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Vascular access surveillance: an ongoing controversy

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Hemodialysis vascular access surveillance continues to be widely recommended despite ongoing controversy as to its benefit in prolonging access patency compared with clinical monitoring alone. The most common screening tests are access blood flow and dialysis venous pressure measurements. When surveillance test results cross a predetermined threshold, accesses are referred for intervention with correction of stenosis to reduce future thrombosis and prolong access survival. Current surveillance strategies have four components: (1) underlying condition; (2) screening test; (3) intervention; and (4) outcomes. However, limitations exist within each component that may prevent achieving the desired outcomes. This review discusses these limitations and their consequences.
To date, randomized controlled trials have not consistently shown that surveillance improves outcomes in grafts, and there is limited evidence that surveillance reduces thrombosis without prolonging the life of native fistulae. In conclusion, current evidence does not support the concept that all accesses should undergo routine surveillance with intervention.

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In the years following the introduction of synthetic arteriovenous grafts, hemodialysis patients frequently experienced graft thrombosis and failure. The cycle of thrombosis, thrombolysis with correction of stenosis, and thrombosis was considered the natural order of things. This changed when two paradigm-altering studies[1,2] reported that dynamic and static dialysis venous pressure (VP) measurements combined with preemptive angioplasty yielded large reductions in thrombosis rates and replacement of vascular accesses.

These reports led the National Kidney Foundation Kidney Disease Outcomes Quality Initiative (NKF-KDOQI) guidelines[3,4] to recommend that grafts and native arteriovenous fistulae undergo routine surveillance for stenosis with preemptive correction of the stenosis. Surveillance includes device-based measurements, such as access blood flow (Qa) and static (or derived static) VP. Surveillance is intended to supplement clinical monitoring, which uses physical examination and other clinical evidence of access dysfunction, such as difficult cannulation or prolonged bleeding post dialysis. Dynamic VP has been largely discredited as a surveillance strategy and is considered to be monitoring.[4]

The KDOQI Vascular Access Work Group[4] applied an evidence-based approach to develop the guidelines; however, the lack of quality evidence in this field has led to considerable debate over these guidelines.[5–11] The controversy follows from a growing body of evidence that surveillance as usually practiced (i.e., monthly Qa measurement) may not improve access outcomes, is costly, and may even be harmful.[6,8,9,12–15] However, there is a general agreement that monitoring of accesses with physical examination and clinical assessment should be part of the standard care of all dialysis patients.

This paper will review the recommendations for access surveillance, its rationale, and the controversy by evaluating the four necessary components of the strategy of access surveillance (Figure 1): the background condition, the screening test, the intervention, and the outcome.

CURRENT RECOMMENDATIONS

In Qa surveillance, the KDOQI Guidelines[4] recommend an intervention referral when graft Qa is <600 ml/min or when Qa has decreased by $>25\%$ and falls below 1000 ml/min. Fistula referral is not recommended until Qa is

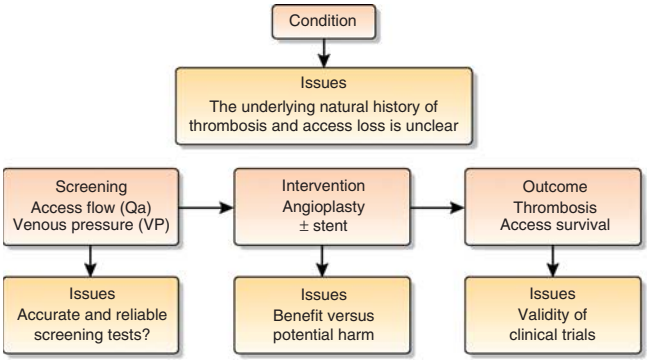

Figure 1 | Components of a surveillance strategy that influence its validity and usefulness. The four components of vascular access surveillance strategy are as follows: the underlying condition, screening tests, referral for intervention procedures, and outcome. Each component has issues that may affect success of the surveillance strategy.

$<$400–500 ml/min because fistulae may remain patent at a lower Qa than grafts. VP is normally adjusted for the mean arterial pressure (VP/MAP) because an increase in MAP causes VP to increase. Grafts or fistulae are referred if static VP/MAP has increased to >0.50 or derived static VP/MAP is >0.55. KDOQI[4] recommends that Qa be measured monthly. VP measurements are less complex. Therefore, static VP measurements are recommended at least every 2 weeks, whereas derived static VP measurements should be taken every 1–2 weeks because the blood pump augments variation in measurements.[4] The Guidelines emphasize the importance of using trend analysis to guide referral decisions rather than relying on a single measurement. It is noteworthy that not all national guidelines recommend the routine use of Qa surveillance.[16]

RATIONALE FOR SURVEILLANCE

Grafts have a low early failure rate. However, they often develop stenosis and subsequent thrombosis in the weeks and months after surgery, most commonly at the venous anastomosis or outflow vein. The stenotic lesion, referred to pathologically as neointimal hyperplasia, is believed to develop because of a combination of blood flow turbulence, low or oscillatory shear stress, and endothelial dysfunction.[17,18] Neointimal hyperplasia includes myointimal proliferation, matrix deposition, infiltration with macrophages, and neovascularization.[17,19] Active participants in this process include vasoactive, thrombogenic, and mitogenic factors, among which reactive oxygen species, inflammation, and platelet aggregation are major components. The lesion results in progressive stenosis and thrombosis and accounts for most graft failure.

