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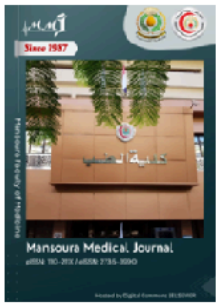

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EFFECT OF TEMPORARY OCCLUSION OF ARTERIOVENOUS FISTULAE ON LEFT VENTRICULAR DIASTOLIC FUNCTION IN PATIENTS WITH CHRONIC RENAL FAILURE

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ABSTRACT

Background : Numerous factors affect the development of left ventricular hypertrophy & dysfunction in end stage renal disease. Among those, arteriovenous fistulae emerged as an important factor. These fistulae undergo surgical closure following successful renal transplantation. However, sudden closure of arterivenous fistulae after its presence for long time may have an implications on left ventricular diastolic performance & may precipitate heart failure.

Objectives : To assess the possible impact of AV closure on diastolic function we prospectively examined 24 patients with ESRD with AV fistulae on regular haemodialysis before & after temporary complete fistulae occlusion. EchoDoppler parameters of diastolic function including transmitral early filling (E) & atrial filling (A) wave acceleration & deceleration times, transmitral E & A wave peak & mean velocities & gradients, transmitral E & A wave velocity time integrals , heart rate .& isovolumic relaxation time.

Results : Transmitral (E) & (A) wave acceleration times were significantly shorter while transmitral (E) & (A) wave deceleration times were significantly longer after than before sudden fistulae occlusion. Heart rate was significantly slower after than before temporary fistulae occlusion. There were non significant trend for E/A peak & mean velocity & gradient decrease or reversal & increase in isovolumic relaxation time after than

before temporary fistulae occlussion.

Conclusion : Temporary changes in left ventricular diastolic filling characteristics induced by sudden & complete temporary occlusion of arteriovenous fistulae in chronic renal failure patients unmask the occult LV diastolic dysfunction & may draw the attention towards a more carefully planned surgical closure of these fistulae.

Key words : Left ventricular diastolic dysfunction, chronic renal failure, arteriovenous fistulae.

INTRODUCTION

The clinical importance of the diastolic function of the heart cannot now be questioned (1). It is increasingly recognized that impairment of left ventricular diastolic function produces significant hemodynamic abnormalities, which may contribute substantially to, or be fully responsible for, the pathophysiology of many cardiac diseases (2-4). In conditions such as left ventricular hypertrophy (LVH) induced by pressure load (hypertensive heart disease or aortic stenosis), diastolic dysfunction may lead to frank cardiac failure even when systolic function is normal [5].

Cardiovascular disease is the leading cause of mortality in patients with end-stage renal disease (ESRD) on maintenance hemodialysis [6]. Although numerous factors affect the development of LVH & dysfunction in ESRF it is principally from a combination of longstanding pressure and volume overload [7-17]. Many of these patients need arteriovenous fistulae to perform regular hemodialysis.

The effect of long-term arteriovenous fistulae on cardiac function has been recognized for many years [18]. There is a reduction in peripheral resistance and blood pressure is maintained through an elevation of cardiac output mediated by an increased heart rate and stroke volume [19]. Acute compression of such fistulae has been shown to produce an immediate reduction in stroke volume and heart rate [20], however neither long-term studies nor controlled intervention studies have been conducted.

To our knowledge, no prospective controlled studies had been conducted to assess the possible impact of transient complete occlusion of arteriovenous fistulae on left ventricular diastolic function in patients with chronic renal failure subjected

to regular hemodialysis.

PATIENTS & METHODS

This prospective controlled study was carried out in Mansoura University Hospital form March 2002 to September 2002. The study population comprised 24 patients 12 males age range (39-55) years & 12 females age range (25-47) years. Patients were obtained form dialysis unit in ward 3 internal medicine in Mansoura university hospital & from intensive care unit in Mansoura emergency hospital.

