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PERITONEAL DIALYSIS IS NOT A RISK FACTOR FOR PRIMARY VASCULAR GRAFT THROMBOSIS AFTER RENAL TRANSPLANTATION

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.*Objective:To* find out if patients undergoing peritoneal dialysis (PD) have an increased risk of primary vascular thrombosis of the renal allograft, compared with patients on hemodialysis (HD).
.*Design:* Observational, retrospective cohort study.
.*Setting:Tertiary* care hospital, covering an approximate population of 2000000. Extensive use of suboptimal donors for renal transplantation.
.*Patients and Methods:The* study included 827 patients receiving a cadaveric renal transplantation (RTx) in our center between 1988 and 1997 (700 on HD and 127 on PD). We searched for a potential difference in the incidence of graft thrombosis, according to the pretransplant dialysis modality and taking into consideration the main reported risk factors for this complication of RTx.
.*Results:* The accumulated incidence of primary graft thrombosis was 4.7% in PD patients, and 6.1% in HD patients (NS). Arterial and venous thrombosis were also similar in both groups. Logistic regression analysis demonstrated that extremes of age of the donor, use of the right kidney, protracted cold ischemia, delayed graft function, and transplantation to a hypersensitized recipient independently predicted graft thrombosis. Peritoneal dialysis was not independently associated with the complication under study (adjusted odds ratio HD/PD = 2.5, 95% CI = 0.8 7.7).
.*Conclusions:* Peritoneal dialysis is not associated with an increased risk of primary vascular thrombosis of the renal allograft.

KEY WORDS: Renal transplantation; graft thrombosis; hemodialysis.

It is generally accepted that the global results of renal transplantation (RTx) are similar in uremic patients, whether they have been previously treated with peritoneal dialysis (PD) or hemodialysis (HD) (1). Early reports ofpoor graft survival in PD patients (2) have not been confirmed (3-9). The incidence of acute rejection is similar in both populations, while it has been claimed that patients on PD present lower rates of delayed graft function (9,10). Peritonitis and catheter-related disorders may complicate the course ofRTx in PD patients, but they do not seem to have a detrimental effect on the overall results of therapy (1).

Vascular thrombosis (V cT) is one of the most feared early complications ofRTx (11), its incidence oscillating between 0.5 and 8% in different studies. It occurs predominantly during the first 2 weeks after RTx, resulting in graft failure in the vast majority of cases, although sporadic, but well documented, cases of graft salvage have been reported, especially after venous thrombosis (11). Graft thrombosis is a natural endpoint in the course of complications such as ongoing, irreversible acute rejection or renal artery stenosis (11,12), but may also present as a seemingly primary complication ofRTx. The list offactors that have been associated with an increased risk of primary V cT of the renal allograft is extensive, including cyclosporine (CsA) (13) and OKT3 (14) therapy, and also technical, immunological, and donor/recipient mismatch-related factors (Table 1) (15-18). The role ofhypercoagulability syndromes (19) in the pathogenesis of this complication has not yet been established, although it has been suggested that thrombophilia may predispose to early graft failure (20).

Recently, at least two studies have associated PD therapy with an increased risk of primary graft thrombosis after RTx (21,22). A preliminary survey, carried out in Spain in 1996 and specifically addressing this question (23), could not confirm this association. We have performed an in-depth analysis of the risk profile for early primary V cT of the renal allograft in a wide population transplanted in our center between 1988 and 1997, with the specific aim of assessing the potential relation between this complication and the pretransplant dialysis modality.

PATIENTS AND METHODS

The study population included 827 cadaveric RTx performed in our unit between January 1988 and July

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TABLE 1
Factors Previously Implicated in the Genesis of Primary Vascular Thrombosis of the Renal Allograft

Cyclosporine	Vascular reconstructive surgery
Antilymphocyte antibodies (OKT3)	Warm ischemia time
Pediatric donor	Cold ischemia time
Elderly donor	Diabetes of the recipient
Female donor (especially if male recipient)	Renal dysplasia as basal disease of the recipient (pediatric transplantation)
Donor hemodynamic instability	Thrombocytosis and hypercoagulability disorders of the recipient
Donor-recipient weight mismatch	Previous thrombotic events of the recipient
Use of the right kidney	Recipient hemodynamic instability soon after transplantation
Vascular anomalies of the graft	Acute tubular necrosis after transplantation

