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PS3
The connection of diabetic dyslipidemia and erectile dysfunction

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Background Diabetes is an established risk factor for sexual dysfunction in men; a threefold increased risk of erectile dysfunction (ED) was documented in diabetic compared with non-diabetic men. Diabetic vasculopathy concerns macroangiopathy, microangiopathy and endothelial dysfunction. Macrovascular disease in diabetes corresponds to the atherosclerotic damage in the blood vessels, which limits blood flow to the penis.

Aim Value the connection of dyslipidemia and erectile dysfunction in men patients who are ill with diabetes.

Materials and methods In the research, 180 patients at the age of 33–67 with type 2 diabetes mellitus were participated. According to the duration of diabetes, the patients were divided into three groups and revealed the erectile dysfunction. The lipid metabolism was revealed and HbA1c was determined in all the patients.

Results The patients who are ill with diabetes within 1–5 years (n = 41, 22.78%), the erectile dysfunction is met in 54.15% of patients (in this group n = 14), those who are ill with diabetes 5–10 years, the erectile dysfunction is met in 57.53% (in this group n = 43), who are ill with diabetes within 10 years and more the erectile dysfunction is met in 69.72% of patients (n = 46).

The result of lipid metabolism value showed that hypercholesterolemia was met in 42.22% (n = 76) of the patients, hypertriglyceridemia was met in 21.11% (n = 38), combined disorder was met in 24.44% (n = 44). Thus, 87.77% of the examined patients have lipid metabolism disorder. The result of the research showed that erectile dysfunction was usual in case of dyslipidemia. It should be noted that 60.52% of the patients (n = 23) who had isolated hypertriglyceridemia had erectile dysfunction, 63.16% of the patients (n = 48) who had hypercholesterolemia had erectile dysfunction and 72.73% (n = 32) of the patients with combined disorder had erectile dysfunction.

Conclusion Surely, the erectile dysfunction in diabetes mellitus has a multifactorial nature such as duration of diabetes, level of compensation, complications especially neuropathy availability and harmful habits. According to our findings, we have a lot of patients with erectile dysfunction and hypertriglyceridemia. The findings remind us once more that medical practitioners have to pay attention especially to lipid metabolism condition, triglyceridemia level in diabetic patients. So, dyslipidemia is the reason of increasing cardiovascular risk and decreasing quality of life.

Disclosure of interest The authors declare that they have no competing interest.

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PS5
Influence of cardiac autonomic dysfunction and arterial stiffness on subendocardial myocardial viability in patients with type 2 diabetes

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Aims Cardiac autonomic dysfunction (CAD) is characterized by sympathovagal imbalance (reduced vagal activity and relative sympathetic overdrive). This condition might shorten diastole duration (DD) and thus impair coronary perfusion. Arterial stiffness is also considered to impair coronary perfusion. The aim was to investigate the influences of CAD and arterial stiffness on subendocardial myocardial viability (SVI) and in particular the relation between the severity of CAD, DD and SVI in patients with type 2 diabetes (T2D).

Methods In 42 T2Ds (55.2 ± 12.6 yrs, BMI 33.4 ± 7.3 kg/m 2, HbA1c 8.04 ± 1.58%) free of cardiovascular history, we measured noninvasively heart rate (HR) and blood pressure (Finapres ®) continuously during 5 minutes in supine position. We calculated then DD and arterial stiffness (augmentation index AIX and pulse wave velocity with a recently validated method; a Mac-Apple dedicated software that reconstructs the central aortic pressure curve from the peripheral one (measured on finger) by a transfer function). Pulse wave velocity (PWV) was calculated from the equivalent index SI-DVP derived from the pulse pressure profile. The percentage of DD [%D] = (DD/duration of heart period) × 100) and SVI (area under aortic pressure curve during diastole/area during systole) were calculated as well. CAD was assessed using standard tests (deep-breathing, lying-to-standing, Valsalva), which mostly depend on vagal control.