The frequent failure of established grafts has led to a preference for fistulae that, once established, have lower failure rates.[3] However, fistulae also develop neointimal hyperplasia,[20–22] although the details of fistula failure differ from grafts. Fistulae are not suitable for cannulation until they have undergone a maturation process of dilatation and remodeling of the vein. Poor vessel elasticity and vessel calcification may cause maturation failure by preventing adequate dilatation,[23] which may be associated with hemodynamic conditions that promote neointimal hyperplasia. Thus, fistulae with maturation failure often develop stenosis at the arteriovenous anastomosis or downstream vein. Additional factors may contribute to stenosis. For example, mobilization of the swing segment of the vein during surgery may disrupt the vasa vasorum, leading to ischemic changes that promote stenosis.[24] Thus, although grafts may fail after initially functioning adequately, as many as 60% of fistulae may never be suitable for dialysis.[25,26]

The rationale for surveillance is based on the hypothesis that progressive stenosis is accurately detected by reduced Qa and increased VP before thrombosis and access loss. Screening of these parameters, which are surrogates for stenosis, is intended to detect stenosis before thrombosis and allows for a corrective procedure such as angioplasty to maintain the patency of the vascular access. Prevention of thrombosis is beneficial because it avoids unscheduled intervention procedures that may be accompanied by central venous dialysis catheters, hospitalizations, and increased costs of care.

SURVEILLANCE STRATEGY

In evaluating the validity and usefulness of a surveillance strategy, it is useful to examine its four components (Figure 1) by applying globally recognized criteria for screening tests according to the World Health Organization.[27]

The condition

The undesired condition, thrombosis in a vascular access, must be an important health issue. The epidemiology and natural history of access thrombosis, including development from latent to declared disease (i.e., stenosis leading to thrombosis), should be adequately understood. In addition, there should be a detectable risk factor, disease marker, latent period, or early symptomatic stage. This latent period is necessary to allow time for an intervention to be applied so as to improve the outcome. Other relevant conditions that should be considered in the surveillance strategy are the natural history of the access following the intervention and the pathophysiology that leads to ultimate access abandonment.

The test

The screening test, Qa or VP measurements, must be simple, safe, precise, unbiased, and validated with adequate reproducibility in detecting stenosis in appropriate populations. The distribution of test values should be known and there should be suitable threshold levels for intervention referral that are well defined and agreed upon. A policy should be agreed upon with regard to the further diagnostic investigation of individuals with a positive test, in this case an angiogram, to confirm the access stenosis.

The intervention

The intervention, angioplasty (+/− stenting) or surgical revision, should be effective for patients identified through early detection. There should be evidence that early treatment leads to better outcomes compared with late or no treatment. The intervention must be clearly defined and standardized to ensure reproducibility.

The outcome

There should be evidence from high-quality randomized controlled trials that the surveillance program is effective both in reducing morbidity and in yielding the desired outcomes. The opportunity cost of the screening strategy's program (including testing, diagnosis and treatment, administration, training, and quality assurance) should be economically balanced in relation to expenditure on medical care as a whole. There should be a plan for managing and monitoring the screening program and an agreed set of quality assurance standards.

THE CONDITION

The natural history of access thrombosis and the impact of intervention (angioplasty or thrombolysis) on the natural history are unclear and under active research.[28,29] However, the conditions of the dialysis environment and its potential impact on the screening test will be discussed.

Impact of access hemodynamics on surveillance

Role of vessel diameter. The screening test attempts to detect stenosis by reliably detecting changes or abnormalities in Qa or VP. Thus, mathematical models have been used to improve the understanding of access hemodynamics and the potential effectiveness of surveillance.[23,30–32]

The graft model, derived from duplex ultrasound studies of 94 patients, includes the inflow artery, arterial and venous anastomoses, graft, stenosis, and outflow vein.[30,31] A key characteristic of the model is the vessel luminal diameters, which vary over a wide range; however, the artery was generally narrower than the vein (median artery/vein diameter ratio = 0.77). A narrow vessel mimics stenosis in that it increases vascular resistance. Examination of these models has shown that vessel diameters in the access circuit predominantly control the relation between Qa or VP and stenosis (Figure 2), and this can impair the accuracy of surveillance.

Figure 3a and b show that the relationship between Qa and stenosis in grafts is sigmoidal: as stenosis progresses, Qa initially remains unchanged but then rapidly decreases as critical stenosis is reached.[30] Moreover, narrower arteries cause a reduction in Qa and shift the curve to the right. Thus, as stenosis progresses, there is a longer delay in the decrease in Qa, followed by an even more rapid decrease in Qa at critical stenosis. When translated into a clinical setting, the duration of the delay, the magnitude of the Qa change, and its relationship to access diameters (i.e., 'the condition') are so poorly understood that it cannot be reliably applied to a surveillance strategy.

