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A new femoral compression device compared with manual compression for bleeding control after coronary diagnostic catheterizations

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Objectives: We evaluated the safety and efficacy after hemostasis with our locally designed Assiut femoral compression device (AFCD) versus manual compression after diagnostic coronary angiography at Assiut University Hospitals.
Background: Previous clinical studies have indicated that FCD can be used for achievement of hemostasis after coronary angiography (CA). This study investigated the safety and efficacy of AFCD in achieving hemostasis after CA compared with manual compression in a cohort of consecutive patients.
Methods: A total of 162 patients were followed after CA was performed with the transfemoral approach. Univariate and multivariate analysis were used to identify the predictors of vascular complications with AFCD (n. 56) or with manual compression (n. 106) as a hemostasis option after sheath removal.
Results: As regard safety, the use of AFCD was associated with similar occurrence of minor bleeding compared with manual compression (25% vs. 26%, p, 0.8). There was no significant difference in occurrence of major bleeding compared with manual compression (1% vs. 2%, p, 0.5). None of our patient had a pseudoaneurysm, site infection or arteriovenous fistulae with either hemostasis technique. The use of AFCD was associated with a more frequent occurrence of vasovagal manifestation compared with manual compression (5.4% vs. 1%, p, 0.04).

As regard efficacy, the device application was easy in 84% with good fixation and stability in 90% of patients. Success in complete hemostasis was 91% with rapid learning curve. There was no difference in total duration of compression to reach full hemostasis with AFCD versus manual compression (13 ± 4 min vs. 12.5 ± 4 min., p, 0.4).
Conclusions: In this early experience with our locally designed AFCD after CA, their use was associated with similar efficacy and safety profile compared with hemostasis using manual compression.

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Hepatitis C Viral (HCV) Infection as a novel risk factor for severe coronary artery disease: A Prospective Angiographic Study

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Background: The link between coronary artery disease (CHD) and chronic hepatitis C virus (HCV) infection has been shown in many studies. However, the effect of chronic HCV infection on the extent of coronary artery disease (CAD) has not been determined so; the aim of the present study is to determine the effect of HCV infection on the severity and the pattern of CAD in Egyptian patients.
Patients and methods: This study group included two groups of patients with angiographically documented CAD; 25 HCV seropositive patients as test group and another 25 HCV seronegative patients as control group. Both groups were comparable as regard, age, sex, hypertension, and diabetes mellitus, and smoking. A detailed qualitative coronary angiographic analysis and SYNTAX score were used to assess the extent and severity of CAD.
Results: The presence of total occlusion was significantly higher in the HCV seropositive group ($p < 0.05$) and the SYNTAX score was higher (14.86 ± 6.64 vs. 10.86 ± 7.28, $p < 0.05$). After adjustment, HCV seropositivity still represented an independent predictor for severity of coronary atherosclerosis demonstrated by higher SYNTAX score ($p < 0.05$).
Conclusion: HCV infection is an independent predictor for severe coronary atherosclerosis, as demonstrated by higher syntax score. It also associated with higher incidence of totally occluded coronaries.

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Angiotensin-converting enzyme insertion/deletion polymorphism in hypertrophic cardiomyopathy: An Egyptian case control study

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Introduction: Hypertrophic Cardiomyopathy (HCM) is a disease characterized by genetic and phenotypic heterogeneity. Renin–angiotensin–aldosteron be system (RAAS) is a potential disease modifier. The aim of the present case control study is evaluation of the controversial role of ***ACE*** I/D polymorphism in HCM among Egyptians.
Subjects and methods: The study comprised 211 unrelated HCM patients (138 sporadic, 73 familial) and 203 age and sex matched ECG screened healthy volunteers. ***ACE*** I/D polymorphism was determined using previously described PCR and gel electrophoresis based method.
Results: Distribution of ***ACE*** genotype among the Egyptian controls was in Hardy-Weinberg equilibrium ($\boldsymbol{P} = 0.778$) but not in HCM patients ($\boldsymbol{P} = 0.0010$). The ***ACE*** DD genotype was significantly higher among HCM patients ($\boldsymbol{P} = 0.049$), particularly in sporadic HCM group compared with familial cases ($\boldsymbol{P} = 0.0001$). In addition, the distribution of D allele was significantly higher in HCM patients carrying sarcomeric mutations in ***TNNT2*** and ***MYH7***, ($\boldsymbol{P} = 0.0476$). There was no observed significant effect of the ACE genotypes on the phenotypic expression of the disease.
Conclusion: The finding of higher frequency of DD genotype among HCM patients compared to healthy volunteers, particularly so, in sporadic cases suggests that HCM expression is possibly influenced by a genetically predisposed milieu partially determined by the ACE I/D variants. Despite the lack of significant correlation between I/D variants and clinicopathologic characteristics of the HCM patients, however, the higher prevalence of D allele among TNNT2 and MYH7 mutation carriers may contribute to the variable disease outcome among sarcomeric gene positive cases, such a correlation can only be proven through long term follow up studies.

