Anatomical correlation of a well-functioning access graft for haemodialysis

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Abstract

Background. While it is widely accepted that vascular access (VA) haemodynamic surveillance should be performed, its real benefit has not yet been established. In addition, controversy still reigns over pre-emptive angioplasty of detected significant VA stenosis. This pilot study was designed to question the rationale of our decision-making process in VA management.

Methods. All 12 adult patients at our centre, dialyzed through a PTFE graft for over 3 months and meeting the clinical and haemodynamic criteria of a well-functioning VA [dynamic venous pressure (DVP) < 150 mmHg and VA flow (Qa) measurement > 800 ml/min], were prospectively selected. The selected patients underwent a baseline diagnostic angiogram at consent. No patient was submitted to endovascular intervention, including those with stenosis. Close clinical and haemodynamic follow-up monitoring were maintained over the next 6 months, using monthly Qa measurement through a haematocrit dilution (optodilutional) technique to record any access morbidity.

Results. The baseline diagnostic angiogram of the 12 patients revealed at the venous anastomosis: (i) stenosis reducing > 75% of the access lumen in three cases; (ii) stenosis of 50–75% in two cases; (iii) stenosis of 25–50% in four cases and (iv) no stenosis in three cases. One patient with stenosis > 75% and another with stenosis between 25 and 50% had graft thromboses during the follow-up period. Neither graft thrombosis could have been predicted from the previous month’s Qa evaluation. All four patients with a stenosis > 50% and who did not thrombose had normal Qa at the end of the follow-up period.

Conclusions. Our data suggest that the presence of what we call a significant stenosis does not necessarily correlate with measured Qa and might not be associated with early thrombosis deserving immediate intervention. Further studies are needed to clarify the best surveillance protocol and the role of pre-emptive intervention in significant stenosis.

Keywords: graft surveillance; haemodialysis; stenosis; thrombosis; vascular access flow

Introduction

Vascular access (VA) malfunction and failure is a leading cause of morbidity in chronic haemodialysis patients, responsible for over 25% of annual hospital admissions and, in the USA, estimated annual VA-related costs of USD 1 billion [1].

In haemodialysis VA management, just as in general medicine, early detection of malfunction and prevention of definite failure is considered the best approach to diminish morbidity and costs. This axiom was strongly suggested in several seminal studies [2,3] and is expressed in most guidelines of scientific societies in this field.

It is recommended that regular monitoring of access function should be performed, preferably by measuring VA flow (Qa) and when access stenosis is present, pre-emptive intervention should be performed percutaneously without further delay. In support of these level 2 recommendations, we can quote: ‘All types of pressure measurement should be abandoned in favour of access flow measurement,’ and ‘Monitoring plus intervention reduces thrombotic rates, morbidity and costs’ [3,4].

Consensual recommendations for pre-emptive intervention in malfunctioning grafts are (a) Qa measurement < 600 ml/min for grafts and (b) a Qa drop > 25% over two consecutive measurements [5].

However, recent and quite relevant information has questioned those recommendations, and scanning through recent prospective randomized controlled trials in this field reveals some discordant opinions.

No matter if we are looking at native fistulas or PTFE grafts, using only Qa measurements in association with Doppler studies or dynamic venous pressure as surveillance techniques, it is believed that VA stenosis is now very effectively detected, and responsible for a large increase in percutaneous VA procedures. Surprisingly, however, it has been found that all these diagnostic and therapeutic procedures fail to reduce the thrombosis rate, or prolong access longevity [6–13].
In fact, we do not really know if a VA defined by us as functioning well based on surveillance data would actually look normal in an angiography. This being the case, it is difficult to really appreciate the sensitivity and specificity of our monitoring indicators to predict and prevent thrombosis and, most of all, the meaning of stenosis in the natural history of each VA.

By default, we continue to monitor our patients’ VAs monthly and perform pre-emptive angiography whenever the values given by the surveillance technique do not meet pre-defined target values. All these diagnostic and intervention procedures are expensive to perform and do not have a well-proven benefit.

A pilot study was designed to question the rationale of our decision-making process in VA management.

Patients and methods

A pilot study was conducted aiming to determine (i) the anatomic correlations of a VA with normal clinical and haemodynamic parameters, and (ii) what happens to a VA with significant stenosis not detected by clinical or haemodynamic parameters.

