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Automated Intravascular Access Pressure Surveillance Reduces Thrombosis Rates

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ABSTRACT

Although monitoring of vascular accesses by physical examination is nearly as sensitive as surveillance measurements by vascular access pressure when performed by examiners, the frequency of examinations is limited by time. We developed intravascular access pressure surveillance as a surrogate to physical examination. Using real-time data from hemodialysis machines, we derived intravascular access pressure ratios for each dialytic procedure. An automated, noninvasive surveillance algorithm that generated a "warning" list of patients at risk for thrombosis was formulated. We hypothesized that this algorithm would reduce access thrombosis frequency. We designed a study comparing thrombosis rates during a baseline 6-month interval to three subsequent 6-month periods of active surveillance. Referrals for interventions during this 18-month period were based on persistently abnormal elevated vascular access pressure ratio tests (VAPRT) >0.55. Thrombosis rates declined progressively for arteriovenous grafts (AVG) during the intervention period compared with the baseline period. Arteriovenous fistula (AVF) thrombosis rates decreased during postintervention months 13–18 during employment of the VAPRT. We conclude that use of VAPRT can reduce thrombosis rates in vascular accesses, and the magnitude of the effect is larger and more consistent in arteriovenous grafts (AVGs) than autologous AVFs.

Hemodialysis (HD) vascular access thromboses result in missed treatments, disrupt patient treatment schedules, and reduce dialysis facility revenues. The value of clinical screening or detection of asymptomatic hemodynamically significant vascular access stenoses was recognized over 15 years ago and anticipated to reduce vascular access thrombosis rates (1) In addition, the adoption of the National Kidney Foundation Kidney Disease Outcomes Quality Initiative-recommended vascular access surveillance guidelines (2), by dialysis centers could reduce vascular access thrombosis rates. Several techniques are used in surveillance. Vascular access surveillance by flow measurement has not generally reduced access thrombosis rates (3–5), although ultrasonic stenosis detection has (6). However, duplex ultrasonography is probably cost prohibitive for large-scale use (7–9).

Elevated venous access pressure (VAP) indicates stenosis in the venous outflow limb. (10–12). Previously, we demonstrated that the vascular access pressure ratio test (VAPRT) (Vasc-Alert™; Vasc-Alert LLC, Chicago, IL) could be used to detect stenosis in the venous outflow tract of an access. VAPRT utilizes derived static venous access pressures (VAPs), taken several times during the dialysis treatment, and simultaneous mean arterial pressure (MAP) measurements to calculate the mean venous access pressure ratio (VAPR = VAP/MAP) for each treatment (13). Calculation of the VAPR ratio compensates for changes in MAP among and within dialytic sessions. The VAPRT analyzes ratios from consecutive (HD) treatments to determine the presence of a stenosis.

The primary objective of this study was to test the hypothesis that VAPRT-based referrals to interventional vascular access centers would reduce access thrombosis rates when added to the routine clinical assessment of vascular access. A secondary objective was to evaluate whether reductions of delivered dialysis dose from access recirculation predicted impending thrombosis, because many centers still use dialysis dose or recirculation as an access surveillance method (14,15). Lastly, the urea reduction ratio (URR), a measure of delivered dialysis dose, may decline as access blood flow declines below the prescribed blood pump flow rate (QB) (16). Therefore, serially declining URR with confirmation by recirculation measurements could detect impending vascular access thrombosis.

In this study, we evaluated the effect of surveillance with VAPRT on access thrombosis rates and monitored URRs as a predictor of thrombosis in vascular accesses.

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Methods

Clinical Setting

This prospective, observational study was carried out at a single in-center 50-station hemodialysis facility, affiliated with Henry Ford Hospital, Detroit, MI. The period of data collection, July 2003 to June 2005, involved 268 patients: 130 female and 138 male with a mean age of 63.2 ± 15 years and 98% African American. The monthly in-center patient census ranged from 246 to 265 patients. Treatments were stratified by vascular access type arteriovenous grafts (AVG) and arteriovenous fistula (AVF) at each dialysis. Subjects who received dialytic therapy through a (HD) catheter were excluded from analysis during periods of catheter usage. All patients with an (AVG) or (AVF) were eligible for vascular access surveillance (VAS) during any given month, irrespective of time of entry into the study. Baseline event data were accrued from the first 6-month period (7/2003 through 12/2003) when VAPR values were collected, but alerts were not provided to the treating staff and were accordingly not used to make referral decisions for evaluation/interventions. In the next three 6-month periods, decisions to refer patients were based on VAPRs, and the effects of VAPRTs on thrombosis rates were analyzed monthly.

Data Elements

Data used for VAPRT included machine blood flows, patient hematocrit (Hct) and venous drip chamber pressures (VDPs) collected when the patient blood pressures were recorded during treatment. URRs were ascertained monthly on all patients. For analysis of thrombosis rates, the date of each patient thrombosis event was recorded.

