Predictors of Arteriovenous Graft Patency After Radiologic Intervention in Hemodialysis Patients

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• **Arteriovenous grafts in hemodialysis patients are prone to recurrent stenosis and thrombosis, requiring frequent radiologic and surgical interventions to optimize their long-term patency. Little is known about the factors that determine graft outcome after a radiologic intervention. The present study examined the clinical and radiologic predictors of intervention-free graft survival after elective angioplasty or thrombectomy. A prospective computerized database was used to determine the outcomes subsequent to all graft angioplasties (n = 330) and thrombectomies (n = 326) performed at the University of Alabama at Birmingham between April 1, 1996, and June 30, 1999. Primary graft survival rates after angioplasty and thrombectomy were 86% versus 43% at 1 month, 71% versus 30% at 3 months, 51% versus 19% at 6 months, and 28% versus 8% at 12 months, respectively. The median intervention-free graft survival time was substantially longer after angioplasty than thrombectomy (6.7 versus 0.6 months; *P* < 0.001). The superior outcome of angioplasty over thrombectomy was observed even for the subset of procedures with no residual stenosis (median survival, 6.9 versus 2.5 months; *P* < 0.001). The median graft survival was inversely related to the magnitude of residual stenosis for both elective angioplasty and thrombectomy. Median intervention-free graft survival after angioplasty was inversely related to the postangioplasty intragraft to systemic systolic pressure ratio (7.6, 6.9, and 5.6 months for ratios <0.4, 0.4 to 0.6, and >0.6, respectively; *P* < 0.001). Intervention-free graft survival after angioplasty or thrombectomy was not affected by graft location (forearm versus upper arm), number of stenotic sites, or presence of diabetes. In conclusion, graft survival is substantially longer after elective angioplasty than thrombectomy, even when the radiologic appearance after the procedure suggests complete resolution of the stenotic lesion. Moreover, the risk for requiring a subsequent graft intervention can be predicted from two simple radiologic measurements: grade of stenosis and intragraft to systemic systolic blood pressure ratio. These parameters may help determine the frequency of monitoring for recurrent stenosis in a given graft.**

• **INDEX WORDS: Hemodialysis (HD); angioaccess; arteriovenous graft; angioplasty; thrombectomy.**

MAINTAINING patent vascular access is critical for providing adequate hemodialysis to patients with end-stage renal disease. The National Kidney Foundation-Dialysis Outcomes Quality Initiative guidelines on vascular access recommend placing a native arteriovenous fistula, with a polytetrafluoroethylene (PTFE) graft reserved as an alternative in patients in whom vascular anatomy is not suitable for fistula creation.[1] Despite these recommendations, approximately 60% of the hemodialysis patients in the United States use grafts rather than fistulas.[2] PTFE dialysis grafts are more prone than fistulas to recurrent stenosis, thrombosis, and infection and have decreased survival.[3-7] The major cause of graft thrombosis is the development of critical stenosis at the venous anastomosis, draining vein, or central vein.[8] Observational studies have found that correction of hemodynamically significant graft stenosis by angioplasty can substantially reduce the frequency of graft thrombosis.[9-12] This has highlighted the need for noninvasive surveillance methods that can be used on an ongoing basis to screen for hemodynamically significant stenosis, permitting timely referral for a fistulogram.

A number of monitoring methods have been validated as screens for graft stenosis, including serial measurements of dynamic dialysis venous pressure,[10] static intragraft venous pressure,[12] Doppler ultrasound measurements of peak systolic velocity,[13] and measurement of access blood flow.[14,15] At the University of Alabama at Birmingham (UAB), we have used aggressive clinical monitoring, including elevated dynamic dialysis venous pressures, prolonged bleeding times from needle sites, abnormal graft inspection or auscultation, and unexplained declines in Kt/V.[9]

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Received November 9, 2000; accepted in revised form January 26, 2001.

