

these sex-differences in stroke risk. However, a more recent study found a persistently higher risk of stroke among women as compared to men despite similar warfarin adherence rates.

Objective: To describe the potential mechanisms behind the increased risk of stroke in AF associated with female sex.

Materials and Methods: General mechanisms of thromboembolism in AF – Rudolf Virchow postulated that thrombosis arises from three co-existing phenomena: abnormalities in the vessel wall, blood stasis, and a hypercoagulable state. Virchow's triad can be applied to thrombogenicity in AF. Structural changes in the left atrium (LA) and left atrial appendage (LAA), blood stasis induced by left atrial dilatation and inhibited forward flow contributes to thrombus formation in patients with and without AF. As a consequence of structural and blood flow changes, prothrombotic conditions develop with activation of coagulation proteins.

Potential mechanisms for higher stroke risk in women with AF Hormone therapy and menopause – the risk for ischemic stroke in women doubles between the ages of 55 and 65, the menopausal transition period during which estradiol levels decrease by about 60%. Endogenous estrogen has favorable outcomes on lipid metabolism, coagulation and vascular tone, and even incident AF. In a meta-analysis of seven major randomized trials analyzing hormone therapy (HT) reported an increased risk of stroke in both combination HT trials and estrogen-only trials.

Conclusion: Sex-related differences in the vasculature and myocardial structure may predispose to alterations in blood flow, shear stress, and altered endothelial function. Further, there is evidence suggesting a potential sex-based increase (especially in the post-menopausal state) in systemic inflammatory and procoagulant markers, thrombogenic particles and platelet aggregation, all of which contribute to a prothrombotic circumstance. Observational data suggest sex-based differences in stroke outcomes are related to differences in stroke risk factor profile and management, in addition to underutilization of anticoagulant therapy in women. However, recent study results demonstrate an increased stroke risk in women despite baseline anticoagulant use.

Keywords: Atrial fibrillation, female sex, thromboembolic stroke

32. ASSOCIATION BETWEEN CARDIAC AUTONOMIC NEUROPATHY AND PERIPHERAL NEUROPATHY IN DIABETES MELLITUS TYPE 1

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Introduction: Cardiovascular autonomic neuropathy (CAN) is defined as the impairment of autonomic control of the cardiovascular system. The prevalence of CAN varies widely from 2.5 to 50%. Neural damage by chronic hyperglycaemia, vascular insufficiency in the vessels supplying the nerves, and autoimmune mechanisms have been suggested as possible causes of CAN, its pathogenesis remains poorly understood. As in the case of diabetic peripheral neuropathies (DPN), disease duration and long-term poor glycaemic control are important risk factors for the development of CAN.

Purpose and Objectives: Evaluation of the correlation between CAN and peripheral neuropathy in type 1 diabetes (T1DM).

Materials and Methods: In study were included 27 patients (10 men and 17 women) with T1DM and CAN (the diagnosis of CAN was established on the basis of changes in heart rate and blood pressure, during cardiovascular reflex tests, Ewing's battery). According to the total score of the CAN severity, patients were divided in 3 groups. Were evaluated: T1DM duration and severity of peripheral neuropathy.

Results: The 1st group included 11 patients (40.7%) with mild CAN, which were discovered DPN mild – 6 patients (22.2%), DPN moderate – 5 patients (18.5%). T1DM average duration was 5.5±2.0 years. After analysis by the statistical method MedCalc 12.7.2 we detect significant correlation with mild DPN ($r=0.645$, $p=0.0003$).

The 2nd group was made by 8 patients (29.6%) with moderate CAN, which were observed the

presence of DPN moderate – 6 patients (22.2%) and DPN severe – 2 patients (7.4%). In this group the T1DM average duration was 13.6 ± 5.5 years. Important correlation with moderate DPN ($r=0.452$, $p=0.017$).

In the 3rd group were added 8 patients (29.6%) with severe CAN, which had severe DPN. The average duration of T1DM was 18.9 ± 7.6 years. In this group were observed more significant correlation with duration of T1DM ($r=0.585$, $p=0.0013$) and severe DPN ($r=0.846$, $p<0.0001$).

Conclusions:

1. Cardiac autonomic neuropathy severity correlates with peripheral neuropathy severity in type 1 diabetes.

2. Cardiac autonomic neuropathy and peripheral neuropathy severity increases with the duration of type 1 diabetes.

Keywords: Cardiac autonomic neuropathy, diabetes type 1, peripheral neuropathy

33. SERUM LIPID PROFILES IN PATIENTS WITH METABOLIC SYNDROME WITH OR WITHOUT CORONARY ARTERY DISEASE

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The purpose of study: To assess serum lipid profiles in patients with metabolic syndrome (MS) and stable angina pectoris (SAP) vs. patients with MS without SAP.

Material and methods: This study included 122 patients with metabolic syndrome (mean age 54.06 ± 0.86 years). The diagnosis of MS was established according to criteria proposed by IDF and AHA/NHLBT in 2009. All patients with MS and clinical signs of SAP undergo bicycle exercise stress test (EST), unless contraindicated. Depending on EST results, there were selected 66 (54.09%) patients with SAP and positive EST (group I) and 56 (45.9%) patients with negative EST (group II, control). Following evaluation included laboratory investigations: total cholesterol (TC), LDL cholesterol (LDL-C), HDL cholesterol (HDL-C), triglycerides (TG) and TC/HDL-C ratio $\geq 4,2$ in both groups.

Results: Lipid profile assessment revealed that the mean value of TC for patients in group I was 5.63 ± 0.14 mmol/l vs. 5.42 ± 0.15 mmol/l for patients in group II ($p>0.05$). The mean LDL-C in group I patients was 3.46 ± 0.11 mmol/l vs. 3.25 ± 0.13 mmol/l in group II patients ($p<0.05$). In group I patients we estimate a mean HDL-C value of 1.23 ± 0.04 mmol/l vs. 1.27 ± 0.04 mmol/l in group II patients ($p>0.05$). As for TG findings, the average value was 2.22 ± 0.1 mmol/l in group I patients vs. 1.95 ± 0.13 mmol/l in group II patients ($p>0.05$). When considering the frequency of dyslipidemia, we found TC values ≥ 4.5 mmol/l in 59 patients (95.16 %) from group I vs. 46 patients (82.14%) from group II ($p<0.05$). Values of LDL-C ≥ 2.5 mmol/l were found in 48 patients (87.27%) from group I vs. 44 patients (78.57%) from group II ($p >0.05$). Analysis of TG levels ≥ 1.7 mmol/l revealed significant higher rates of hypertriglyceridemia in group I patients (82.26%, $n=51$) vs. group II patients (48.21 %, $n=27$) ($p<0.001$). HDL-C assessment demonstrated values <1.0 mmol/l in men and <1.3 mmol/l in women in 22 patients (36.02%) with MS and SAP and 28 patients (50.0%), MS without SAP ($p >0.05$). Also an increased atherogenic index, as determined by the ratio of TC / HDL-C, was proven in both groups (group I - 4.7 ± 0.17 vs. group II - 4.3 ± 0.12 , $p >0.05$). In group I we determined values of TC/HDL-C ratio ≥ 4.2 in 36 patients (58.06%) vs. 23 patients (41.07%) in group II ($p>0.05$).

Conclusion: In both groups of patients we determined abnormal lipid profiles. To be also mentioned the presence of a larger number of patients with TC and TG values exceeding the allowable limits in the group with metabolic syndrome and stable angina pectoris.

Key words: lipid profile, metabolic syndrome, coronary artery disease