18-FDG-PET, MES MONITORING, CIRCULATING AND HISTOLOGICAL MARKERS OF INFLAMMATION IN CAROTID STENOSIS.

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Background and Goal of study: Carotid plaque inflammation is thought to be a crucial event for plaque vulnerability and increased risk of ischemic stroke. 2-deoxy-2-[18F] fluoro-Dglucose positron emission tomography (18F-FDG-PET) has been used to evaluate atherosclerotic plaque metabolic activity, and through its uptake by macrophages is believed to have the potential to identify vulnerable plaques.

The main aim of this study is to assess the correlation between



18F- FDG uptake on PET scan of carotid plaques in symptomatic and asymptomatic patients, with other vulnerability markers, such as echogenicity of plaque on ultrasound, microembolic signals (MES), histological assessments of plaque inflammation and peripheral blood markers of inflammation.

Materials and Methods: A total of 44 consecutive patients with carotid stenosis, 17 symptomatic and 27 asymptomatic, underwent Colour Duplex ultrasound, transcranial Doppler for MES monitoring, 18F-FDG-PET (Fig. 1 A-B) and blood tests. Patients were stratified into two groups on the basis of presence or absence of previous stroke. Plaques were defined symptomatic when associated with ipsilateral cerebral ischemic symptoms within 15 days prior to inclusion. In symptomatic patients PET evaluation was made considering ipsylateral and contralateral sides. Plaques were assessed histologically following endarterectomy (Fig. 1 C-D). The level of agreement between 18F-FDG uptake (TLG: Total Lesion Glicolisys), and target-to-background ratio, symptoms, blood and histological

Fig. 1: Device for Transcranial Doppler MES
monitoring (A), 18-FDG-PET (B),
endoarterectomy (C), histological assessment post
TEA (D).



evidence of inflammation has been assessed.

Results and Discussion: Analysis of variance with the oneway anova was performed. Symptomatic patients have a higher ipsilateral plaque metabolic rate than asymptomatic patients (P < 0,05) (Fig. 2 A); they have a LCR (lipid core ratio) > 40%, a more presence of erosion, a maior presence of plasmacell and a maior presence of lipids on histological examination, higher than in symptomatic patients (p < 0,05). There is, also, a direct correlation between PCR and ac. uric, on blood, and symptomaticy (p> 0,05). No statistically significant correlation was found with the presence of MES between symptomatic and asymptomatic patients.

Fig.2: Symptomatic patients have a higher ipsilateral plaque metabolic rate than asymptomatic patients (P < 0,05) (A); symptomatic patiente have a higher presence of lipids than asymptomatic and a less presence of calcium on histological assessmente (B1-B2); there is, also, a direct correlation between PCR and ac. uric, on blood, and symptomaticy (p > 0,05)(C1-C2).

Conclusion: Our data shows that 18F-FDG uptake on PET is higher in patients with symptomatic carotid stenosis. Novelty of our study is in the approach to assess plaque inflammation, faced by both ultrasound and hemodynamic point of view (MES), from a diagnostic point of view (18F-FDG-PET) and, finally, from bio-histochemical research of inflammatory blood markers and plaque. Other studies, with a larger number of patients, are therefore needed, to confirm the data.



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