We prospectively selected all 12 adult patients from a single dialysis unit (Dialysis Unit of Hospital Garcia de Orta), with polytetrafluoroethylene (PTFE) grafts in use for more than three months, appearing normal on physical examination [14], routinely monitored by Qa measurements, using a haematocrit dilution (optodilution) technique (Crit-line III[15], Hemametrics, Salt Lake City, UT, USA) [15], and meeting the following criteria: (a) Qa > 800 ml/min, with a variation of <25% over the last 2 monthly measurements, (b) dynamic venous pressure (DVP) < 150 mmHg, measured with 15G needles and a circuit blood flow (Qb) of 200 ml/min, and (c) allowing an effective circuit blood flow > 350 ml/min.

Seventeen percent of all patients in this unit were dialyzed through PTFE grafts, and 9% used tunnelled catheters.

The 12 patients in our study have an average age of 63 years (42–79) and had used their graft for 1217.5 days ± 786. 4 days with no previous intervention. Five patients (41.6%) were female, and none had diabetes mellitus or congestive heart failure.

Patients were informed about the procedure and gave their written consent. They then underwent a diagnostic angiogram in the week following baseline Qa measurement, performed always by the same experienced intervention nephrologist. The results were analysed by two independent observers, searching in particular for stenosis occluding >50% of the access lumen around the venous anastomosis. The arterial inflow, the graft itself and central vein drainage were also investigated in order to exclude other possible causes for low Qa and graft thrombosis.

No endovascular intervention was considered, independent of the angiogram results, as to all intents and purposes the grafts were well functioning.

All patients received close clinical and haemodynamic follow-up over the next 6 months, repeating Qa measurement monthly and recording any access morbidity. Measurements were performed within 30 min of the dialysis treatment, repeated twice and averaged. As Qa measurements with Crit-Line III are on average 73% of the transonic device, our threshold for selection of well-functioning grafts to go to angiography is higher, favouring normal findings in our baseline study [15,16].

We selected only PTFE grafts, as a recent series has shown the cost-benefit and quality improvement linked to surveillance to be limited to patients with AV grafts and not observed in patients with native fistulas [17]. In addition, the definition of a well-functioning graft is better standardized and its morbidity much higher, generating more events in a short follow-up period.

We did not elect to use Doppler/ultrasound studies as a surveillance tool in our protocol as access flow measurement usually outperformed duplex ultrasonography in predicting incipient thrombosis [12]. Although less invasive, duplex ultrasonography is extremely operator dependent and not as reproducible.

Results

At the beginning of the study, access flow was, as per protocol, > 800 ml/min, in all our 12 patients.

The baseline diagnostic angiogram revealed (i) a venous anastomosis stenosis reducing > 75% of the access lumen in three patients, (ii) a stenosis of 50–75% of the access lumen in two patients, (iii) a stenosis of 25–50% of the access lumen in four patients and (iv) no stenosis in three patients.

Two patients suffered a graft thrombosis during the follow-up period, but in neither of the two cases could it have been predicted from the previous monthly Qa determinations, which were 2565 and 2517 ml/min, respectively. Only one of these patients had a stenosis reducing > 75% of the access lumen.

Four of the five patients with a stenosis > 50% of the access lumen and no thrombosis had as their last Qa values at the end of the follow-up period 1950, 940, 2502 and 1653 ml/min, respectively.

It was found that none of the 12 patients had arterial inflow stenosis or central venous drainage problems.

There was no correlation between the degree of lumen stenosis and access flow. All three patients with lumen stenosis > 75% had a Qa measurement always > 1100 ml/min, and the one that thrombosed had a Qa of 2517 ml/min, 9 days before that event.

Table 1 summarizes the haemodynamic results, angiogram findings and patient outcomes.

We resolved both thromboses in the angiography suite, using pharmacomechanical thrombolysis. The major finding was stenosis of the venous anastomosis > 75% in both cases.

Discussion

All presently approved clinical guidelines recommend performing surveillance of VA quality and performance, aiming at early detection of access stenosis.
Table 1. Access haemodynamics, angiogram findings and outcomes