Procedures

For any venous tubing set, VDP is a function of access pressure in needle insertion site, needle gauge, MAP, dialyzer blood flow, tubing length, blood viscosity as a function of hematocrit Hct, and the pressure differential attributable to the vertical distance between the needle insertion site and the VDP transducer. Coefficients that normalize these effects specific to particular needles and tubing sets at blood pump flows of 200–500 ml/min and Hct 15–40% to calculate true VAP were derived as previously described (13). A unique VAP is calculated each time the BP is measured during a treatment and the mean VAPR is calculated. The mean treatment VAPR represents the mean of at least three readings. Notably, single treatment VAPRT does not interrupt HD therapy, and the algorithm using predefined data limits eliminates spurious readings caused by poor needle placement. VAPRT is empirically derived using trend analysis of VAPRs and identifies patients with consistently elevated or progressively increasing intra-access pressures. From previous work, VAPRs >0.55 represented the optimal balance between sensitivity and specificity for diagnosing vascular access thrombosis (13) in AVGs, and this value has been independently validated by Paulson et al. for AVGs (17). To eliminate spurious effects from poor needle insertion, the algorithm operationally defined an abnormal VAPRT whenever the average treatment VAPR exceeded 0.55 during three consecutive HD treatments. Patients were then placed on a monthly "Access Warning List" prompting further evaluation and possible intervention. In the absence of intervention, alerts would continue.

Event Rates

Events per patient-year were calculated from documented treatment events that involved AVFs or AVGs. Throughout the study period, clinical staff documented the inability to conduct HD by failure to obtain sufficient blood flow. These events were "labeled" as possible thrombotic events, with actual thrombosis confirmed by subsequent physical examination or at endovascular evaluation.

Clinical Assessments (Monitoring)

Treatment length reductions from access clotting or missed treatments were recorded with HD duration, swelling of the extremity ipsilateral to the access, cannulation difficulties, and events of prolonged bleeding, infection or infiltration, and aneurysm formation. Patients who experienced such events were examined for possible vascular stenosis (Table 1) and vascular access evaluations were scheduled when clinical suspicion was high. Lastly, URR values were reviewed monthly to evaluate trends in dialysis adequacy.

Data Presentation

All patients with an AVF or AVG in the 2-year study period were included in the analysis. The thrombosis rate was calculated for each access type in four 6-month periods as the ratio of the number of thrombotic events per total person-years of follow-up. Each patient's cumulative follow-up for a 6-month period was calculated and summed over patients with the same access

TABLE 1. Vascular access monitoring sheet

Patient name	Yes	No
Recorded by clinical staff	O	O
Dialysis duration, not achieved	O	O
Extremity swelling	O	O
Difficult cannulation	O	O
Prolonged bleeding	O	O
Decrease in Kt/V or URR	O	O
Unable to achieve adequate blood pump flow	O	O
Vascular "steal" symptoms or pain	O	O
Signs or symptoms of infection	O	O
Signs or symptoms of infiltration	O	O
Aneurysm formation	O	O
Other_____	O	O
Analyzed by physician/access coordinator	O	O
"Warning List" (Increased VAPRT)	O	O

Data input is conducted during clinical rounds. The last two items were added in the last three 6-month periods after active surveillance was implemented.

type. The first 6-month period was considered the baseline, and no alerts were generated during this period. Vascular access surveillance using VAPRT with alerts to the respective health-care providers was introduced and continued from months 7 through 24.

Six-month rates were recalculated and included all individuals receiving dialysis during those periods. To examine longitudinal differences in thrombosis rates, a generalized estimating equation (GEE) was used that produced a negative binomial regression model with total person-years of follow-up for an interval. This value represented the value as the offset variable in the model. This approach modeled the count nature of the data and adjusted for correlations within the same individuals over the study duration.

URRs were analyzed by ANOVA for repeated measures. Statistical analysis was performed with SAS v 9.2 (SAS v9.2, Cary, NC), and presented as means ± SEM, with the level of significance set at 0.05.

Results

Hemodialysis treatments were analyzed when an AVG or AVF was utilized for treatment. More than 146,000 data samples for VAPR determinations were evaluated during this 24-month study in which a total of 91 thrombotic events occurred, 56 in AVGs and 35 in AVFs (Table 2). On average, during the entire study period, there were 117 patients with AVGs and 89 patients with AVFs. Patients were in the study group for 92% of the total possible time, with the missing 8% due to a change to a different access, death or loss to follow-up.

