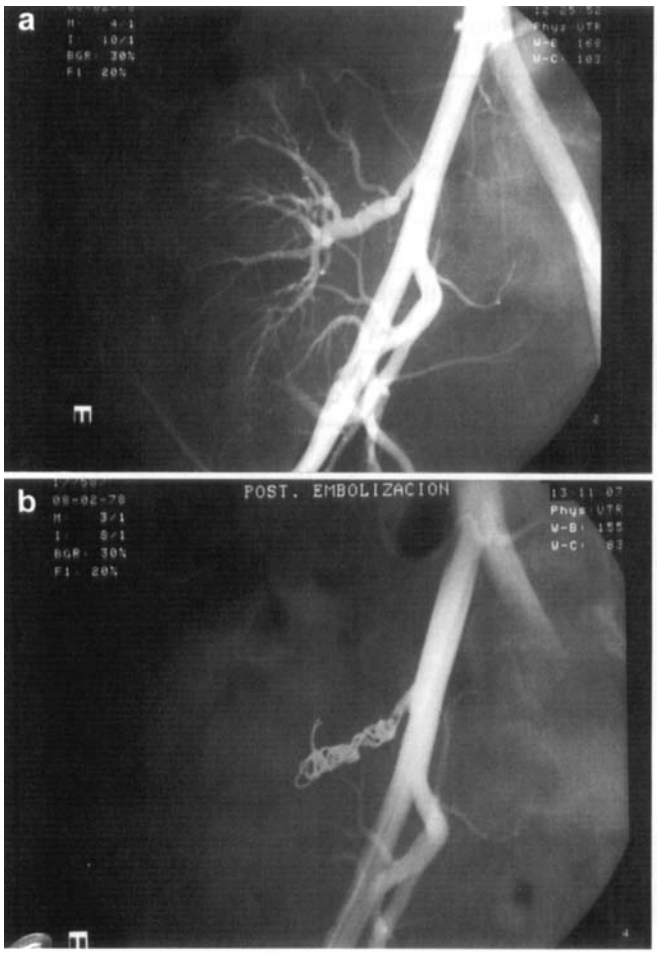

Fig. 1. **a** Intra-arterial digital subtraction angiography of the right aorto-iliac region. Renal artery patency with end-to-side anastomosis with the iliac artery. **b** Post-embolisation angiography. Total occlusion of the allograft renal artery and the intrarenal branches. Visualisation of stainless steel coil in main branch of the renal artery

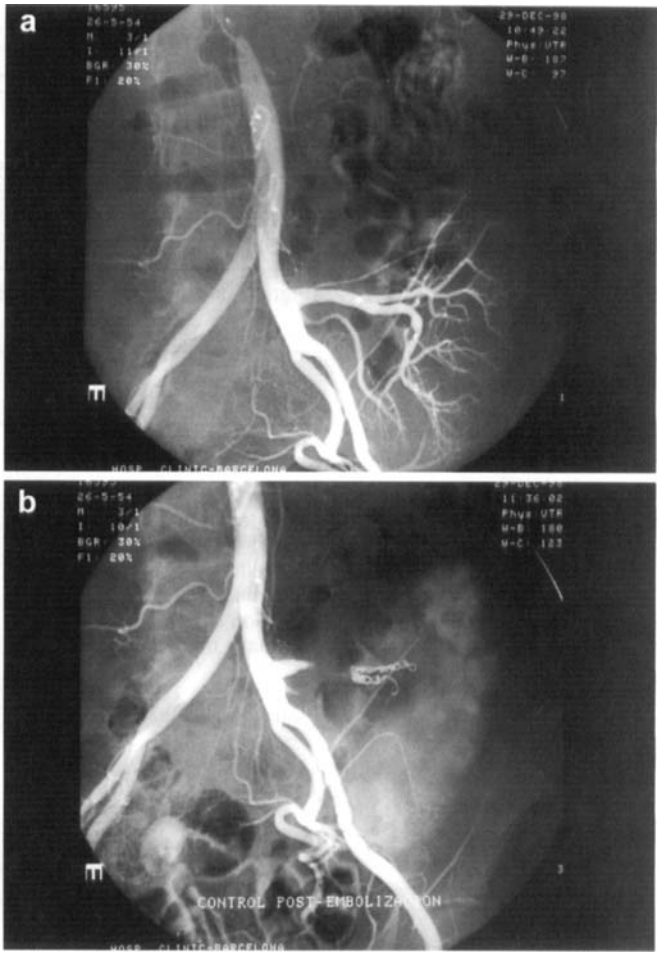

Fig. 2. **a** Angiography of the left aorto-iliac region. Allograft renal artery patency with two small polar arteries can be seen. **b** Post-embolisation angiography. Complete occlusion of the allograft renal artery and two small polar arteries. Tungsten coils can be seen in the bifurcation branches

(index 1–1.5), due to recent steroid therapy prior to the test.

