

Perimyocarditis may coexist with acute myocardial infarction

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Abstract: The relation of acute myocardial infarction (AMI) with ST elevation (STEMI) and perimyocarditis has been viewed simply and exclusively as differential diagnostic one. The aim of the paper is to show arguments for the case that perimyocarditis may coexist with AMI: The same infective / inflammatory cause of perimyocarditis may affect coronary artery (“coronaritis”), leading to AMI. In patients with perimyocarditis tachycardia, hypotension, acute heart failure, etc. may occur, which are well-known pathophysiologic mechanisms capable of leading to type two MI. Perimyocarditis can lead to left ventricle (and atrium in atrial fibrillation) thrombus formation. Such thrombus can embolize in coronary artery, producing AMI. Coronary artery spasm can also occur in a patient with perimyocarditis, due to stress, smoking, cocaine, methamphetamine, etc. and result in AMI (if lasting enough). Conclusion: Acute perimyocarditis is an important differential diagnosis of STEMI, because inappropriate administration of thrombolytic (fibrinolytic) may lead to fatal outcome. There is no analysis of the possibility of concomitant occurrence of acute perimyocarditis and AMI in the available literature. In this paper arguments are presented for the case that perimyocarditis might coexist with AMI. Diagnostic workup and therapeutic approach for this situation have been studied.

Key words: Acute perimyocarditis, Myocarditis, Pericarditis, Acute myocardial infarction, Diagnosis, Therapy.

1 Introduction

The problem of *differential diagnosis* between acute myocardial infarction (AMI) and perimyocarditis has been commonly encountered in practice [1]. Although both AMI with ST elevation (STEMI) patients and perimyocarditis patients have chest pain and ST elevation on ECG, these diseases are the opposite - as far as *thrombolytic therapy* is concerned. Namely, in obvious perimyocarditis with ST elevation thrombolysis is “vitium artis” (serious doctor’s error) [2]. On the contrary, in the world today, millions of patients with STEMI receive thrombolytic therapy annually. Thus, it is very important to differentiate between STEMI (thrombolysis may be life-saving) and “pure” perimyocarditis – without coronary artery thrombosis- (thrombolysis is contraindicated and may be even fatal.

2 Problem Formulation

The relation of STEMI (type of AMI) and perimyocarditis has been viewed simply and exclusively as differential diagnostic one. The aim of the paper is to show arguments for the case that perimyocarditis may coexist with AMI.

3 Problem solution

Generally, patients with perimyocarditis are younger, have a different type of chest pain, have infective syndrome, have more diffuse both ECG and echocardiographic abnormalities, etc. On the other hand, there is a certain group of patients with perimyocarditis in whom ST elevation corresponds to a single coronary artery territory, resembling STEMI even more. Such patients may have risk factors for AMI, indeed, making the differential diagnosis even more troublesome. Doctors responsible for the above mentioned patients may easily make a mistake if infective syndrome (which suggests perimyocarditis) is not apparent. Rarely,

perimyocarditis can be obvious, but it may be very difficult to confirm or reject the possibility of **concomitant STEMI**. Myocarditis may coexist with AMI [3].

Some of the arguments follow:

1. The same infective / inflammatory cause of perimyocarditis may affect coronary artery (“coronaritis”), leading to AMI. It has been well known (both from literature and from practice) that **inflammation** represents a pathophysiologic factor for AMI.

2. In patients with perimyocarditis tachycardia, hypotension, acute heart failure, etc. may occur, which are well-known pathophysiologic mechanisms capable of leading to **type two MI** (according to Universal MI definition), especially if patients have coronary artery stenosis.

3. Perimyocarditis can lead to left ventricle (and atrium in atrial fibrillation) thrombus formation. Such **thrombus** can **embolize** in coronary artery (this mechanism has been known for over a century and a half – since Virchow (Virchow R. Ueber capilare Embolie. Virchows Arch Path Anat 1856;9: 307-308.)

4. Coronary artery **spasm** also can occur in a patient with perimyocarditis, due to stress, smoking, cocaine, methamphetamine, etc. - as in previously healthy individuals and probably even easier.

Therefore, the question remains whether it is “only a perimyocarditis” or if there is also a coronary artery thrombosis present. If available, **echocardiography** can help, but not always: sometimes even left ventricular (LV) wall motions abnormalities are not diffuse (which is suggestive of perimyocarditis), but fit to LV regions supplied by one coronary artery. Chest pain at rest occurs in 26% of patients with perimyocarditis, with segmental wall motion abnormalities [4].

