Opinion

When Is Arteriovenous Fistula Dangerous for Hemodialysis Patients?

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Abstract: Hemodialysis arteriovenous fistula is a shortcut of the systemic circulation and sometimes fistula flow exceeds 2 L/min. Possible hemodynamic and clinical consequences are discussed.

Keywords: arteriovenous fistula; heart failure; pulmonary hypertension

1. Introduction

Functioning vascular access is a condition of functioning hemodialysis. Nowadays, the following three types of access into the blood stream are used: hemodialysis catheter, naive arteriovenous fistula (AVF), or arteriovenous graft (AVG). The blood flow through the hemodialysis device is usually set to 300–400 mL/min; the blood flow volume at entry must be even higher to prevent recirculation (>500 mL/min). The creation of both AVFs and AVGs increases the flow through the extremity by 10–30 times, and it reaches 500–2000 mL/min, sometimes even more. When we consider the normal resting cardiac output (i.e., the amount of blood pumped by the heart in a minute), 4–6 L/min, it then becomes apparent that an AVF or AVG graft could interfere with the human circulation.

Considering the hemodynamic impact of hemodialysis access only, a catheter may seem to be the safest type. This, however, is not true; several studies revealed higher mortality in patients hemodialyzed through catheters [1]. Infective complications are responsible for the higher mortality, and also include life-threatening bacterial endocarditis [2]. The optimal blood flow of an AVF is between 500 and 1500 mL/min; for AVGs, the lower limit is 600 mL/min [3]. Arteriovenous access blood flow (Qa) is determined by the systemic arterial pressure and the access resistance. The latter is mainly influenced by the size of the arteriovenous anastomosis and the feeding artery (or of a stenosis, if present). Therefore, upper-arm AVFs, especially brachio-cephalic AVFs, usually have higher Qa [4]. The flow volume increases dramatically immediately after the creation of any of these arteriovenous accesses, and reaches a plateau within 3–6 months. While the flow through the AVG is usually stable, thanks to the rigid graft, this is not true for AVFs. In AVFs, continuous dilatation of the feeding artery (remodeling), driven by the high wall shear stress, leads to dilatation of the arteriovenous anastomosis, and, thus, to a decrease in access resistance. Indeed, flow over 2000 mL/min develops almost exclusively in AVFs. These high-flow fistulas are known to be associated with heart failure in some patients [4].

There are the following two important questions: What is the safe upper limit of Qa? Do the high, or very high, Qa values matter if the patient is asymptomatic?

2. Safe Upper Limit of Qa

AVF creation with “normal” Qa leads to worsening of the left ventricular diastolic function, and to an increase in B-type natriuretic peptide (BNP, a marker of heart failure) [5,6]. An increase in BNP depends on the Qa value. An interesting study, published by B. Dundon et al. [7], used cardiac magnetic resonance before and 6 months after AVF creation. At the follow-up visit, the patients had a mean Qa value of 1158 mL/min, and
significantly higher left ventricular mass and dilatation of all cardiac chambers. A recent study reported similar results [8].

The term “high-flow AVF” is not uniformly used for patients with signs of heart failure (e.g., leg oedema, ascites, and hypotension), especially those with symptoms that develop sooner than signs (e.g., shortness of breath, bendopnoea, and fatigue), or for those with $Q_a > 1500–2000 \text{ mL/min}$ or when the ratio of $Q_a$ and cardiac output exceeds 30% [4]. These AVFs bring a higher risk of heart failure, but are frequently asymptomatic, or the patients suffer from orthopedic limitations, frailty, neuropathy, etc., that prevent the manifestation of exercise-induced symptoms. Even sooner than exercise-induced complaints, we could observe echocardiographical changes that mirror altered hemodynamics, such as the dilatation of heart cavities, and the development of secondary valvular regurgitation or of diastolic dysfunction. Although they are evidently a mild stage of heart failure, the current guidelines reserve the term “heart failure” for symptomatic patients only (at least temporarily), and call these abnormalities “heart failure precursors”. Several studies tested the effects of $Q_a$ reduction, or even AVF ligation [9,10]. They reported decreases in left ventricular mass, the size of cardiac chambers, and pulmonary hypertension, and improvements in left ventricular diastolic function, and even slightly improved brain oxygenation after $Q_a$ reduction.

Some trials reported a prediction of higher mortality in hemodialysis patients with left ventricular hypertrophy [11], left atrial dilatation [12], or with pulmonary hypertension [13]. All these parameters are influenced by AVF, especially when $Q_a$ is high. However, we have no formal proof of the concept that high $Q_a$ increases mortality.

There is general agreement that symptomatic, high-flow AVFs should be treated by surgical flow reduction in hemodialysis patients [4], and some experts recommend AVF ligation after successful kidney transplant. Surgical $Q_a$ reduction in asymptomatic, high-flow AVF patients on hemodialysis has not been reported, except for in our two articles. We indicated flow reduction, even in asymptomatic patients who have left ventricular hypertrophy or pulmonary hypertension. The effects on the heart were the same in symptomatic and asymptomatic patients. This could also be important in light of the finding that AVF is frequently created too early before the real need for hemodialysis, and, in some subjects, it is never used at all.

The $Q_a/CO$ ratio seems to increase exponentially when $Q_a$ exceeds 2 L/min. We can cautiously consider that this is the upper safe $Q_a$ limit for most patients. However, the limit is significantly lower in patients with heart failure, and this is very frequent in the hemodialysis population.

The current guidelines are not in agreement regarding the indication of flow-reducing surgery. The European Society of Vascular Surgery guidelines recommend that dialysis patients with an access flow above 1500 mL/min should be regularly monitored by means of flow measurements, echocardiography, and evaluating the clinical signs of heart failure [14]. On the other hand, the Spanish guidelines suggest AVF flow reduction when $Q_a$ is $>2000 \text{ mL/min}$ or CPR $>30\%$, to reduce the risk of high-output heart failure [15]. Importantly, AVF creation must be thoroughly considered, especially in patients with very low ejection fractions and shortness of breath [16].

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References


