My Acute Myocardial Infarction Strategy

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To patients who taught me
the management strategy of acute
myocardial infarction
and prompted to write my book
and prepare this presentation

Definition

- Acute myocardial infarction is a disease or a clinical syndrome accompanying other diseases, which is represented by acute coronarogenous aseptic inflammation of the part of a heart wall, and clinically correlates with stress reactions of body control systems and is determined by the extent, localization, nature, and stage of structural transformations in the infarction zone, as well as circulation changes, and their consequences.

Etiology

- As follows from the definition of acute myocardial infarction, it develops, when local coronary circulation disturbances as a triggering mechanism cross the time threshold and make the ischemia irreversible. Strictly speaking, acute ischemia that already crossed the threshold is just the formal cause of a further chain of events that results, if you like, in pathology and the clinical picture of the disease .

Mechanisms

- The mechanisms of acute myocardial infarction should naturally be examined at systemic and local levels. They were selected by evolution. They are aimed at providing most favourable of possible courses of the disease. Therefore, its complications can be understood only in terms of philosophy of failure of these mechanisms.
- We should pray that, as often as possible, acute myocardial infarction followed mechanisms established genetically and selected by Nature. And the number of disturbances in them was as small as possible.

Mechanisms: systemic level

- The response of organism systems to acute myocardial infarction is realized through stress and clinically manifests itself as brain mediated sympathetic activation and increased functional activity of a hypothalamo-pituitary an adrenal system with the change of functions of all target organs. For a favourable course and outcome of the disease, all other conditions being equal, an adequate organization of stress (eustress) becomes of primary importance.
- Leukocyte reactions are important for the further development of the process, though they are usually considered to be of secondary importance. These reactions are triggered by the ejection of leukocytes from the depot to the systemic blood flow. Since the depot mainly contains neutrophils, leukocytosis appears as the shift in cell count, neutrophils are activated and migrate to the infarction zone by positive chemotaxis. Infarction zone products getting in the blood flow play the role of attractants for them. The activation of neutrophils appears as hyperenzymemia (transaminase, *etc*.), higher contents of eicosanoids, leukotrienes in particular, protein-carbohydrate complexes, and other biologically active agents.
- Stress is changing as the process develops. Nervous and humoral regulations are regained. Leukocytosis declines, and the leukogram changes. Granulocyte counts decrease, and their functional activity is suppressed, while agranulocyte counts and their activity increase. The structural change of the leukogram is the result of controlling effects of infarction zone products getting in the blood flow. Thus, neutrophil decay products from the infarction zone are repellents for neutrophils and attractants for agranulocytes. An enzyme level in blood falls, while the proteins and protein-carbohydrate complexes content grows.
- These are the systemic manifestations of an inflammation process in the infarction zone. Thus, even such a special case as hyperfibrinogenemia is not a sign of the activation of blood coagulation system but is the an evidence that the situation with myocardial infarction takes a satisfactory turn. Here, it is important to remember that great hopes, placed in anticoagulant therapy, were not justified. These lost hopes cost life to many patients. It was long ago…
- With the termination of an acute myocardial infarction phase, regulation problems disappear and not a trace remains of the stress. I would ask to take notice of the fact that we speak of a natural uncomplicated pathologic process

Mechanisms: local level

- The local level is the heart. The components of a pathologic process at the local level are not only changes in the infarction zone and recovery of heart shape and sizs but also adaptive changes of biomechanics of heart functions.

Local level: *infarction zone*

- Here everything is clear. All that happens is inflammation. Special, aseptic, alterative but still inflammation.
- Let us look at phase processes. The first one is ischemia, reversible myocardial changes. Strictly speaking, it is the precursor of acute myocardial infarction, the state of pre-infarction. It is fully reversible . But if…. Myocardial infarction, of course.
- The transfer to irreversible changes marks the onset of necrosis. Necrosis is a lesion. General pathology teaches that in the location of the lesion there is always inflammation. The lesion is the cause of inflammation. Thus, with the transfer from ischemic changes in the infarction zone to the necrotic ones, the inflammation starts in accordance with its traditional scheme. The necrotized myocardium undergoes destruction, and decay products are removed through the peri-infarction zone. It is specifically destroyed by neutrophil getting by chemotaxis from blood into the infarction zone and produsing cathepsins. Their migration rate is rather high, about 2-4 mm per hour. It is easy to calculate that even the lugest possible infarction is infiltrated by neutrophils in 6 hours at the most. At the same time, fibroblast precursors enter the infarction zone and the recovery process begins. It is impossible to separate necrotic and recovery processes, to look at them as the individual ones. They are synchronized and interconnected not only at the level of the infarction zone itself but also at the systemic blood level. I described this phenomenon in the previous section, illustrating by the mechanisms of leukocytic blood reaction changes.
- An absolutely false view raised to the level of dogma exists: necrotic and recovery processes in acute myocardial infarction are separated, the recovery processes following the necrotic ones. If this were true, all patients would be dead. Most likely, because of heart rupture. If not because of heart rupture, then because of acute vast aneurysm, for sure.
- Necrosis is destruction, tissue gangrene. The tissue loses its functions, not only contractile characteristics but also strength properties. If infarction were necrosis, the heart wall in the infarction zone would have raveled out as a rotten shirt. But it does not happen in the case of an uncomplicated process. The strength properties of the mycrocardium in the infarction zone do not decrease. Moreover, for various reasons they even increase during the acute period. The retained strength of the heart wall in the infarction zone demonstrates that infarction in survived patients is not necrosis but inflammation accompanied with synchronized necrotic and reparative processes.
- The result of a natural inflammation course in the infarction zone is the formation of a full-fledged scar in the place of necrosis . Maturation of a granulation tissue results in its consolidation followed by a decrease in infarction zone sizes. Depending on conditions, they can decrease by 25% or more.
- We should remind those who want to strongly intervene in the infarction zone that the phenomena occurring there (inflammation, compensatory and adaptive responses) are protective reactions originated as the result of evolution. On the whole, it is clear. We may intervene but carefully.

