

NT- PRO BNP, IMPORTANT MARKER FOR EVOLUTION. MONITORING ACUTE CORONARY SYNDROMES WITH NO ST INCREASE

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Abstract: Brain natriuretic peptide (BNP), a neurohormone peptide synthesized in ventricular myocardium and effused into circulation in response to ventricular dilatation and pressure overload, leading to an increase in natriuresis, vasodilatation, inhibition of angiotensin-aldosterone axis and sympathetic activity, becomes elevated in heart failure. It is possible to differentiate it by dosing inspiratory dyspnea due to cardiac infarction normal ejection fraction (EF) without left ventricular (LV) dysfunction. BNP superior to a prohormone ANP is then cleaved at the N-terminus to produce the BNP peptide and N-terminal proBNP (NT-proBNP). This hormone has been incriminated as present in the acute coronary syndromes with ST which increased in the first 24 hours after myocardial infarction (MI).

Cuvinte cheie: sindrom coronarian acut, brain natriuretic peptid, infarct miocardic

Rezumat: Peptidul natriuretic de tip P (BNP), un neurohormon peptidic sintetizat în miocardul ventricular și vărsat în circulație ca răspuns la dilatarea ventriculară și la supraîncărcarea de presiune, având ca efect o creștere a natriurezei, vasodilatație, inhibiția axului angiotensină-aldosteron și a activității simpatice, având valori crescute în insuficiența cardiacă. Este posibil prin dozarea sa diferențierea dispneei inspiratorii de cauză cardiacă față de cauză extracardiacă în infarctul miocardic cu fracție de ejeție (FE) normală și fără disfuncție de ventricul stâng (VS). BNP superior față de Peptid natriuretic atrial (ANP) este ca un prohormon, ce este apoi clivat la capătul N terminal pentru a produce BNP și peptidul N terminal proBNP (NT-proBNP). Acest hormon a fost incriminat ca prezent în sindroamele coronariene acute cu supradenivelare de S-T, unde a crescut în primele 24 ore după infarct.

INTRODUCTION

It is known that for more than 20 years, Ross Russell (1999) has been defining the atherosclerotic process as an inflammatory disease. This process occurs in the vascular endothelium, with a final response in the transformation of the stable atherosclerotic plaque into an unstable plaque (cracked, with parts broken), event that is perceived as occurring under the influence of the well-known risk factors, the occurrence of an endothelial dysfunction that parallels nitric oxide (NO) decrease in cells and at the same time increase in the oxygen free radicals, increase in the concentration of oxidised LDL which are perceived as precursors to trigger the inflammatory cascade in vascular endothelium, this argues for the increase in the clinical inflammatory markers such as: fibrinogen, C-reactive protein (I-5), as low levels of serum amyloid A, interleukin 6, interleukin 18, alpha-TNF (tumour necrosis factor), pregnancy-associated plasma protein (PAPP-A), leukocyte adhesion molecules ICAM-1, VCAM, selectins, etc., facts supported by epidemiological data, by the increase in these factors during different stages of infection.

In the process of atherogenesis, there have been studied other factors such as the von Willebrand factor (FWV) which activates platelets, as well as the signalling complex identified CD40/CD40α identified at the level of the lymphocytes α, β and platelets activated beside with the hemodynamic stress markers. Brain natriuretic peptide (BNP), neurohormone peptide synthesized in the ventricular

myocardium and effused into circulation in response to ventricular dilatation and pressure overload, leading to an increase in natriuresis, vasodilatation, inhibition of angiotensin-aldosterone axis and sympathetic activity, have increased in heart failure. It is possible to differentiate it by dosing inspiratory dyspnea due to cardiac infarction normal EF without LV dysfunction. BNP is superior to a prohormone ANP, which is then cleaved at the N-terminus to produce the peptide BNP and N-terminal proBNP (NT-proBNP). This hormone has been incriminated as present in the acute coronary syndromes with ST which increased in the first 24 hours after MI, being able to be increased to 5-6 days after MI (second peak) due to the remodeling process.(6) In recent years, the BNP marker is also used to define coronary syndromes without ST elevation. Analysis of mortality after myocardial infarction has shown that BNP association with mortality is independent of age, heart or kidney failure, troponin I and CRP, which correlates to long-term mortality also for NT -pro BNT.(6,9) Omland and the collaborators researched on NT- BNP and found that mortality in acute coronary syndromes is independent of the stage of the heart failure or of LV ejection fraction. This hormone also increases in the absence of the ischemic myocardial necrosis reflecting its size (gravity). Values of 348 or 260 pg / ml were found suggestive of acute myocardial infarction (MI) complications (dyspnea, arrhythmias, conduction disturbances, cardiogenic shock, heart break, sudden death).(6,4)