TABLE 2. Thrombosis data for each 6-month period are shown

Access	Period	Patients	Clots	Follow-up patient years	Thrombosis rate per person-year
Graft	Preintervention (6 months)	115	21	50.9	0.413
	Postintervention (0–6 months)	138	19	63.3	0.300
	Postintervention (7–12 months)	116	8	54.5	0.147
	Postintervention (13–18 months)	99	8	45.5	0.176
			56		
Fistula	Preintervention (6 months)	85	5	36.5	0.137
	Postintervention (0–6 months)	104	12	48.3	0.249
	Postintervention (7–12 months)	93	15	42.6	0.352
	Postintervention (13–18 months)	75	3	37.2	0.081
			35		

During the 24-month monitoring study, 56 arteriovenous grafts and 35 arteriovenous fistulas thrombosed during the 18-month interval. The baseline vascular access thrombosis rate (thrombosis rate) was 0.41 events per person-year prior to the completion of systematic implementation of VAPRT, and declined to 0.18 in the last post intervention period. In AVG, a persistent and steady decline of thrombosis rate is evident during 3, successive 6-month intervals of utilizing the vascular access pressure ratio test (VAPRT). In AVF, the pattern differs and a decrease in thrombosis rate effect is not seen until the last 6 month period. For AVG and AVF combined 6-month thrombosis data, the baseline thrombosis rate of 0.297 events per person-year prior declined to 0.133 at the completion of VAPRT implementation.

The vascular access thrombosis rates are summarized (Table 2). In AVG, the thrombosis rate declined progressively from a preintervention rate of 0.41 to 0.18 events per person-year, during the final postintervention period (Fig. 1). The AVF thrombosis rate unexpectedly increased during the first year before falling to a lower event rate of 0.081 events per person-year during postintervention months 13–18 (Fig. 1). Combining both access types, the rate/person-year declined consistently from a preintervention rate of 0.297 events per person-year to 0.13 events per person-year (Table 2 and Fig. 2) in the last postintervention period. This represents a 57% decrease overall. The number of clotting episodes within each 6-month period decreased from 26 in the control period to 11 events in the last.

The AVG relative risk (RR) for thrombosis demonstrated a statistically significant decline from months 7 to 12 months, with a minimal decline during months 13 to 18. AVG access had a relative risk for thrombosis of 0.44 relative to baseline confidence interval (CI: 0.18–1.09), during months 13 to 18 (Table 3). By contrast, AVFs had a near-significant increase in thrombosis rate during postintervention months 7 to 12 compared with baseline. In postintervention months 13 to 18, the rate decreased, relative to baseline (RR 0.61; CI 0.19–1.92), but this result did not reach statistical significance (Table 3).

During the 24-month study period, we determined VAPRTs and the number of interventional vascular procedures. Table 4 delineates the decreased number of alerts from the first 6-month control period to the first vascular access surveillance period, which then remained stable during the last 2, 6-month periods. On average, the number of total alerts per access decreased from 2.3 (range 0–5) to 1.3 alerts (range 0–3) per 6 month period. During the active surveillance period (periods 3–4), attempts were made to obtain referrals done after the first alert was confirmed in the next month. The fraction of accesses on alert decreased from 38% to 21–23% in the surveillance period. As shown in Fig. 3, the number of angioplasties per 6-month period tended to remain constant after vascular access surveillance was introduced, but the ratio of procedures requiring thrombectomy/thrombolysis/PTA compared with simple PTA declined.

We retrospectively examined changes in URR in the 6-month interval pre-and post-vascular access thrombosis. No significant changes occurred in URR during the 6 months before or after AVG thrombosis (Fig. 4). By contrast, URR decreased significantly in AVF by nearly 6% from 73% to 67% in the month preceding thrombosis and then improved only minimally after angioplasty. The mean nadir values for both access types were still above the minimum considered "adequate" dialysis. There was no change in URR in the 2 months before thrombosis in AVGs, but there was an abrupt onset of decreased dialysis dose delivery in the month before thrombosis in AVFs using repeated measures ANOVA

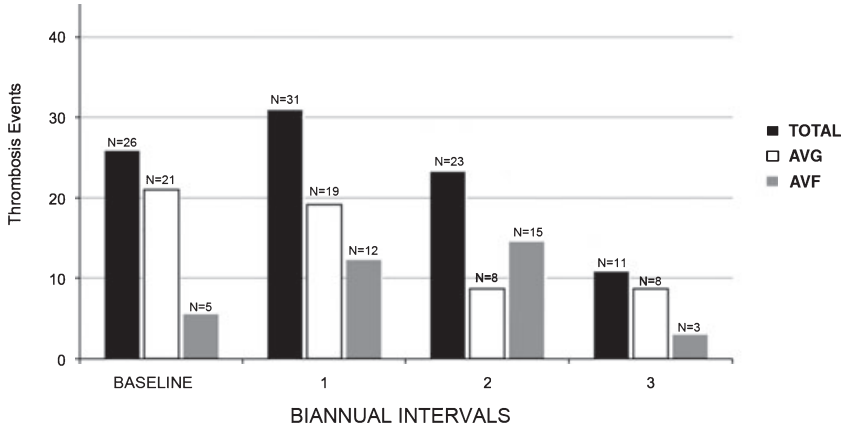

FIG. 1. Raw thrombosis events by vascular access type. Thrombosis events for arteriovenous grafts (AVGs) and arteriovenous fistulas (AVFs) are displayed in the baseline period when VAPRT alerts were not provided and during three, consecutive 6-month intervals of active monitoring. VAPRT was the sole method of vascular access surveillance and was associated with a progressive decline in thrombosis rates of AVGs. For AVFs, a reduction did not occur until the last interval.