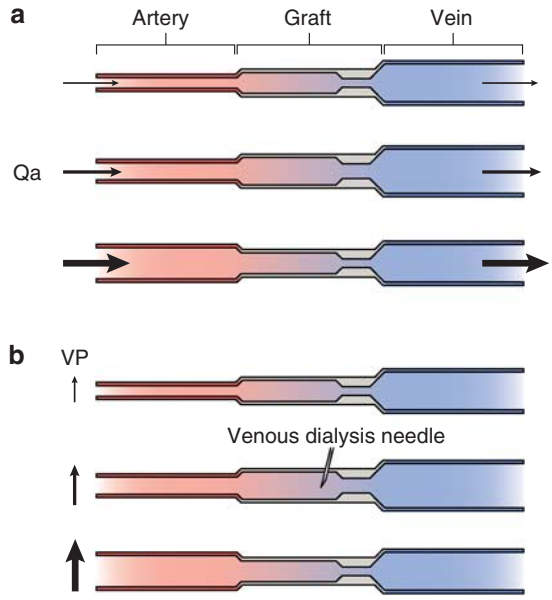

Figure 2 | Luminal diameters strongly influence blood flow (Qa) and dialysis venous pressure (VP) in the vascular access circuit. In (**a**), increasing artery luminal diameter reduces overall circuit resistance, and thus graft Qa increases despite no change in stenosis. Similarly, in (**b**), increasing artery luminal diameter allows transmission of vascular pressure to the dialysis needle, and thus VP increases. Thicker arrows represent larger values for Qa and VP, but do not indicate relative numerical values.

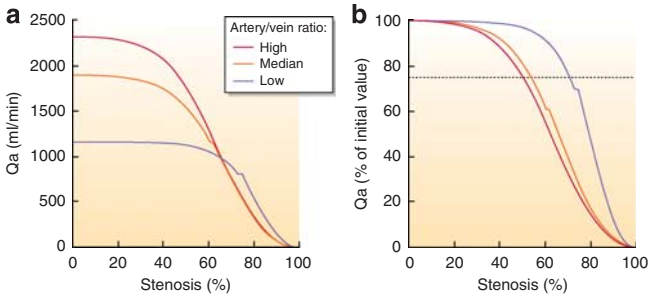

Figure 3 | Vessel diameters control relation between blood flow (Qa) and stenosis. (**a**) Relationship between graft (Qa) and stenosis at venous anastomosis.[30] (**b**) Relationship between graft Qa and stenosis at venous anastomosis, with Qa plotted as a percentage of the initial value. For narrower arteries (lower artery/vein diameter ratios), the sigmoid curve is flattened and shifted to the right, causing delay and then rapid reduction in flow as stenosis progresses. Dashed line shows that by the time Qa has decreased by 25% (the Kidney Disease Outcomes Quality Initiative (KDOQI) referral threshold[4]), flow is on the rapidly falling part of the curve. Thus, standard monthly surveillance may fail to detect a decrease in flow before thrombosis. High (1.28), median (0.77), and low (0.40) artery/vein ratios derived from 94 patients; low and high ratios enclosed 95% of patients.

Similar concerns and results are obtained with static VP.[31] A relatively narrow artery will cause a delay in the increase in VP with progressive stenosis, followed by a more rapid increase in VP at critical stenosis. KDOQI recommends referral for intervention when VP/MAP increases to >0.50,[4] but this threshold occurs at stenoses ranging from 38 to 73%

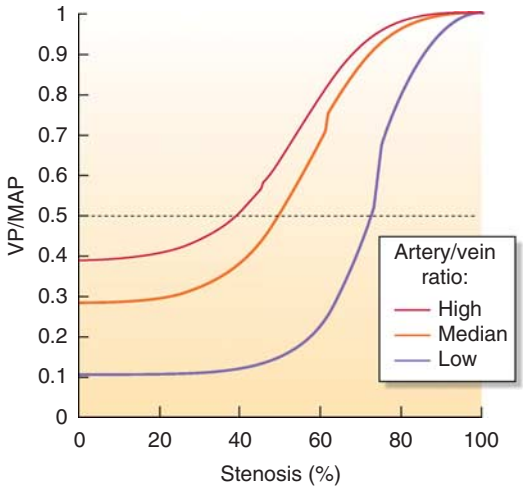

Figure 4 | Vessel diameters control relation between venous pressure and stenosis. Relationship between static dialysis venous pressure adjusted for mean arterial pressure (VP/MAP) and stenosis at graft venous anastomosis.[31] Dotted line at VP/MAP = 0.50 indicates the Kidney Disease Outcomes Quality Initiative (KDOQI) referral threshold.[4] There is no consistent relationship between VP/MAP and stenosis, and note the rapid increase in VP/MAP when artery is narrower than vein. Artery/vein ratios defined in Figure 3.

(average 50%; Figure 4). Thus, VP does not indicate a particular level of stenosis. Moreover, similar to Qa, a relatively narrow artery will cause a delay in change followed by a more rapid increase in VP/MAP at critical stenosis.

Hence, as vessel diameters control the relationship between Qa or VP and stenosis, relatively narrow arteries dominate circuit resistance until stenosis is well advanced. Assuming stenosis progresses at a constant rate, Qa or VP may change so rapidly that monthly Qa or twice monthly VP measurements may not detect a change before thrombosis.