Each patient served as a control for himself. All patients underwent complete transthoracic echoDoppler study with special stress on diastolic function parameters before & immediately after transient occlusion of the functioning surgically created arteriovenous fistulae. Transient occlusion of the fistulae was done after measurement of the systolic blood pressure & then inflating the sphygmomanometer cuff 20 mmHg above the recorded systolic blood pressure for at least 1 minute before repeating the echoDoppler study. The following left ventricular diastolic performance parameters were obtained:- transmitral early filling (E) & atrial filling (A) wave acceleration & deceleration times, transmitral E & A wave peak & mean velocities & gradients, transmitral E & A wave velocity time integrals & heart rate. Isovolumic relaxation time was obtained by simultaneous recording of mitral & aortic flow via sample volume placement mid way between the LVOT & anterior mitral leaflet then measuring the time interval between the end of aortic flow & the beginning of mitral flow.

STATISTICAL ANALYSIS

Statistical analysis was done using computer software SPSS version 8. Data were expressed as mean & standard deviation. Non parametric 2 related samples Wilcoxon rank test was done to compare between echoDoppler data before & just after occlusion.

RESULTS

EchoDoppler data were shown in table 1.

The table clearly showed that transient occlusion of AV fistulae produced statistically significant shortening of acceleration times of transmitral E & A waves, where at the same moment produce statistically significant lengthening of their deceleration times. Heart rate was statistically

slower after than before fistulae occlusion. There is non significant change regarding the isovolumic relaxation time, transmitral E & A wave peak & mean velocities, gradients & velocity time integrals.

Table 1

EchoDoppler data	Before occlusion of AV fistulae	After occlussion of AV fistulae	P value
Transmitral E wave peak velocity	0.7167 + 0.149	0.7413 + 0.2628	0.699
Transmitral E wave peak gradient	2.1487 + 0.8565	2.1988 + 1.2075	0.519
Transmitral E wave mean velocity	0.4729 + 0.06528	0.4737 + 0.1303	0.864
Transmitral E wave mean gradient	1.1 + 0.3282	1.115 + 0.5863	0.917
Transmitral E wave acceleration time	113.2071 + 16.1267	93.3087 + 10.9876	< 0.0001
Transmitral E wave deceleration time	160.2888 + 32.5273	169.345 + 48.4913	0.02
E wave velocity time integral	0.09793 + 0.03097	0.09625 + 0.03308	0.731
Transmitral A wave peak velocity	0.7967 + 0.1158	0.815 + 0.1239	0.112
Transmitral A wave peak gradient	2.0989 + 1.2418	2.735 + 0.7916	0.229
Transmitral A wave mean velocity	0.5241 + 0.07476	0.5325 + 0.07759	0.295
Transmitral A wave mean gradient	1.1733 B+ 0.5481	1.3638 + 0.3813	0.497
Transmitral A wave acceleration time	93.4125 + 16.9936	82.2488 + 11.4426	0.001
Transmitral A wave deceleration time	78.5784 + 14.7471	87.7438 + 24.3653	0.008
Transmitral A wave velocity time integral	0.0825 + 0.09891	0.08375 + 0.1345	0.317
E/A ratio	0.9061 + 0.2191	0.9449 + 0.4713	0.303
Isovolumic relaxation time	75.1133 + 30.2023	87.11 + 15.0735	0.335
Heart rate	89.5714 + 13.9089	86.5714 + 10.6751	0.012

DISCUSSION

Relation between mitral flow characteristic and hemodynamics :

A pressure gradient between the left atrium and the left ventricle is a prerequisite for blood to flow into the left ventricle during diastole. Many factors affect the amplitude and duration of pressure gradients. In addition, for any given pressure gradient, the passive-elastic properties of the chambers will determine how much blood moves from the atrium into the ventricle (1). Thus left ventricular filling is strongly modulated by the prevailing hemodynamic situation (21). Preload is a very significant determinant of the filling pattern, as, to a lesser extent, is afterload (1,22) . Similar hemodynamic profiles may occur in many different types of cardiac disease or different physiological conditions. Therefore, a left ventricular filling pattern cannot be equated with any one disease state, nor can a given diastolic abnormality (such as slowed left ventricular relaxation, for example) be equated directly with a single filling pattern. Because the hemodynamic situation associated with any cardiac disorder may change from day to day, or minute to minute, the transmitral Doppler flow pattern must be expected to change accordingly.

