



Heart Disease New Hypotesys: Under Endogenous Toxicological Aspect



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Introduction

To suggest new pathogenetic hypothesizing some heart disease we think is interesting to observe some biomedical literature: Can we think some endogenous toxicologic moves in some heart pathologies?

"In past centuries physicians in some cardiologic PATHOLOGY were used to prescribe to the patient a long rest in order to recuperate health status. (There weren't available highly efficient drugs strategies and so a long rest without physical and-or psychological high stress contributes to this process). In example in oriental medicine we can see body balances strategies, and in some psychologic techniques we can see that time is relevant to re-equilibrate some conditions (acute stress).

We can think that the main factors involved are the TIME and LONG REST condition in order to have a right balance the physiologic functions. it was observed complete resolve in some pathologic situations as CHF. So we can think that a metabolic-catabolic unbalances can create this situation and the time make possible to adequately restore.

In example we can see what happen in sudden death heart syndrome in untrained? Why in this condition physical training can reduce this kind of event? We can think a condition of endogen intra-toxicity time related" [1].

"We think in our opinion that in this condition is relevant to deep observe the endogen time related intra- local toxicity situation involved in some Metabolic- catabolic-electrical cell membrane-immune status and other. (KINETICS). In example involved in some heart aritmia, epileptic status conditions, septic shock and other pathologic situation high time related (ischemic coronaric spasm et other).

In embryology, oncology, toxicology field, some heart or brain pathologies time is relevant added to local micro environment and inters-cellular communication (acute -crisis). We can consider an endogen intra- local toxicology aspect time related to deep verify some pathologic process under a new light [1].

Katritsis et al. [2] write that "CAD is the predominant causes of SCD in older athletes. Vigorous exertion can trigger cardiac arrest or SCD sudden cardiac death, especially in the untrained persons, but habitual vigorous physical exercise diminishes the risk of sudden death during the vigorous exertion.

According the recent paper "Brain and Transmission Signal Modulation "2017 "As observed in other scientific or research bio-medical discipline controlling some non-physiological cellular activity can results in reducing of abnormal tissue-organ activation [3]". "There is a circadian variation in SCD sudden cardiac death. The peak incidence of SCD occurs between 6 am and noon (and is blunted by beta-blockers drugs), with a smaller peak occurring in the late afternoon for out-of-hospital VF arrests. The incidence in SCD is highest on Mondays.

In the <35 years, the most common cause of sudden cardiac death is arrhythmia, mostly in an apparently normal heart situation. The most common causes of Sudden cardiac death are congenital abnormalities in those aged 0-13 years, primary arrhythmia in the 14-24-year age group, and CAD in those >25 years. In 5-20% of cases no significant cardiac abnormality is found at autopsy procedure [4].

In a recent Danish registry report on individuals aged <50 years, sudden death was caused by non-cardiac pathology, such as pulmonary embolism, meningitis and cerebrovascular bleeding, in 28% of cases [5]".

According to Hung et al. [4] “the Coronary Artery Spasm (CAS), a pathology with an intense vasoconstriction of coronary arteries (that causes total or subtotal vessel occlusion), plays an important role in myocardial ischemic syndromes including stable and unstable angina, acute myocardial infarction, and SCD sudden cardiac death.

Coronary angiography and provocative tests usually are required to establish a definitive diagnosis. But the mechanisms underlying the development of CAS coronary artery spasm are still poorly understood, CAS appears to be a multifactorial pathology but is not associated with the traditional risk factors for coronary artery disease.

The diagnosis has important therapeutic implications, as calcium antagonists, not β -blockers, are the cornerstone of medical therapy. The prognosis is generally benign; however, recurrent episodes of angina are frequently observed [4] and “Precipitating factors may contribute to the onset of coronary artery spasm CAS and act in the same patient to cause angina in different Conditions and situations. CAS can be precipitated by physical and/or mental stress, magnesium deficiency status, alcohol consumption, the cold pressure test, hyperventilation, the Valsalva physicians maneuver, remnant lipoproteins, and the administration of pharmacological agents such as cocaine or drugs, sympathomimetic agents (epinephrine, norepinephrine), beta-blocking agents parasympathomimetic agents (methacholine, pilocarpine), and ergot alkaloids particularly in the morning when spontaneous CAS is most likely to occur. Activated platelets may trigger CAS by releasing vasoconstrictor substances, including thromboxane and serotonin, both of which are found to be associated with CAS.”

Discussion and Conclusion

Can we think some endogenous toxicologic moves in some heart pathologies? Metabolic-catabolic Kinetics is relevant factor in balancing some cellular-tissue responses? Some endogenous gradients time related can be responsible in some organ failure? In toxicology field usually are high considered the external environmental factors as iatrogenic substantia but we think we

must observe under toxicological methods also the endogenous intra-extra cellular local microenvironment (in para physiologic-pathologic situations).

In some cardio vascular pathology, the time is relevant added to endogenous local microenvironment and inters cellular communication status. We must consider an endogenous intra-local toxicology aspect time related to better verify some pathologic process under a new light.

In some time related cardiovascular local metabolic-catabolic-toxic status we can observe some cellular effect resulting in global organ failure. The time involved and kinetics aspect in resolve some temporary metabolic- catabolic gradients or the velocity involved in this process can be fundamental. The same effect related to too much rapid evolution or too slow reduction(kinetics) in balancing equilibrates some physiologic systems.

(In example the reduced effect showed in sports trained in SCD vs not trained, activate platelets in trigger coronary artery spasms or other relevant examples). Need us to introduce more toxicological methods in some pathologies to BETTER clear some relevant aspect in etiology, diagnosis and therapy?

Clarifications

This paper was not writing for any diagnostic or therapeutic intent, only to produce new research hypothesis..

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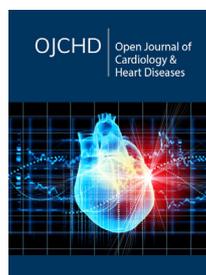
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