1997. Of these, 700 patients (84.6%) were treated with HD, and 127 (15.4%) with PD. Immunosuppression was based on CsA in all cases, although four different protocols were used during the study period: CsA + prednisone (Pred) (15.5%), CsA + Pred + azathioprine (66.3%), CsA + Pred + mycophenolate mofetil (10.5%), and OKT3 (5 mg/day for 10 days) + CsA + Pred + azathioprine (7. 7%). Our center takes extensive advantage of suboptimal donors, including non-heartbeating (12.6%), pediatric (5.4% 5 years old or younger), and elderly (9.1% older than 60 years) donors.

Primary V cT of the renal allograft was diagnosed according to standard criteria (11). Clinical evaluation, isotopic scan, duplex-Doppler sonography and, in some cases, arteriography were the usual steps in diagnosis. Surgical exploration was carried out as soon as a presumptive diagnosis was established. In spite of this, graft salvage was not possible in any instance, and graft nephrectomy specimens confirmed the di agnosis in all cases. We excluded from the analysis cases of definite secondary graft thrombosis (basically due to irreversible rejection), and also episodes occurring beyond the first month after RTx.

The following variables were considered for analysis:

Donor-related: age, sex, blood group, cause of death, hemodynamic instability, non-heartbeating, multiorgan involvement (versus renal), and serum creatinine.

Recipient-related: age, sex, blood group, basal disease, time on dialysis, residual diuresis, basal hypertension (antihypertensive drugs required), significant atherosclerosis (clinical diagnosis), pretransplant blood transfusions, degree of sensitization against the lymphocyte pannel (current), first versus second/third graft, and hematocrit, platelet count, and cholesterol and albumin values at the time ofRTx.

Transplant-related: cold and warm ischemia, vascular anomalies of the graft, need for vascular reconstructive surgery, use of right/left kidney, HLA (human leukocyte antigen) mismatches, immuno suppression schedule, preservation fluid, recipient instability during the peritransplant period [estimated by performance of a hemodialysis session immediately before RTx, peritransplant blood transfusion, first central venous and systolic blood pressures after RTx surgery, and severe complications (infections, bleeding) during the first week after RTx], immediate versus delayed graft function (dialysis required during the first week after RTx), and CsA profile during the first days after RTx, with special emphasis on delays in achieving therapeutic CsA levels after RTx surgery.

STATISTIC

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Univariate analysis was based on chi-square test, Student's t-test (two-tailed, unpaired), and the Mann-Whitney test. Independent predictors of primary V cT were identified using enter, forward, and stepwise logistic regression analysis. Dialysis modality was included in the general, best theoretical, and best-fit models, correcting for confounding variables and significant interactions. Risk measures are presented as odds ratios (OR), with 95% confidence intervals (CI). The SPSS 6.1.3 software (SPSS Inc, Chicago, IL, U.S.A.) was used for analysis.

RESULTS

There were 49 episodes of primary V cT of the renal allograft (accumulated incidence 5.9%). Thrombosis was arterial in 19 cases, venous in 28 cases, and arterial and venous in 2 cases. As previously stated, graft nephrectomy specimens confirmed the diagnosis of V cT in all cases. However, in 13 cases associated rejection could not be positively excluded, due to massive graft infarction and, in 2 other cases, findings were equivocal, disclosing mild signs ofrejection, which were considered not to be severe enough to establish a diagnosis of immunologic V cT.

Graft thrombosis was diagnosed a mean of 6.1 days after RTx (range 1 30), 41% of the episodes occur

ring during the first 3 days, and 90% during the first 10 days, after surgery. Arterial thrombosis occurred later than venous thrombosis (mean 7 vs 5 days, p = 0.015, Mann-Whitney). Twenty-four patients suffering V cT were retransplanted later, and the complication recurred in only one case (4.2%).

The accumulated incidence ofVcT was 4.7% in PD patients (n = 6), and 6.1% in HD patients (n = 43) (NS). Also, there was no significant difference in the incidence of arterial (2.3% PD vs 2.3% HD) or venous thrombosis (2.4% PD vs 3.6% HD) between both groups.