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0272-6386/01/3705-0009$35.00/0
doi:10.1053/ajkd.2001.23615

Referral of dialysis patients with any one of these abnormalities for a fistulogram resulted in a 60% decrease in graft thrombosis compared with our historical controls.[9]

When graft stenosis is missed despite ongoing surveillance, the graft usually clots. The patency of such grafts can frequently be restored radiologically by a combination of angioplasty of the stenotic lesion in conjunction with pharmacomechanical or mechanical thrombolysis.[16-20] If the angioplasty is not successful or the graft rethromboses within a brief period, the patient is typically referred for surgical revision and thrombectomy of the graft. When the latter intervention fails, patients typically require placement of a new vascular access at a different anatomic location.

Because graft stenosis is a recurrent phenomenon, dialysis patients typically require multiple radiologic or surgical interventions over time to maintain long-term graft patency.[8,21-23] However, the frequency of graft intervention varies markedly among hemodialysis patients. Little is known about the clinical or radiologic factors that predispose patients to accelerated recurrent stenosis and thus to more frequent salvage procedures. A better understanding of the factors that affect the duration of intervention-free graft survival after a radiologic intervention may be useful in stratifying patients into different risk groups. Such an approach may result in prophylactic intervention in high-risk patients or decreased frequency of monitoring for recurrent graft stenosis among low-risk patients.

In the current study, we analyzed intervention-free graft survival after radiologic graft interventions among all hemodialysis patients at a single institution. The predictive value of several clinical and radiologic factors on subsequent graft survival was evaluated. In addition to the standard items included in a radiologic evaluation, we also measured intragraft to systemic systolic pressure ratio. This ratio, which is equivalent to static intragraft venous pressure, has been reported to correlate with hemodynamically significant graft stenosis.[12,24]

METHODS

Patient Population

UAB provides chronic dialysis to approximately 500 patients, of whom 85% receive in-center hemodialysis. The total number of dialysis patients followed up by our medical center has increased by approximately 5% annually. There are seven outpatient dialysis units, including a hospital-based dialysis unit and six satellite units. The demographics of the patient population are as follows: 28% of the patients are aged 65 years or older, 49% are women, 82% are black, 18% are white, and 37% have diabetes mellitus. As of January 1998, approximately 26% of the hemodialysis patients were dialyzing with arteriovenous fistulas, 60% were dialyzing with PTFE arteriovenous grafts, and 14% used temporary dialysis catheters. More than 95% of the grafts had a loop configuration. The medical care of these patients is provided by eight clinical nephrologists, all full-time UAB faculty in the Division of Nephrology. All patient hospitalizations, surgical procedures, and radiologic procedures are performed at UAB Hospital. Dialysis access procedures are performed by the renal transplant surgeons. Members of the Division of Interventional Radiology perform radiologic diagnostic tests and interventions for vascular access.

Screening for Graft Stenosis

To increase the longevity of dialysis grafts, an aggressive clinical monitoring protocol for early detection of graft stenosis was established at our dialysis units.[9] Grafts were referred for a fistulogram with possible angioplasty if any one of the following abnormalities was noted: (1) abnormal graft inspection or auscultation (before each dialysis session, nurses checked the graft for the presence of a thrill, distal edema, a high-pitched bruit, or a discontinuous bruit); (2) prolonged bleeding from the graft needle sites (>30 minutes on two of three consecutive dialysis sessions); (3) persistent elevation of dynamic dialysis venous pressures at a low blood flow, described by Schwab et al[10]; or (4) an unexplained decline in Kt/V (>0.20 units on 2 consecutive months). We previously reported that the aggressive clinical monitoring program decreased graft thrombosis by 60%.[9]

Radiologic Management of Graft Stenosis

A 4 F or 5 F Microstick system (Cook Inc, Bloomington, IN) was used to access the graft, most commonly in the midgraft or venous limb. After injection of contrast into the venous limb, the graft, outflow veins, and central veins were imaged using rapid-sequence digital subtraction angiography. The arterial limb and arterial anastomosis were visualized by manual compression of the graft during injection of contrast. Angioplasty was performed on all lesions with a 50% or greater stenosis because these were considered hemodynamically significant.[12] A 6 F or 7 F vascular sheath was placed at the angiographic puncture site, and 2,000 to 5,000 U of heparin was injected directly into the graft. The area of stenosis or occlusion was crossed using a 4 F or 5 F catheter and an angled hydrophilic guidewire. An appropriate balloon catheter dimension was chosen based on the area to be treated (most commonly a 7-mm × 4-cm Tru Trac balloon; Bard Urological, Covington, GA). The balloon was placed across the lesion and inflated until there was no remaining waist or the maximal rated pressure for the balloon was reached (usually 14 to 15 atm). If necessary, balloon angioplasty was repeated using a larger diameter or

a high-pressure balloon. The balloon catheter was then withdrawn with the guidewire left in place, and repeated imaging was performed by injection of contrast through the sheath. The procedure was ended when further balloon dilatations did not reduce the stenosis further.