From a clinical standpoint, it would seem ideal for the clinician to know the access' diameter in relation to the rate of change of Qa. Yet, how would this be practically known, interpreted, and used? This highlights the complex relationship between vessel diameter and Qa and our lack of true understanding of the condition's natural history, a requisite in the criteria for using a screening test.

Hemodynamic variation during dialysis. Another issue affecting surveillance measurements is the large hemodynamic variation that occurs during dialysis when measurements are taken.[33] Consider that Qa is determined by MAP, central venous pressure (CVP), and vascular resistance of the access circuit (R) according to the following equation: $Qa = (MAP - CVP)/R$.[30] All three variables rapidly change during dialysis; therefore, Qa, the most common screening measurement, is unstable. Figure 5 shows large variations in MAP in two patients who were typical of a group of 51 patients.[33] These hemodynamic variations explain the large Qa changes that often occur within a single dialysis session (Figure 6), and these variations may be even greater when measured from session to session.[34–37] Thus, a change in Qa must be >33% in order to be significantly different from

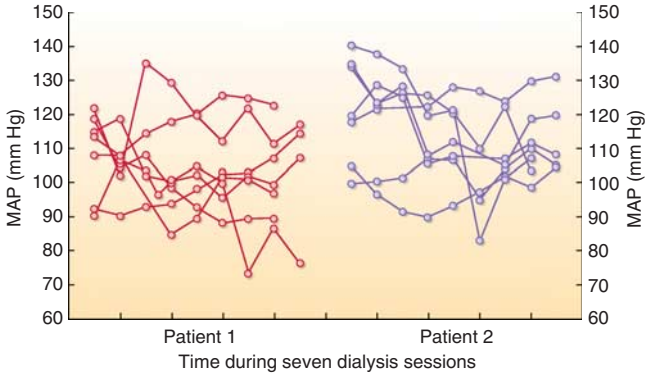

Figure 5 | Blood pressure varies widely within and between dialysis sessions. Mean arterial pressures (MAPs) of two representative patients with grafts during seven consecutive dialysis sessions (reprinted with permission from the National Kidney Foundation).[33] Note that large MAP variations occurred throughout the sessions.

background Qa variation at $P<0.05$.[34] A reduction in Qa of >33% may be an adequate justification for an intervention referral if the result is consistent with other clinical information.

Timing of surveillance measurements

The KDOQI[4] recommends that surveillance measurements be taken early in dialysis before ultrafiltration causes hemodynamic changes in Qa or VP. However, hemodynamic variation is generally not reduced early in a session;[33,34] hence, this approach may be flawed. Moreover, higher ultrafiltration volumes increase the risk of thrombosis in the period between sessions when thrombosis usually occurs.[38] Hence, one could argue that measurements should be taken late in a session when hemodynamics most closely approximate the period following a session. In our opinion, there are insufficient data to make a recommendation with regard to timing of measurements.

To summarize, in terms of the World Health Organization criteria, the conditions are imperfect. The needed latent period that allows time for intervention is not always available and the natural history of the access is not well understood. Given the patient characteristics (e.g., vessel sizes) and dialysis environments (e.g., hemodynamic variability), the screening test is not reliably reproducible. Even when surveillance threshold values are met, some accesses will not thrombose, whereas others will thrombose much earlier than the threshold value, highlighting our lack of understanding of the pathophysiology and our inability to appropriately intervene, in order to reliably prevent access thrombosis.

THE TESTS

The foregoing discussion indicates that access hemodynamics may impair the ability of Qa and VP measurements to detect stenosis and impair the ability to avoid false indications of stenosis. How do monthly Qa and twice monthly VP measurements hold up to the current standards for screening tests?[27,39]

