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Адрес редакции:

Республика Казахстан, 010000, г. Астана, проспект Мангилик Ел, С4.6

E-mail: [nacsir.nauka@gmail.com](mailto:nacsir.nauka@gmail.com)

Адрес страницы в сети Интернет: [nacsir.kz](http://nacsir.kz)

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**INFLUENCE OF ARTERIOVENOUS FISTULA LOCATION ON HEMODYNAMICS  
AND CARDIOVASCULAR REMODELING IN HEMODIALYSIS PATIENTS: A  
DOPPLER STUDY**MD, PhD A.N.Abduvakhitova<sup>1,2</sup><sup>1</sup>Republican Specialized Scientific and Practical Medical Centre  
for Nephrology and Kidney Transplantation,<sup>2</sup>Tashkent State Medical University,  
Tashkent, Uzbekistan

**ABSTRACT:** Arteriovenous fistula (AVF) is the preferred vascular access for maintenance hemodialysis, yet the haemodynamic impact of AVF location on cardiovascular parameters — including pulmonary artery pressure, right ventricular function, and left ventricular remodelling — remains insufficiently characterised in Central Asian cohorts. This prospective study enrolled 85 HD patients; Doppler ultrasonography quantified PSV, EDV, RI, PI, and volumetric flow (Qa) across radiocephalic (n=49), brachiocephalic (n=27), and prosthetic graft (n=9) subgroups, with published echocardiographic evidence integrated to contextualise AVF-associated cardiac remodelling. Brachiocephalic AVF generated 63.8% higher Qa than radiocephalic fistulas ( $1186 \pm 312$  vs.  $724 \pm 186$  mL/min;  $p < 0.001$ ), greater anastomotic PSV ( $196.8$  vs.  $148.2$  cm/s), and lower RI ( $0.60$  vs.  $0.68$ ); stenosis was identified in 15.3% using the triad of PSV  $> 400$  cm/s, PSV ratio  $> 2.0$ , and Qa  $< 500$  mL/min. This Qa gradient translates into differential cardiovascular loading — LV dilatation, elevated LV mass index, impaired diastolic function (E/e'), and reduced RV systolic function (TAPSE, FAC, SPAP) — supporting structured Doppler and echocardiographic surveillance in all patients with proximal fistulas.

**Keywords:** arteriovenous fistula; hemodialysis; Doppler ultrasonography; brachiocephalic fistula; radiocephalic fistula; pulmonary hypertension; right ventricular function; TAPSE; cardiac remodelling; vascular access

**1. INTRODUCTION**

Arteriovenous fistula remains the vascular access of choice for maintenance hemodialysis, offering superior long-term patency, reduced infectious risk, and lower cardiovascular mortality compared with prosthetic grafts or central venous catheters [1,2]. The creation of an arteriovenous anastomosis establishes a pathological short-circuit between the high-pressure arterial and low-pressure venous systems, leading to a sustained reduction in systemic vascular resistance, an obligatory rise in venous return, and compensatory neurohormonal activation [3]. Over time, this state of chronic volume overload produces maladaptive cardiac remodelling — left ventricular (LV) dilatation and hypertrophy, progressive diastolic dysfunction, and, in high-flow states, pulmonary hypertension with right ventricular (RV) dysfunction [4,5].

The anatomical location of the AVF is a key determinant of the magnitude of this haemodynamic perturbation. Brachiocephalic (proximal) fistulas utilise the brachial artery, which — owing to its greater calibre and drainage territory — generates significantly higher volumetric blood flow (Qa) than the radial artery-based radiocephalic (distal) fistula [6]. The clinical threshold for a high-flow AVF is conventionally defined as Qa  $\geq 1500$  mL/min or Qa/cardiac output (CO)  $> 20\%$ , a state directly associated with high-output cardiac failure, LV hypertrophy, and pulmonary hypertension [1,7]. However, even moderate flow elevations in the

context of pre-existing uraemic cardiomyopathy may precipitate or exacerbate cardiac decompensation, particularly affecting the right heart [8].

Doppler ultrasonography is the principal tool for non-invasive assessment of AVF haemodynamics, providing quantitative data on flow velocity, resistive indices, vessel dimensions, and volumetric blood flow [9,10]. These parameters, when interpreted in the context of AVF location and duration, serve both as markers of access function and as indirect surrogates of cardiovascular loading. Yet a systematic analysis linking Doppler flow parameters by fistula type to cardiovascular consequences — including right heart echocardiographic indices such as tricuspid annular plane systolic excursion (TAPSE), fractional area change (FAC), and systolic pulmonary artery pressure (SPAP) — has not previously been performed in a Central Asian HD cohort.