The radiologist estimated the grade of the stenotic lesion visually by comparing the area of greatest narrowing to the adjacent normal graft or blood vessel. The radiologist documented the location and visual grade of stenosis before and after angioplasty. Each stenotic lesion was graded semiquantitatively from 1 to 5 (1 = no [<10%] stenosis; 2 = mild [10% to 49%] stenosis; 3 = moderate [50% to 69%] stenosis; 4 = severe [70% to 99%] stenosis, and 5 = total [100%] occlusion) on the basis of visual inspection by the radiologist before and after angioplasty. If there were two or more coexisting stenotic lesions, the highest grade of stenosis was recorded.

In late 1997, the radiologists also began to record intragraft and systemic systolic blood pressures before and after the angioplasty. The rationale for obtaining these pressure ratios was as follows. In grafts without stenosis, approximately 60% of the systemic pressure is lost between the artery and the venous end of the graft, such that the intragraft pressure is approximately 40% of the systemic pressure.[24] Moreover, an intragraft pressure ratio greater than 0.4 is predictive of high-grade graft stenosis by angiography.[12] The intragraft systolic pressure was measured in the venous limb with an electronic transducer (Witt Biomedical Corp, Melbourne, FL), and the systemic systolic pressure was measured with an automated blood pressure cuff. Although the pressures were measured and recorded during the radiologic procedures, the pressure ratios were not used prospectively to determine the need for future interventions.

Radiologic Management of Graft Thrombosis

Grafts that clotted despite aggressive clinical monitoring were referred to Interventional Radiology for thrombectomy and angioplasty. Venous outflow was imaged initially. If venous outflow was so compromised as to preclude successful thrombectomy, a dialysis catheter was placed and the patient was referred for surgical revision. If the graft was amenable to thrombectomy, angioplasty of the venous anastomosis and other visualized venous outflow lesions was performed. The thrombectomy was achieved by mechanical or pharmacomechanical thrombolysis, involving a combination of clot dissolution with urokinase and mechanical disruption of the thrombus.[16-20] The procedure was considered complete when all the thrombus was removed, a palpable thrill was present throughout the graft, and all significant lesions were treated. Failure of the declotting procedure was almost always caused by the inability to correct an underlying stenosis. Intragraft pressures were not measured after graft thrombectomy.

Data Analysis

To optimize the management of vascular access in hemodialysis patients, we instituted a multidisciplinary team approach in April 1996, including nephrologists, radiologists, and vascular surgeons.[9] A full-time dialysis access coordinator (D.C.) scheduled all vascular access procedures with Interventional Radiology and Renal Transplant Surgery and maintained a prospective computerized record of all the procedures. The prospective vascular access database was used to evaluate the outcomes of all grafts undergoing either angioplasty or thrombectomy during the period from April 1, 1996, to June 30, 1999. The subsequent outcome of each graft was determined from the database. Intervention-free graft survival was defined as the period from the angioplasty or declotting to the date of the next graft intervention (declotting, angioplasty, or surgical revision). Fistulograms not accompanied by an angioplasty were not considered a graft event for the purpose of this analysis.

The radiology procedure reports were reviewed retrospectively to obtain the following information: number of stenotic sites, location of stenosis, grade of stenosis, and intragraft to systemic systolic pressure ratio. Patient events, including death, transplantation, or transfer to another dialysis facility, were obtained from the medical records, dialysis computer database, dialysis units, or family contact.

Statistical Analysis

Survival analysis techniques were used to model the time until graft failure. The graft outcomes were censored for patient death, transplantation, or transfer to a nonparticipating dialysis unit. Functioning grafts were censored for the date of analysis (September 30, 1999). Univariate Cox proportional hazard models were used to evaluate the significance of the independent variables in predicting intervention-free graft survival. Hazard ratios for significant terms were calculated with 95% confidence intervals. Survival distributions were plotted using the Kaplan-Meier method.