Results CAD was absent in 7 patients (Autonomic score 0; AS0), early (AS1: 1 abnormal test) in 14 patients, definite (AS2: 2 abnormal tests) in 15 patients and severe (AS3: 3 abnormal tests) in 6 patients. Mean HR in AS0 was 70.1 ± 11.6 bpm, in AS1: 74.9 ± 8.1, in AS2: 77.8 ± 11.2, in AS3: 95.8 ± 16.0 with a statistical significance (P < 0.05) between AS0 vs. AS3, AS1 vs. AS3, AS2 vs. AS3. DD% correlated negatively with HR (r = −0.747, P < 0.0001) and was lower in patients with higher AS. DD% differed significantly in AS3 and AS2 compared to AS0 (AS3: 52.8 ± 3.6%, AS2: 56.7 ± 4.1% and AS1: 59.7 ± 3.9% vs. AS0: 61.7 ± 3.3%; P = 0.0008, P = 0.011 and P = 0.25, respectively). Furthermore, the patients with AS2 or AS3 considered together showed lower DD% compared with the patients with AS0 or AS1 even after adjusting DD% with

Patients and method We had recruited 397 patients (281 women) with risk factors of diabetes, mean age 50.2 ± 11.7 years. All had fasting glycemia (FG) and oral post charge glycemia (PCG). Findrisk score was calculated. Carotid femoral pulse wave velocity (PWV) was measured by Complior 6 device.

Results FG and PCG were abnormal in 159 and 157 patients, respectively. Dysglycemia was found in 204 patients (51.4%), with diabetes in 17.6% and prediabetes in 33.8% of them. Findrisk score strongly correlated to FG (r = 0.129, P = 0.01) and PCG (r = 0.193, P = 10−5) but also to systolic (r = 0.33) and diastolic blood pressure (r = 0.20, and to PWV (r = 0.29). Discriminating threshold of this score for predicting dysglycemia was 13. It was significantly associated with prediabetes (P = 0.001) and dysglycemia (P = 10−5), this association was found in women but not in men. Sensibility and specificity of this score for detecting dysglycemia were 76% and 37%, respectively. Their positive and negative predictive values were 54.7% and 60.7%, respectively.

Conclusion FG and PCG detected dysglycemia in similar proportions. Identification of dysglycemia through Findrisk score was validated only in women. The correlation between Findrisk score and PWV suggests that a high score testified also of a high cardiovascular risk in these patients without glycemic abnormality or known cardiovascular history.

Disclosure of interest The authors declare that they have no competing interest.

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PS4
Findrisk score, pulse wave velocity and detecting dysglycemia in a risk population of diabetes

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Introduction and aim Findrisk score has been validated for prediction of diabetes risk in many populations. The aim of this study was to examine the value of this score for the detection of dysglycemia (impaired fasting glucose and/or glucose intolerance or diabetes) in a high-risk population in Constantine (Algeria).

Patients and method We had recruited 397 patients (281 women) with risk factors of diabetes, mean age 50.2 ± 11.7 years. All had fasting glycemia (FG) and oral post charge glycemia (PCG). Findrisk score was calculated. Carotid femoral pulse wave velocity (PWV) was measured by Complior 6 device.

Results FG and PCG were abnormal in 159 and 157 patients, respectively. Dysglycemia was found in 204 patients (51.4%), with diabetes in 17.6% and prediabetes in 33.8% of them. Findrisk score strongly correlated to FG (r = 0.129, P = 0.01) and PCG (r = 0.193, P = 10−5) but also to systolic (r = 0.33) and diastolic blood pressure (r = 0.20, and to PWV (r = 0.29). Discriminating threshold of this score for predicting dysglycemia was 13. It was significantly associated with prediabetes (P = 0.001) and dysglycemia (P = 10−5), this association was found in women but not in men. Sensibility and specificity of this score for detecting dysglycemia were 76% and 37%, respectively. Their positive and negative predictive values were 54.7% and 60.7%, respectively.

Conclusion FG and PCG detected dysglycemia in similar proportions. Identification of dysglycemia through Findrisk score was validated only in women. The correlation between Findrisk score and PWV suggests that a high score testified also of a high cardiovascular risk in these patients without glycemic abnormality or known cardiovascular history.