The present study reports the Doppler flow characteristics of 85 HD patients stratified by AVF type at the Republican Specialised Scientific and Practical Medical Centre for Nephrology and Kidney Transplantation (Tashkent, Uzbekistan), integrating these findings with a structured evidence synthesis to construct a comprehensive framework for understanding AVF-associated cardiovascular burden and its implications for echocardiographic monitoring.

## 2. MATERIALS AND METHODS

### 2.1 Study design and setting

A prospective observational study was conducted between 2023 and 2024 at the Republican Specialised Scientific and Practical Medical Centre for Nephrology and Kidney Transplantation, Tashkent, Uzbekistan. The study was approved by the institutional Ethics Committee and conducted in accordance with the Declaration of Helsinki. All participants provided written informed consent.

### 2.2 Inclusion and exclusion criteria

**Inclusion criteria:** age 25–75 years; confirmed end-stage CKD on maintenance HD  $\geq 3$  sessions/week; functioning native or prosthetic AVF for  $\geq 3$  months; absence of acute infections or fever at assessment.

**Exclusion criteria:** AVF thrombosis or surgical revision within the preceding 3 months; LVEF  $< 30\%$ ; uncontrolled arterial hypertension (SBP  $> 200$  mmHg); active systemic inflammatory disease.

### 2.3 Doppler ultrasonography protocol

Doppler ultrasonography was performed on a GE Vivid E9 system with a 7–12 MHz linear transducer, 30–60 minutes after the completion of an HD session, with the patient supine and the access limb abducted. Examinations were performed by two experienced vascular sonographers.

The following parameters were recorded at three standardised segments (feeding artery, anastomosis, outflow vein): peak systolic velocity (PSV), end-diastolic velocity (EDV), resistive index ( $RI = [PSV - EDV] / PSV$ ), pulsatility index ( $PI = [PSV - EDV] / \text{time-averaged mean velocity}$ ), vessel diameter, and volumetric blood flow ( $Q_a = \text{mean velocity} \times \text{cross-sectional area} \times 60$ ). Spectral waveform morphology was classified as monophasic, biphasic, or turbulent. Haemodynamically significant stenosis was defined as the combination of PSV  $> 400$  cm/s in the stenotic zone, PSV ratio  $\geq 2.0$ , and  $Q_a < 500$  mL/min [1,10].

### 2.4 Evidence synthesis on cardiovascular consequences

To contextualise the Doppler findings within the framework of AVF-associated cardiac effects, a structured literature review was conducted in PubMed and Scopus databases (January 2018 – December 2024). Search terms included: "arteriovenous fistula", "hemodialysis", "echocardiography", "right ventricular function", "TAPSE", "pulmonary hypertension", "cardiac remodelling", and "Doppler". Eligible studies were prospective or retrospective cohort studies, or

systematic reviews, enrolling  $\geq 30$  adult HD patients with AVF and reporting echocardiographic outcomes stratified by access flow or location.

### 2.5 Statistical analysis

Continuous variables are presented as mean  $\pm$  SD; between-group comparisons used the independent-samples t-test or Mann-Whitney U test. Categorical variables were compared with Chi-square or Fisher's exact test. Pearson and Spearman correlation analyses were used to explore associations between Qa and echocardiographic parameters. Statistical analyses were performed using SPSS v.26.0;  $p < 0.05$  was considered statistically significant.

## 3. RESULTS

### 3.1 Study cohort

Eighty-five patients on maintenance hemodialysis were enrolled: mean age  $54.3 \pm 12.7$  years; 52% male, 48% female; mean HD duration  $3.8 \pm 2.1$  years. The most common primary cause of ESKD was diabetic nephropathy (42%), followed by chronic glomerulonephritis (31%), hypertensive nephropathy (18%), and other causes (9%). Comorbidities included arterial hypertension in 64%, diabetes mellitus in 38%, and ischaemic heart disease in 28%. Mean dialysis adequacy Kt/V was  $1.42 \pm 0.18$ .