RESULTS

During the 39-month period from April 1, 1996, to June 30, 1999, a total of 330 angioplasties were performed in patients referred for radiologic evaluation because of suspected graft stenosis based on aggressive clinical monitoring. In addition, 326 radiologic thrombectomies were performed in grafts that clotted before an elective intervention despite the surveillance program. Approximately 75% of the stenoses were at either the venous anastomosis or the draining vein (Table 1). A smaller number of stenoses were intragraft or in the central vein, and rarely, a stenosis was found at the arterial anastomosis. In 60% to 70% of cases, there was a single stenotic lesion, and in 30% to 35%, there were two discrete stenotic lesions. Three separate stenotic lesions were observed rarely.

Subsequent graft outcomes were compared between patent grafts that underwent elective angioplasty of a stenotic lesion and grafts that were already clotted and underwent thrombectomy with concomitant angioplasty. Primary graft survival rates after angioplasty and thrombec-

Table 1. Location and Number of Graft Stenoses

	Angioplasty	Thrombectomy
Site of stenosis* (%)		
Venous anastomosis	55.2	59.7
Venous outlet	21.5	14.2
Central vein	14.9	9.0
Intragraft	6.0	10.4
Arterial anastomosis	2.4	6.7
No. of stenotic sites† (%)		
1	70.2	59.8
2	28.2	34.8
3	1.6	5.4

*Percent of all stenotic sites.
†Percent of all grafts.

tomy were 86% versus 43% at 1 month, 71% versus 30% at 3 months, 51% versus 19% at 6 months, and 28% versus 8% at 12 months, respectively (Fig 1). The median intervention-free graft survival time was substantially longer after elective angioplasty than after thrombectomy (6.7 versus 0.6 months; $P < 0.001$). When the comparison was restricted to those radiologic procedures in which there was no residual stenosis after the intervention, graft survival was still substantially longer after elective angioplasty than thrombectomy (6.9 versus 2.5 months; $P < 0.001$; Fig 2).

The magnitude of graft stenosis decreased significantly after both elective angioplasty and thrombectomy (Table 2). Intervention-free graft survival was inversely related to the magnitude of residual stenosis after the intervention for both elective angioplasty and thrombectomy. Specifically, median graft survival was longer postangioplasty when there was no residual stenosis than if there was any degree of residual stenosis (6.9 versus 4.6 months; $P < 0.001$; Fig 3). Similarly, median graft survival after thrombectomy was 2.5 months when there was no residual stenosis, 1.6 months when there was mild residual stenosis, and 0.3 months when there was moderate to severe residual stenosis ($P < 0.05$; Fig 4).

The intragraft to systemic systolic pressure ratio could be calculated from the information available in the reports of 179 graft angioplasties. The mean pressure ratio decreased significantly after angioplasty of the stenotic lesions (Table 2). The postangioplasty pressure ratio was less than 0.4 in 50% of the procedures, between 0.4 and 0.6 in 36% of the procedures, and greater than 0.6 in 14% of the procedures. Median intervention-free graft survival was inversely related to the postangioplasty pressure ratio (7.6, 6.9, and 5.6 months for ratios <0.4, 0.4 to 0.6, and >0.6, respectively; $P < 0.001$; Fig 5). Graft survival curves after angioplasty did not differ

