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32PS Triglyceridy, polymorfizmy a riziko akutního koronárního syndromu v české populaci

Todorovová V[1], Hubáček JA[2], Dlouhá L[2], Šatný M[1], Adámková V[2], Piťha J[2], Češka R[1], Vrablík M[1]
[1]Centrum preventivní kardiologie, III. interní klinika – endokrinologie a metabolismu 1. LF UK a VFN v Praze
[2]Centrum experimentální medicíny IKEM, Praha

Úvod: Zvýšená hladina plazmatických triglyceridů (TG) je považována za rizikový faktor rozvoje kardiovaskulárních onemocnění, včetně akutního koronárního syndromu (AKS). Finální hladiny TG jsou do značné míry ovlivněny genetickými faktory. V české populaci jsou mezi nejvýznamnějšími genetickými faktory ovlivňujícími hladiny TG polymorfizmy v genech pro APOA5, GCKR, MAP3K1, CTF1, CYP26A1, LRP1, CILP2, LIPC, APOE, GALNT2 a LPL. **Metodika**: V naší studii jsme analyzovali varianty ve výše zmíněných genech u 929 pacientů s AKS a u 936 zdravých kontrol (studie post-MONICA). Do studie byli zahrnuti pouze dospělí muži ve věku do 65 let. **Výsledky**: Hladiny plazmatických triglyceridů se významně nelišily mezi pacienty a kontrolami (1,96 ± 1,30 mmol/l vs 2,06 ± 1,47 mmol/l). Nositelé alely GG (*rs2068888*, *CYP26A1*) se častěji (P < 0,05; OR; 95% CI – 1,24; 1,01–1,54) vyskytovali mezi pacienty. Rozdíly ve frekvencích ostatních variant nebyly statisticky významné, nicméně, s výjimkou GCKR, LRP1, GALNT2 a LPL, byly na základě vyšší hodnoty OR (> 1,15) využity pro výpočet rizikového genetického skóre. Jedinci se skóre ≥ 2 se vyskytovali častěji mezi pacienty s AKS než mezi kontrolami (47 % vs 40 %, P = 0,002; OR; 95% CI – 1.34; 1,11–1,60). **Závěr**: Genetické skóre složené ze 7 vybraných variant spojovaných s plazmatickými hladinami triglyceridů je signifikantním prediktorem AKS u českých mužů.

33PS Endoglin blockage is essential in hypercholesterolemia and hyperglycemia induced endothelial dysfunction in HAECs

Tripská K[1], Igreja e Sá IC[1], Vicen M[1], Havelek R[2], Eissazadeh S[1], Vitverová B[1], Nachtigal P[1]
[1]Department of Biological and Medical Sciences, Faculty of Pharmacy in Hradec Králové, Charles University, Czech Republic
[2]Department of Medical Biochemistry, Faculty of Medicine in Hradec Králové, Charles University, Czech Republic

Introduction: It has been demonstrated that exposure of endothelial cells to hypercholesterolemia and hyperglycemia leads to increased levels of endoglin and cell adhesion molecules, as well as increased adhesion and transmigration of monocytes through endothelial monolayer. Carotuximab (TRC105), a monoclonal antibody that binds to endoglin, was originally developed for the use in oncology. However, there are no studies available to elucidate potential role of carotuximab treatment on Eng expression, signaling and function with respect to endothelial dysfunction development and/or prevention. **Aim**: Therefore, in this study, we hypothesized that carotuximab treatment prevents 7-ketocholesterol and hyperglycemia induced development of endothelial dysfunction by direct effect on Eng expression, signaling and function. **Material and Methods**: Human aortic endothelial cells (HAECs), passage 5, were cultured in EGM-2 media with appropriate supplements and 10% FBS until reaching 80% confluence. In hypercholesterolemia studies, cells were treated with carotuximab (300 µg/mL) for 1 hour, followed by addition of 7-ketocholesterol (10 µg/mL) for another 12 h. In hyperglycemia studies, cells were exposed to high glucose (45 mM) for 60 hours, followed by addition of carotuximab for another 12 hours and cells treated with 5 mM glucose and 40 mM mannitol served as osmotic control. Gene expression was measured by qRT-PCR. Protein levels, adhesion and transmigration of monocytes were assessed by flow cytometry. **Results**: Carotuximab pretreatment reduced endoglin protein expression and signaling in both hypercholesterolemia and hyperglycemia induced endothelial dysfunction. Despite increased expression of cell adhesion molecules carotuximab blockage of endoglin prevented increase of adhesion and transmigration of monocytes through endothelial monolayer in both hypercholesterolemia and hyperglycemia induced endothelial dysfunction. **Conclusion**: These results suggest that carotuximab-mediated endoglin blockage is essential in hypercholesterolemia and hyperglycemia induced endothelial dysfunction in HAECs and therefore that endoglin might be an interesting therapeutical target in diseases characterized by elevated cholesterol and glucose.