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Assessment of left atrial function by volumetric indices and tissue Doppler imaging in ischaemic and idiopathic dilated cardiomyopathy

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Background: Left atrial (LA) contractility plays an important role in maintenance of cardiac output in patients with left ventricular systolic dysfunction. Although left atrial contractile dysfunction has been reported in dilated cardiomyopathy of ischemic and non-ischemic etiology, the mechanism of LA dysfunction and the pathophysiologic determinants of left atrial size and function have not been adequately investigated in these patients.
Aim of the work: The aim of this study was to evaluate LA size and contractile function in patients with dilated cardiomyopathy of ischemic and idiopathic etiology and to explore the mechanism and determinants of LA dilation and contractile dysfunction in these patients.
Methods: 35 patients with ischemic dilated cardiomyopathy, 15 patients with idiopathic dilated cardiomyopathy and 30 control subjects were studied with transthoracic conventional echocardiography, tissue Doppler imaging (TDI) and coronary angiography (CA). Left ventricular (LV) size, systolic and diastolic functions as well as mitral regurgitation (MR) were evaluated. Left atrial volume at mitral valve opening (Vmax), onset of atrial systole, determined by onset of the P wave of the electrocardiogram (Vp) and mitral valve closure (Vmin) was determined with two-dimensional echocardiography. The left atrial contractile function was assessed by means of active emptying fraction (ACTEF = {Vp − Vmin}/Vp%) and TDI for assessment of late diastolic velocity of the mitral annulus and left atrial free wall.
Results: Left atrial Vmax was greater while ACTEF and left atrial wall velocity were lower in cardiomyopathy patients compared with the control subjects (79 ± 32 vs. 59 ± 18; $P < 0.05$, 27.6 ± 13 vs. 42 ± 15; $P < 0.05$ and 10.2 ± 4.7 vs. 16.2 ± 5.4; $P < 0.05$, respectively). Vmax , ACTEF and left atrial wall velocity were similarly affected in both types of cardiomyopathy, ischaemic and idiopathic, under the same loading conditions (74 ± 24 vs. 91 ± 46; $P > 0.05$, 29 ± 12 vs. 27 ± 13; $P > 0.05$ and 10 ± 5 vs. 11 ± 4; $P > 0.05$, respectively). The determinants of ACTEF were left atrial volume, left ventricular ejection fraction (EF), E/e' and MR severity.
Conclusion: Left atrial enlargement and contractile dysfunction are common in patients of dilated cardiomyopathy regardless of its aetiology, with the same degree of contractile dysfunction in both ischemic and idiopathic cardiomyopathies under similar loading conditions. This dysfunction is related to worse LV systolic and diastolic function, more severe mitral regurgitation and larger LA volume rather than to the aetiology of cardiomyopathy.

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Assessment of left ventricular long axis contraction in patients with ischemic mitral regurgitation after acute myocardial infarction

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Introduction: The development of ischemic mitral regurgitation (MR) after myocardial infarction (MI) may impose hemodynamic load during a period of active left ventricular remodeling and promote heart failure (HF). The aim of our study was to evaluate left ventricular (LV) long axis contraction assessed by both mitral annular plane systolic excursion (MAPSE) and peak systolic velocity in patients with ischemic MR after acute MI.
Methods: Thirty eight patients with a first attack of acute MI were classified into two groups. Group I comprised 18 patients with MI and ischemic MR, and group II comprised 20 patients with MI without ischemic MR. Twenty subjects without acute MI were considered as the control group (group III). Measurement of MAPSE from M-mode tracing of the mitral annulus from apical 4- and 2-chamber, and Pulsed wave tissue Doppler imaging (TDI) of the 4 sides of the mitral annulus for assessment of peak systolic (Sa) and diastolic (Ea and Aa) velocities were done.
Results: Significant decrease of MAPSE in 4 sides in patients with acute MI with MR compared to MI without MR and control group ($P < 0.05$). Peak systolic velocity (Sa) in septal, anterior, inferior sides of mitral annulus was significantly decreased in MI patients compared with control group ($P < 0.05$); and not in lateral side ($P > 0.05$). Significant correlation between MAPSE on anterior side of mitral annulus and LV EF ($P < 0.001$) in patients with ischemic MR after acute MI.
Conclusion: mitral annular displacement is a useful parameter for assessment of longitudinal LV function in patients with ischemic MR after MI.

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