Local level: *peri-infarction zone*

- Systemic mechanisms and the infarction zone are interconnected through the peri-infarction zone, first of all, through its microcirculatory bed. Wastes from the infarction zone are removed through it, and the products necessary for reparative processes enter there the same way.
- The larger the infarction–peri-infarction zone interface, the better the mutual effect of the infarction zone and systemic control. The peri-infarction zone cannot be smaller than that required for uncomplicated healing of the infarction zone].
- Therefore all the efforts to restrict it were doomed to failure.

Local level: *heart shape and size*

- In the case of infarction, a part of a mycrocardium becomes disabled. Disabled forever. And the functions of this part should be compensated. Hypertrophy of an intact myocardium develops. Healing of the infarction zone accompanied with the consolidation of scar leads to a decrease in its size. At the same time, corresponding changes occur in the intact myocardium. The heart shape is remodelled in such a way that its anatomic proportions are restored to favour its pumping functions. In the best case, the traces of infarction are difficult to reveal, even after thorough investigations.

Local level: *heart biomechanics*

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Complications: frequency and immediate cause

- **Frequency**
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- Acute myocardial infarction is a complex and vulnerable process. In the most optimistic estimations, its complications are observed somewhere in a quarter of cases [10, 14, 22]. Of course, if it is diagnosed and treated correctly.
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- **Immediate cause**
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- Lets start with predisposing factors. However many we can name, all of them hit one target. The immediate cause of complications is inadequate stress. In other words, distress. Have little patience, and we would see how distress is realized as complications.

Complications: risk factors

- Acute myocardial infarction can happen in a practically healthy person. Then, there is a good chance that it would take a favourable course. If such serious factors as vast infarction zone size, involvement of a cardiac conduction system, and serious cardiac rhythm disturbances do not appear.
- Vast infarction determines hyperreactive distress with possible torpent phase of true cardiac shock, or it causes an initially hyporeactive course, most likely with a short and therefore missed hyporeactive phase. Small infarction induces systemic wave reactions, and there is danger of its spread.
- But it is not all. Even under most favourable conditions, a natural cataclysm (*e.g.* magnetic storm at the Sun) may happen, and as a result, stress outsteps the limits of adaptive changes.
- Risk of complications is higher in young and elderly persons. The coronary system of young patients is not trained, thus, vigorous stress reactions are highly possible, and it is not so difficult for them to turn into hyperreactive distress. On the contrary, elderly patients have problems with regulatory processes and, thus, with sufficiency of stress.

Standard mechanism of complications

- We would remind that the cause of complications is distress. It is hyperreactive, hyporeactive or intermittent. The mechanism of complications, irrespective of a distress type, is always the same, *viz*. desynchronization of necrotic and reparative processes.
- Desynchronization of necrosis and reparation leads to the loss of heart wall strength in the infarction zone. It is but a step from here to the stretching with cardiac aneurysm or heart ruture outcomes. 'High arterial pressure is a threat of rhexis', we are frightened. The threat, of course, if the heart wall strength in the infarction zone falls to a critical level. The strength of a heart in the case of uncomplicated myocardial infarction as in healthy people is 'a hard nut to crack', even for the highest pressure. Since biomechanical properties of the infarction zone differ from those of the intact myocardium, healing of the infarction zone is complicated not only due to the loss of heart wall strength but also due to the concentration of stresses along its boundary. Their magnitude grows when other unfavourable factors are superimposed. Thus, the areas of marked curvature of inner heart ventricular surfaces (transition of an interventricular septum onto other ventricular walls, transition of ventricular walls onto papillary muscles, *etc*.) are anatomic stress concentrators. If the infarction zone boundary is close to such a concentrator, the loss of strength contributes to its easier rupture .

