Conclusion: Our data showed that the level of hs-CRP of 2.5 mg/L was that discriminative value that indicates the risk for development and/or progression of PAD in patients with diabetes type 2. Furthermore, our investigation has found that the hs-CRP value greater than 2.5 mg/L increase the risk for PAD approximately three times.

P 3: Insulin sensitivity and related atherogenic conditions in ischemic stroke: comparison between type 2 diabetics and nondiabetics

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Background and aims: The role and importance of insulin sensitivity (IS) and related atherogenic conditions in ischemic stroke have not yet been elucidated. Therefore, our study was aimed to analyze (a) IS and plasma insulin (PI) (b) lipoproteins (c) plasminogen activator inhibitor 1 (PAI-1) levels (d) inflammatory markers, hs-C reactive protein (hs CRP) and interleukin-6 (IL-6) levels (e) abdominal obesity, in 40 type 2 diabetics (T2D) with ischemic stroke (group A), 35 T2D without ischemic stroke (group B), 35 nondiabetics with ischemic stroke (group C) and 34 healthy controls (group D).

Material and Methods: Ischemic stroke was confirmed by clinical and neuroimaging criteria. IS levels were determined by the frequently sampled intravenous glucose tolerance (FSIGT) test with minimal model analysis (Si index). Total cholesterol, HDL-cholesterol (HDL-c), and tryglicerides concentration were determined with the chromatography method. LDL-cholesterol (LDL-c) concentrations were calculated using the Friedewald formula. Plasma PAI-1 activity was determined by plasminogen chromogenic plasin substrate assay. Hs-CRP was determined by Olympus Analyzer and interleukin 6 (IL-6) levels were measured by ELISA method. Waist circumference was measured at the midpoint between the lower border of the rib cage and the iliac crest.

Results: Si levels were significantly lower in group A vs B (1.15 +/- 0.46 vs 2.81 +/- 0.65 min-1/mU/lx104; p<0.001) and in C vs D (3.12 +/- 0.77 vs 6.10 +/- 1.64 min-1/mU/lx104; p<0.001). However, PI levels were higher in group A vs B (19.96 +/- 4.10 vs 14.84 +/- 1.73 mU/l; p<0.001) and in C vs D (16.14 +/- 2.20 vs 7.76 +/- 2.08 mU/l; p<0.001). Also, LDL-c and PAI-1 were significantly higher in group A vs B (5.21 +/- 0.42 vs 4.12 +/- 0.53 mmol/l; p<0.001), (7.73 +/- 1.04 vs 4.58 +/- 0.66 mmol/l; p<0.001) and in C vs D (4.24 +/- 0.53 vs 3.66 +/- 0.52 mmol/l; p<0.001), (4.60 +/- 0.64 vs 3.40 +/- 1.23 mmol/l; p<0.001). Simultaneously, hs CRP and IL-6 levels were significantly higher in group A vs B (5.21 +/- 0.42 vs 4.12 +/- 0.53 mmol/l; p<0.001), (7.73 +/- 1.04 vs 4.58 +/- 0.66 mmol/l; p<0.001) and in C vs D (4.24 +/- 0.53 vs 3.66 +/- 0.52 mmol/l; p<0.001), (4.60 +/- 0.64 vs 3.40 +/- 1.23 mmol/l; p<0.001). Simultaneously, hs CRP and IL-6 levels were significantly higher in group A vs B (5.21 +/- 0.42 vs 4.12 +/- 0.53 mmol/l; p<0.001), (7.73 +/- 1.04 vs 4.58 +/- 0.66 mmol/l; p<0.001) and in C vs D (4.24 +/- 0.53 vs 3.66 +/- 0.52 mmol/l; p<0.001), (4.60 +/- 0.64 vs 3.40 +/- 1.23 mmol/l; p<0.001). Also, waist circumference was higher in group A vs B (16.22 +/- 2.33 vs 9.78 +/- 1.95 g/l; p<0.05; 20.14 +/- 4.56 vs 14.98 +/- 5.04 pg/ml; p<0.05) and in C vs D (7.54 +/- 0.67 vs 2.50 +/- 0.32 g/l; p<0.01; 11.45 +/- 2.26 vs 3.36 +/- 1.44 pg/ml; p<0.01). Also, waist circumference was higher in group A vs B (103.26 +/- 2.56 vs 93.97 +/- 9.51; p<0.01), and in C vs D (101.00 +/- 1.27 vs 84.19 +/- 1.45; p<0.001). The changes in SI significantly correlated with LDL-c, PAI-1, hs-CRP, IL-6 levels and waist circumference, both in T2D (r=0.388 r=0.376 r=-0.368 r=-0.413 r=-0.403 p<0.05) and nondiabetics (r=-0.398 r=-0.369 r=-0.372 r=-0.432 r=-0.394 p<0.05).