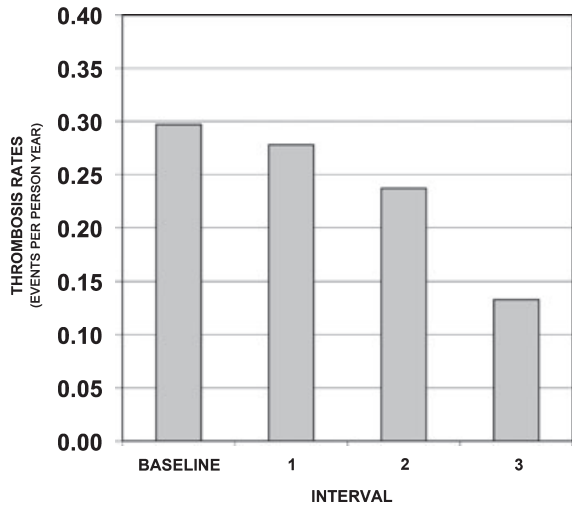

FIG. 2. Thrombosis event rate reduction of vascular accesses after implementation of VAPRT. The 24-month study was divided into a 6-month control interval followed by three, 6-month intervals at a single in-center hemodialysis unit with a mean patient census of 227. VAPRT was not utilized during the baseline period.

TABLE 3. Thrombosis risk by vascular access type

Comparison	RR (95% CI)	*p*-value
Graft		
Interval 2 vs. Interval 1	0.74 (0.35, 1.57)	0.439
Interval 3 vs. Interval 1	0.37 (0.15, 0.90)	0.028
Interval 4 vs. Interval 1	0.44 (0.18, 1.09)	0.078
Fistula		
Interval 2 vs. Interval 1	1.78 (0.69, 4.61)	0.237
Interval 3 vs. Interval 1	2.55 (0.96, 6.76)	0.061
Interval 4 vs. Interval 1	0.61 (0.19, 1.92)	0.398

All access relative risk (RR) estimates. The arteriovenous graft rate demonstrated statistically significant decline from months 7 to 12, with a minimal decline during months 13 to 18. A subject with an AVG access had a RR for thrombosis of 0.44 relative to baseline confidence interval (CI: 0.18–1.09). In the postintervention period from (13–18) months, reduction in rate/patient year was seen in the 4th 6-month period. The AVF group showed an increase in thrombosis rate during postintervention months 7–12 month compared with baseline. At postintervention months (13–18), the rate was decreased, with a RR 0.61 (0.19–1.92) relative to baseline.

(Fig. 5). This reduction was evident in AVFs at −30 and +30 days. $*p < 0.05$ compared to 60 days. URR remained depressed by nearly 8.2% during the succeeding several months postthrombosis in patients with thrombosed AVFs. In some patients, the lower URR was the results of a greater requirement for dialysis catheter use as salvage with thrombolysis/PTA was unsuccessful.

Discussion

In this study, systematic use of VAPRT surveillance technology reduced the thrombosis rate by generating a simple-to-read "warning list" of patients with possible access dysfunction. No special equipment or clinical staff was required by VAPRT because it is an electronically automated process. After 18 months of application, the overall thrombosis rate decreased 57% from 0.29 to 0.13 events per patient-access-year. Concordantly, the number of interventions performed to maintain access patency, and the number of "emergency" procedures was reduced. During the control period, 115 procedures were performed, and 55 of these included a thrombectomy prior to angioplasty. During the last two study intervals, thrombectomies in permanent accesses (both AVG and AVF) decreased by half, while frequency of simple angioplasties did not increase. In fact, the total number of procedures decreased from 180 at baseline to 129, 115, 112 in each subsequent period. However, there was a shift to a greater proportion of elective PTA as opposed to thrombolysis/thrombectomy with PTA.The fraction of procedures triggered by a thrombosis decreased from 31% at baseline to 21% in the last period. These results are similar to those reported by Besarab et al. (10) who evaluated static pressure ratios, and McCarley et al. (18) whose study involved flow-based technology.