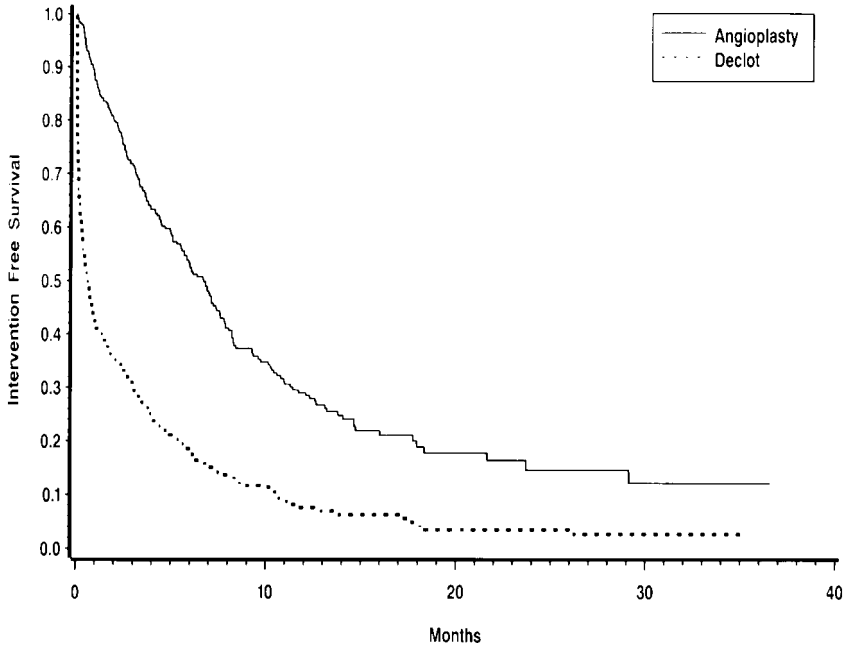

Fig 1. Intervention-free graft survival after elective angioplasty (solid line) or thrombectomy plus angioplasty (dotted line). Graft survival was calculated from the date of the initial intervention to the date of the next intervention (angioplasty, declotting, or surgical revision). $P < 0.001$ for the comparison between the two groups.

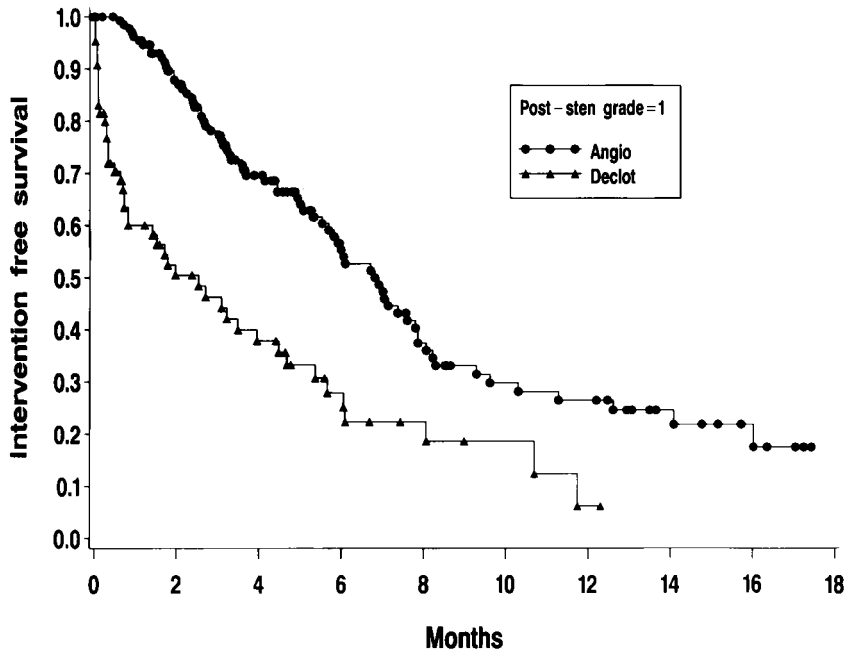

Fig 2. Intervention-free graft survival after elective angioplasty (Angio) or thrombectomy plus angioplasty (Declot) in the subset of procedures with no residual stenosis. Graft survival was calculated from the date of the initial intervention to the date of the next intervention (angioplasty, declotting, or surgical revision). *P* < 0.001 for the comparison between the two groups.

significantly between grafts in which pressure measurements were obtained and those in which pressures were not measured.

The intervention-free graft survival after angioplasty was not significantly associated with the location of the graft (forearm versus upper arm), number of stenotic sites, or presence of diabetes (Table 3). Similarly, graft outcome after thrombectomy was not related to graft location, number of stenotic sites, or diabetic status.

