Infarct Combat Project

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Effects of the Cardiotonic + Coronary Dilator in Unstable Angina

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Summary

Disclosure of the therapeutic conduct of unstable angina, by the association of cardiotonic + coronary dilator, recommended through the Myogenic Theory for correction of Regional Myocardial Insufficiency, presented as the pathophysiological mechanism triggering this alarming clinical syndrome usually characterizing the pre-infarction.

Introduction

The ideal therapeutic should be based on knowledge of the true cause of the disease and of the pathophysiological mechanism of the real clinical picture, at each stage, in order to achieve complete success.

However, despite this perfect aphorism that should govern medical practice, it is not always fully respected and what is observed very often is the use of therapeutic methods based on logical conjectures and apparent findings only circumstantial and secondary, taken as true; but that can be interpreted in other ways and with results incomparably superior, as has happened in our experience with the use of the cardiotonic in the treatment of unstable angina, since 1972 (1-9).

In our study of coronary-myocardial disease presented with evolutionary and successive stages, starting from the stage of stable myocardial and symptomatic: stable angina or silent coronary disease in front of exertion (1st stage), will result, over time, in myocardial and symptomatic instability: unstable angina (UA) or resting UA (2nd stage), which precedes the myocardial infarction with and without Q wave (3rd stage), the latter being considered since some time ago, as a process still evolving for the myocardial infarction with Q-wave, seeming that, this very frequent hapenning, perfectly fits with the sense of the Myogenic Theory.

We register our disagreement with the pathophysiological precepts of the Thrombogenic Theory, in relation to clinical conditions that fall under the myocardial and symptomatic instability based in coronary vasospasm and / or coronary thrombosis.

In 1944, when we expected to interrupt the evolutionary unstable angina picture, in crescendo, for the myocardial infarction, with the use of oral anticoagulants and heparin (IV), we recorded successive and frustrating failures. Therefore, recognizing the failure of these agents we stopped its use in 1954 (11). Our impression was confirmed later (1969), with the abandonment of these drugs by the orthodox cardiology.

With the advent of coronary angiography and ventriculography and through the notable subsidies provided about the commitment of coronary/dependent segments, we were led to the development of the myogenic theory of myocardial infarction (1972), advocating the pathophysiology of unstable angina, in crescendo, as a result of primary Regional Myocardial Insufficiency and a secondary Regional Myocardial Ischemia (Table 1), spontaneously reversible, justifying the need for the cardiotonic use as the specific drug, because it is a structurally compromised myocardial region and faltering in their contractility.

Table 1

Ischemic Coronary-Cardiomyopathy
Unstable Angina Pathophysiology

1 – Regional Myocardial Insufficiency, Primary
(Regional circulatory stagnation, platelet aggregates.
Possible vasospasm in situ or remotely. Increased volumes and residual systolic and diastolic pressures and in pulmonary capillary pressure. Decreased ejection fraction and ejection of left ventricular phase. Increased parietal tension and stroke volume)

2 – Regional Myocardial Ischemia, Secondary

3 – Spontaneous Reversibility: Unstable Angina Characteristic

Casuistry and Method

The casuistry of unstable angina, exclusively under medical treatment, advocated by the Myogenic Theory consisting of 199 pts, with emphasis: Age, Sex, Personal History and distribution of cases, according to the interval marking the beginning of unstable angina until the admission of the patient into the Coronary Care Unit (Table 2).

Table 2

Unstable Angina
Important Casuistry Information:
199 Patients

Age:	Male	Mean: 56y (27-86y):	150 pts
	Fem.	Mean: 59y (32-78y):	49 pts

Coronary History

Previous Infarction: 43 pts (21.6%)
Stable Angina: 49 pts (24.6%)
Assymptomatic: 107 pts (53.7%)

Interval: Start of Unstable Angina/Hospitalization

First 24 hours:	116 pts (58.2%)
Inside 1 month:	77 pts (38.6%)
Inside 2 months:	3 pts (1.5%)
Inside 3 months:	1 pt (0.5%)
Inside 4 months:	1 pt (0.5%)
Inside 9 months:	1 pt (0.5%)

Obs.: 24 patients were treated in their own homes

The new therapeutic routine (Table 3), is characterized by the association of the cardiotonic plus coronary dilator by intravenous route (IV), as therapeutic attack during 6 days, followed by maintenance treatment with cardiotonic plus coronary dilator by oral route(OR). At this point the patients become recognized as cases of restored myocardial and symptomatic stability and then continuously maintained by this therapeutic way.

The intravenous (IV) strophanthin-K or G was used in 150 pts, Digitalis (IV) in 30 pts and exceptionally, by oral route, the methyldigoxin in 1 patient and Proscillaridin-A in 18 pts.