TABLE 4. Vascular access warning list summary

Period	Total warnings (*N*)	Alerts per access in period	Monthly percent alert rate
1	386	2.3 ± 1.2	38.0 ± 3.5
2	196	1.3 ± 0.5	21.2 ± 3.0
3	220	1.4 ± 0.6	22.7 ± 1.3
4	223	1.3 ± 0.6	22.1 ± 2.2

Rates are expressed as number of warnings for all vascular accesses, arteriovenous grafts, and fistulas combined during the control and surveillance periods.

The VAPRT surveillance technology can be used to prioritize patients with the greatest expectation of access failure. Vascular access warning reports triage patients with VAPRT's >0.55 their accompanying individualized trend reports/graphs of VAPRs and pump blood flows (Fig. 6) delineate those accesses at risk for clotting. Monthly reports highlight previous months on the warning list, interventions, clotting incidents with blood flows, and historical VAPRTs. A nurse practitioner or physician conducts a careful physical inspection of vascular accesses for patients who are "on alert," and completes a patient-specific Access Information Sheet (Table 1) prior to referral for intervention.

An impact of the warning list on the AVG thrombosis rate was evident within the first 6-month period of systematic use. Graft thrombosis rate progressively decreased during the three designated interventional periods with the rate stabilizing in the last two periods. The gradual rather than the anticipated stepwise functional decrement may reflect the transition period for training staff and referring patients for vascular access evaluation intervention. Impediments to further rate-lowering include patient refusal to undergo access evaluation and a small proportion of vascular accesses that have previously and repeatedly failed interventions.

Most individuals do not develop an access thrombosis within a month of the appearance of their vascular access on the "warning" list. The persistence of a high proportion of alerts in the first 6 month control period reflects cumulative time on this list of the same accesses until corrective actions were taken. In the absence of referral and therefore endoluminal/surgical intervention, there is no reason for the alert rate to fall. Only successful detection and intervention on stenosis should reduce the alert rate by correcting the stenosis that lead to the initial alert leading to its subsequent absence in the months thereafter. Implementation of VAPRT process did exactly that. It lowered the total number of monthly alerts, reducing the fraction of accesses on alert from 38% in the control period to ~21–22% in the three subsequent active surveillance periods.

Static access pressures are generally lower in AVFs compared to AVGs (12,19,20), especially in forearm fistulas. The common occurrence of inflow segment stenosis further lowers the pressures within the access (19,20). This differs from AVGs where stenosis is

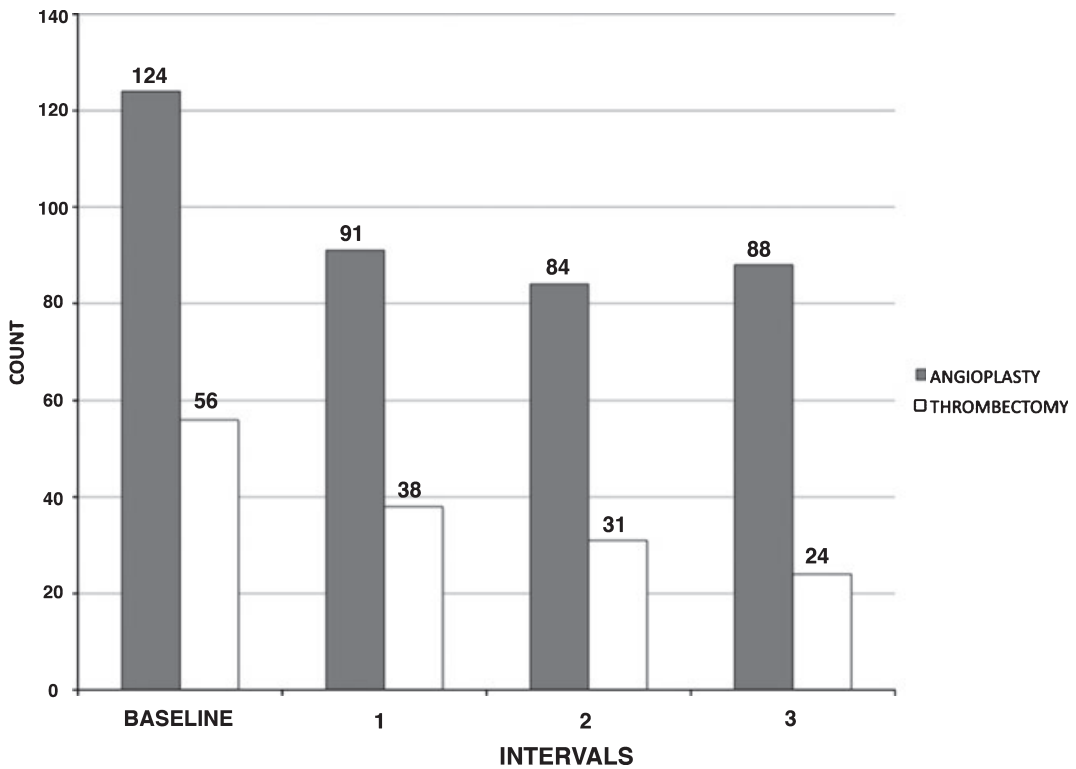

FIG. 3. Vascular access interventions decrease after VAPRT implementation. The 24-month study was divided into a 6-month baseline interval followed by three, 6-month consecutive intervals at a single in-center hemodialysis unit with a mean patient census of 227. VAPRT was used for vascular access surveillance during the 18 months following the baseline interval. Absolute numbers of vascular access interventions, represented as angioplasties ("left panel") or thrombectomies ("right panel"), declined from respective control periods at all follow-up intervals.