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CLINICAL ASPECTS

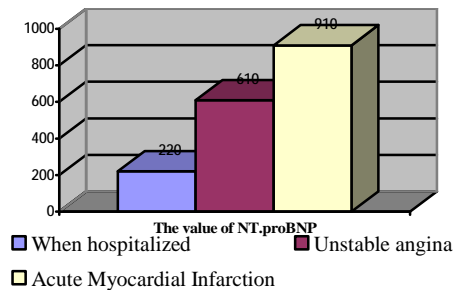
METHODS

We studied a number of 48 patients with acute coronary syndrome with no ST increase in whom the biological chart (troponine, fibrinogen, reactive C protein), except for the sternocardiac crises, presented no convincing increase.

RESULTS

NT- proBNP dosage at hospitalization was increased - 220 pg /ml, compared to the normal value of 7 pg/ml (figure no. 1).

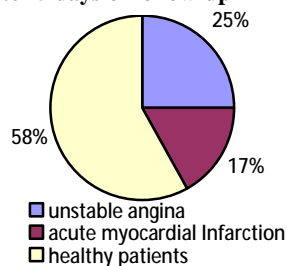
Figure no. 1. The values of NT pro BNP



Dosing was done by ELISA with special kits for this determination.

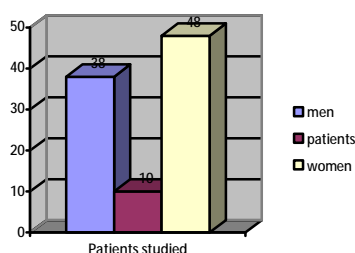
Patients have been closely kept an eye on by clinical examination, by ECG series of five days, dosages of necrosis, enzymes (including troponin was not increased at this stage), the ECG after 5 days (figure no. 1) out of 48 patients a number of 12 patients (25%) have had unstable angina (characteristic pain during irradiation, the response to nitroglycerin, variability ECG and NT-pro BNP value increased to 610pg/ml). A total of 8 patients progressed to MI (myocardial infarction) (17%) changed the nature of pain, ECG changes the appearance of the lesion, necrosis and ischemia, creatinphosphokinase MB, troponin increased also in this phase of ischemia was great, and the value of NT-pro BNP 910pg/ml was reached (figure no. 2).

Figure no. 2. After 5 days of follow-up



Patients in the study were 38 men of 48 and 10 women (figure no. 3).

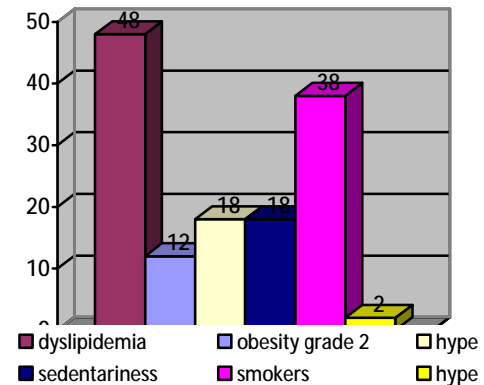
Figure no. 3. Patients studied



Age = 31-40, 41-50, 51-60 over 60 years.

All patients had a history of dyslipidemia present, two of them presented high blood sugar, 12 of them, obesity stage II, 18 of them presented high blood pressure, smoking was present in 38 of them and sedentary life in 18 of them. Clinical course was good, except for 2 patients (4.16%) with MI who died of cardiogenic pulmonary edema (figure no. 4).

Figure no.4. Personal pathological antecedents



CONCLUSIONS

1. From the present inflammatory markers the atherogenic process along with the C reactive protein, 6 and 18 interleukin, the tumoral necrotic factor, NT-BNP proved to be an important predictive factor because its value increased from the angina phase to the intermediary syndrome phase and then MI.
2. It is a marker that does not allow us to make the difference between inspiratory dyspnea present in the coronary syndromes from the extracardiac dyspnea.
3. It helps to monitor the chronic cardiac failure.
4. It is an index of the physical evolution and electrochographic severity of the acute sufferings.

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