Table 2 shows the main differences between PD and HD patients, regarding the variables studied. Table 3 presents the main results of the univariate analysis of risk factors for the complication under study.

There were 49 patients on PD (39%) who did not present immediate graft function; 25 of these (51 %) were treated with HD, and 20 (41 %) with PD, during the first days after RTx, while in 4 cases (8%) irreversible graft failure was diagnosed before posttransplant dialysis was initiated. Graft V ct occurred in 4 patients (16%) while being treated with HD, in 1 while being treated with PD (5%), and in 1 before any dialysis was performed (NS). No instance ofVcT affected initially functioning grafts in the PD group.

Multivariate analysis (Table 4) confirmed previous reports on the association of graft thrombosis with factors such as extremes of age of the donor, use of the right kidney, protracted cold ischemia, or delayed graft function. The excess risk detected in hypersensitized recipients may indicate a role for undetected rejection in some cases of apparently primary V cT.

TABLE 2
General Variable Profile in Peritoneal Dialysis (PD) and Hemodialysis (HD) Patients
(Only significant or almost significant differences are presented.)

	PD (n=127)	HD (n=700)	p Value
Recipient's age (years)	42	45	0.07
Recipient's sex (% males)	54	62	0.06
Recipient's diabetes (%)	11	4	0.02
Time on dialysis (months)	24	41	0.001
Pretransplant blood transfusions (units)	3	6	0.001
Previous renal transplant (%)	8	17	0.01
Residual diuresis (mL/day)	766	412	0.001
HLA-A mismatches	1.4	1.2	0.03
Basal albumin (g/L)	33	36	0.005
Basal cholesterol (mg/dL)	173	147	0.001
HD before RTx surgery (%)	15	72	0.001
Immediate graft function (%)	61	44	0.001

RTx = renal transplantation.

Pretransplant poly transfusion and mycophenolate mofetil-based immunosuppression did not reach statistical significance as independent predictors of VcT, but were maintained in the final equation as confounding variables because they improved the predictive power of the model and influenced the regression coefficient of the study variable (dialysis modality). Most importantly for this study, PD was not an independent predictor of graft thrombosis. In fact, the risk was somewhat, albeit not significantly, higher in HD patients.

DISCUSSION

The results of our study do not support those of Murphy *et al.* (21) or van der Vliet *et al.* (22). Although we are not aware of other studies specifically addressing this question, other groups have found a similar risk of graft thrombosis in patients treated with PD and HD (18). The reasons for this discrepancy are not clear. The study by Murphy *et al.* (21) was carried out on a relatively small population, and a multivariate statistical approach was not attempted, which limits the reliability of their conclusions. Sample size and statistic methodology provide more consistent support to the results ofvan der Vliet *et al.* (22), although it is surprising that pretransplant PD therapy was the only independent predictor of graft V cT. Our study has taken into consideration all factors commonly considered to playa potential pathogenic role in graft thrombosis (Table 1), and the multivariate approach did not detect even a minor trend to a higher incidence ofVcT in our patients on PD.

Frequent transfusions before RTx exerted some protection against graft VcT in our study (Table 3). This may reflect a potential role for concealed rejection in the genesis of some cases of so-called primary V cT of the renal allograft. As patients on HD were more frequently transfused (Table 2), this factor may have provided some protection against V cT in this group. However, the magnitude of the effect is probably small, and the introduction of erythropoietin therapy has minimized the differences in the transfusion rates between PD and HD patients. On the other hand, and in agreement with previous reports (18), we found that delayed graft function was strongly associated with graft V cT. Peritoneal dialysis has been associated with a lower incidence of acute renal failure after RTx compared with HD (Table 2) (9,10). This could confer on PD patients some advantage against VcT. However, the pathogenic significance of this factor is unclear because the relationship between delayed graft function and V cT may be bidirectional. (Delayed function could favor graft thrombosis, but the latter may also be a cause of acute renal failure after RTx.)

TABLE 3
Risk Profile for Primary Graft Thrombosis. Univariate
(Only significant or almost significant differences are presented.)