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Quadro 3

Therapeutic Attack in Unstable Angina

Cardiotonics:

Strophantin-K	: 0,25-0,34 mg/day, IV
Strophantin-G	: 0,25-0,50 mg/day, IV
Lanatoside-C	: 0,40 mg/day, IV
Digoxin	: 0,50 mg/day, IV
Methyldigoxin	: 0,20-0,30 mg/day, OR
Proscillaridin-A	: 1,50-2,0 mg/day, OR

Coronary Dilators

Dipyridamole	: 20 mg/day, IV
Verapamil	: 240 mg/day, OR
Prenylamine	: 180 mg/day, OR
Nifedipine	: 30 mg/day, OR

IV: Intravenous OR: Oral Route

Results

With the use of the combination of the cardiotonic + coronary dilator was recorded immediate suspension of this alarming clinical syndrome, always admitted as a prognostic of next infarction.

In unstable angina, the first time submitted to the cardiotonic, especially to the action of strophanthin-K, we found:

- Perfect tolerance for the drug.
- Immediate disappearance of the spontaneous painful episodes from the first injection of the cardiotonic.
- Interruption of the clinical syndrome in 199 pts, with the registry of only 1 case that progressed to the myocardial infarction in the eighth day of evolution.
- No deaths.
- ECG graphic alterations with rapid disappearance.
- Benign arrhythmic events (20.5%) are easily resolved, transient and without hemodynamic repercussions.
- Meaningless enzymatic alterations in the first 24 hours.

Discussion

Our main purpose in this paper is the dissemination of the Myogenic Theory with new concepts of pathophysiology and therapeutics (1-9) in comparison with the concepts of the Thrombogenic Theory and its therapeutic precepts. After 31 years of frustrating failures with all medications recommended by orthodoxy, including our attempts carried out with oral anticoagulants and heparin (IV) between 1944-1954 (11), we initiated in 1972 a new routine therapy based on the use of

cardiotonic as specific, indicating electively and preferably the strophanthin K or G (IV), until then never used in this clinical syndrome.

We demonstrate that a simple therapy available to physicians and patients, eluding entirely the orthodox principles, seems able to easily halt and readily the unstable angina, in crescendo, and prevent myocardial infarction.

In the treatment of unstable angina, our pioneer clinical experience has demonstrated that the association of cardiotonic plus the coronary dilator is the unmatched clinical solution, producing an immediate cessation of spontaneous anginal episodes that characterize the alarming myocardial and symptomatic instability, especially when manifested, in crescendo -- stage of pre-infarction - providing quick return to myocardial and symptomatic stability.

In our view, these results confirm the pathophysiological mechanism of unstable angina, that assign to the primary Regional Myocardial Insufficiency, episodic and spontaneous, the triggering cause of the secondary Regional Myocardial Ischemia, based on the myocardial region structurally and severely compromised.

The myocardial and symptomatic instability is easily identified by electrocardiography, magnetic resonance imaging (12), echocardiography (13-14), myocardial scintigraphy (15), and hemodynamic (10), especially during spontaneous anginal episodes, long lasting, of unstable angina and even in the intervals in such a crisis, configuring in this way the preceding stage to the myocardial infarction (Table 1).

The current case series serves to prove the principles advocated by the Myogenic Theory, in all cases of unstable angina, in crescendo and also in symptomatic cases with long observation periods, generally regarded as resistant to the therapeutic recommended by the orthodox cardiology.

In our experience, the cardiotonic + coronary dilator has been showed as the only treatment able to reverse the myocardial and symptomatic instability ensuring again the permanence of myocardial and symptomatic stability since not interrupting the use of these drugs.

Within the focus of the Myogenic Theory (1, 4, 5, 8, 9), the therapeutic success reached, fits perfectly to the pathophysiological mechanism advocated (Table 1), whereas, according to the Thrombogenic Theory, which attributes to the coronary Spasm and / or coronary thrombosis as responsible for unstable angina, especially when in the condition in crescendo, we allow ourselves to ask:

How else one would explain and justify the immediate restraining of this alarming clinical syndrome and precursor of acute myocardial infarction after the exclusive administration of cardiotonic + coronary dilator?

Furthermore, with regard to the effects of thrombolytics administered in unstable angina, according to the precepts of Thrombogenic Theory, the results have shown improvement in coronary arterial events and ineffectiveness regarding clinical events (16-19). Consequently, the authors of the referenced studies recommend the use of thrombolytics in unstable angina. As it turns out, it seems that the failure of anticoagulants in UA and now the failure of thrombolytic also in UA are significantly conflicting with the Thrombogenic Theory and reinforce the Myogenic Theory, mainly faced with the results achieved with the cardiotonic.

It seems to us, also, that the incidence of intracoronary thrombi observed in the acute phase of unstable angina in 52-85% of cases, within a few hours and up to 2 weeks, after the active symptoms; contrasting to the chronic phase with an incidence of 0 -12% of cases, giving us the impression that in unstable angina episodes of Regional Myocardial Insufficiency and prolonged myocardial ischemia, favor the platelet hyperaggregation and the formation of unstable

intracoronary thrombi, which are reduced or disappear within 30-90 days after the last attack of unstable angina. Therefore, these manifestations have been considered by us as effects and not the cause of unstable angina.

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