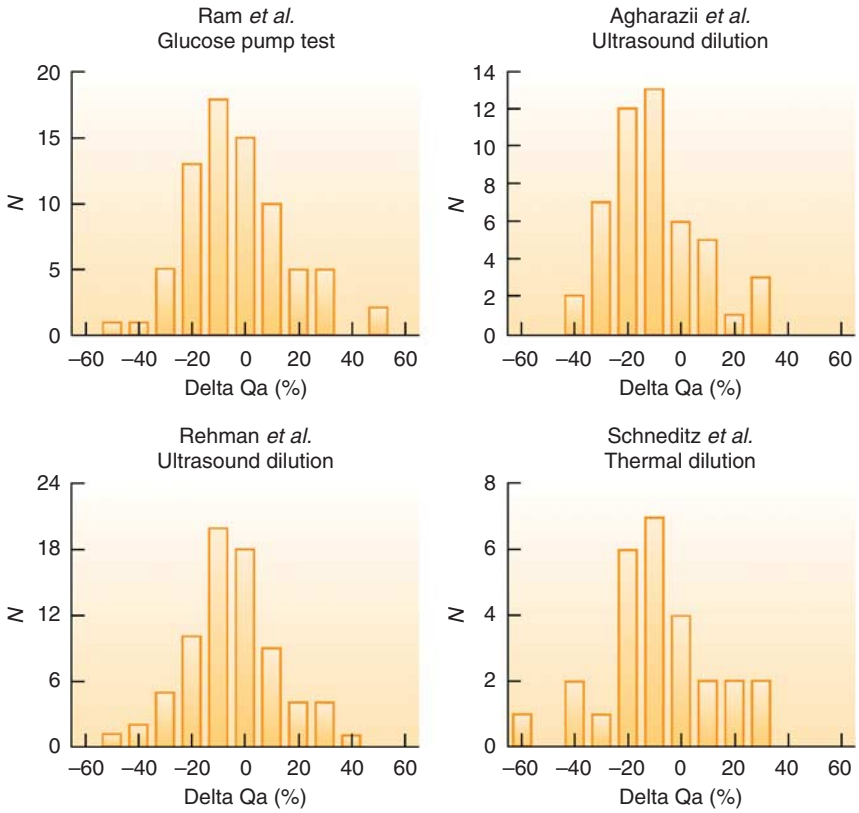

Figure 6 | Blood flow (Qa) varies widely within dialysis sessions. Histograms computed with data from four studies[34–37] show large percentage change in Qa (delta Qa) in grafts within single dialysis sessions.[34] Changes were measured over periods ranging from 1 to 3 h by three measurement methods. Positive values indicate decrease in flow; negative values indicate increase in blood flow. *N*, number.

Recommendations for screening tests are on a strong ground if they are based on randomized controlled trials in which a screened group is compared with a control group that undergoes usual care.[39] The problem with nonrandomized or observational studies is that, for a number of reasons, they are biased toward finding a treatment benefit.[40–42] For example, historical control groups may not be comparable to contemporary treatment groups that may benefit from recent improvements in care. Also, more interest and attention may be given to access issues in a treatment group when clinicians are aware that they are working with a new or potentially beneficial treatment under study.

The paradigm-altering studies[1,2] were not randomized controlled trials, and subsequent KDOQI surveillance recommendations[3] have been based upon studies that used historical or nonconcurrent control groups. Consequently, their statistical methods were not always appropriate for evaluating the usefulness of screening tests. For example, the influence of Qa on the relative risk of thrombosis was used to justify surveillance.[43–45] Although a low Qa is associated with an increased risk of thrombosis, this association does not ensure that the test has adequate accuracy in predicting thrombosis.

In contrast, when receiver operating characteristic (ROC) curves were used, Qa and VP surveillances were found to be inaccurate predictors of graft thrombosis.[46–50] For example, Ram *et al.*[50] studied 176 patients who underwent a total of 1957 monthly Qa measurements over 6 years. They evaluated the accuracy of monthly Qa measurements, or percentage decrease in Qa (ΔQa), in predicting thrombosis within the next month. They found that Qa had a sensitivity of 80% at a false-positive rate of 60% and ΔQa had a sensitivity of 81% at a false-positive rate of 50%. The mean Qa in grafts that did not thrombose the next month was 1345 ml/min (range 90–4000), and the mean in grafts that did thrombose was 895 ml/min (range 105–2115; Figure 7); hence, values overlapped widely. Moreover, the majority of thromboses were not preceded by a ΔQa measurement, usually because thrombosis occurred before a second measurement could be taken. Thus, this study did not support routine application of Qa surveillance to predict graft thrombosis.

It is important to avoid a 'one size fits all' approach to screening tests as the risks and benefits of tests may vary among different groups of patients and/or their accesses. Thus, surveillance guidelines should consider differences in risk of thrombosis. For example, Ram *et al.*[50] found that older grafts were unlikely to thrombose even at a low Qa, indicating that a low Qa is not always associated with a high risk of thrombosis. Older grafts with a large ΔQa were also unlikely to thrombose, suggesting that these large decreases

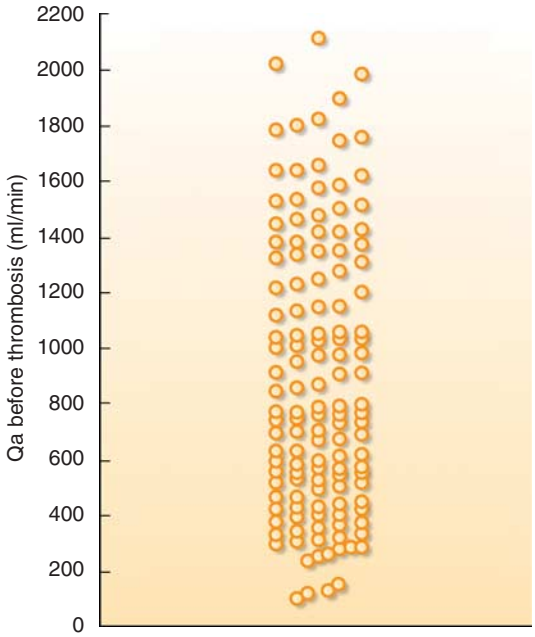

Figure 7 | Graft blood flow (Qa) values measured within 1 month before thrombosis (132 thromboses in 108 grafts). Many thromboses occurred at Qa values well above the Kidney Disease Outcomes Quality Initiative (KDOQI) referral threshold of <600 ml/min.[4] Database of ref. 50 was used to create the figure.

were likely caused by hemodynamic variation rather than by increased stenosis. On the other hand, new grafts were far more likely to thrombose in the near future.

Thus, the screening test should take into account the risks associated with a particular population. In this example, graft age should be considered when deciding whether to refer for intervention, and referrals should not be based solely upon a single Qa measurement. Further studies to determine high-risk populations and how the surveillance strategy performs in these populations will help focus the use of limited resources.