DISCUSSION

The recurrent nature of dialysis graft stenosis has been observed repeatedly and is the primary cause of graft thrombosis.[8] Primary (intervention-free) graft survival rates after angioplasty have been reported to range from 53% to 85% at 3 months and 27% to 65% at 6 months.[21,22,25,26] Conversely, primary graft survival rates after thrombectomy ranged from 35% to 58% at 3 months and 11% to 39% at 6 months.[16-19,27,28] Whereas comparison of graft outcomes among various series after thrombectomy or elective angioplasty suggests shorter survival after thrombectomy, these comparisons are limited by the potential differences in patient populations, criteria for referral, and radiologic techniques. The present study offers a direct comparison of graft outcomes after the two radiologic procedures performed during the same period in dialysis patients at a single clinical center by a small number of experienced interventional radiologists and using well-defined criteria for screening for graft stenosis and referral for radiologic procedures. The results clearly show a much shorter median intervention-free graft survival after thrombectomy compared with angioplasty (Fig 1). Primary graft survival rates at 3 months were 30% after thrombectomy versus 71% after elective angioplasty. Similarly, intervention-free graft survival rates at 6 months were 19% after declotting versus 51% after elective angioplasty. Importantly, even in the subset of cases in which

Table 2. Effect of Radiologic Graft Procedures on Graft Stenosis and Intragraft Pressure Ratio

	Preprocedure	Postprocedure	*P*
Degree of stenosis*			
Angioplasty	3.4 ± 0.7	1.6 ± 0.8	<0.001
Thrombectomy	4.0 ± 0.7	1.8 ± 0.9	<0.001
Pressure ratio†			
Angioplasty	0.59 ± 0.24	0.41 ± 0.18	<0.001
Thrombectomy	—	N/A	

NOTE. Values expressed as mean ± SD.

Abbreviation: N/A, not available.

*Degree of stenosis was estimated on a semiquantitative scale from 1 to 5 (see Methods).

†Ratio of intragraft to systemic systolic pressure measured during the radiologic procedure.

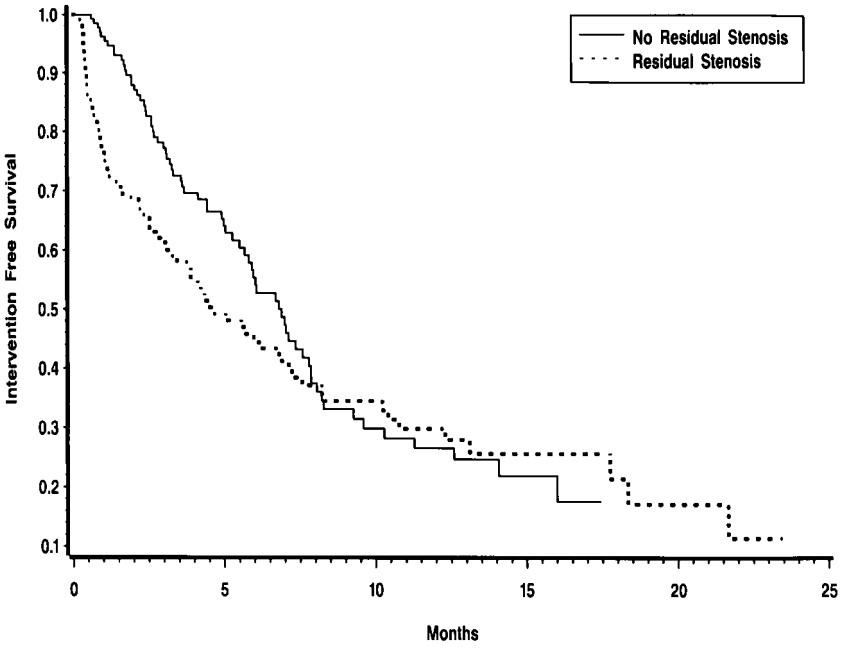

Fig 3. Intervention-free graft survival after elective angioplasty for procedures in which there was no residual stenosis and residual stenosis. Graft survival was calculated from the date of the initial intervention to the date of the next intervention (angioplasty, declotting, or surgical revision). $P < 0.001$ for the comparison between the two groups.

there appeared to be complete radiologic resolution of the stenosis, subsequent graft survival was still substantially shorter after thrombectomy than angioplasty (Fig 2). The discrepancy in outcomes after the two radiologic interventions highlights the critical importance of prospective noninvasive monitoring of grafts for stenosis and elective referral of suspicious grafts for fistulogram with possible angioplasty.

