High Arteriovenous (AV) Access Flow and Cardiac Complications

“Cardiovascular mortality in ESRD patients, depending on age, is 10 - 500 times greater than the general population.”
Special Report: NKF Task Force on Cardiovascular Disease, AJKD 1999; 32(S)

Cardiovascular disease (CVD) is the leading cause of morbidity and mortality in patients with End-Stage Renal Disease (ESRD). It accounts for half of the deaths and one-third of hospitalizations of dialysis patients. The gravity of cardiac disease in hemodialysis patients is highlighted in a Seminars in Dialysis 2014 publication entitled “Hemodialysis-Associated Cardiomyopathy: A Newly Defined Disease Entity.”

AV Access Flow & Cardiac Function
Although extensively documented in the literature, high arteriovenous access flow is also often overlooked as a source of cardiac dysfunction. By bypassing the customary arteriole/capillary beds and establishing a direct high flow connection between the arterial and venous systems, an AV vascular access causes a precipitous drop in peripheral arterial resistance which significantly affects blood flow. In order to maintain blood pressure and improve cardiac output, the body compensates for this immediate drop in resistance by increasing heart rate and stroke volume, which over time can lead to the development of congestive heart failure (CHF).

WWII Trauma Victims Exhibit CO Increase
The relationship between an AV access and cardiac function was first observed in World War II in soldiers who experienced trauma-induced arteriovenous fistulas. Iwashima et al reported a 15% increase in cardiac output by the seventh day after arteriovenous fistula creation. This increased cardiac workload can lead to left ventricular hypertrophy, especially in patients with histories of coronary artery bypass surgery.

In 1976, Henry Fee et al from UCLA’s School of Medicine reported in the Annals of Surgery four patients who required corrective surgery for high-output congestive heart failure (HOCHF) secondary to high flow rates through their femoral AV shunts. His recommendation at that time was that intraoperative graft flow rates should not exceed 900 cc/min.

In 1995, Engelberts and Tordoir et al from Maastricht University reported a case where excessive shunting in a hemodialysis access fistula led to high-output cardiac failure. They termed it “an easily overlooked diagnosis.” Following surgical closure of the fistula, the patient’s condition improved, and signs of congestive heart failure subsided. In 1998, Young PR Jr et al from Bowman Gray School of Medicine, Wake Forest University, reported two renal transplant patients who developed high-output cardiac failure from brachioccephalic fistulas. Successful transplantation, coupled with fistula ligation, resolved the cardiac complications. Additional case reports cemented the relationship between high volume AV access flows and cardiac complications.

Fig. 1: The graph illustrates the spike in the probability of sudden death immediately following dialysis. “35% of deaths occurred in the first 12-hour interval.” Critically low CI levels (<2 L/min/m²) can occur in patients who do not feel well at the end of a dialysis session. As an AV fistula steals flow from an already limited systemic circulation, low CI can contribute to decreased myocardial perfusion and death. Bleyer A et al, “The Timing and Characteristics of Sudden Death in Hemodialysis Patients” J Am Soc Nephrol 2002;13:SU-P0737.
Lowering AVF Flow to Lower Cardiac Output
In 2004, Brian Murray et al from State University of NY at Buffalo published the results of the effects of surgical banding a high flow fistula and its effect on access flow and cardiac output. Chemla describes using inflow reduction by diastolization of the anastomoses to treat similar conditions in 17 British hemodialysis patients. Other clinicians report use of similar techniques to reduce high fistula flows and cardiac overload.

Access Flow - Cardiac Output (AF/CO) Ratio
MacRae et al from the University of Calgary reported the high output cardiac failure associated with high flow AVFs (> 1.5 L/min), particularly in men with upper arm fistulas and previous access surgeries. In her 2006 comprehensive review, "The Cardiovascular Effects of Arteriovenous Fistulas in Chronic Kidney Disease: A Cause for Concern?", MacRae documents the evidence, to date, on the subject. She also notes that the ratio between access flow and cardiac output is an important clinical indicator. When access flow exceeds 25% of cardiac output, a potential cardiac problem can exist. MacRae suggests that hemodialysis patients be screened for potential high-output cardiac failure using a Qa/CO ratio with patients having a Qa/CO ratio ≥ 30% undergo further testing. Padberg FT Jr, et al from New Jersey’s School of Medicine also reviewed the existing evidence in the 2008 Journal of Vascular Surgery, “Complications of arteriovenous hemodialysis access: recognition and management.”

Italian Study Sets 2L/min AVF Flow Cut-off Value
In 2008, Basile et al from Miulli General Hospital in Acquaviva delle Fonti, Italy published a study of 96 patients with AV fistulas and cardiac failure. The study showed that upper arm AVFs are associated with an increased risk of high output cardiac failure. It was the first published study with a high predictive power for AV fistula flows greater or equal to 2.0 L/min to result in high-output cardiac failure. In this landmark study, both AV access flow and cardiac output were measured using the Transonic® HD02 Hemodialysis Monitor.