The latency time between graft loss and renal embolisation was 184 ± 227 days (17–1,181). Seventy-four percent ($n = 22$) of the embolisations were performed in the first 6 months and 13% ($n = 4$) from 6 to 12 months after graft loss. It must be noted that four patients (13%) underwent embolisation after a longer interval, with GIS appearing at 1 year or more (12, 20, 12 and 39 months) after graft loss. After embolisation with microspheres, steel coils were left in the artery branches in 26 patients (87%) and in the renal artery in 4 patients (13%) (Fig. 1a, b) and (Fig. 2a, b). Arteriographic evaluation immediately after vascular occlusion showed that embolisation was complete in all the patients. Eleven patients (37%) had a prominent collateral circulation, which we were able to embolise in only three patients, since catheterisation was technically very difficult to perform.

The long-term results of allograft embolisation were good, with sustained absence of clinical intolerance in 24 patients (80%). The six remaining patients (20%) showed initial improvement of symptoms, but GIS reappeared at 40 ± 18 days (13–66) and required graft nephrectomy. The average follow-up period of the group not requiring nephrectomy ($n = 24$) was 38 ± 14 months. No long-term complications were observed in this group. It is noteworthy that embolisation was clinically successful in all the patients presenting early graft failure, including one with primary non-function who was embolised 17 days after renal transplantation.

There were no deaths and no major complications in any of the patients. Only four patients (13%) experienced slight post-embolisation pain, and one patient presented a haematoma at the puncture site that did not

Table 3. Post-embolisation syndrome (PES): Influence of steroid therapy

	Overall series $n=30$	Steroid therapy $n=16$	No steroids $n=14$	P
Frequency	47% ($n=14$)	25% ($n=4$)	71% ($n=10$)*	$P<0.001$
Latency of fever (hours)	29 ± 13	22 ± 8	32 ± 14	NS
Duration of fever (days)	4.2 ± 2.4	3.2 ± 1.5	4.6 ± 2.7	NS
Length of stay (days)	5.5 ± 3.0	3.6 ± 0.8	7.7 ± 3.1*	$P<0.05$
≤ 4 days	50% ($n=15$)	81% ($n=13$)	14% ($n=2$)*	$P<0.01$
5–8 days	33% ($n=10$)	19% ($n=3$)	50% ($n=7$)*	$P<0.05$
> 8 days	17% ($n=5$)	–	36% ($n=5$)*	$P<0.001$

*Significance between steroid therapy versus non-steroid therapy

Table 4. Factors influencing graft nephrectomy. *KT* kidney transplantation, *Latency of embolisation* time between graft loss and renal embolisation, *Tx* nephrectomy

	Graft Tx $n=6$	No graft Tx $n=24$	P
Mean age (years)	34 ± 10	47 ± 16	NS
Sex (men/women)	67% ($n=4$)	58% ($n=14$)	NS
	33% ($n=2$)	42% ($n=10$)	
Renal location			
Right iliac	66% ($n=4$)	38% ($n=9$)	NS
Left iliac	17% ($n=1$)	50% ($n=12$)	NS
Lumbar region	17% ($n=1$)	12% ($n=3$)	NS
Duration of KT			
Primary non-function	–	4% ($n=1$)	NS
< 24	–	33% ($n=8$)	$P<0.05$
24–60	17% ($n=1$)	21% ($n=5$)	NS
60–120	83% ($n=5$)	25% ($n=6$)	$P<0.001$
> 120	–	17% ($n=4$)	$P<0.05$
Latency of embolisation*			
< 6 months	67% ($n=4$)	67% ($n=16$)	NS
> 6 months	33% ($n=2$)	33% ($n=8$)	NS
Collateral circulation	83% ($n=5$)	25% ($n=6$)	$P<0.001$
Embolisation of collateral in first arteriography	–	50% ($n=3$)	$P<0.05$
Steroid therapy	67% ($n=4$)	50% ($n=12$)	NS
Post-embolisation syndrome	67% ($n=4$)	42% ($n=10$)	NS

require transfusion. Post-embolisation syndrome, characterised mainly by fever, was observed in 14 cases (47%) with early onset, latency of 29 ± 13 h and duration of 4.2 ± 2.4 days. Infection was ruled out in all cases (Table 3). The initial treatment was symptomatic. More recently, we have introduced prophylactic therapy for post-embolisation syndrome, consisting of the administration of a short course of steroids immediately prior to embolisation and for 5–7 days afterward. With the use of this therapy, the incidence of post-embolisation syndrome has significantly decreased from 71% in the non-steroid era (10 out of 14 patients) to 25% in the steroid era (4 out of 16 patients) ($P<0.01$) (Table 3).