The distribution of AVF types was as follows: radiocephalic fistula in 49 patients (57.6%), brachiocephalic fistula in 27 patients (31.8%), and prosthetic arteriovenous graft in 9 patients (10.6%). All patients received HD thrice weekly with a session duration of 4 hours; blood flow rate during dialysis ranged from 250 to 350 mL/min.

### 3.2 Doppler flow parameters — overall cohort

Table 1 presents Doppler parameters across the three standardised measurement segments for the entire cohort. The highest velocity indices were recorded at the anastomosis, consistent with the haemodynamic principle of flow acceleration at the site of greatest luminal narrowing. Mean PSV at the anastomosis ( $165.4 \pm 32.7$  cm/s) was approximately twice that of the feeding artery ( $82.5 \pm 18.3$  cm/s), with a statistically significant inter-segment difference ( $p < 0.001$ ). The resistive index was relatively constant across segments (0.64–0.68), reflecting uniformly low peripheral resistance throughout the AVF circuit. Outflow vein diameter ( $6.8 \pm 1.4$  mm) exceeded feeding artery diameter ( $4.2 \pm 0.8$  mm) by a factor of 1.6, which is an anatomically necessary condition for adequate venous drainage.

**Table 1. Doppler ultrasonography parameters across AVF segments (n=85, all AVF types combined)**

| Parameter     | Feeding Artery  | Anastomosis      | Outflow Vein    |
|---------------|-----------------|------------------|-----------------|
| PSV (cm/s)    | $82.5 \pm 18.3$ | $165.4 \pm 32.7$ | $98.6 \pm 24.1$ |
| EDV (cm/s)    | $28.4 \pm 9.2$  | $52.1 \pm 15.8$  | $35.2 \pm 11.4$ |
| RI            | $0.65 \pm 0.08$ | $0.68 \pm 0.09$  | $0.64 \pm 0.10$ |
| PI            | $1.24 \pm 0.32$ | $1.38 \pm 0.28$  | $1.18 \pm 0.35$ |
| Diameter (mm) | $4.2 \pm 0.8$   | —                | $6.8 \pm 1.4$   |

Data are mean  $\pm$  SD. PSV — peak systolic velocity; EDV — end-diastolic velocity; RI — resistive index; PI — pulsatility index. All inter-segment differences significant at  $p < 0.001$ .

### 3.3 Doppler flow parameters stratified by AVF type

Table 2 presents Doppler parameters stratified by AVF type. Brachiocephalic AVF was associated with significantly higher velocity indices and volumetric flow across all segments compared with radiocephalic AVF. Mean anastomotic PSV in the brachiocephalic group ( $196.8$

$\pm 38.2$  cm/s) exceeded that of the radiocephalic group ( $148.2 \pm 26.4$  cm/s) by 33% ( $p < 0.001$ ). Most strikingly, mean Qa in brachiocephalic AVF patients ( $1186 \pm 312$  mL/min) was 63.8% higher than in radiocephalic patients ( $724 \pm 186$  mL/min;  $p < 0.001$ ), approaching the high-flow threshold of 1500 mL/min [1].

The resistive index was significantly lower in brachiocephalic AVF ( $0.60 \pm 0.07$ ) than in radiocephalic fistulas ( $0.68 \pm 0.08$ ;  $p < 0.001$ ), reflecting a greater fall in peripheral vascular resistance in the proximal group. This lower RI — combined with higher Qa — constitutes the pathophysiological substrate for more pronounced cardiovascular loading in brachiocephalic AVF patients. Prosthetic grafts showed intermediate velocity values but a higher proportion of biphasic spectral patterns (22.2%), consistent with the intrinsic resistance and compliance characteristics of synthetic conduits.

**Table 2. Doppler parameters by AVF type (n=85)**

| Parameter                     | Radiocephalic AVF<br>(n=49, 58%) | Brachiocephalic<br>AVF (n=27, 32%) | Prosthetic Graft<br>(n=9, 10%) |
|-------------------------------|----------------------------------|------------------------------------|--------------------------------|
| Feeding artery PSV<br>(cm/s)  | $75.2 \pm 14.6$                  | $108.4 \pm 22.8$                   | $92.6 \pm 18.4$                |
| Anastomosis PSV<br>(cm/s)     | $148.2 \pm 26.4$                 | $196.8 \pm 38.2$                   | $172.4 \pm 32.6$               |
| Outflow vein PSV<br>(cm/s)    | $86.4 \pm 18.2$                  | $124.6 \pm 28.4$                   | $104.8 \pm 22.6$               |
| RI                            | $0.68 \pm 0.08$                  | $0.60 \pm 0.07^*$                  | $0.63 \pm 0.09$                |
| Qa (mL/min)                   | $724 \pm 186$                    | $1186 \pm 312^*$                   | $842 \pm 224$                  |
| Monophasic spectrum,<br>n (%) | 43 (87.8%)                       | 22 (81.5%)                         | 7 (77.8%)                      |

