



Arteriovenous fistulae and cardiovascular function: the relationship between brain natriuretic peptide, cardiac index and access flow

Short communication

The Vascular Access Society defines “high-flow” AVF as those with $Qa > 1000\text{--}1500\text{ mL/min}$ [1]. High vascular access blood flow (Qa) is believed to increase cardiac output and may lead to high output cardiac failure [2]. There is anecdotal evidence that high flow arteriovenous fistulae (AVF) can cause symptomatic heart failure with dyspnea, orthopnoea, paroxysmal nocturnal dyspnea and peripheral oedema [2,3].

However there is little evidence in the literature attempting to quantify the cardiovascular effects of an arteriovenous fistula. Several years ago, Suttie and colleagues (2012) measured BNP in patients with maturing de novo AVF finding no effect on brain natriuretic peptide (BNP) levels [4]. The authors report difficulties measuring BNP, which is renally excreted, in patients in varying degrees of renal failure and dialysis dependence. They also highlight that the cardiovascular effects of a maturing AVF are likely to be small and occur slowly enough to permit adaptive response. Recently we have published work on the cardiovascular changes in stroke volume and systemic vascular resistance that occur with temporary occlusion of a mature AVF [5]. We now describe our experience following permanent occlusion of an AVF.

The aim of this study was to evaluate the relationship between BNP, cardiac index (CI) and brachial artery blood flow (BA flow) in patients undergoing AVF ligation.

14 asymptomatic patients with well functioning renal transplants ($eGFR > 60\text{ mL/min/1.73m}^2$) (in order to exclude any confounding effect of renal function on BNP levels) undergoing ligation of a brachiocephalic fistula (BCF) were identified. BA flow was measured via Doppler ultrasound (4-10Hz Linear Probe, Mindray™). CI was also measured pre- and immediately

post-ligation using thoracic bioimpedance technique (NICCOMO™, Medis GmbH). Serum BNP (CardioRenal Panel, Alere) was also measured pre-, immediately post- and 6 weeks post-ligation.

The mean patient age was 47.9 ± 14.6 years (57.1% male). Mean BA flow was $1329.8 \pm 793.7\text{ mL/min}$. Mean CI pre-ligation was $3.5 \pm 0.4\text{ l/min/m}^2$. There was a significant reduction in cardiac index following ligation of the AVF (Mean ΔCI was $-0.31 \pm 0.47\text{ l/min/m}^2$ [$p=0.02$]) (FIGURE 1). Mean BNP pre-ligation was $111.2 \pm 23.7\text{ pg/ml}$. This reduced to $94.1 \pm 13.9\text{ pg/ml}$ immediately post ligation and $91.9 \pm 13.8\text{ pg/ml}$ after 6 weeks ($p=0.04$). There was good linear correlation between ΔBNP and both BA flow and ΔCI ($r^2=0.56$ and 0.53 respectively).

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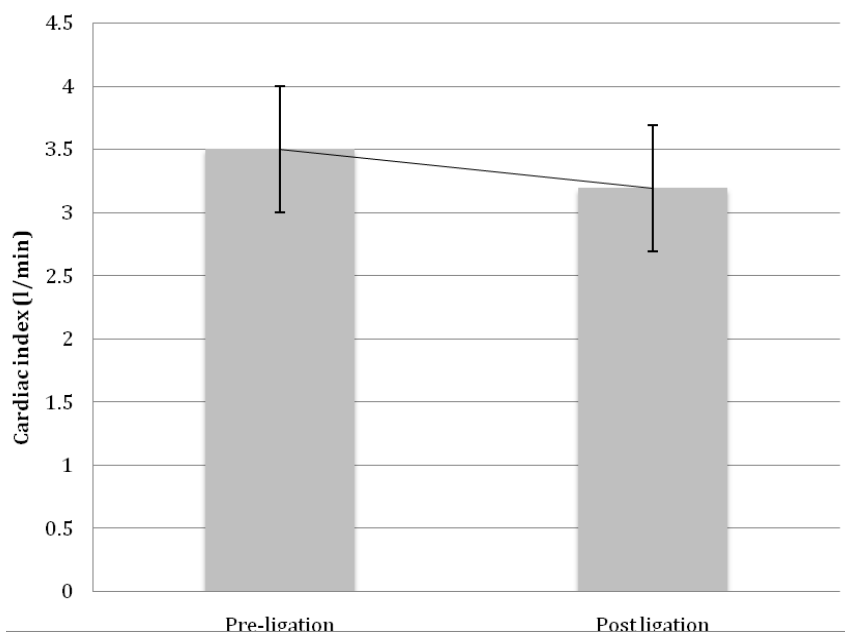


Figure 1. Cardiac index reduced following ligation of the AVF.

This work supports our previous ascertain that AVF exert significant haemodynamic effects on the cardiovascular system even in asymptomatic patients. These effects are rapidly reversible upon ligation of the AVF. Consideration of the cardiovascular effects

should be given prior to creation of an AVF, particularly in an increasingly elderly, co-morbid haemodialysis population.

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