tricular systolic and diastolic functions, mass and geometry by using conventional echocardiography and pulsed wave tissue Doppler imaging in individuals with MS compared to healthy subjects.

Methods: The study included 120 subjects divided by half into control and case group: 60 subjects (30 males and 20 females) with MS (diagnosed according to consensus statement from the International Diabetes Federation (platinum standard)) aged 54.4 ± 9.5 years and 60 healthy subjects (32 males and 28 females) aged 50.3 ± 13.7 years. Left ventricular ejection fraction (LVEF) was measured with the Teich method in the short axis parasternal view by averaging measurements from five consecutive cardiac cycles. The LV diastolic function was estimated with Doppler measurement of transmitral blood flow and determination of the maximal rates for E and A peaks, E/A ratio, delay time (DT) for early diastolic filling rate, E', A' and E/A' ratio according to criteria of the European Study Group on diastolic heart failure. IMP was be calculated as the sum of IVRT and IVCT divided by ventricular ET (IMP = IVRT + IVCT/ET). LV mass was determined using the corrected cube formula as $(0.80 \times (1.04 \times [(septal thickness + LV internal diameter + posterior wall thickness)^3] - (LV internal diameter)^3)] + 0.6$.

Results: The assessed parameters using conventional and tissue Doppler echocardiography showing that those with metabolic syndrome had greater LV mass and significantly lower E/A ratio; increased LVH and significantly higher IMP, indicating impaired global left ventricular functions in patients with MS compared with control subjects without MS. There were no statistically significant difference in EF percentage.

Conclusions: Our results demonstrate that LV functions are impaired in patients with metabolic syndrome using IMP and E/A ratio. LVH incidence are higher in metabolic syndrome patients. To conclude metabolic syndrome are associated with significant cardiac functions deterioration and should be prevented and treated in proper manner.

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HCV Cardiomyopathy; raising a regional alert
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Introduction: The global prevalence of Hepatitis C Virus “HCV” carriers is estimated to average 3%, ranging from 0.1% to ≥10% among different countries, 17–30% of them complain of HCV Cardiomyopathy.

Objectives: To raise regional awareness of HCV as a cardiotropic virus through sharing four stages of our HCV Cardiomyopathy research.

Methodology: Fifty HCV positive patients with 50 controls were enrolled in first stage where conventional echocardiography was used to evaluate LV systolic and diastolic function. Tissue Doppler imaging was used in the second stage to evaluate 30 HCV patients and 30 controls. Forty five HCV positive patients with 45 controls were evaluated by Strain Rate Imaging to assess left atrial tissue dynamics in addition to measuring NTproBNP. At our fourth stage, we evaluated combined antiviral and anti-inflammatory therapy in 13 patients complaining of HCV Cardiomyopathy. Speckle tracking echocardiography was our tool to assess myocardial function before and after administration of therapy.

Results: HCV group showed significant increase in QTc interval, significant increase in A wave, Deceleration time in addition to highly significant decrease in tissue Doppler $E_{s}$, highly significant decrease in $A_{s}$ and highly significant increased $E/E_{s}$ ratio. Evaluation of Left atrial function revealed significant increase in left atrial ejection fraction, ejection time, ejection force. Significant improvement of global myocardial strain was showed after administration of therapy in patients with HCV Cardiomyopathy.

Conclusion: HCV is a cardiotropic virus. HCV derived heart diseases are chronic and devastating diseases. The combined anti-viral and anti-inflammatory treatment may cure HCV derived cardiomyopathy.

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Left main stenting, a short and intermediate follow-up
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Background: Significant left main coronary artery disease occurs in about 5–7% of patients undergoing coronary angiography. Coronary artery bypass graft (CABG) surgery was the standard acceptable way in treating these lesions. The introduction of drug eluting stents (DES) in mid 2003 has pushed the rate of clinical restenosis below 5% for most lesion types. This encouraged the cardiologists to treat the LMCA with DES.

Methods and results: We implanted DES in 30 patients with denovo LMCA-Stenosis (middle age 69.9 ± 12.5 83% men). 25 patients (83%) had hypertension, 5 diabetes mellitus (17%), 22 hyperlipidemia (73.3%) and 16 were smokers (53.3%). We implanted Paclitaxel-Stents in 26 (87%) and 4 Sirolimus-Stents (13%). 11 patients (37%) with Stents <16 mm. In 14 patients (47%) were distal LM (Bifurkation) and in 16 (53%) ostium or schaft. Details of intervention technique (T-Stenting, V-Stenting, Culotte and Y-Stenting) was left to the examiner. During the followup (middle duration 6 months; range 4–12 months) were no myocardial infarctions, Stent thrombose or death documented. In 4 patients (13.3%) was a re-revascularisation procedure mandatory (1 CABG, 3 re-interventionen), without significant difference between distal or non-distal lesions, type or length of stent.

Conclusion: LMCA intervention is in the hands of experienced interventionalist technically possible and sure (without MACE), connected with an accepted rate of re-intervention.

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