THE INTERVENTION

The goal of a surveillance strategy is to correct access dysfunction while avoiding significant harm. However, Ram *et al.*[50] showed that a high sensitivity in predicting thrombosis requires a high false-positive rate that likely yields many unnecessary intervention procedures. Furthermore, it has now been recognized that unnecessary angioplasty of a stable or slowly growing stenotic lesion may impair access survival. For example, Chang *et al.*[51] showed that angioplasty causes an increase in cellular proliferation that is associated with neointimal hyperplasia. In fact, angioplasty is used to induce vessel wall intimal hyperplasia and stenosis in order to study its pathophysiology.[52] Thus, surveillance-induced angioplasty may stimulate aggressive re-stenosis.[53–57] It is possible that the failure of surveillance to prolong access survival in randomized controlled trials has been caused by false-positive referrals with unnecessary angioplasty procedures. Thus, surveillance tests may fail the requirement that they do no harm.

Clarification of this issue will require standardization of angioplasty procedures and documentation of outcomes. However, it is unclear that such standardization has been achieved among interventional nephrologists, radiologists, and surgeons.[58,59] Furthermore, studies that evaluate surveillance should document the effectiveness of consequent angioplasty.[7] They should confirm that angioplasty has yielded improvements in both access anatomy and in the screening test measurements that have led to the intervention. Moreover, they should define what constitutes a successful angioplasty treatment. In the future, this may be guided by research that validates clinically important procedure-related outcomes.

In the meantime, new techniques are being evaluated that may limit the challenges posed by angioplasty-related re-stenosis and problematic elasticity of stenotic lesions. For example, stent grafts are based on the concept that treatment of stenosis with a stent covered with graft material may prevent elastic recoil and tissue in-growth. Such an approach appears to yield improved short-term patency of the stenotic lesion when compared with angioplasty alone,[60] particularly if they are heparin coated.[61] It is currently unknown whether the application of concepts borrowed from the cardiovascular literature on drug-eluting stents[62,63] to hemodialysis vascular access is valid. Early animal models of antiproliferative agents such as sirolimus and paclitaxel on grafts appear promising[64–66] but require further research. The ultimate role of stent grafts within the 'surveillance strategy' has not yet been defined. However, it appears they are susceptible to damage by cannulation during intervention procedures,[67] migration, and infection.[67] Finally, their cost-effectiveness has not been established.[68]

THE OUTCOMES

Tonelli *et al.*[15] published a systematic review and meta-analysis of available randomized controlled trials that have evaluated Qa or duplex ultrasound surveillance in fistulae ($n=4$) and grafts ($n=7$). The primary outcome was access thrombosis. In addition to the small number of studies, their quality was not high and statistical power was generally low.

Qa surveillance of fistulae was associated with a significantly reduced relative risk of thrombosis, but no significant improvement in fistula survival.[15] The positive result for fistula thrombosis should be considered tentative given that it is based upon only four studies of 360 subjects. By contrast, there was no evidence that graft surveillance by flow or duplex ultrasound reduced thrombosis or improved graft survival.

Some have argued that the randomized controlled trials had inadequate power and would have shown a surveillance benefit if they had included more patients.[7] However, given the foregoing factors that impair the success of surveillance, it seems unlikely that larger studies would have changed these negative outcomes. It is interesting that the