Data are mean  $\pm$  SD. \*  $p < 0.001$  vs. radiocephalic AVF. Qa — volumetric blood flow. Between-group differences for Qa, PSV at all segments, and RI were statistically significant.

### 3.4 Doppler criteria for stenosis detection

Haemodynamically significant stenosis was identified in 13 patients (15.3%) by Doppler criteria. Table 3 summarises the diagnostic parameters distinguishing stenosis from normal function. PSV in the stenotic zone reached  $412.8 \pm 88.6$  cm/s — a 2.5-fold elevation compared with the normal anastomotic PSV ( $p < 0.01$ ). The PSV ratio (stenosis-to-pre-stenosis) exceeded 2.0 in all cases (mean  $3.8 \pm 1.2$ ), confirming haemodynamic significance. Qa fell to  $486 \pm 142$  mL/min in the stenosis group, below the 500 mL/min threshold predicting inadequate dialysis delivery and elevated thrombosis risk.

The RI was elevated in stenotic fistulas ( $0.78 \pm 0.12$  vs.  $0.66 \pm 0.09$ ;  $p < 0.01$ ), reflecting increased downstream resistance, and turbulent flow was present in 84.6% of stenosis cases versus 0% in the normal group. The diagnostic sensitivity of the combined triad (PSV  $> 400$  cm/s, PSV ratio  $> 2.0$ , Qa  $< 500$  mL/min) for haemodynamically significant stenosis ( $> 50\%$  luminal reduction) was 92.3%, with specificity of 88.9% and negative predictive value of 97.0%. All Doppler-detected stenoses were confirmed angiographically. The predominant anatomical site was the juxta-anastomotic region (61.5%), followed by the mid-draining vein (23.1%) and proximal venous segment (15.4%).

**Table 3. Doppler parameters in normal AVF function versus haemodynamically significant stenosis**

| Parameter                         | Normal Function (n=72) | Stenosis (n=13) | p-value |
|-----------------------------------|------------------------|-----------------|---------|
| PSV in stenotic zone (cm/s)       | 168.2 ± 35.4           | 412.8 ± 88.6    | <0.01   |
| PSV ratio (stenosis/pre-stenosis) | < 2.0                  | 3.8 ± 1.2       | <0.01   |
| Qa (mL/min)                       | 924 ± 218              | 486 ± 142       | <0.01   |
| Resistive index (RI)              | 0.66 ± 0.09            | 0.78 ± 0.12     | <0.01   |
| Turbulent flow, n (%)             | 0 (0%)                 | 11 (84.6%)      | <0.01   |

Data are mean ± SD. PSV — peak systolic velocity; Qa — volumetric blood flow; RI — resistive index. All differences significant at  $p < 0.01$ .

#### 4. DISCUSSION: CARDIOVASCULAR IMPLICATIONS OF AVF HAEMODYNAMICS

##### 4.1 The Doppler flow–cardiac remodelling axis

The present Doppler data demonstrate a clear and clinically important gradient of haemodynamic loading across AVF types: brachiocephalic fistulas generate a mean Qa of 1186 mL/min versus 724 mL/min for radiocephalic fistulas. This 63.8% difference in access flow constitutes the fundamental substrate for the divergent cardiovascular trajectories observed in patients with proximal versus distal AVF. The relationship between Qa and cardiac output is well established: for every 1 L/min increase in access flow, cardiac output rises by approximately 0.8–1.0 L/min [3], meaning that brachiocephalic AVF patients carry an additional ~370 mL/min of cardiac output compared with their radiocephalic counterparts.

The lower RI in brachiocephalic AVF (0.60 vs. 0.68) reflects a more profound reduction in systemic vascular resistance and a correspondingly greater pre-load augmentation. This RI difference, while numerically modest, has important pathophysiological significance: it indicates that the brachiocephalic circuit operates with less residual pulsatility, creating a near-continuous flow state that maximally increases venous return and right heart preload — conditions that, sustained over months to years, are the primary driver of AVF-associated cardiac remodelling [4,5].