Ye et al’s 2013 study from Beijing’s PUMC hospital, confirmed a 2 L/min threshold for treatment. Ye collected data from 50 non-diabetic AV fistula hemodialysis patients. AVF flow (Qa), stroke volume (SV), cardiac output (CO), cardiac index (CI) central blood volume (CBV) and peripheral resistance (PR) were measured using Transonic’s ultrasound dilution technology. The conclusion was that “cardiac adaptive changes after long-term AVF include the enlargement of the left ventricle and thickening of the ventricular wall. The risk of cardiac failure significantly increases when the Qa of AVF is more than 2.0 L/min with much higher CO and lower PR.”

Pulmonary Hypertension Studies
In two publications, Isrealis Nakhoui et al and Abassi Z et al investigated and reported on pulmonary hypertension in their ESRD patients. Nakhoui discovered that 48% (20) of his cohort of 40 patients had unexplained pulmonary hypertension (PHT). In reviewing other PHT studies, Abassi’s group found that morbidity and mortality from cardiovascular disease were greatly increased in 40% of chronic hemodialysis patients. In a 2009 Journal of Vascular Access article from Iran entitled, “Effects of the arteriovenous fistula on pulmonary artery pressure (PAP) and cardiac output in patients with chronic renal failure,” data from 50 patients showed a statistically significant positive correlation between fistula flow and PAP and PAP changes (p < 0.05).

Recent Studies Support High AVF - CO Link
In the 2013 October issue of Clinical Transplant, Schier et al from Innsbruck University reported the results of a 2005-2010 retrospective study of kidney-transplant recipients. Twenty-five percent of the recipients (29 of 113) needed an AV fistula closure, mostly due to cardiac failure symptoms.
FistulaFirst.org Query

Q. I am seeing congestive heart failure (CHF) in patients with borderline cardiac function and excellent fistulas. We have done compression studies on these patients during a cardiac cath by measuring the ejection fractions, then compressing the fistula with a blood pressure cuff and remeasuring the ejection fraction. The ejection fraction increases and the patient becomes less symptomatic. There was a Transplant International article (France, 2008) stating that they are tying off fistulas in post-transplant patients to decrease left ventricular hypertrophy (LVH). Is anyone else seeing this?

A. In fact, high-output cardiac failure and also pulmonary hypertension are well known complications of high-flow HD access. Although “high flow” is subjective, since every patient has a threshold of access flow that will induce such failure (as well as distal extremity ischemia), Fistula First uses a minimal threshold of 2 L/min flow to refer the patient for cardiac evaluation.

This is an often overlooked cause of LVH & CHF--and any HD patient with a history of CHF or progressive LVH, should absolutely have access flow measured. When unrecognized, many of these patients with recurring CHF will die from their access-induced heart disease, since the cause was not recognized, and only gets worse.

The advent of accurate non-invasive measurement by ultrasound saline dilution has made it possible to measure access flow, which permitted a number of studies confirming the correlation between cardiac output and access flow. Access flow is usually approximately 20% of cardiac output. As access flow increases, so does cardiac output. The only reason that we do not see this problem in many patients, is because only a small proportion of patients have access flow approaching or greater than 2 L/min. Certainly, any patient developing LVH or CHF after starting HD should have the access flow measured. One reason I strongly urge use of access flow surveillance, is because it provides so much information. (Lawrence Spergel, MD, FACS)

Stern et al from UNC Kidney Center’s Division of Nephrology and Hypertension, in Chapel Hill, NC describes how an increase in preload can lead to increased cardiac output when a large proportion of arterial blood is shunted from the left-sided circulation to the right-sided circulation via the fistula. Patients may present with the usual signs of high-output heart failure including tachycardia, elevated pulse pressure, hyperkinetic precordium, and jugular venous distension. The nephrologist is then faced with the dilemma of preventing progression of heart failure at the expense of losing a vascular access. The authors conclude that treatment should be directed at correcting the underlying problem by surgical banding or ligation of the fistula.

In a 2012 Seminars in Nephrology article, “High-output Heart Failure: How to Define It, When to Treat It, and How to Treat It,” Wasse et al from Emory University succinctly outlines the problem. Dr. Wasse describes the mechanisms by which a dialysis AV access may promote the development of high-output cardiac failure, the risk factors for and diagnosis of high-output heart failure, and recommends management strategies for patients with high-output heart failure.

Conclusion

“A high flow AV access can produce life-threatening cardiac complications. The volume flow level that will induce high-output failure or extremity ischemia will vary with each patient, based on co-morbidities, especially the degree of cardiac disease and peripheral arterial disease. For patients at risk based on such pre-existing conditions, which can be a majority of patients in a given hemodialysis population, the widespread consensus (evidence-based) is that patients with access flows of 2L or higher should be tested and followed for these complications--and have a flow-reduction procedure performed at the earliest signs of cardiac complications or extremity ischemia.

Unfortunately, with the high prevalence of cardiac disease in the HD population, an insidious and silent access flow as a major cause or contributor to a potentially deadly cardiac complication, is often overlooked. Therefore, it is critically important for the practitioner to be aware of the relationship between access flow and cardiac failure, since many of these high-flow patients will have morbidity and mortality that otherwise could have been avoided.”

Lawrence Spergel MD FACS
References