Three general abnormalities in transmitral flow patterns have been described, slow relaxation, 'pseudonormal' and 'restrictive. It is believed that, at least in some disorders, there is an evolution from one pattern to the next as hemodynamic decompensation progresses, and, correspondingly, that patterns can also 'regress' from more abnormal to more normal if hemodynamic conditions improve.

The rate of isovolumic relaxation (IVRT) is liable to be slowed with almost any form of cardiac pathology, including, ischemia, increased afterload (e.g. hypertension) or myocardial hypertrophy (3,23). Thus, a mitral inflow pattern reflecting slowed left ventricular relaxation may be a very early indicator of diastolic dysfunction. Slowing of left ventricular relaxation will reduce the atrioventricular pressure gradient and tend to delay mitral valve opening and, therefore, prolong the IVRT. The peak pressure gradient between left atrium and left ventricle during early diastole will also be smaller; so that the peak E velocity will be diminished. The acceleration and deceleration of the early filling wave will be markedly diminished be-

cause of the reduction in the transmitral pressure gradient both at mitral valve opening and throughout the rapid filling phase. Finally, the atrial contribution to ventricular filling will usually increase (peak A velocity), probably as a compensatory mechanism.

Progressive cardiac dysfunction may lead to an elevation of left atrial pressure by a variety of recognized mechanisms. That elevation tends to restore the early diastolic atrioventricular pressure gradient that has been diminished by impaired left ventricular relaxation. This may have a number of consequences: (i) the mitral valve opens earlier, which (ii) shortens the IVRT to the extent that it may actually be shorter than normal, and then (iii) restore the E wave to a more normal size and shape, though the deceleration of the early filling may be more rapid than normal. Thus, elevation of left atrial pressure may result in 'pseudonormalization' of the transmitral flow pattern, despite a continued abnormality of left ventricular relaxation [24,25]. The pseudonormal pattern may be distinguished from normal by the findings of shortened IVRT, rapid E wave deceleration, and increased flow reversal in the pulmonary veins following atrial contraction.

Further progression of myocardial dysfunction may cause marked elevations of preload leading to an increased left ventricular distension and stiffness. The filling pattern that characterizes 'restriction' or increased ventricular stiffness, has a tall and deep E wave with very rapid acceleration and deceleration. This is because of blood entering the noncompliant left ventricle under high pressure [4,24,26]. The IVRT is generally shorter than normal because the mitral valve will open earlier in the presence of increased left atrial pressure. There is usually a marked reduction in the amplitude and time-velocity integral (area) of the transmitral A wave, while the A wave of reversed flow in the pulmonary veins is clearly augmented .

Effect of ESRD on cardivascular function:

Cardiovascular disease is the leading cause of mortality in patients with end-stage renal disease (ESRD) on maintenance hemodialysis (6). Although numerous factors affect the development of LVH & dysfunction in ESRF it is principally from a combination of longstanding pressure and vol-

ume overload. The main contributors to pressure overload include an accelerated pulse wave velocity (PWV) - associated with decreased arterial compliance and an early return of arterial wave reflections [7,8], hypertension [9,10] and aortic stenosis [11].

Left ventricular dilatation is observed in 32-38% of patients with ESRF: most patients on echocardiographs demonstrating internal LV diameters around the upper limits of normal (12). Dilatation (and eccentric hypertrophy) results from a sustained increase in blood volume or flow in association with a high output state as occurs with plasma volume expansion, anemia and arteriovenous fistula formation.

Left ventricular dilatation may also occur in response to programmed myocyte death, possibly exacerbated by diminished coronary reserve and perfusion [13,14], ischemic heart disease, malnutrition, hyperparathyroidism[15,16]or inadequate dialysis [17].

Impact of arteriovenous fistulae on cardiovascular function :

Many patients with ESRD undergo regular hemodialysis through surgically created arteriovenous fistulae. The effect of long-term arterio-venous fistulae on cardiac function has been recognized for many years[18]. There is a reduction in peripheral resistance and blood pressure is maintained through an elevation of cardiac output mediated by an increased heart rate and stroke volume [19] . Acute compression of such fistulae has been shown to produce an immediate reduction in stroke volume and heart rate [20], however neither long-term studies nor controlled intervention studies have been conducted.