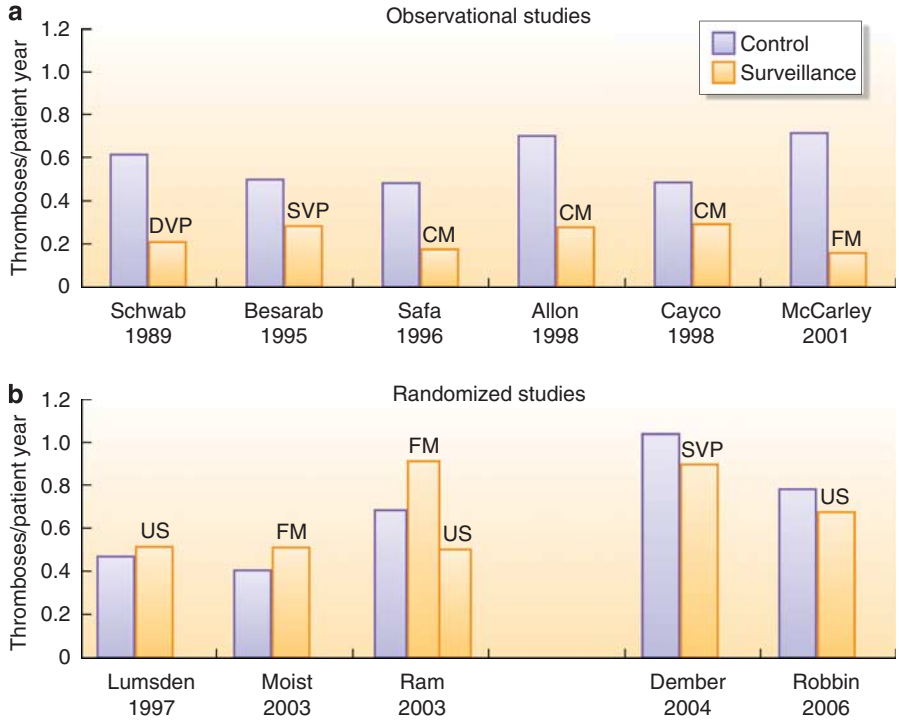

Figure 8 | Surveillance benefit disappears under rigorous conditions of randomized controlled trials (RCTs). (**a**) Nonrandomized studies that reported graft thrombosis rates during a historical control period and after implementing monitoring or surveillance. All studies reported that thrombosis decreased during monitoring or surveillance. (**b**) Rates of graft thrombosis in graft surveillance group and control group in RCTs. Thrombosis rates were not significantly different between groups. CM, clinical monitoring; DVP, dynamic venous pressure; FM, flow monitoring; SVP, static dialysis venous pressure; US, duplex ultrasound. Reprinted with permission from the American Society of Nephrology.[13]

paradigm-altering surveillance studies[1,2] were also underpowered. Nevertheless, the observed large improvements in access outcomes would suggest the presence of bias.

A comparison of the results of nonrandomized versus randomized studies further shows why randomized clinical trials are needed to limit bias and confounding when establishing guidelines. Allon[13] reviewed six nonrandomized studies of graft thrombosis that evaluated monitoring or surveillance by various methods. These studies evaluated a historical control period, followed by a monitoring or surveillance period in which grafts were referred for preemptive angioplasty when stenosis was suspected. All six studies reported a substantial reduction in thrombosis rates (Figure 8). In contrast, five randomized clinical trials found no reduction in thrombosis.

IMPLICATIONS OF A SURVEILLANCE MANDATE

Despite the recent data from randomized controlled trials, surveillance continues to be widely supported as a routine part of access maintenance. For example, ESRD Networks in the United States continue to promote access surveillance. Moreover, the Centers for Medicare and Medicaid Services (the US Government-sponsored insurance payer for dialysis) has issued new rules[69] that state 'The [dialysis] facility must have an ongoing program for vascular access monitoring *and* surveillance (italics added) for early detection of failure and to allow timely referral of patients for intervention when indications of significant stenosis are present.' This new requirement is intended to promote continuous quality improvement and reflect advances in technology and standard care practices.

However, the Centers for Medicare and Medicaid Services surveillance mandate has been criticized for not being evidence based[6,9] and may ultimately have the unintended effect of limiting quality improvement. At the core of quality improvement is ongoing research with reevaluation of standard practices as new knowledge becomes available. In the United States, however, the mandate has made it impossible to conduct the studies (e.g., randomized controlled trials) that are needed to determine the proper role of surveillance. Specifically, because of this mandate, dialysis units are unwilling to participate if patients are randomized into a control group that consists solely of clinical monitoring.[7] Loss of such a control group also means that we cannot properly study whether clinical monitoring has similar, or possibly even better, outcomes when compared with surveillance. If so, then failure to make this determination would lead to continued application of surveillance and a missed opportunity to avoid unnecessary costs, intervention procedures, and patient inconvenience.

Indeed, an important issue is the effect of the mandate on the overall cost of care. The Centers for Medicare and

Medicaid Services does not reimburse for the cost of screening tests. Thus, dialysis units are expected to bear these costs in the absence of strong evidence of efficacy. The cost of surveillance ultimately depends on whether it can prevent thrombosis and access loss, which is costly because of unscheduled intervention procedures, catheter use, and hospitalizations. There are limited data available on the costs of access surveillance. Some have reported reduced costs with Qa surveillance [70,71]; however, these analyses were based on observational or retrospective data and subject to the biases inherent in such studies. Duplex ultrasound surveillance of stenosis reduced costs in one randomized study,[72] but not in another.[73] Randomized trials with no reduction in thrombosis have reported that surveillance was associated with increased rates of intervention procedures,[54,74,75] which would be expected to increase costs. This result was supported by a Canadian cost analysis that was based on a simulation of results from two screening (surveillance) strategies.[76] They found that screening would not reduce expected access-related costs under any clinically plausible scenario, and many unnecessary angiography procedures would be required. This Canadian analysis was performed in an environment in which, unlike the USA, surveillance is not mandated and reimbursement is applied through a universal healthcare system. Thus, there is real concern that a surveillance mandate will increase costs without yielding significant benefits.

CURRENT OPTIONS

An ideal surveillance method should quickly, accurately, noninvasively, and economically evaluate access anatomy (eg., stenosis) and function. One basis of the surveillance controversy is that the tests that were originally selected for surveillance, Qa and VP, were surrogates for stenosis rather than direct measurements. Although these tests are associated with thrombosis, they lack the predictive accuracy needed to be the sole basis for intervention referrals. Thus, Qa and VP should be emphasized as ancillary tests to be used in combination with information obtained from clinical monitoring.

Qa and VP surveillance might improve outcomes if measurements are taken more frequently. A key issue is that patients have wide hemodynamic variation during dialysis; hence, the reproducibility of measurements from session to session is poor and may either fail to detect or falsely suggest stenosis. Moreover, Qa and VP may change so rapidly that there may be insufficient time to detect the change before thrombosis. More frequent measurements would allow calculation of average values that may neutralize hemodynamic variation and might make it easier to detect rapid changes in Qa or VP before thrombosis. However, Qa measurements take significant time; hence, it is probably impractical to increase measurement frequency unless there is adequate reimbursement for staff time. In contrast, online methods are available that allow frequently derived static VP measurements.[77] Our recommended approach to access maintenance is listed in Table 1.