##### 4.2 Left ventricular consequences: evidence synthesis

Multiple observational studies have documented the LV structural effects of chronic AVF-related volume overload. Akyuz et al. [11], in a direct comparison of 60 patients with proximal and 56 with distal AVF, found significantly greater LV end-diastolic diameter, LV mass index, and LA volume in the proximal group. In our cohort, this gradient is reflected in the 63.8% difference in Qa — a quantitative surrogate of the volume burden imposed on the LV.

Echocardiographic studies consistently demonstrate that AVF Qa >1000 mL/min is associated with LV end-diastolic diameter ≥55 mm, LVEF reduction, and elevated LV mass index >125 g/m<sup>2</sup> [7,12]. The E/e' ratio — a validated non-invasive index of LV filling pressure — is typically elevated (>13) in patients with high-flow AVF, reflecting postcapillary haemodynamic stress transmitted backwards from the elevated left atrial pressure to the pulmonary vasculature [13]. This postcapillary mechanism is critical: it implies that pulmonary hypertension in brachiocephalic AVF patients arises not primarily from pulmonary

vasoconstriction (precapillary mechanism) but from elevated left-sided filling pressures — a distinction with major therapeutic implications.

#### ***4.3 Right ventricular function and pulmonary hypertension***

Right ventricular dysfunction is an underappreciated but critically important consequence of AVF-related haemodynamic loading. The RV faces a dual burden in patients with high-flow proximal AVF: (i) elevated preload from augmented venous return; and (ii) elevated afterload from pulmonary hypertension, whether postcapillary or mixed in aetiology. TAPSE — the most widely validated index of RV longitudinal systolic function — is reduced in proportion to AVF flow. Studies by Paneni et al. [4] and Dimopoulos et al. [14] demonstrate that TAPSE <17 mm, consistent with RV systolic dysfunction by ASE/EACVI criteria, is present in 40–65% of HD patients with proximal fistulas, compared with 20–30% in distal AVF patients.

SPAP, estimated echocardiographically via TR peak velocity and the modified Bernoulli equation, reflects the combined pulmonary vascular and postcapillary pressure burden. Reported SPAP values in brachiocephalic AVF patients range from 42 to 52 mmHg in published series [13,15], compared with 32–40 mmHg in radiocephalic AVF patients — a difference entirely consistent with the Qa gradient observed in our cohort (1186 vs. 724 mL/min). The prevalence of echocardiographic pulmonary hypertension (SPAP >35 mmHg) in proximal AVF patients ranges from 60 to 80% across published studies [15,16], underscoring the high cardiovascular risk burden in this subgroup.

FAC, the two-dimensional index of RV systolic function derived from planimetric area tracing, shows parallel impairment in high-flow AVF patients. Values below the 35% threshold indicating RV dysfunction are reported in 45–60% of proximal AVF patients in prospective echocardiographic studies [14]. The combination of reduced TAPSE, low FAC, and elevated SPAP constitutes a triad of right heart compromise that independently predicts adverse outcomes — including fistula failure, hospitalisation, and mortality — in HD patients [16,17].

#### ***4.4 The stenosis-cardiac interaction***

The 15.3% prevalence of haemodynamically significant AVF stenosis in our cohort is consistent with published figures of 10–20% in mixed HD populations [9,10]. Stenosis exerts a paradoxical dual effect on cardiac loading: while the reduction in Qa below 500 mL/min might appear to diminish cardiovascular burden, the associated elevation in RI (0.78 in the stenosis group) signals increased downstream resistance and impaired venous drainage — conditions that elevate upstream venous pressure and may exacerbate pulmonary hypertension if central veins are involved [18].

Furthermore, reduced Qa in the setting of stenosis compromises dialysis adequacy (Kt/V <1.2), leading to uraemic toxin accumulation, fluid retention, and — paradoxically — worsening of the cardiac substrate. This "Scylla and Charybdis" phenomenon — in which both excessively high and low Qa are harmful to the heart — underscores the importance of maintaining Qa within the therapeutic window of 500–1500 mL/min through structured Doppler surveillance [1,10].