Potential advantages of renal transplantation on cardiovascular system can not be questioned. Cardiovascular improvement is expected to occur via abolition of many of factors contributing to cardiovascular dysfunction [27]. However, many physicians may be reluctant for the mode of arteriovenous fistulae closure & its immediate impact on the cardiovascular system especially if it is functioning for long time. So it is critical to investigate the possible hemodynamic effect of sudden & complete occlusion of AV fistulae on left ventricular diastolic performance.

In our study, temporary AV fistulae occlusion produced statistically signifi-

cant lengthening of E wave deceleration time together with a non significant trend towards E/A decrease or reversal & increase in isovolumic relaxation time. These findings could be explained by 2 factors. First, the reduction of rapid escape of blood to the venous side of the circulation produced by temporary fistulae occlusion may contribute significantly to preload reduction thus decreasing E wave amplitude that is higly dependent on preload (1, 22). Second, the sudden increase in after load with increased pulse wave reflection may significantly slow the left ventricular relaxation(1, 22).

We conclude that Temporary changes in left ventricular diastolic filling characteristics induced by sudden & complete temporary occlusion of arteriovenous fistulae in chronic renal failure patients unmask occult left ventricular diastolic dysfunction in those patients & may draw the attention towards a more carefully planned surgical closure of these fistulae (e.g., staged or gradual closure) in patients with chronic renal failure after renal transplantation has been successfully conducted to allow for gradual accommodation of the left ventricle to the new hemodynamic environment since sudden transition of the left ventricle from lower impedance systemic circuit to a high impedance one may precipitate left ventricular diastolic dysfunction & possibly heart failure despite adequate systolic function.

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تأثير الغلق المؤقت للوصلة الشريانية الوريدية على الوظيفة الانبساطية للبطين الأيسر للقلب فى حالات مرضى الفشل الكلوى المزمن

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هناك عوامل كثيرة تؤثر على تضخم البطين الأيسر للقلب فى حالات الفشل الكلوى المزمن ومن ضمن هذه العوامل الوصلة الشريانية الوريدية التى يتم من خلالها عملية الغسيل الكلوى .

الغلق المفاجئ للوصلة بعد استمرارها وبقاؤها لفترة طويلة قد يؤدى إلى التأثير على وظائف البطين الأيسر مما قد يؤدى إلى فشل فى وظائف القلب.

وقد هدفت هذه الدراسة إلى توضيح دور غلق الوصلة الشريانية الوريدية على الوظيفة الانبساطية للبطين الأيسر.

وقد شملت هذه الدراسة ٢٤ مريضاً من حالات الفشل الكلوى المزمن مع وجود وصلة شريانية وريدية حيث يتم لهم عملية الغسيل الكلوى بصورة منتظمة من خلال هذه الوصلة

ويتم تقسيم المرضى إلى مجموعتين ١٢ مريضا من الذكور ١٢ مريضا من الإناث وكان نطاق عمر المجموعة الأولى يتراوح من ٣٩-٥٥ سنة ونطاق عمر المجموعة الثانية يتراوح بين ٢٥-٤٧ سنة وقد تمت هذه الدراسة فى وحدة الغسيل الكلوى بمستشفى المنصورة الجامعى ووحة العناية المركزة بمستشفى الطوارئ الجامعى.

وقد تم عمل موجات فوق الصوتية على القلب بالدوبلر لمجموعات البحث قبل وبعد غلق الوصلة مباشرة عن طريق رفع الضغط أعلى من ضغط الدم المقاس بواسطة جهاز الضغط فى الجزء الشريانى أعلى مستوى الوصلة مع أخذ بعض القياسات الخاصة بالوظائف الانبساطية .

وقد استخلص البحث على أنه يوجد اختلال فى وظائف القلب الانبساطية بعد الغلق المؤقت للوصلة الشريانية الوريدية. ولذلك فإنه يجب التخطيط لاختيار الطريقة المثلى لغلق الوصلة الشريانية الوريدية فى مثل هؤلاء المرضى وذلك منعاً لحدوث هبوط القلب الناتج عن الغلق المفاجئ لها.