	Thrombosis	No thrombosis	p Value
Donor <6 years (%)	19	5	0.001
Donor >60 years (%)	21	8	0.001
Donor serum creatinine (mg/dL)	0.9 (SD 0.3)	1.0 (SD 0.4)	0.05
Time on dialysis (months)	30 (SD 31)	39 (SD 41)	0.07
Recipient's hypertension (%)	51	64	0.07
Pretransplant blood transfusions (units)	3 (SD 5)	6 (SD 8)	0.002
Hypersensitized recipient (%)	14	6	0.02
Right kidney (%)	67	46	0.01
Cold ischemia time (hours)	23 (SD 6)	21 (SD 6)	0.002
Immunosuppression			0.05
CsA + prednisone	4	16	
CsA + prednisone + MMF	16	10	
Immediate graft function (%)	6	49	0.0005
Delay to first in-range CsA (days)	4.1 (SD 1.7)	6.1 (SD 3.0)	0.02
Basal albumin (g/L)	32 (SD 4)	34 (SD 4)	0.04
Basal cholesterol (mg/dL)	132 (SD 30)	151 (SD 42)	0.005

CsA = cyclosporine; MMF = mycophenolate mofetil; SD = standard deviation.

TABLE 4
Risk Profile for Primary Graft Thrombosis. Multivariate

	Odds ratio	CI 95% Odds ratio	p Value
Donor age (21–50 years)			0.007
< 6 years	8.4	2.4–29.1	
6–20 years	2.5	0.9–6.6	
51–60 years	2.4	0.8–7.0	
>60 years	4.4	1.6–12.2	
Immunosuppression schedule (double)			0.07
CsA + Aza + Pred	4.1	0.5–32.8	
CsA + MMF + Pred	12.1	1.3–112.1	
OKT3 + CsA + Aza + Pred	3.5	0.4–33.8	
Hypersensitized recipient (N)	6.7	2.2–20.5	0.001
Blood transfusions (0)			0.09
1–5 units	1.2	0.5–2.7	
>5 units	0.4	0.1–1.1	
Kidney used (left)	1.9	1.04–3.7	0.05
Cold ischemia time (<20 hours)			0.04
20–24 hours	1.1	0.5–2.5	
25–30 hours	1.0	0.4–2.5	
>30 hours	3.8	1.1–14.3	
Early graft function (immediate function)	14.6	4.2–51.4	0.0005
Dialysis modality (PD)	2.5	0.8–7.7	0.11

CsA = cyclosporine; Aza = azathioprine, Pred = prednisone; MMF = mycophenolate mofetil; PD = peritoneal dialysis.
Best fit model, including dialysis modality. Among brackets, reference category. Global significance $p < 0.0005$. 94.7% of cases correctly classified.

Why should PD therapy induce an increased risk of graft VcT? Nothing indicates that patients undergoing PD may receive a more thrombogenic immunosuppression schedule (with higher doses of CsA or OKT3) (13,14), or present more technical or donor-recipient mismatch problems, than their counterparts undergoing HD. On the other hand, even if we exclude from analysis graft thrombosis secondary to

acute rejection, immunologic events may yet playa role in this complication, as suggested by the association of seemingly primary VcT with factors such as pretransplant blood transfusions, hypersensitization, or retransplant (17, present study). However, it is now clear that, in disagreement with earlier reports (2), the incidence of acute rejection is not increased in patients on PD (1).

Does PD therapy result in a more thrombogenic hemostatic pattern than HD? Hemostasis is, seemingly, better preserved in PD than in HD patients. Peritoneal dialysis therapy has been classically associated with a lower spontaneous tendency to anemia and a higher degree of hemoconcentration, as also with higher blood levels of some procoagulant factors, such as fibrinogen and apolipoprotein (a), compared with HD (24). However, the real thrombogenic potential of these differences is unclear and, moreover, some of them may have been eliminated or minimized by the introduction of erythropoietin therapy (25). Furthermore, blood levels of natural coagulation inhibitors may be higher in PD patients than in HD patients (26,27) leading, theoretically, to a lower risk of thrombosis in the former. Overall, there is no evidence linking PD to an increased incidence of thrombotic events, compared with HD.

We are not aware of studies showing differences in the coagulation patterns of PD and HD patients after RTx. Even if there were differences, the post transplant dialysis technique (in the case of delayed graft function) could have at least as much impact on the post transplant hemostatic pattern (and, eventually, on the risk of graft thrombosis) as did the pre transplant mode of treatment. Our results do not support this hypothesis; PD patients with delayed graft function and treated temporarily with HD showed a trend to suffer more V cT than those treated throughout with PD, although the difference was not significant.