Table 4 shows the results of a comparison between the clinical and angiographic characteristics of the group that required graft nephrectomy and the group that did not. Perirenal collateral circulation was found to be a risk factor for the reappearance of graft intolerance syndrome ($P<0.001$). In a retrospective analysis of the group requiring graft nephrectomy, perirenal collateral circulation, which could not be embolised in the initial procedure because of the small diameter of the blood vessels, was noted in five patients. The collateral circulation originated in the ileo-lumbar artery, circumflex iliac artery or fourth lumbar artery (Fig. 3). In these five patients, Doppler study showed no flow either in the renal artery or the intrarenal arteries, demonstrating that the vascular embolisation remained intact. Radionuclide angiography using [99mTc] MAG-3 renogram with a study of the vascular sequence (one image per s for 1 min) demonstrated the absence of renal perfusion in all these patients (photopenic image). However, a slight peripheral ring-shaped uptake was observed, suggesting perirenal perfusion through collateral circulation. A second arteriography performed in three of these five patients demonstrated extensive collateral perirenal circulation. An incomplete embolisation of this collateral circulation was carried out in two patients, but it proved unsuccessful. The duration of renal allograft function between 5 and 10 years was another factor favouring the reappearance of graft intolerance syndrome ($P<0.001$).

The improvement of post-embolisation syndrome with the use of steroid therapy has also significantly

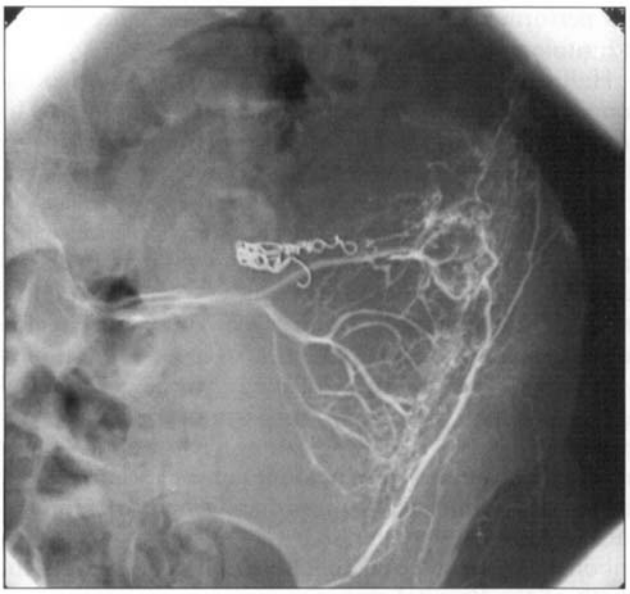

Fig. 3. Patient with relapse of clinical intolerance (the same patient as in Fig. 2). Angiography shows total occlusion of the main renal artery, with cortical repatency originating in the circumflex iliac artery and visualisation of venous return

reduced the length of hospitalisation. Now the average hospital stay is 3.6 ± 0.8 days. After steroids were introduced for this purpose, 81% of the patients were hospitalised for less than 4 days, as compared to 14% before steroid use ($P < 0.05$) (Table 3).

Discussion

Graft intolerance syndrome can occur in patients with a non-functioning renal allograft after immunosuppressive therapy has been discontinued. The incidence of GIS is not well documented. The development of this syndrome seems to depend on the duration of graft function and the speed with which steroids are withdrawn. It usually appears shortly after graft loss, although it may develop long after immunosuppressant therapy has been discontinued [3, 10, 14]. In our series, 13% of the cases occurred 1 year after renal failure. Fever was the constant clinical feature in all the patients, followed by graft pain and hematuria. The diagnosis was confirmed in the majority of our patients by positive uptake on 111In-labeled platelet scintigraphy [22]. The scan was negative in three patients, because they had received steroids shortly before the test was performed.

The criteria for nephrectomy of a non-functioning renal allograft are not well established [15, 18, 23]. At present, the absolute indications for surgical transplant removal are hyperacute rejection, irreversible arterial or venous thrombosis, kidney rupture, irreparable urological complications and neoplasm (de novo or transplanted) [4, 21]. In cases of asymptomatic non-functioning grafts, nephrectomy may not be necessary. The classic therapy for non-functioning renal allografts with clinical intolerance has been surgical nephrectomy. Some authors have indicated systematic preemptive nephrectomy for asymptomatic patients with early allograft failure (less than 1 year after transplantation) combined with the immediate withdrawal of immunosuppression, since they are much more likely to develop graft-related complications [23]. However, the surgical procedure can be very difficult because of the development of fibrosis around the kidney. Morbidity is frequent, with severe potential complications such as haemorrhage, haematoma, abscess, wound infection and vascular lesions. In late transplant nephrectomy, a subcapsular technique is recommended to reduce postoperative complications. Although the mortality related to transplant nephrectomy has decreased, it is still significant, with a frequency that varies from 0.7 to 5% [1, 3, 12, 24].