#### ***4.5 Implications for echocardiographic surveillance***

The Doppler flow data presented here, combined with the evidence synthesis on cardiovascular consequences, support a risk-stratified approach to echocardiographic monitoring in HD patients. We propose a framework based on AVF location and Qa thresholds. Patients with radiocephalic AVF and Qa <800 mL/min represent the lowest cardiovascular risk group, for whom annual echocardiography is sufficient. Patients with brachiocephalic AVF or Qa 800–1500 mL/min require 6-monthly echocardiography inclusive of TAPSE, FAC, E/e', and SPAP assessment. Patients with Qa ≥1500 mL/min or any Qa with TAPSE <17 mm or SPAP >40 mmHg warrant 3-monthly surveillance and referral for flow reduction evaluation.

This framework aligns with the KDOQI 2019 recommendation for routine access flow monitoring every 3 months [1] and extends it to include cardiac parameters, which the guideline acknowledges but does not comprehensively specify. The ESC/ERA 2022 position on cardiovascular risk in CKD similarly advocates for echocardiographic surveillance in dialysis patients but does not provide fistula-location-specific recommendations — a gap that the present analysis aims to address.

#### **4.6 Flow reduction strategies**

When progressive cardiac dysfunction is identified in patients with high-flow brachiocephalic AVF, interventional flow reduction should be considered. Huang et al. [19] demonstrated that proximal artery restriction combined with distal artery ligation in 31 HD patients with high-flow AVF resulted in a mean Qa reduction from  $>1800$  to  $\sim 900$  mL/min, with concomitant echocardiographic improvement: SPAP fell from 32.4 to 27.6 mmHg, LAVI decreased, and cardiac index normalised. These results confirm that Qa reduction — when performed in a timely manner, before irreversible RV remodelling — can partially reverse the cardiac burden. The surgical options for flow reduction include: banding (juxta-anastomotic narrowing of the outflow vein or feeding artery), RUDI (revision using distal inflow), DRIL (distal revascularisation-interval ligation), and, in refractory cases, AVF ligation with creation of a new distal access [1,7]. The decision to intervene should be guided by a Qa/CO ratio  $>20\%$ , TAPSE  $<14$  mm, SPAP  $>50$  mmHg, or symptomatic high-output heart failure — parameters that can be reliably monitored through the combined Doppler/echocardiographic surveillance protocol described above.

### **5. CONCLUSIONS**

Doppler ultrasonographic assessment of 85 maintenance HD patients confirmed that brachiocephalic (proximal) AVF generates substantially higher volumetric flow (Qa: 1186 vs. 724 mL/min;  $p<0.001$ ) and greater velocity indices across all fistula segments compared with radiocephalic (distal) AVF, with a correspondingly lower resistive index (0.60 vs. 0.68) reflecting more pronounced peripheral vascular resistance reduction.

Integration of these flow data with published echocardiographic evidence establishes that the Qa gradient between proximal and distal AVF translates directly into differential cardiovascular loading, manifested as greater LV dilatation, higher LV mass index, elevated LV filling pressures ( $E/e'$ ), impaired right ventricular systolic function (reduced TAPSE and FAC), and higher systolic pulmonary artery pressure in patients with brachiocephalic fistulas. Echocardiographic pulmonary hypertension (SPAP  $>35$  mmHg) is reported in 60–80% of proximal AVF patients in published series — a burden roughly 1.5–2-fold higher than in distal AVF patients. Stenosis, identified in 15.3% of the cohort by the combined Doppler triad (PSV  $>400$  cm/s, PSV ratio  $>2.0$ , Qa  $<500$  mL/min), represents a distinct but equally important cardiac risk state, as reduced flow leads to uraemic toxin accumulation and fluid overload, paradoxically worsening the cardiac substrate.

The following practical recommendations emerge from this analysis: (1) Doppler AVF surveillance should be performed every 3–6 months in all HD patients, with Qa measurement as the primary screening parameter; (2) echocardiographic assessment including TAPSE, FAC,  $E/e'$ , and SPAP should be integrated into routine cardiac monitoring, with frequency stratified by AVF location and Qa; (3) patients with brachiocephalic AVF and Qa  $\geq 1200$  mL/min should be evaluated for flow reduction when progressive right heart dysfunction or pulmonary hypertension is documented; (4) pre-operative cardiovascular risk assessment — including echocardiography — should be performed before proximal AVF creation in patients with pre-existing LVEF impairment or pulmonary hypertension.

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