The Cardiovascular Effects of Arteriovenous Fistulas (AVFs) in Chronic Kidney Disease: A Cause for Concern?

This paper reviews the hemodynamic and cardiovascular consequences of AVFs in chronic kidney disease.

**SUMMARY**

Immediate hemodynamic effects of AVF creation
- Increase in Cardiac Output (10-20%).
- Increase in Sympathetic nervous system activity (increasing contractility).
- Increase in Stroke volume and heart rate.
- Decrease in Peripheral Resistance.

Hemodynamic changes within one week of AVF creation
- Increase in Circulating Blood Volume resulting in increased left atrial, inferior vena cava, and left ventricle end-diastolic volume (LVEDV).
- Increase in Neuro-hormones: atrial natriuretic peptide (ANP) and brain natriuretic peptide (BNP) implying atrial and ventricular filling pressure are increased. (ANP is a powerful vasodilator and BNP is correlated with echocardiographic features).
- Decrease in Plasma renin and aldosterone levels.
- Decrease in Systemic vascular resistance and systolic/diastolic blood pressure.

Consequences of long-term AVF creation

Left Ventricular Hypertrophy (LVH):
An adaptive response to increased cardiac workload caused by volume or pressure overload.

High-Output Cardiac Failure
- Patients with high-flow AVFs are most likely have a greater risk of developing CHF and greater increase in LVEDV.
- AVFs in HD patients may contribute to the development of heart failure.
- Left ventricle enlargement at the start of HD is very common and progressive left ventricle dilation with hypertrophy continues over time. Most of the left ventricle growth occurs during the first year of dialysis.

Exacerbation of Coronary Ischemia
- AVF placement is associated with increased myocardial O2 demand that may not be met, especially in patients with established coronary artery disease (CAD) or left ventricle hypertrophy (LHV).
- Increased O2 consumption may have clinical manifestations in dialysis patients who have had CABG. A decrease in coronary perfusion that occurred with the onset of HD was demonstrated by the reduction in graft flow and reversible hypokinesis of the anterior left ventricle wall.
- High-flow AVFs with an associated high cardiac output may increase O2 demand and reduce supply more than a smaller AVF.

Central Vein Stenosis
- The endothelium plays an active role in vascular remodeling by secreting vasoactive substances and growth factors in response to alterations in flow and shear stress.
- Increased blood flow due to AVF creation alters the shear stress on the endothelium and promotes production of substances like transforming growth factor (TGF)-ß and NO which dilate the vessel lumen.
- A majority of central vein stenosis occurs at the junction of the cephalic and subclavian veins. There was a high correlation between the location of a central vein stenosis and ipsilateral AVF. It suggests that altered flow hemodynamics due to a fistula may result in endothelial damage and vascular remodeling, leading to stenosis.

**REVIEW’S CONCLUSIONS**

- A thorough cardiac assessment should be performed in patients with CAD prior to placing an AVF.
- Regular careful evaluations are necessary in patients with cardiac disease and AVFs.
- Patients with a large AVF and high flow should be followed with serial echocardiography to watch for changes in the LVEDV and monitor for LVH.
- Patients with high flow fistulas (flow greater than 2L/min) and increasing LVEDV are recommended to have a flow reduction procedure on their AVF.
- Patients with preexisting severe ischemic heart disease (class III unstable angina) should avoid AVF placement if the underlying ischemia cannot be treated.
- AVFs are superior to catheters and grafts due to fewer thrombogenic and infectious complications.

**Reference:**