Complete transcatheter artery embolisation of native kidneys has been used in dialysis patients with refractory hypertension in the untreatable nephrotic syndrome and in non-operable renal carcinoma [6, 9, 17]. The application of this endovascular treatment in kidney transplantation has mainly been in selective arterial embolisation of post-biopsy arteriovenous fistulae or pseudoaneurysms [2, 8, 16, 20]. A case of selective arterial embolisation of a renal allograft to control haemorrhage secondary to percutaneous nephropyelostomy has also been described [5].

Our series demonstrated efficacy in 80% of the cases with no significant morbidity or mortality. The technique permits non-surgical ablation of a non-tolerated graft in a significant proportion of patients and avoids the potential complications of surgical nephrectomy. All the patients with early allograft loss presented a favourable evolution after embolisation. It should be remembered that nephrectomy is the classic treatment for patients presenting symptoms after the withdrawal of immunosuppression. Thus, surgical therapy can be avoided in eight out of ten patients with the use of embolisation.

Lorenzo et al. have published the only report in the literature of transcatheter artery embolisation of non-functioning renal allografts, in this case with absolute ethanol [13]. The results in this series of 14 patients were favourable, and there were no severe complications. However, only half of the patients were embolised with ethanol alone. In the others, the embolisation procedure was combined with the insertion of a stainless steel coil, which was left in the renal artery following ethanol injection. Thus, the sclerosing effect of ethanol may not be evaluated accurately in this series. The results from this work (79% efficacy) were similar to ours; however, the mean follow-up time was not reported, leaving the

question of long-term efficacy. Embolisation with ethanol is not without risk, since there is a danger of an inadvertent passage of ethanol into the bloodstream. The retrograde reflux of ethanol should be avoided through the use of a balloon occlusion catheter. A fatal outcome after ethanol renal ablation has been reported in a child with end-stage kidneys [7].

Our results indicate that collateral supply predisposes the patient to a recurrence of clinical intolerance. Careful embolisation of the collateral circulation is required to avoid the reappearance of graft intolerance. However, embolisation of collateral vessels is complex for two main reasons: (1) it is extremely difficult to locate the origin of the collateral supply (several projections and injections of contrast medium are needed for correct identification on angiography) and (2) the small diameter of the vessels makes superselective embolisation technically challenging. It is important to achieve the greatest possible distal diffusion of polyvinyl alcohol particles during embolisation for the complete occlusion of intrarenal distal circulation. This prevents collateral circulation from peripherally perfusing the renal allograft and will probably improve the embolisation results.

In the group that required graft nephrectomy, we observed a ring-shaped peripheral uptake on radionuclide imaging. The significance of this finding is unclear, but it might have been secondary to perirenal perfusion through collateral blood supply. However, in that case, the effect of collateral circulation was not uniform, since not all the patients with collateral blood supply showed a recurrence of GIS. The size and extension of the collateral circulation may be decisive factors in this regard. Moreover, there are no conclusive clinical data to predict which patients will likely do well with embolisation. Patients with early graft failure and those that develop late graft failure do not show clear differences. One might think that longer duration of the transplant would facilitate the development of collateral circulation and thus the recurrence of GIS. However, in four patients the renal transplant lasted 10 years before embolisation was performed, and they showed no recurrence of clinical intolerance after the procedure.

Half of our patients were affected by post-embolisation syndrome, a lower rate than that reported by Lorenzo et al. [13]. The clinical picture was mainly characterised by the development of fever 24 to 48 h after the procedure in the absence of any infectious disease. The pathogenesis and treatment of this syndrome have not been well documented. It is probably related to tissue necrosis with the release of cytokines and other inflammatory molecules. There was an important change in the frequency of post-embolisation syndrome over the period of study. Initially, the incidence was high, treatment was symptomatic and response was poor, with the prolonged duration of fever and hospitalisation. The introduction of a short course of steroids administered immediately before embolisation dramatically decreased the frequency of post-embolisation syndrome and considerably reduced the length of the hospital stay.

In summary, allograft renal artery embolisation was effective in a high percentage of patients, with no significant short- or long-term complications. This procedure, involving an angioradiological technique with local anaesthesia, avoids the potentially serious complications of more invasive surgical nephrectomy in dialysis patients, whose general condition is often poor. The presence of non-embolised collateral circulation seems to increase the risk of recurrence of graft intolerance syndrome treated by this method. We believe that future experience will confirm that vascular embolisation can be used as the treatment of choice for patients with non-functioning renal allograft and clinical signs of graft intolerance. Surgical graft removal would then be reserved for the relatively low percentage of patients with the reappearance of the intolerance syndrome.

In conclusion, renal artery embolisation is a simple, safe and effective technique for treating renal allograft intolerance syndrome and could be a feasible alternative for the first-line treatment.

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