Traumatic Arterio-Venous Fistula Causing Cardiac Failure —A Case Report

*SULTAN MAHMOOD**
*ZAIR MOHAMMAD***
*M. TARIQ NISHTAR****

In most cases the underlying cause of Cardiac Failure is clinically apparent when it is due to ischaemic heart disease, valvular heart disease, Hypertension, advanced pulmonary disease and pericardial disease. However, when the underlying cause is a less common condition like various congenital heart lesions with left to right shunt, Cardiomyopathy, specific heart muscle disease and myocarditis, early stages of constrictive pericarditis, occult pulmonary disease, connective tissue disorders, myocardial involvement by tumors and granulomas and the various conditions causing high output states, the diagnosis is usually clinically not apparent and needs specific investigations for confirmation or exclusion of these conditions.

In high output cardiac failure the heart is faced with volume overload and is forced to pump abnormally large quantities of blood in order to deliver the required amount of oxygen to rapidly metabolising tissues.

Among its causes include thyrotoxicosis, anaemia, arteriovenous fistula, Beriberi, pregnancy, Paget's disease of bone, corpulrmonale, polycythemia, erythrodermas, septiceamia, cirrhosis liver and renal diseases. Some evidence of these conditions may be clinically apparent if specifically sought. Most of these are potentially curable conditions, if treated in time.

We report a case of post-Traumatic Arterio-Venous fistula causing Cardiac failure which was successfully surgically treated.

Case Report:

Fiftyfive years old, house wife from Bannu, presented in September 1987, with six months history of dyspnoea and palpitation. Initially the dyspnoea was on exertion but during the last one month, she was short of breath at rest and used to feel forceful thumping of her heart at rest. There was no history of chest pain, cough, sputum, wheez or fever but she admitted to have anorexia for the last one month.

In the past she has been well apart from a swelling above the forehead which she developed following an injury with a broken glass in childhood. She had no addiction and was taking valium and mucaine.

Fig. 1

On Examination, she was breathless, sitting up in bed and had pitting ankle oedema. Her pulse was 110/mint: large volume, collapsing and regular and B.P. 110/30. There was no anaemia, lymphadenopathy, jaundice or cyanosis. Her hands were warm but there was no goitre or tremors and JVP was raised at 4 cm. Precardium revealed diffuse forceful cardiac pulsations and cardiac impulse was felt in the 5th intercostal space at the anterior axillary line. Ist and 2nd heart sounds were normally audible and there was a short ejection systolic murmur at left sternal border. Liver

* Assistant Professor,
** Associate Professor,
*** Professor of Medicine, Dept. of Medicine, Khyber Medical College, Peshawar.

was palpable 3 finger breadth below the right costal margin, smooth and tender and bilateral basal crepitations were present in her chest.

There was pulsatile swelling above the forehead, the skin over the swelling was warm with varicose veins radiating from the swelling. Fig 1. Over the swelling a palpable thrill and loud bruit was present and (Branham's sign was positive). CNS and Fundi were normal. Laboratory studies showed.