Conclusion: Our results demonstrated that decreased IS are associated with increased LDL-c, decreased PAI-1, together with higher hs-CRP, IL-6 levels and waist circumference, both in T2D and nondiabetics. These results imply that decreased IS in association with compensatory hyperinsulinemia, underlying the development of the ischemic stroke, through potentiation of dislipidemia, hypo fibrinolisis, low-grade inflammation and abdominal obesity.

P 4: Association of insulin sensitivity level and atherogenic lipid profile in mild cognitive impairment

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Background and aims: As an early stage of cognitive decline (mild cognitive impairment (MCI)) has been linked to changes in cholesterol profiles, previous studies have suggested that decreased insulin sensitivity and dyslipidemia are consistently indicated as essential in the pathophysiology, and possibly the pathogenesis of Alzheimer disease (AD), most common form of age-related non vascular dementia. As diagnosis of MCI, the intermediate cognitive disorder between normal aging and AD, are becoming more reliable, the aim of this study was to analyze in patient with MCI the levels of (a) insulin sensitivity (IS), (b) plasma insulin (PI) and (c) lipid parameters comprising total cholesterol (Ch), low-density (LDL) and high density (HDL) Ch, triglycerides and apolipoproteins (apoAI, apoAII, apoB, Lp(a) and apoE) potentially involved the pathogenesis of the disease.

Materials and Methods: We included 40 normoglycemic patients with MCI (group A; BMI: 24.43 +/- 0.52 kg/m2, age: 69.36 +/- 7.31 years), and 30 matched controls (group B; BMI: 24.29 +/- 0.61 kg/m2, age: 65.38 +/- 6.69 years). IS was evaluated by using euglycemic hyperinsulinemic clamp method with insulin infusion rate of 1 mU/kgbw/min during 120 min and glucose infusion adjusted manually, at 5 min intervals, to maintain target euglycemia. Total glucose uptake (M value) was calculated on the basis of the amount of glucose infused during steady state period (80-120 min). PI levels were determined by radioimmunossay. Total cholesterol, HDL-Ch, and triglycerides levels were determined by using enzymatic method, and LDL-Ch was calculated using the formula of Friedewald. Apolipoproteins ApoAI, ApoAII, Lp(a), ApoB and ApoE were determined by using nephelometry method.

Results: We found that total glucose uptake was significantly lower in group A vs group B (M value; A: 7.51 +/- 0.57; B: 8.53 +/- 0.45 mg/min/kg, p<0.01). In addition, basal PI levels were higher in group A vs group B (A: 11.37 +/- 1.35; B: 7.39 +/- 0.70 mU/l, p<0.01). Moreover, the levels of total Ch and LDL-Ch were significantly higher in group A vs group B (total-Ch: A: 6.34 +/- 0.26; B: 5.38 +/- 0.35; LDL-Ch: A: 4.28 +/- 0.33; B: 3.19 +/- 0.22 mmol/l, p<0.01). The HDL-Ch levels were significantly lower in group A vs B (A: 1.34 +/- 0.11; B: 1.54 +/- 0.14 mmol/l, p<0.01). Simultaneously, the levels of ApoAI were significantly lower in group A vs B (A: 1.69 +/- 0.12; B: 2.06 +/- 0.43 g/l, p<0.01). The levels of triglycerides and other apolipoproteins, ApoAI, ApoB, Lp(a) and ApoE, did not differ significantly between the groups.

Conclusions: Our results have demonstrated that the presence of MCI in patients was associated with decreased IS and increases in peripheral insulin levels. Moreover, our results has been suggested overlap in pathogenic influence of factors like insulin resistance and abnormal cholesterol metabolism, especially the increases in LDL-Ch, decreases in HDL-Ch and ApoAI levels in cognitive decline, which emphasise that appropriate changes to diets and lifestyles will likely reduce MCI risk, and also improve the prognosis for people already suffering from conditions like insulin resistance and dyslipidemia.