<table>
<thead>
<tr>
<th>Patients</th>
<th>First Qa (ml/min)</th>
<th>Last Qa (ml/min)</th>
<th>Angiogram</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>1830</td>
<td>1950</td>
<td>Stenosis &gt;75%</td>
<td>No problem</td>
</tr>
<tr>
<td>2</td>
<td>950</td>
<td>790</td>
<td>Stenosis 50–75%</td>
<td>No problem</td>
</tr>
<tr>
<td>3</td>
<td>1750</td>
<td>1620</td>
<td>No stenosis</td>
<td>No problem</td>
</tr>
<tr>
<td>4</td>
<td>950</td>
<td>910</td>
<td>Stenosis 25–50%</td>
<td>No problem</td>
</tr>
<tr>
<td>5</td>
<td>950</td>
<td>1300</td>
<td>No stenosis</td>
<td>No problem</td>
</tr>
<tr>
<td>6</td>
<td>1403</td>
<td>989</td>
<td>Stenosis 25–50%</td>
<td>No problem</td>
</tr>
<tr>
<td>7</td>
<td>1481</td>
<td>1196</td>
<td>Stenosis 25–50%</td>
<td>No problem</td>
</tr>
<tr>
<td>8</td>
<td>1900</td>
<td>2502</td>
<td>Stenosis 50–75%</td>
<td>No problem</td>
</tr>
<tr>
<td>9</td>
<td>1362</td>
<td>1433</td>
<td>No stenosis</td>
<td>No problem</td>
</tr>
<tr>
<td>10</td>
<td>1290</td>
<td>1653</td>
<td>Stenosis &gt;75%</td>
<td>PTA fourth month</td>
</tr>
<tr>
<td>11</td>
<td>1730</td>
<td>2565</td>
<td>Stenosis 25–50%</td>
<td>Thrombosis sixth month</td>
</tr>
<tr>
<td>12</td>
<td>1199</td>
<td>2517</td>
<td>Stenosis &gt;75%</td>
<td>Thrombosis third month</td>
</tr>
</tbody>
</table>

This is the most prevalent anatomic defect in VAs and considered to be the direct cause of >85% of all VA thrombosis and definite failure. If a stenotic lesion is detected, the next step usually is pre-emptive percutaneous endovascular intervention to repair it.

This recommendation assumes Paulson’s Dysfunction Hypothesis, which states that stenosis (i) is the dysfunction that always leads to thrombosis, (ii) progresses at a slow and predictable rate, giving enough time to detect dysfunction to further intervene before thrombosis occurs, (iii) trends can be accurately detected from Qa, as Qa measurement of access dysfunction is reproducible, (iv) accesses that will thrombose had prior dysfunction and those that do not thrombose had no prior dysfunction, and finally, (v) there are no factors other than the abovementioned implicated in thrombosis [18].

The literature of the last decade abundantly supports the fact that we have developed fairly accurate tools to predict the presence of stenosis and quite effective ways to correct it percutaneously. Unfortunately, however, our capability to predict and prevent thrombosis and therefore prolong access survival remains dismal [6–13].

The key measurement of effective surveillance is the avoidance of thrombosis; no surrogate to this is acceptable.

A successful surveillance programme should aim to reduce thrombosis rate by an amount similar to the angioplasty rate it induces. Evidence shows that we are certainly still lagging well behind this desideratum.

Confirming our worst fears, our short pilot study suggests that almost half of the grafts considered optimal by standard monitoring surveillance criteria have what is usually considered a significant stenosis, deserving immediate intervention. On the other hand, what we consider a significant stenosis has neither a direct correlation with the measured Qa nor an accurate thrombosis prediction capacity. Indeed, thrombosis occurred unexpectedly in two cases.

Both our protocol and our daily practice use a 50% stenosis as the threshold for endovascular intervention. This is the most commonly adopted figure, in line with the recommendations of the updated 2006 NKF K/DOQI guidelines and the 2007 European Best Practice Guidelines. These define a haemodynamically significant stenosis as a reduction in normal vessel diameter ≥50%, accompanied by a haemodynamic, clinical or functional abnormality (CPG 4) [19]. In this study, we do not address the question of whether a 50% stenosis should be dilated, but rather the accuracy of our present surveillance guidelines and what should trigger referring a patient to angiography.

Apparently, not all stenoses are alike. Their progression is erratic and not all of them carry the same risk of thrombosis. It was found that neither the anatomical degree of stenosis nor access blood flow was a predictor of thrombosis occurrence, meaning that we are probably missing other, more determinant, characteristics of the stenotic lesions.

We would like to know the sensitivity and specificity of our haemodynamic surveillance criteria to predict thrombosis, and the sensitivity and specificity of stenosis found in an angiogram to predict thrombosis. We suggest that determining the sensitivity and specificity of haemodynamic parameters to detect stenosis is probably not very useful information; there are a lot of false negatives, meaning low sensitivity.

Our pilot study certainly recommends a full scale study to investigate the natural history of a VA from its beginning, and how monitoring or pre-emptive intervention can change its fate and at what cost.

Until then, we remain with prospective trend analysis of our favourite haemodynamic parameter, always correlated with VA physical examination, looking for persistent swelling of the arm, presence of collateral veins, or altered characteristics of its pulse or thrill.

Conflict of interest statement: None declared.
References


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