In the current study, we evaluated the value of a number of clinical and radiologic parameters in predicting intervention-free graft survival subsequent to a radiologic intervention. Our retrospective analysis found that the visual grade of graft

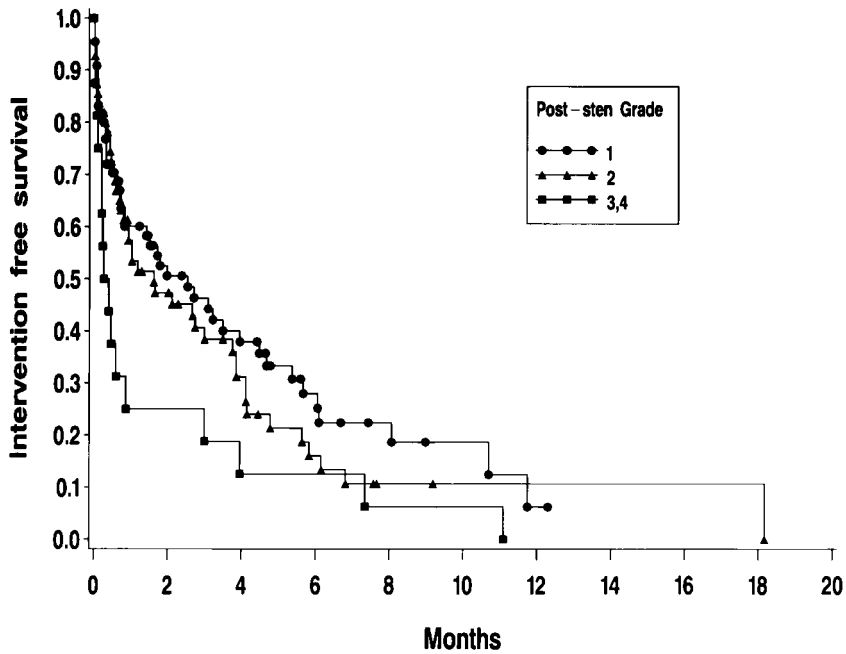

Fig 4. Intervention-free graft survival after thrombectomy for procedures in which there was no residual stenosis (●), mild residual stenosis (▲), or moderate-to-severe residual stenosis (■). Graft survival was calculated from the date of the initial intervention to the date of the next intervention (angioplasty, declotting, or surgical revision). $P < 0.05$ for the comparison between the two groups.

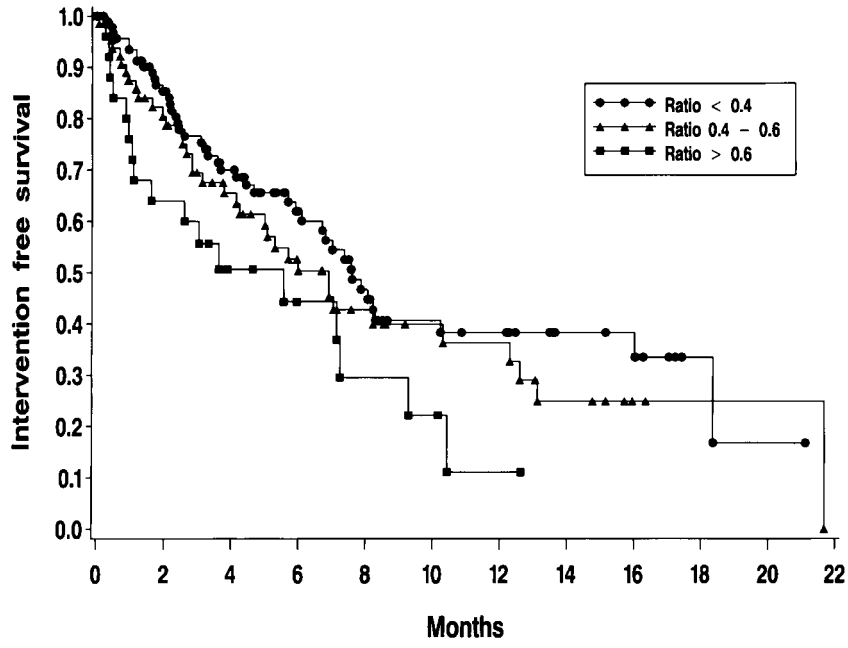

Fig 5. Intervention-free graft survival after elective angioplasty for procedures in which postangioplasty intragraft to systemic systolic pressure ratio was less than 0.4, 0.4 to 0.6, or greater than 0.6. Graft survival was calculated from the date of the initial intervention to the date of the next intervention (angioplasty, declotting, or surgical revision). *P* < 0.001 for the comparison between the groups.