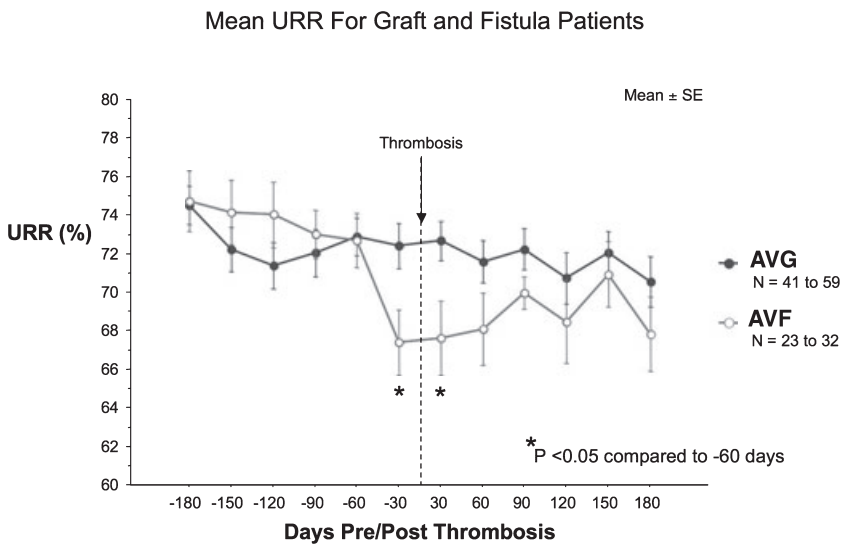

FIG. 4. Time-varying urea reduction rates (URRs) during the vascular perithrombosis period. Mean URRs are only altered during the progression of thrombosis of arteriovenous fistulas (AVFs), compared with arteriovenous grafts (AVGs). The reduction of URRs in AVFs is evident within 30 days of fistula thrombosis and within 30 days of having achieved stability (Day 60). After development of thromboses, AVF URRs remained lower, necessitating hemodialysis catheter placement in patients with AVFs ($n = 6$) and patients with AVGs ($n = 13$).

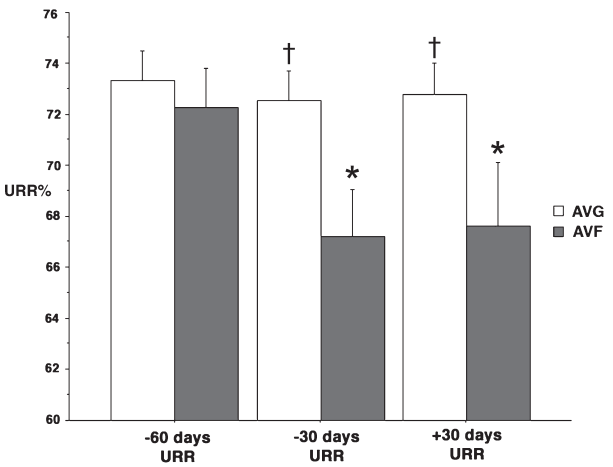

FIG. 5. Time-varying urea reduction ratios (URRs) in thrombosed vascular accesses. Time = 0 is defined as the day of vascular access thrombosis. URRs declined in arteriovenous fistulas (AVFs) but not in arteriovenous grafts (AVGs). This reduction was evident in AVFs at −30 and +30 days. *$p < 0.05$ compared to −60 days. †$p < 0.05$ compared between grafts and fistulas (repeated measures, two-way analysis of variance).

typically in the outlet or mid-portion (21) elevating the pressures. Thus, the probability of achieving the critical value for VAPR of 0.55 is higher in AVG than AVF.