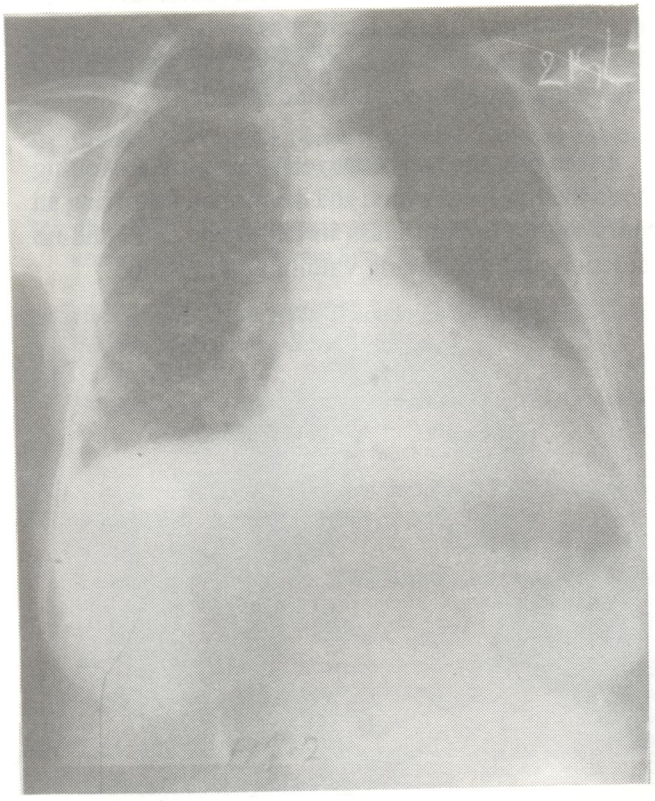

Fig. 2

Hb: of 12.4 Gm%; TLC: of 7000/cmm^3; DLC: P: 68%; L: 30%; E: 2%. Urinalysis and LFT's were normal. Blood urea was 24 mg % and Blood sugar was 110 mg %. Chest X-Ray showed enlargement of Cardiac shadow with congested lung fields. Fig. 2. Serum Electrolytes were normal. E.C.G showed sinus rhythm, 112/minute, and no evidence of Ischaemia and no arrhythmias or signs of chamber hypertrophy. Echocardiogram was reported a dilated left ventricle. X-Ray skull and Thyroid function tests were normal. Angiogram of the fistula was not done.

She was treated with frusemide, Potassium chloride and Digoxin. A week later she felt better, dyspnoea had decreased and Exercise tolerance had improved.

Two months later she underwent surgical excision of the fistula and her post-operative course was uneventful.

She was seen four months after her surgery when she had no symptoms, her E.C.G and chest X-Ray were normal and Echocardiogram was normal except minimal dilatation of left ventricular cavity.

Discussion:

Abnormal direct communication between arteries and veins is called Arteriovenous fistula. Acquired arteriovenous fistula is a rare but curable cause of Cardiac failure and should be considered and sought specifically in any case with high output Cardiac failure in the absence of regurgitant valvular lesions and thyrotoxicosis. The high output states are recognised by their characteristic physical signs which are tachycardia proportionate to the degree of failure, large volume collapsing pulse, warm-extremities, large difference between systolic and diastolic blood pressures, hyperdynamic Cardiac impulse, clinical Cardiomegaly and systolic ejection flow murmurs.

The systemic arteriovenous fistulae may be congenital and acquired. Acquired fistulae are due to trauma; accidental or surgical, usually involving the large peripheral arteries they may be visceral, e.g., following nephrectomy or musculoskeletal, e.g., laminectomy. Arteriovenous fistulae are created surgically in patients with renal disease for haemodialysis.

Although such fistulae are well tolerated by patients with normal heart, they may cause heart failure in older patients as illustrated by our case.

When situated over exposed area, the fistula appears as pulsatile mass, the skin over the mass is warm with continuous thrill and bruit over it. There are signs of chronic venous insufficiency with incompetent varicose veins distal to the fistula (well shown in the photograph of our case). Branham's bradycardia sign is elicited by obstruction of the fistula by manual compression of the fistula or its afferent artery which is promptly followed by a sharp decrease is pulse rate. This sign suggests that the fistula is large enough to be a potential cause of heart failure.

Arteriography is essential for diagnosis only for

those fistulae that are situated so deeply that the pulsatile mass with thrill and bruit cannot be detected and Branham's sign can not be elicited.

The only satisfactory treatment of systemic Arterio-venous fistula causing heart failure is the excision of the fistula with restoration of the arterial continuity whenever possible. Repair of the fistula may result in the return of the heart size and function to normal as was documented in our case.

***Address for Correspondence:**

Medical B Unit,
Hayat Shaheed Teaching Hospital,
Peshawar.

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