CIGARETTE SMOKE AND ENDOTHELIAL DYSFUNCTION: NGAL A NEW BIOMARKER

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Objectives:

NGAL (neutrophil gelatinase-associated lipocalin) is a small protein belonging to the superfamily of lipocalins, whose gene is under transcriptional regulation by several factors including the NF-kB, involved in the response and in the cellular defense to different insults such as ischemia and oxidative stress. In such conditions, the vascular wall in all its components is able to overexpress NGAL. Cigarette smoking is an important cardiovascular risk factor able to induce acute vascular changes (after a single exposure) and chronic (in smokers.) The aim of this study is to demonstrate, through the effects of cigarette smoke, such as NGAL may represent a novel biomarker of endothelial dysfunction implicated in the physiopatology of different diseases, including hypertension, heart failure, kidney failure.

Methods:

The study was conducted on 20 subjects: 10 healthy smokers (HS) and 10 smokers with hypertension and CKD grade II (CKD). After a 30-min rest were controlled blood pressure (BP) and heart rate (HR) and performed sampling baseline for NGAL assay (T0) and then were made to smoke two cigarettes without filter (with a nicotine content of 0.8 mg/sig). Were then made levies NGAL dosages and measured BP and HR after smoking (T1), 10' (T2), 30' (T3) and 60 '(T4).

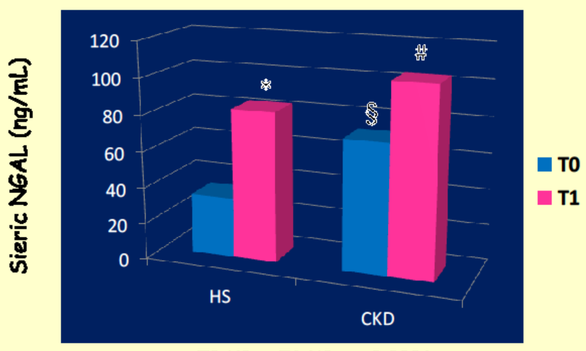

Fig 1. Increased serum levels of NGAL immediately after smoking in healthy subjects (HS) and in patients with CKD.

	HS		CKD	
	Basal	*After smoking*	*Basal*	*After smoking*
SBP (mmHg)	110 ± 10	135 ± 15	130 ± 10	140 ± 10
DBP (mmHg)	60 ± 5	80 ± 10	80 ± 8	90 ± 10
HR (b/m')	70 ± 3	80 ± 4	75 ± 5	90 ±4

Tab 1. Increase in systolic blood pressure (SBP) and diastolic blood pressure (DBP) and heart rate (HR) immediately after smoking in healthy subjects (HS) and in patients with CKD.

Results:

The results showed in the HS group a statistically significant increase of NGAL in T1 compared to T0 ($p < 0.0001$). The same trend was also seen in the group CKD highlighting how NGAL levels were still higher than in group HS (CKD T0 vs HS T0 <0.0001). We also found a statistically significant increase in BP and HR in T1 compared to T0 in both the HS and CKD. [Fig.1, Tab.1]

Conclusions:

In our patients, cigarette smoking, causing endothelial dysfunction, determines a widespread vascular stress, responsible for the increase of the values of NGAL, BP and HR.The different behavior in patients with CKD indicate the presence of a state of chronic dysfunction present, reported alongside the persistent high level of NGAL compared to healthy subjects. The highest levels of NGAL found in the CKD group indicate an exacerbation of chronic endothelial dysfunction after stimulation of smoke, but not completely resolved as in the healthy patient.

References:

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