High Output Cardiac Failure Secondary to a Brescia-Cimino Fistula

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A patient with high output cardiac failure secondary to a Brescia-Cimino fistula is presented. The heart failure responded to a reduction in the fistula size. The literature on this complication is reviewed.

Congestive heart failure is a well documented complication of systemic arteriovenous (AV) fistulas.[1] The introduction of the surgically created AV fistula for vascular access by Brescia[2] has simplified chronic dialysis by eliminating many complications associated with Quinton-Schribner shunts. Although cardiac failure was considered a possible complication of the Brescia-Cimino fistula, only a few such reports have appeared.[3-10]

We wish to report a single patient who developed high output cardiac failure 22 months after creation of a Brescia-Cimino fistula and to review the literature on this complication.

Case Presentation

The patient is a 55-year-old man who was first seen by the Renal Service in February 1972. He had a five-year history of hypertension and chronic sinusitis but was otherwise well. He was taking antihypertensives and diuretics.

Physical examination revealed a healthy appearing black man. The only abnormal finding was arteriolar narrowing and AV nicking seen in the optic fundi.

The following laboratory results were either normal or negative—CBC, differential, electrolytes, total protein, albumin, calcium, glucose, bilirubin, alkaline phosphatase, LDH, SGOT, and complement. His BUN was 22 mg/100 ml, creatinine 2.3 mg/100 ml, and creatinine clearance 52 ml/min. Urinalysis was unremarkable except for 1+ proteinuria. A 24-hour protein excretion was 1.8 gm. An intravenous pyelogram was unremarkable. Renal biopsy was performed and the results were consistent with nephrosclerosis.

He was seen again in September 1974. In the interval, his blood pressure had become more difficult to control. His serum creatinine increased to 11.5 mg/100 ml and he had developed uremic symptoms. Chronic hemodialysis was begun in November 1974. He initially responded well with normalization of blood pressure and relief of uremic symptoms. However, hypertension returned after about five months of dialysis and was difficult to control because of extreme sensitivity to ultrafilitration. He subsequently developed chronic congestive heart failure which was unresponsive to drugs, fluid restriction, and ultrafiltration.

He was admitted to the hospital in October 1976, where congestive heart failure was documented by appropriate studies. Because of our suspicion that his large AV fistula may have been contributing to his heart failure, he was referred to the Cardiology Service for hemodynamic studies. On September 8, 1976, right heart catherization was performed. Cardiac output determined by dye dilution was 14.9 L/min and determined by thermodilution was 14.7 L/min. Total blood volume was 88 ml/KG (upper limit of normal) with decreased red blood cell volume and increased plasma volume consistent with his anemia. His fistula was subsequently reduced to approximately one third of its preoperative diameter by banding with umbilical tape. Postoperatively his cardiac output was again measured by thermodilution and was 4.5 L/min. Clinically, the symptoms of congestive heart failure cleared and he subsequently did well on chronic hemodialysis.

Discussion

The Brescia-Cimino fistula for hemodialysis vascular access represented a major improvement over the Quinton-Schribner shunt. It allows freedom from an external appliance and a markedly reduced incidence of infection and clotting. Its major disadvantages are the need for repeated venipunctures with large bore needles and the possible development of high output cardiac failure. The first drawback, while not trivial, is apparently well tolerated, whether the needles are inserted by professional staff or by a trained dialysis partner in the home. The second remains a theoretical possi-

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Table 1. Summary of Reported Occurrences of High Output Cardiac Failure from Upper Extremity Hemodialysis Fistula