stenosis was a significant predictor of graft outcomes after both elective angioplasty (Fig 3) and thrombectomy (Fig 4). Similarly, the intragraft to systemic systolic pressure ratio was inversely related to graft outcome after elective angioplasty (Fig 5). Because the pressure ratios were not obtained after graft thrombectomy, it is not known whether they are predictive of graft outcomes after that procedure. Finally, intervention-free graft survival after angioplasty was not significantly associated with graft location, number of stenotic lesions, or diabetic status of the patient (Table 3). Windus et al[29] observed lower graft survival in patients with diabetes compared with those without diabetes, whereas three other studies reported no significant differences.[7,23,30]

Table 3. Clinical Predictors of Intervention-Free Graft Survival After Radiologic Procedures

Variable	Hazard Ratio	95% CI	*P*
Angioplasty			
Graft location*	1.18	0.89-1.56	0.25
No stenotic lesions†	1.26	0.88-1.80	0.21
Diabetes‡	1.04	0.78-1.39	0.78
Thrombectomy			
Graft location*	1.14	0.88-1.46	0.32
No stenotic lesions†	0.75	0.54-1.05	0.10
Diabetes‡	1.09	0.85-1.39	0.50

Abbreviation: CI, confidence interval.
*Upper arm versus forearm graft.
†Two or more versus one stenotic site.
‡Diabetes present versus absent.

The duration of primary graft survival after elective angioplasty or thrombectomy might have been different if an alternate method of monitoring for graft stenosis had been used. Referral of patients for elective fistulogram and angioplasty on the basis of screening with serial measurements of dynamic dialysis venous pressure,[10] static intragraft venous pressure,[12] Doppler ultrasound measurements of peak systolic velocity,[13] measurement of access blood flow,[14,15] or aggressive clinical monitoring[9] have each reduced the frequency of graft thrombosis by 50% to 60% compared with the respective historical controls. However, none of these screening methods eliminates graft thrombosis entirely. In the current study, all grafts were screened for stenosis by the same aggressive clinical monitoring protocol. Therefore, the enhanced intervention-free graft survival after elective angioplasty compared with thrombectomy likely reflects the difference in the natural history of the grafts after the respective procedures rather than the specific monitoring technique for graft stenosis.

Grafts undergoing thrombectomy tended to have more stenotic sites than grafts undergoing elective angioplasty (Table 1). Because graft

thrombosis is usually a consequence of preexisting severe stenosis, it is likely that the thrombosed grafts had more time to develop stenosis at more than one site. The number of stenotic lesions did not influence graft survival after elective angioplasty or thrombectomy (Table 3). It is likely that the outcome of the graft was determined by that lesion with the highest grade of stenosis. Thus, coexistence of additional lesions with a lower grade of stenosis would not be expected to affect graft outcome after a radiologic intervention.

The duration of graft patency after angioplasty varies substantially among patients. It would be useful to stratify the risk for subsequent intervention after an angioplasty to guide the medical management of the patients. Accurate stratification of the grafts requires identification of those clinical or radiologic parameters that predict the duration of intervention-free graft survival after an angioplasty. Thus, high-risk grafts might need to be monitored for evidence of recurrent stenosis at frequent intervals or even referred promptly for a surgical revision, whereas low-risk grafts may require less frequent monitoring. Our retrospective analysis suggests that measurement of the pressure ratio and degree of stenosis after angioplasty may be useful in stratifying subsequent graft survival. Both parameters can be measured easily and with minimal expense. Quantitative rather than semiquantitative measurement of the degree of stenosis may further increase the predictive value of this parameter. A prospective clinical trial would be helpful to validate whether measuring these two parameters after radiologic graft interventions can be used to stratify the likelihood of subsequent intervention-free graft survival.

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