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Brief review

Cardiac complications of arteriovenous fistulas in patients with end-stage renal disease

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ABSTRACT

Cardiovascular disease is the leading cause of the death in dialysis patients. Arteriovenous fistulas (AVFs) are associated with lower mortality and are viewed as the desired access option in most patients with advanced kidney disease needing dialysis. However, AVFs have significant and potentially deleterious effects on cardiac functions particularly in the setting of preexisting heart disease. This article provides a comprehensive and contemporary review to what is known about the impact of AVFs on: congestive heart failure, left ventricular hypertrophy, pulmonary hypertension, right ventricular dysfunction, coronary artery disease and valvular heart disease.

Complicaciones cardiacas de las fístulas arteriovenosas en pacientes con enfermedad renal terminal

Palabras clave:
Acceso vascular para hemodiálisis
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RESUMEN

La enfermedad cardiovascular es la principal causa de muerte en los pacientes dializados. Las fístulas arteriovenosas (FAV) se asocian a una menor mortalidad y se consideran la opción preferible de vía de acceso en la mayor parte de los pacientes con enfermedad renal avanzada que requieren diálisis. Sin embargo, las FAV tienen efectos importantes y potencialmente nocivos sobre las funciones cardíacas, en especial en presencia de una cardiopatía preexistente. En este artículo se presenta una revisión completa y actualizada de

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los conocimientos existentes sobre las repercusiones que tienen las FAV en los trastornos de: insuficiencia cardiaca congestiva, hipertrofia ventricular izquierda, hipertensión pulmonar, disfunción ventricular derecha, enfermedad coronaria y valvulopatías cardíacas.

Cardiovascular disease is the leading cause of the death in patients receiving chronic renal replacement therapy.[1–3] Arteriovenous fistulas (AVFs) have superior longevity, lower infection and mortality rates and are associated with lower cost, and hence have become the vascular access of choice for patients needing dialysis.[4] Indeed, the prevalence of AVFs in the United States increased from 32% of all dialysis access in 2003 to 61% in 2012.[5,6] Despite their association with a lower mortality, AVFs have significant effects on cardiac functions predominantly related to the increase in preload and cardiac output (CO). This article reviews the potential effects of the creation and the ligation of AVFs on cardiac function and their mechanisms.

It should be emphasized, at the outset, that determining the exact effects of AVFs on cardiac functions is fraught with problems for a couple of reasons: patients with end stage renal disease (ESRD) requiring dialysis almost invariably have volume overload due to water and salt retention. They also have pressure load due to arterial sclerosis and hypertension, and increased CO secondary to chronic anemia. In addition, many hemodialysis patients have significant pre-existing myocardial, valvular or coronary heart disease. It is, therefore, often difficult to tease out the exact contribution of an AVF to cardiac dysfunction in hemodialysis patients. Nevertheless, worsening in cardiac functions soon after AVF creation has been observed favoring a causative effect of the AVF on certain cardiac functions. The current literature suggests that the creation of AVF can cause or exacerbate the following conditions: congestive heart failure, left ventricular hypertrophy, pulmonary hypertension, right ventricular dysfunction, coronary artery disease, and valvular dysfunction.

AVFs and congestive heart failure

Congestive heart failure (CHF) is highly prevalent among patients with ESRD. Approximately 35–40% of patients with ESRD have an established CHF diagnosis at initiation of hemodialysis.[1,3,7–9] Patients with ESRD and CHF have a far worse prognosis than those without CHF.[3,10] Since hemodynamic optimization is the corner stone of managing patients with ESRD as well as those with CHF, studying the hemodynamic effects of AVFs in patients with ESRD with and without CHF is a sensible task.

Long before we utilized AVFs for hemodialysis access, the hemodynamic effects of AVFs were studied in patients who developed AVFs secondary to trauma AVFs. In these patients, the development of an AVF was noted to be associated with an apparent increase in CO.[11,12] The introduction of the 'man-made' AVFs for hemodialysis access provided more insight into the hemodynamic effects of these fistulas: First, the creation of an AVF leads to shunting of blood flow from the high resistance arterial system into the low resistance venous system, with a subsequent rise in venous return and CO.[13] Second, the presence of an AVF decreases arterial impedance and thus lessens the left ventricular afterload. The lowering of arterial impendence may also reduce the effective circulating volume of the systemic circulation, activating arterial baroreceptors, and leading to secondary increase in cardiac sympathetic tone, contractility, and CO.[14–16] The net effect of AVFs is a significant increase in CO.

Many studies investigated the impact of AVFs on echocardiographic indices of cardiac morphology and function.[13,14,16–21] These studies consistently showed an increase in LV end-diastolic dimension (LVEDD), contractility, stroke volume and CO within 7–10 days after the surgical construction of AVF.[13,14,18] Diastolic filling parameters (E to A ratio) were also impaired, indicative of worsening diastolic functions. On average, the creation of an AVF increases CO by 15–20% and left ventricular end-diastolic pressure by 5–10%.[16] Additionally, biomarkers secreted in response to hypervolemia such as atrial naturietic peptide (ANP) and brain naturietic peptide (BNP), are both substantially increased,[13,14] suggesting the presence of an cardiac volume overload despite an optimal overall body volume status.

The impact of these physiological effects of AVF on the cardiac function is controversial. While many studies suggested that the decreased vascular resistance and the increased CO are predisposing factors for the development or the worsening of heart failure,[9] others suggested that the decrease in peripheral resistance and blood pressure with a parallel increase in ejection fraction could be potentially beneficial.[22]

Risk of worsening heart failure after AVF

There is no standard definition for high output CHF. The literature is inconclusive with regards to the incidence of worsening CHF after AVF creation. Most authors believe that the incidence of high output CHF among hemodialysis patients with AVFs is low, and that most patients with ESRD tolerate AVFs.[23,24] This belief is supported by the fact that the literature on high output CHF in ESRD patients is limited to case reports and small series[25–28] and that corrective measures (AVF banding or surgical ligation) due to AVF-related cardiac derangement are uncommon. Dixon et al. noted that the rate of AVF banding due to worsening CHF in a cohort of 204 patients (322 accesses) was only 2.6%.[29] On the other hand, some authors suggest that high output CHF is not uncommon but is often overlooked.[26,30] These authors argue that when cardiac deterioration occur in hemodialysis patients, it is usually attributed to the many risk factors that are highly prevalent in this population, and that the exact contribution of