Hypercoagulability syndromes are not rare in the general population, representing a significant risk factor for venous and arterial thrombosis (28). Factor V Leiden mutation is probably the most frequent of these disorders and has been implicated in the pathogenesis of native renal artery thrombosis (29). In dialysis patients, more attention has been given to anti cardiolipin antibodies. These are frequently present in certain diseases, potentially causing renal failure, particularly systemic lupus erythematosus, but have also been detected with increased frequency in the general population on dialysis (30). The presence of anticardiolipin antibodies has been associated with an increased risk of thrombosis of the vascular access for HD (31). The role ofhypercoagulability syndromes in the pathogenesis of renal allograft thrombosis, and their comparative prevalence in patients on PD and HD, have not yet been established. These points may deserve consideration, because recurrent vascular access thrombosis is a cause for switching from HD to PD, and thus patients with hypercoagulability disorders could be overrepresented in PD units. Given the small overall incidence of graft thrombosis after RTx, a minor difference in the prevalence of hypercoagulability syndromes between PD and HD could have a significant impact on the comparative incidence ofVcT.

In conclusion, our results do not support the contention that peritoneal dialysis is associated with an increased risk of primary vascular thrombosis of the renal allograft.

REFERENCES

1. Winchester JF, Rotellar C, Goggins M, Robino D, Alijani MR, Rakowski TA, *et al.* Transplantation in peritoneal dialysis and hemodialysis. *Kidney Int* 1993; 43(Suppl 40):101-5.
2. Guillou PJ, Will EJ, Davison AM, Giles GR. CAPD -A risk factor in renal transplantation? *Br J Surg* 1984; 71:878-80.
3. Donnelly PK, Lennard TW, Proud G, Taylor RMR, Henderson R, Fletcher K, *et al.* Continuous ambulatory peritoneal dialysis and renal transplantation: a five-year experience. *Br Med J* 1985; 291:1001-4.
4. Evangelista JB, Bennett-Jones D, Cameron JS, Williams DG, Taube DH, Neild G, *et al.* Renal transplantation in patients treated with hemodialysis and short term and long term CAPD. *Br Med J* 1985; 291:1004 7.
5. Diaz-Buxo JA, Walker PJ, Burgess WP, Farmer CD, Chandler JT, Faircloth W, *et al.* The influence ofperitoneal dialysis on the outcome of transplantation. *Int J Artif Organs* 1986; 9:359-62.
6. Heyka R, Schreiber MJ, Steinmuller DR, Novick AC, Streem SB, Cunningham RJ, *et al.* Renal transplantation in patients on CAPD. In: Khanna R, Nolph KD, Prowant BF, Twardowski ZJ, Oreopoulos DG, eds. Advances in CAPD. Toronto: Peritoneal Dialysis Bulletin, 1987; 3:49-55.
7. O'Donoghue D, ManosJ, PearsonR, ScottP, BakranA, Johnson R, *et al.* CAPD and renal transplantation: a ten-year experience in a single center. *Perit Dial Int* 1992; 12:242-9.
8. Kyllönen L, Helanterä A, Salmela K, Ahonen J. Dialysis method and kidney graft survival. *Transplant Proc* 1992; 24:354.
9. Perez Fontán M, Rodríguez-Carmona A, Falcon TG, Moncalián J, Oliver J, Valdés F. Renal transplantation in patients undergoing chronic peritoneal dialysis. *Perit Dial Int* 1996; 16:48-51.
10. Lambert MC, Bernaert P, Vijt D, de Smet R, Lameire N. CAPD -Arisk factor in renal transplantation? ARF after transplantation. *Perit Dial Int* 1996; 16(Suppl 1):495-8.
11. Gray DWR. Vascular and lymphatic complications af ter renal transplantation. In: Morris PJ, ed. Kidney