Author and Date		Age	Fistula Type	Duration (mos)	Size (mm)	Fistula before surgery L/min	Flow after surgery L/min	Cardiac before surgery L/min	Output after surgery L/min	Treatment	Outcome
McMillan and Evans 1968	1	41	Radial S-S	—	5	—	—	5.8	5.5	Closure	
Ahern and Maher 1972	2	37	Radial S-S	6	7	0.6	0.3	11.2	8.4	Narrowing	Improvement
		39	Radial E-E	5	10	0.75	0.3	—	—	Narrowing	Improvement
George et al 1973	1	51	Radial	35	5	—	—	12.3	6.3	Excision Replaced by Shunt	Improvement
Zerbino et al 1974	4	Adults	not specified	not specified	not specified	1.83	—	—	—	Narrowing -1 Closure -1 Medical -2	Improvement
Draur 1973	3	not specified	Radial S-S	not specified	not specified			16.5 6.8 9.9	5.5 5.7 8.0	not specified	not specified
Anderson et al 1976	6	48	Radial S-S	6	9	1.4	0.7	—	—	Narrowing	Improvement
		51	Radial E-E	30	6	2.9	0.6	—	—	Narrowing	Improvement
		45	Radial E-S	48	8	2.1	0.5	7.0	5.5	Narrowing	Improvement
		42	Brachial S-S	18	10	—	—	—	—	Closure	Improvement
		25	Radial E-S	13	8	—	—	5.5	4.6	Medical	Improvement
		45	Radial E-S	2	7	1.0	0	7.9	6.1	Closure	Improvement
Langescheid et al 1977	1	21	not specified	not specified	not specified	1.56	0.7	—	—	Narrowing	Improvement
Dillard and Alexander	1	55	Radial S-S	23	5	—	—	14.8	4.5	Narrowing	Improvement

bility, but the very small number of reported cases of proven high output failure following creation of Brescia-Cimino fistulas indicates the actual risk is very small.

Congestive heart failure is a documented complication of AV fistulas. It occurs most frequently following traumatic injury to large vessels. In the report by Pate and associates,[1] the incidence of heart failure following traumatic fistulas involving central vessels was 55 percent, while the rate was 28 percent when peripheral vessels were involved. Arteriovenous fistulas acutely produce a decrease in systemic resistance with a compensatory increase in cardiac output and heart rate.[11-12] Subsequently the intravascular volume expands increasing cardiac stroke volume. This maintains the cardiac output at an elevated level while permitting a fall in the heart rate. The cardiovascular system remains compensated so long as myocardial reserve is adequate. When the demand for increased stroke volume or heart rate exceeds the ability of the heart to respond, symptoms of congestive failure appear.

Published reports from several centers (Table 1) indicate that upper extremity AV fistulas for hemodialysis are generally well tolerated.[6,13] Direct measurements show that flows generally range from 200 to 600 ml/min. Investigators specifically emphasize the absence of symptomatic heart failure following creation of the fistula. However, detailed studies reveal a significant increase in heart rate and cardiac output.[14]

Occasionally, the flow in fistulas increases to levels which are well above average. An extrapolation of published reports suggests that fistula flows which exceed 1 L/min, or 25 percent of the cardiac output, carry a greater risk of producing congestive heart failure.[9,10] When such findings are documented in a patient with heart failure, and in whom other contributing causes have been eliminated, the appropriate therapy would depend on the patient's status. Simple ligation is curative in patients who have a functioning transplant.[3] If the patient is still receiving chronic hemodialysis, banding with tape[9] or an adjustable clip[10] is effective. Anastomoses of 4 mm[4] to 10 mm[9] have been suggested, but the fistula flows should be checked with a flow meter to ensure adequacy. Flows ranging between 300 to 600 ml/min have been associated with good clinical results.

A bradycardia produced by compression of a fistula is referred to as the Nicholadoni-Branham sign. This sign has been proposed as a simple test to distinguish hemodynamically significant fistulas. It was negative in our patient in spite of a cardiac output that was greater than twice normal. Similarly, it was positive in only four of the nine reported patients in whom it was sought.[4,5,9] The fistula flows in these nine patients ranged from 0.6 to 2.5 L/min (av 1.5 L/min). All had resolution of their heart failure following surgical reduction of their fistulas. Thus, the absence of a positive Nicholadoni-Branham sign should not be accepted as evidence that the fistula flow is not producing cardiac failure.

Overhydration, anemia, uncontrolled hypertension, and coronary artery disease are common in chronic dialysis patients. All contribute to the production of congestive heart failure and should be sought and appropriately treated. In the great majority of patients, their correction will relieve the heart failure. Only a rare patient will require further investigation for high output failure due to marked increases in fistula blood flow. These rare patients respond well to appropriate reduction in fistula size.

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