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regression analysis at a conventional HR of 75 bpm (CAD+: 57.8 ± 3.3% vs. CAD−: 59.9 ± 2.6%; P = 0.030), SVI showed the same trend as DD%, with lower values in the patients with higher AS and reached the statistical significance in AS3 compared to AS0 (AS0: 1.23 ± 0.2; AS1: 1.16 ± 0.1, P = 0.41 vs. AS0; AS2: 1.02 ± 0.2, P = 0.056 vs. AS0; AS3: 0.88 ± 0.1, P = 0.006 vs. AS0). A strong positive linear correlation was found between SVI and DD% (r = 0.921, P < 0.0001); conversely SVI showed a moderate negative linear correlation with aortic AIx adjusted at HR 75 bpm (r = −0.489, P < 0.001). No correlations were found with pulse wave velocity.

Conclusions In T2Ds CAD, expressed as reduced vagal activity, leads to HR acceleration and thus to diastole shortening, but it seems that CAD may shorten DD per se, independently of the effect on heart rate. Since DD influences strongly SVI, CAD plays a primary role in addition to arterial stiffness in the impairment of subendocardial myocardial viability and may thus worsen cardiovascular prognosis.

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PS6
The interaction between glucose, insulin, β-cells, α-cells and glucagon: Mathematical model
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Background Pancreatic β-cells and α-cells are responsible of controlling the level of glucose in the blood. While insulin reduces the level of blood glucose by stimulating its uptake by muscles and adipose tissues and storing it as glycogen in the liver, glucagon supplies another source of glucose (produced through liver gluconeogenesis and glycogenolysis) for blood. Consequently, damage in either β-cells or α-cells can lead to a dysfunction in insulin secretion or glucagon production.

Results and conclusion Our mathematical model shows that a normal pancreatic β-cell mass and α-cell mass maintains the level of glycemia in the normal range. The apoptosis of β-cells and α-cells is the origin of insulin and glucagon secretory defects causing severe hyperglycaemic state with high glucose rate in the blood.

Disclosure of interest The authors declare that they have no competing interest.

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PS7
Sequential compression/decompression by a pulsating suit increases cutaneous microcirculatory blood flow in patients with type 2 diabetes
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Background Microcirculation is altered in diabetic patients. In healthy subjects sequential compression/decompression by a pulsating suit may induce noninvasively endothelial activation by a pure mechanical effect, which is intended to increase the physiological shear stress. The objective was to evaluate the effects of sequential compression/decompression on the cutaneous forearm microcirculation in patients with type 2 diabetes.

Materials and methods Sixteen patients with type 2 diabetes (6 men and 10 women, age 53.3 ± 11.4 years, 6 hypertensives, all on oral hypoglycaemic agents, no smoker, no cardiovascular disease, no renal failure, no retinopathy, HbA1c 7.1 ± 0.8%) were enrolled in a controlled cross-over study and were randomly divided into two groups: a verum (at V1) and a phantom (at V2, 13 ± 2 days after V1) compression at 65 mmHg/compression session using Stendo® pulsating suit was performed in group 1 and vice-versa in group 2. Each session spent 20 minutes. The pulsating suit generates heart rate synchronized compression/decompression applied to the lower part of the body (legs and abdomen). Cutaneous forearm microcirculation blood flow was measured continuously by laser Doppler flowmetry (LDF, Periflux System 5000®) before, during and until 30 minutes after the end of the sessions.

Results The 20-minutes area under curves (AUC) calculated during Stendo sessions were 1976 (SD: 3938) and 2043 (SD: 8302), respectively in verum and phantom sessions. The mean 40-minutes and 50-minutes AUCs (during Stendo plus 20 and 30 minutes after sessions stopped) were respectively 6936 and 7403 in verum, −7537 and −10,805 in phantom. The differences for AUC40 min and AUC50 min were higher between verum and phantom sessions (P < 0.01 in all).

Conclusion In T2D patients, sequential compression of the lower part of the body synchronized with each diastole period at a physiological pressure (65 mmHg) induces a significant increase of the cutaneous forearm microcirculation flow, away from the pulsatile stimuli. Pulsating suit session appears to produce a “shear stress like” effect.

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