Duplex ultrasound has the advantage of directly visualizing stenosis while providing flow and velocity measurements that help determine the physiological significance of stenosis. Thus, ultrasound may avoid inaccuracies inherent in surrogate measurements. However, the few available randomized controlled trials have not established whether duplex ultrasound can improve outcomes in grafts. Malik *et al.*[78] reported that duplex ultrasound surveillance prolonged graft survival, but their study is difficult to interpret because it lacks documentation such as thrombosis rates. Dossabhoy *et al.*[72] found that hospitalizations and cost of care were significantly reduced, but their study was a secondary analysis of a randomized trial. They only found a trend of reduced thrombosis, possibly because the study was underpowered. Finally, Robbin *et al.*[74] did not find a benefit; however, as ultrasound was only applied every 4 months, measurements might not have been frequent enough. Thus, further study by properly designed and powered randomized controlled trials with associated economic evaluations are needed to establish the role of duplex ultrasound, which requires far more skill than needed for Qa or VP measurements.

With regard to fistulae, although surveillance may potentially reduce thrombosis,[15] the major problem with

Table 1 | Recommendations for using Qa and VP measurements in access maintenance

1. Regular physical examination and clinical assessment are the keys to access maintenance.[90–92]
2. Measurements of Qa or static (or derived static) VP/MAP may be helpful ancillary tests that should be correlated with physical examination and other clinical data. They are not suited to be the sole basis for intervention referrals.
3. Because hemodynamic variability causes large changes in Qa and VP/MAP, it is important to confirm changes in measurements before making intervention referrals.
4. Fistula Qa $<$400–500 ml/min and graft Qa $<$600 ml/min are associated with stenosis; when such values are confirmed and correlated with clinical monitoring information, they may assist in deciding whether to make an intervention referral.[4]
Note: a decrease in Qa should be $>$33% to be statistically significant with $P<0.05$ (ref. 34); smaller decreases in Qa are often caused by hemodynamic variation rather than an increase in stenosis.
5. Trend analysis is key to using static VP/MAP to detect a significant stenosis. The traditional threshold of VP/MAP $>$0.50 (or derived static VP/MAP $>$0.55) is an unreliable indicator of stenosis and should not be the basis of an intervention referral.[31]
6. It may be best to direct surveillance resources to new accesses, which are most likely to thrombose and fail.[50]
7. Although it is often recommended that Qa and VP should be measured early in a dialysis session, there is no evidence to support this restriction.[50]

Abbreviations: MAP, mean arterial pressure; Qa, blood flow; VP, venous pressure.

fistulae is the high maturation failure rate, which surveillance cannot address.

As currently practiced, the 'access surveillance strategy' uses costly radiology resources and requires significant patient time and inconvenience.[76,79] We need to be able to define and target high risk populations for surveillance, find a validated test to predict those who will succeed or fail after an intervention,[80,81] and determine which interventions will facilitate increased access patency without harm. Targeting measurements to the newest accesses, which are most likely to fail,[50] appears to be a logical step. In contrast, attempts to restore patency after thrombosis of new grafts may be short lived and less successful than preemptive intervention.[81–83] Optimal timing of both screening and intervention is critical yet poorly established, and cannot be achieved without a better understanding of the underlying condition. This cannot occur without further research and validation, both of which are at risk because of seemingly unjustified mandates. It must be emphasized that all components of the access–surveillance strategy need to be securely established to be cost-effective.

Until further progress is made, monitoring by physical examination should be emphasized as a cost-effective and proven method to detect access abnormalities.[84–89] However, Besarab *et al.*[7] have observed that nephrologists and dialysis staff generally have limited knowledge of access anatomy and function, and regular physical examination of accesses is generally not carried out in dialysis units. This trend must be reversed by emphasizing proper vascular access training and clinical assessment in dialysis units.[90] Given that vascular access is the 'Achilles heel' of dialysis, it is understandable why physicians might be eager to adopt new treatments or strategies that seem hopeful without the necessary supportive evaluation from well-designed and conducted studies. It is incumbent on the nephrology community to incorporate a balanced, evidence-based scientific approach to vascular access care.

CONCLUSION

We understand the limitations of surveillance much better now than when the surveillance controversy began nearly two decades ago. Evaluation of the surveillance strategy by applying established criteria (e.g., World Health Organization)[27] shows little evidence that surveillance as currently practiced (e.g., monthly Qa measurements) provides a significant benefit. The current surveillance strategy (Figure 1) fails at each component.

Physical examination and clinical assessment are the keys to access maintenance and should be a part of the standard care of dialysis patients. Qa and VP measurements may be useful ancillary tests that can help confirm clinical suspicion of stenosis or access dysfunction. The goal should be to accurately identify those accesses that are most likely to benefit from a preemptive intervention, while avoiding procedures in accesses that are unlikely to benefit. However, this remains an elusive goal that will only be reached when properly designed studies are conducted.

DISCLOSURE

All the authors declared no competing interests.

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