The dilemma is troublesome especially in young perimyocarditis patients (with lower risk for atherothrombosis), but who smoke cigarettes or use cocaine /methamphetamine (which substantially increase the risk for AMI).

Which drugs to use for obvious perimyocarditis and suspected **concomitant STEMI**? It seems logical to administer **aspirin** as soon as possible, preferably intravenously, because aspirin is useful for AMI as well as for perimyocarditis. **Morphine** is also expected to help, whatever causes the strong pain. **Nitroglycerine** (NTG) is useful for AMI, but not for perimyocarditis (in absence of

heart failure). As many AMI patients respond poorly to NTG, this response can not be used as indicative of AMI *ex iuvantibus*. Nevertheless, it is sensible to titrate up the NTG while tolerated [with frequent blood pressure (BP) control], because it may help to solve coronary artery spasm and it decreases preload. Unfortunately, prolonged vasospasm may lead to thrombosis.

If dilemma continues and there is no coronary angiography available, one may proceed to **anticoagulant** therapy while waiting laboratory results and transportation to the institution with the catheter laboratory (cath lab). Fondaparinux may be suitable, because it is good in STEMI and – if it turns to be perimyocarditis- Fondaparinux is expected to do less harm (less bleeding), in terms of inducing/ worsening pericardial effusion.

To solve: A) differential diagnosis dilemma “AMI versus perimyocarditis”, as well as B) “are there both perimyocarditis and STEMI” **troponin** is not helpful, because it is raised in cardiomyocyte necrosis of various etiologies, including both AMI and myocarditis [3]. **D-dimer** (DD) can also be obtained bedside and relatively quickly, but DD can be elevated in infections and many other conditions, unrelated to myocarditis and AMI. Even if there is a thrombus, DD is not specific for its coronary artery or any other location, including left ventricle- it may result from diminished LV function in myocarditis. Therefore, DD is not absolutely specific in this situation. Negative **ELISA DD** has been successfully used to exclude venous thromboembolism. In attempt to treat best a patient with perimyocarditis with suspected STEMI, one might hope to exclude the need for thrombolysis in case of normal ELISA DD (if there is time to perform the analysis at all). To complicate the situation, DD is not always increased in AMI, although AMI usually follows thrombosis [5]. Intuitively, doctor may avoid fibrinolytic if ELISA DD is normal, but this decision would not be justified by evidence based medicine. **Multislice computer tomography (MSCT)** can be useful, but it is not universally available and it takes some time to arrange and perform it, with additional risk if MSCT is not located close to coronary care unit.

In spite of the administration of aspirin, morphine, nitroglycerine, iv/sc anticoagulant, oxygen, and control of heart rhythm and rate abnormalities, as well as regulation of BP, a very strong retrosternal pain and (pronounced) ST elevation may both continue.

If there is cath lab, it is easier to solve the dilemma whether there is or is not a thrombotic occlusion of the coronary artery. Nevertheless, catheterization is not harmless, because of the possibility to worsen pericardial effusion with high blood heparin level.

In case there is not a cath lab available, the decision whether to give thrombolytic or not is very stressful, although we may know many rules and tips. If there is thrombotic occlusion of the coronary artery, delaying thrombolysis is harmful because "time is muscle" in AMI. If there is not, thrombolysis is useless /senseless, and fibrinolytics can worsen pericardial effusion up to tamponade, in addition to imminent bleeding risk of thrombolysis.

4 Conclusions:

1. **Differential diagnosis** between perimyocarditis and STEMI can be sometimes very difficult, especially if ST elevation and wall motion abnormalities on echocardiography point to the single coronary artery territory.

2. Another trouble is to confirm /exclude **additional coronary artery thrombosis** (and STEMI) in certain group of patients with perimyocarditis. Diagnostic protocol is needed to exclude concomitant STEMI in some patients with perimyocarditis.

3. Failure to manage such patients properly may put them (or leave them) in a **life-threatening** condition.

4. Series with **coronary angiographic** results of patients with perimyocarditis and possible STEMI should be obtained.

5. In the absence of coronary angiography, it can be very difficult to decide whether to **thrombolyse** or not a patient with perimyocarditis and possible STEMI.

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