Standard mechanism of complications

- Hyperreactive distress is excessive activation of all its determining systems. The result is intensive and rapid migration of polynuclears to the infarction zone. Necrosis and destruction are accelerated. Reparative processes lag behind. There is the desynchronization. If the infiltration of polynuclears to the infarction zone is excessive, their decay products retard the entry of reparative agents. In such cases a pathologist finds classical myocardial infarction: nothing but coronarogenic necrosis. Fast destruction of myocardium in the infarction zone has one more consequence, *viz*. high concentration gradients of all products formed in it along the boundary with the peri-infarction zone. Why not a condition for the electronic instability of a heart? As a result, arrhythmia. Whatever you like, with a frequency, deserving respect.
- In the case of hyporeactive distress, everything develops slowly. Systemic reactions are sluggish or are absent at all. Thus, there are problems with the infiltration of polynuclears to the infarction zone. As a result, the necrotic phase is slow, which leads to the delay of reparative processes and their slow development. So, the desynchronization.
- Hyperreactive distress leads to more commonly seen arrhythmia, earlier cardiac aneurysm and heart rupture. Hyporeactive distress with a sluggish disease pattern determines large sizes of aneurysm. As a rule, heart rupture occurs in the thinnest area of aneurysm.

Standard mechanism of complications

- *'Intermittent distress, what happens?'*
- *'Superposition of the above mechanisms by the formula: 'out of the frying-pan into the fire'. Everything becomes much more dangerous.'*
- The result of healing complications accompanied with distress of any type, as it was already mentioned, is aneurysm (acute and chronic, naturally) and heart rupture. They happen earlier upon hyperreactive distress but cover much larger areas in other stress cases. Acute aneurysm is most often acute cardiac insufficiency, and chronic one is chronic cardiac insufficiency. It is difficult to imagine heart rupture without cardiac shock. Arrhythmia upon hyperreactive distress, especially in the hyperreactive phase of intermittent distress, is quite natural.
- During subendocardial myocardial infarction, healing complications with retarded necrotic processes are ready to turn into thromboendocarditis. Just dare say that it is not a patch, protection from the loss of strength in the infarction zone? Thrombus formation disturbances give rise to thromboembolism.
- We can continue this topic. But it is already clear that the share of healing complications is growing like a 'snow ball' in the complications of the disease in general.

Compensatory processes

- Acute myocardial infarction is a disease or a clinical syndrome accompanying other diseases. All that occurs simultaneously with infarction is highly pathologic but not physiologic. But it, in terms of Voino-Yasenetsky's philosophy, corresponds to current requirements and, therefore, is normal. Inflammation, the typical pathologic process underlying the disease, is just that by definition.
- Let us remember transitional atrial flutter accompanying large acute myocardial infarction. Whatever they say, a decrease in heart preload is an extremely precise mechanism. Of course, if this arrhythmia is normosystolic, and what is more, at a rate within upper limits of the norm. Try to restore sinus rhythm in such a patient, and his heart would start to 'choke'. Have you ever met such a situation?
- We have already discussed compensatory functions of naturally formed thromboendocarditis. But if this process is disturbed? Of course, when we actively intervene in blood coagulability with the purpose of treatment. Or let us remember coronaroactive drugs for improving the blood supply in the peri-infarction zone. It appeared that they robbed it. Simply, because Nature did it the way that in the case of infarction, the vessels of the infarction zone are dilated as much as possible. There is nowhere to dilate them any more. We administer coronaroactive drugs and dilate the vessels of an intact myocardium. Blood flows out of the peri-infarction zone. It is an old story.
- The conclusion is simple. Before improving something, one should first think for a while. And no more than that.

Clinical picture and some my publications

- Clinical picture in future presentation
- Some my publications:
- G. G. Avtandilov, N. I. Yabluchansky, K. D. Salbiev, and L. M. Nepomnyashchikh. Quantitative Morphology and Mathematical Simulation of Myocardial Infarction (in Russian), Nauka, Moscow, 1984.
- B. Ya. Kantor, N. I. Yabluchansky, and V. E. Shlyakhover. Nonlinear Cardiobiomechanics of a Left Ventricle (in Russian), Naukova Dumka, Kiev, 1991.
- L. T. Malaya, N. I. Yabluchansky, and M. A. Vlasenko. Uncomplicated and Complicated Forms of Myocardial Infarction Healing (in Russian), Zdorovie, Kiev, 1992.
- N. I. Yabluchansky. Optimum Management of Somatic Patients (General Approach) (in Russian), Osnova, Kharkov, 1995.
- N. I. Yabluchansky. Acute Myocardial Infarction Strategy, Osnova, Kharkov, 2000.