This difference in hemodynamics alone may have resulted in the obvious difference in thrombotic event rates between AVG and AVF. The data clearly show that reduction in vascular access thrombosis occurred promptly in AVG but only after a protracted period of more than 12 months in AVF. This was initially puzzling and several possible explanations were looked for. Like most programs, we attempted to construct more wrist AVF during the initial stages of the Fistula First Initiative (note the increases in AVF years at risk in periods 2 and 3 compared to 1 and 4, Table 2). At the beginning of the baseline period, 41.3% of 179 accesses were AVF. The total number of AVFs increased during the baseline period and 42.5% of all permanent accesses used at any point in the period were AVF (Table 2). During the 18-month intervention period, 54% of 92 "new" permanent accesses were AVF, up from the 42.5% in the baseline period. Of the 31 new AVFs used in period 1 and 2, 22 were constructed by our surgeon; 19 were forearm AVFs. In the last period, he constructed 11 of 15 new AVF, 8 were at the elbow level. The surgical records for the other patients are external to the health system.

The initial increase in thrombosis rate may have resulted from attempts to salvage inadequately functioning, nonmature AVF in which the most common lesion is an inflow stenosis PTA balloon pressures required for successful lasting dilatation are larger in such AVF than in AVG, but our interventionist did not exceed 15 atm of pressure during these periods. It is also known that the PTA procedure itself may injure the vessels and predispose it to thrombosis if there is inadequate long-lasting correction of the stenosis. The lack of efficacy may explain the inability to decrease thrombosis rate initially. The absence of improvement in URR, which remained depressed even after interventional procedures (see Figs. 4 and 5), suggests inadequate correction of the underlying stenosis.

Subsequently, the emphasis of Fistula First Initiative led to the construction of more elbow-level fistulas, many of which are prone to the development of stenoses at both the inflow and outlet of the access. Elbow level accesses are subject to outlet stenosis at the "cephalic arch" if cephalic vein and the outflow "swing segment" if basilic. Stenosis in these downstream segments elevates intra-access pressure, and the pressure profile begins to mimic that of AVGs permitting earlier detection of these lesions in the outflow. In addition, we began to trend

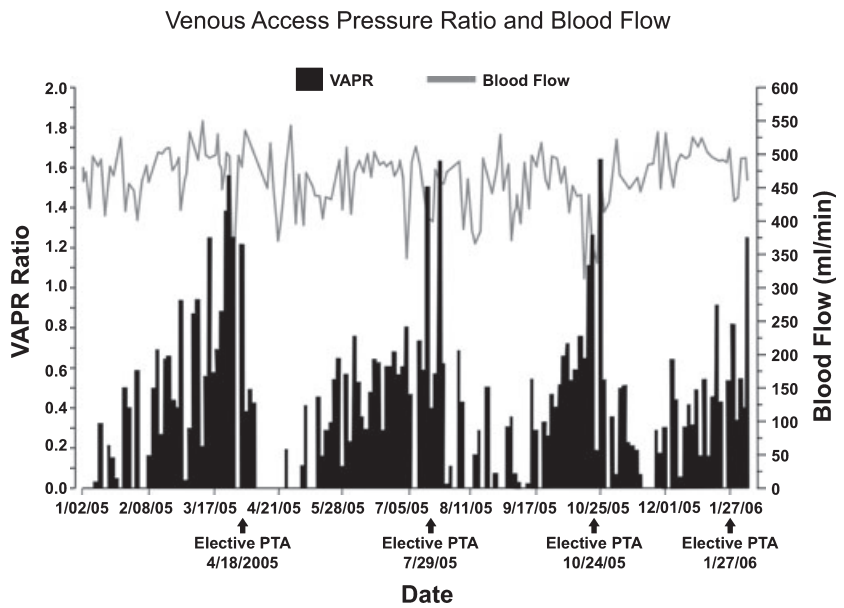

FIG. 6. Time-varying vascular access pressure ratios (“lower plot, VAPRs”) and vascular access blood flows (“upper plot, Qb”) from a single patient AVG. Surgical intervention dates correspond with increasing VAPR, defined as the vascular access pressure measured at the venous limb of a vascular access divided by mean arterial pressure, derived from hemodialysis machine blood pressure measurements.

delivered blood pump flow along with the VAPR to detect inflow lesions (Fig. 6). Our subsequent experience is that combining the two parameters permits detection of AVF stenoses wherever their location is. Once all the elements were in place, we were able to demonstrate a reduction in thrombosis rate below basal in the last 6-month period although the previous upward variations in periods 2 and 3 precluded demonstration of a statistically significant effect. With the small absolute number of five thromboses in the basal period, it would require a much larger study in AVF to demonstrate that the 40% reduction to three thrombotic events in the final period was clinically important.

Thrombosis rates for both access types from 2006–2008 have remained at the levels seen during the last 6-month period (G. Zasuwa, S. Frinak, A. Besarab, and J. Yee, unpublished data). The rate of formation of stenosis is not influenced by our surveillance technique.