transplantation. Philadelphia: WB Saunders, 1994:314-29.
12. Kooijmans-Coutinho MF, Bruijn JA, Hermans J , Schindler R, Frei U, Schrama E, *et d.* Interstitial rejection, vascular rejection and diffuse thrombosis of renal allografts. Predisposing factors, histology, immunohistochemistry and relation to outcome. *Transplantation* 1996; 61:1338-44.
13. Remuzzi G, Bertani T. Renal vascular and thrombotic effects of cyclosporine. *Am J Kidney Dis* 1989; 13:261-72.
14. Abramowicz D, Goldman M, Mat O, Estermans G, CrusiauxA, Vanherweghem JL, *et al.* OKT3 serum levels as a guide for prophylactic therapy: A pilot study in kidney transplant recipients. *Transpl Int* 1994; 7:258-63.
15. Brunkwall J, Bergqvist D, Bergentz SE, Bornmyr S, Husberg B. Postoperative deep venous thrombosis after renal transplantation. *Transplantation* 1987; 43:647-9.
16. Penny MJ, N ankivell BJ, Disney APS, Byth K, Chapman JR. Renal graft thrombosis. *Transplantation* 1994; 58:565-9.
17. SinghA, Stablein D, TejaniA. Risk factors for vascular thrombosis in pediatric renal transplantation. *Transplantation* 1997; 63:1263-7.
18. Bakir N, Sluiter WJ, Ploeg RJ, van Son WJ, Tegzess AM. Primary renal allograft thrombosis. *Nephrol Dial Transplant* 1996; 11:140-7.
19. Thomas DP, Roberts HR. Hypercoagulability in venous and arterial thrombosis. *Ann Intern Med* 1997; 126:638-44.
20. Fischereder M, Göhring P, Schneeberger H, Lohse P, von Appen K, Samtleben W, *et al.* Early loss of renal transplants in patients with thrombophilia (Abstract). *J Am Soc Nephrol1997;* 8:682A.
21. Murphy BG, Hill CM, Douglas JF, McNamee PT. Increased renal allograft thrombosis in CAPD patients. *Nephrol Dial Transplant* 1994; 9:1166-9.
22. van der Vliet JA, Barendregt WB, HoitsmaAJ , Buskens FGM. Increased incidence of renal allograft thrombosis after continuous ambulatory peritoneal dialysis. *Clin Transpl1996;* 10:51-4.
23. Escuín F, del Peso G, perez Fontán M, Carmona AR, Martinez A, Lanuza M, *et al.* A comparative survey of the incidence of kidney graft primary vascular thrombosis among CAPD and haemodialysis patients (Letter). *Nephrol Dial Transplant* 1996; 11:1896-7.
24. Sagripanti A, Barsotti G. Bleeding and thrombosis in chronic uremia. *Nephron* 1997; 75:125-39.
25. Eschbach JW. Hematological problems ofrenal failure. In: Jacobs J, Kjellstrand CM, Koch KM, and Winchester JF, eds. Replacement of renal function by dialysis. Dordrecht: Kluwer Academic, 1996:1059 76.
26. Vaziri ND, Shah GM, Winer RL, Gonzales E, Patel B, Alikhani S, *et al.* Coagulation cascade, fibrinolytic system antithrombin III, protein C and protein S in patients maintained on continuous ambulatory peritonal dialysis. *Thromb Res* 1989; 53:173-80.
27. Bertoli SV, di Belgiojoso GB, Trezzi M, Scorza D, Borone MT, Poggio M, *et al.* Reduced blood levels of coagulation inhibitors in chronic hemodialysis compared with CAPD. In: Khanna R, ed. Advances in peritoneal dialysis. Toronto: Peritoneal Dialysis Publications, 1995; 11:127-30.
28. Thomas DP, Roberts HR. Hypercoagulability in venous and arterial thrombosis. *Ann Intern Med* 1997; 126:638-44.
29. Le Moine A, Chauveau D, Grünfeld JP. Acute renal artery thrombosis associated with factor V Leiden mutation. *Nephrol Dial Transplant* 1996; 11:2067-9.
30. García-Martín F, de Arriba G, Carrascosa T, Moldenhauer F, Martín-Escobar E, Val J, *et al.* Anticardiolipin antibodies and lupus anticoagulant in endstage renal disease. *Nephrol Dial Transplant* 1991; 6:543 7.
31. Prakash R, Miller CC, Suki WN. Anticardiolipin antibody in patients on maintenance hemodialysis and its association with recurrent arteriovenous graft thrombosis. *Am J Kidney Dis* 1995; 26:347-52.