

## Unilateral breast enlargement due to a high-flux ipsilateral hemodialysis fistula

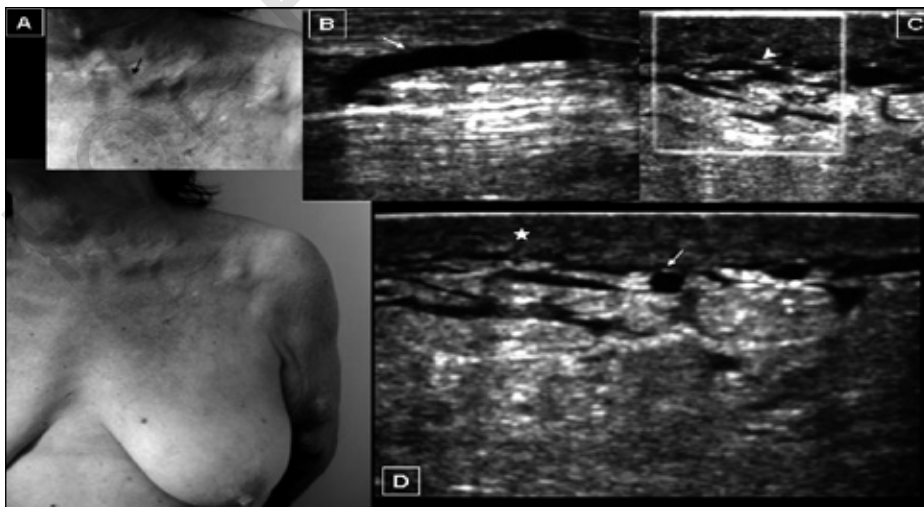
Editor,

A 61 year-old female on hemodialysis, started with mild but frequent dyspnea. A coronary catheterism was normal. Physical examination revealed a prominent radiocephalic fistula in her left arm. The left breast was homogeneously enlarged and swollen. A mammography suggested gland enlargement was due to increased breast vascularization (Fig. 1). A doppler ultrasound of the vascular access revealed an increased flux (blood flow rate: 7 liters/minute); humeral artery blood flow: 2.8 liters/minute (L/min); pre-clavicular subclavian vein blood flow rate: 4.8 L/min. Dyspnea was assumed to be due to high output heart failure. After surgical reduction of the fistula diameter with a polytetrafluoroethylene banding, blood flow decreased to 3 L/min albeit breast size remained basically unchanged.

A unilateral homogeneous breast enlargement is an infrequent physical finding, being one of the causes of it an accumulation of soft tissue edema secondary to an increase in hydrostatic vascular pressures. This imbalance in Starling forces can be due to venous obstruction or to an increase in vascular flow inputs associated with a chronically increased vascular bed. The main mechanism of this phenomenon could have resulted from an increase in venous return flow from the mammary vein into the subclavian vein. The lymphatic system could have also played a role: The lymphatic drainage of the breast is through superficial and deep lymphatic vessels, lymph flowing unidirectionally from the superficial to the deep plexus. Lymph flow from the deep subcutaneous and intra-mam-

mary vessels moves centrifugally toward the axillary and internal mammary lymph nodes. Approximately 3 percent of the lymph from the breast flows to the internal mammary chain, whereas 97 percent directs to the axillary nodes (1). Thus, an imbalance between a highly vascularized arteriovenous system and poorly developed lymphatic tree could have also contributed to the edema.

There still exists no clear definition when a fistula is considered of high-flow. The concept of using the ratio access flow to cardiac output (CO) has been proposed by Pandeya and Lindsay (2); they found that in chronic dialysis subjects, the average access blood flow (Qa) was 1.6 L/min and the average CO was 7.2 L/min, thus describing a normal average Qa /CO ratio of 22 %. Despite surgical correction of the access, the blood flow remained high at 3 L/minute. Published case reports describing high-output cardiac failure all consistently show high-flow AVF with Qa > 2 L/min and Qa/CO ratios greater than 30% to 35 %. More information is required to confirm the plausible concept that high-access flows with elevated Qa/CO ratios may represent a risk factor for heart failure. Most of the reported banding and surgical closure of AVF have occurred when patients developed symptoms of cardiac failure with Qa/CO ratios of > 40%. (3). Studies in upper arm fistulae suggest a normal mean Qa ranging from 1.13 to 1.72 L/min, 15% of patients presenting Qa > 2-2.5 L/min (2). However, a high flux fistula can result in a hyper-dynamic state that can cause a steal syndrome and end up in high-output heart failure. Steal phenomena can occur in approximately 73% of fistulae and is usually asymptomatic, symptoms appearing when collateral or direct flow is not developed to offset the steal. In a hemodialysis AVF, blood is shunted from a high pressure artery into a low pressure vein, increasing systemic vascular volume (4). The increased cardiac output associated with these fistulae depends upon the size of the communication and the



**Fig. 1 - A,B,C,D.** (A) Patient's chest shows upper limb left veins and colaterals, which drain the arteriovenous fistula, increased in size and number. (B-D): Left breast ultrasound, depicting increased hypodermic vasculature (B), fat tissue edema (white arrow, C) and dermis proliferation (10 MHz transducer).

magnitude of the resultant reduction in systemic vascular capillary perfusion. Left ventricular hypertrophy, prevalent in patients with end-stage renal disease, can certainly increase the susceptibility to myocardial stress imposed by an elevated cardiac output. There is no clear indication for the timing or cut-off of a particular Qa/CO ratio to undertake an intervention. We suggest that patients with intractable or worsening heart failure symptoms despite transiently successful medical therapy should undergo assessment of flux vascular access.

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