There are limitations to this study. The first is the absence of a control group where only physical or clinical evaluations would have been performed for the entire 2 year duration of the study and event rates tabulated. Vascular access examination is important and capable of delineating access dysfunction (22–24), and it has been touted as equivalent to static intra-access pressure measurement (25). In AVGs, the positive predictive value by physical examination or clinical clues for stenosis varies from 69–70% (26,27) to 98% (28). A mean positive predictive value of 75% can be calculated form available data. As recently reported, accuracy is highest for detecting stenosis within the AVG, followed by the outlet, and least for the inflow (sensitivity of only 33%) (29). Similarly physical examination of AVF despite its high specificity of 90% in detecting access dysfunction has a highly variable sensitivity varying greatly from 38% to 96% (30). Achievement of superior accuracy requires uncommon expertise and multiple examinations by properly trained, skilled, and experienced individuals, both uncommon circumstances in USA dialysis centers today. We felt that having such a control group for the entire 2 year period would have been a violation of clinical responsibility.

Sequential monthly URR values even if performed for months in advance of thrombosis were not predictive of developing stenosis in grafts and therefore of impending access thrombosis. We noted that the mean nadir URR values for both access types was still above the minimum URR value of 65% indicative of “adequate” dialysis. Development of recirculation producing a significant decrease in URR requires that access flow be much less than prescribed blood pump flow, which averaged 450 ml/min for AVGs and 420 ml/min for AVFs (31). Most AVG will thrombose at access flows < 500 ml/min (2). The test is therefore useless in AVG. As access flow in grafts is almost never limiting for URR, it is not surprising that it did not change after intervention. By contrast, up to one-third of dysfunctional AVFs will show access recirculation (32) that may be subclinically manifested as a decrease in urea reduction ratio (URR) or Kt/V (but not always to frankly inadequate levels). Recirculation could proceed for months as AVF patency can be maintained at blood flows < 500 ml/min. Our data contend that for AVF, decreases in URR or KT/V from their respective baselines are present for just 1–2 months before thrombosis. Therefore, in AVFs, a sudden decrease in URR from baseline, even when the URR remains above a minimally acceptable value, warrants more urgent evaluation. However, the lead time is short and the stenosis usually so advanced that many AVF, particularly wrist level, cannot be salvaged or improved sufficiently. As a result, lower URRs tend to persist after intervention.

Our center has used ultrasonic dilution blood flow techniques to detect stenosis (33–35). We abandoned this methodology because it required dedicated staff, accuracy was operator-dependent, and the time requirement made it impractical for use in our dialysis units. However, the utility of access flow measurements when performed according to the Kidney Disease Outcome Quality Initiative was clearly documented by Wijnen et al. (35). Thrombectomy procedures were reduced by over 60%, whereas the number of radiological interventions more than doubled, from 0.33 to 0.88 events/patient-year, following addition of flow surveillance to "routine" clinical monitoring. Access survival was unaffected.

Randomized clinical trials (RCT) have failed to show that surveillance improved access survival. In RCTs, the addition of a surveillance technique (pressures or flow) was compared with an established regimen of monitoring by physical examination and clinical manifestations of dysfunction. As previously discussed, physical examination is an effective technique when carried out by experienced staff. By contrast, most observational studies were carried out in centers where monitoring was ineffective. In our study, we did not attempt to certify or attest to the "quality" of the monitoring conducted by the clinical staff. Nevertheless, our results obtained in the course of a busy, real-world practice; demonstrate access thrombosis rate reductions are possible in such settings, particularly in AVG, by adding our surveillance method. We do encourage future, sufficiently powered, double blinded, and randomized study of vascular access surveillance methods effects on meaningful outcomes such as thrombosis rate, useful access lifespan till abandonment, or economic costs. Such studies may need to be much larger than those previously performed and involve multi-center cooperation. The sample size for two recent Dialysis Access Consortium studies to demonstrate a 25% difference in event rates between drug and no-drug required $\sim$1000 subjects (36,37).

In conclusion, we document that the access thrombosis rate can be reduced by access surveillance using the VAPRT algorithm to detect temporal changes in derived static pressure ratios for AVFs and AVGs. The effect is most consistent in grafts and influenced by the type of AVF. We also demonstrate that alterations of URR inadequately predict impending vascular access thrombosis, particularly in grafts. The latter should be abandoned as a surveillance technique. Use of the surveillance technique seems to produce a shift from PTA usage for salvage of thrombosed graft to more elective PTA of stenosis in patent grafts, suggesting perhaps "more effective" selection of accesses for intervention. In grafts, more than 60% of accesses may have stenosis, but only some of these will produce clinical dysfunction. Clinical monitoring alone may result in referrals wherein many unnecessary PTA's are performed. We believe that the VAPRT surveillance methodology may more selectively identify those stenoses producing operative dysfunction, which if left unattended eventuate in thrombosis. Additional studies would be needed to confirm this.

Disclosures

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Consultant: Vasc-Alert
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Consultant: Vasc-Alert, Rockwell International
Anatole Besarab:
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Stockholder: None.
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