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Review Article

Diagnosis, Definition and Classification of Myocarditis. Isn't it time to Start Thinking about Changing?

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Abstract

The paper presents historical changes in the understanding of myocarditis by the medical community. Changes in the priorities and informative value of instrumental methods such as endomyocardial biopsy and gadolinium-enhanced cardiac magnetic resonance imaging in myocarditis are shown.

Changes in the diagnosis and classification of myocarditis are presented. The new staging classification (stages A, B, C and D) according to the American College of Cardiology report (2024) is presented. Changes to the definition and classification of myocarditis are also being proposed for discussion.

Kew Words: myocarditis; definition; classification; endomyocardial biopsy; gadolinium-enhanced cardiac magnetic resonance

Introduction

Myocarditis is an enigmatic condition that continues to elude comprehensive understanding; posing numerous unanswered questions.

The study of this pathology was initiated in the latter half of the seventeenth century; with the seminal work of the renowned French physician Jean-Baptiste Sénac in Traité de la structure du cœur; de son action et de ses maladies (1749) marking a pivotal moment. This seminal work provided the first fundamental description of inflammatory heart disease and the challenges associated with its diagnosis. Subsequently; in 1806; the founder of clinical cardiology; Professor Jean-Nicolas Corvisart; advanced the hypothesis that the root of pathogenic infection lies in the body's inherent variability; and the human organism is inherently vulnerable to succumbing to ailments that pose a threat to its well-being (Karamanou et al.; 2010). And in 1873; J. F. Sobernheim was the first coiner of the term "myocarditis" (Pandey et al.; 2023).

Since that time; there has been a considerable advancement in the field; with the diagnostic process for this disease evolving from a post-mortem examination to a clinical and instrumental lifetime diagnosis.

The present-day diagnosis of the condition is based on the presence of clinical manifestations (e.g. chest pain; heart rhythm and conduction disturbances; symptoms of heart failure (HF)) that can be interpreted as manifestations of myocarditis. Changes in the results of non-invasive and laboratory tests of the "first line" (e.g. electrocardiogram; echocardiogram; inflammatory markers [e.g. the erythrocyte sedimentation rate (ESR); CRP]; levels of highly sensitive cardiac-specific troponins (cTn) I or T; levels of brain natriuretic peptides [BNP]; serum cardiac autoantibodies) may also be indicative of myocarditis (Caforio et al.; 2013).

The primary instrumental methodologies employed for the establishment (confirmation) of a diagnosis of myocarditis encompass magnetic resonance imaging (MRI) with gadolinium and endomyocardial biopsy (EMB).

Endomyocardial biopsy.

The development of this method was a major turning point in the study of myocarditis. It made it possible for the first time to diagnose of myocarditis during life. In the late 20th century; Japanese researchers S. Sakakibara and S. Konno published studies demonstrating the feasibility of using a flexible bioptome and performing endomyocardial sampling (EMB) with forceps (Sakakibara & Konno; 1962).

Further modifications of the bioptome (Stanford Caves-Shulz bioptome) by P.K. Caves et al. (1973) rendered it feasible to perform percutaneous biopsies through the right internal jugular vein with only local anaesthesia. This development subsequently became a standard device for EBM for a period of approximately two decades.

In 1987; the “Dallas criteria” were developed by a group of leading American morphologists (Aretz et al.; 1987). They were the first (and for

a long time the main) method of myocarditis identifying and developed a histopathological classification of the disease (Aretz et al.; 1987).

In 1995; the Working Group of the World Health Organization/International Society and Federation of Cardiology Task Force on the Definition and Classification of Cardiomyopathies among other sources; identified a group of "specific cardiomyopathies"; which included inflammatory cardiomyopathy and myocarditis (Richardson et al.; 1996). The term “inflammatory cardiomyopathy” was defined as myocarditis in combination with cardiac dysfunction. Myocarditis was interpreted as an *inflammatory myocardial disease; and it was diagnosed using established histological; immunological and immunohistochemical criteria* (Richardson et al.; 1996).

The "Dallas criteria" were in use for almost a 10-year period. However; it was subsequently determined that these histological criteria exhibited high specificity but low sensitivity. L.H. Chow et al. (1989) found that only 25% of autopsy specimens showed histologic signs of myocarditis; and only 10% of patients with HF "de novo" met these criteria (Chow et al.; 1989; Baughman; 2006).

In 1997; two committees of experts were convened for the purpose of formulating a consensus on the definition of myocarditis; which was referred to as the "Marburg Consensus" (WHF Classification and Consensus Conference; 1997). *The Committee defined myocarditis as a process characterized by an inflammatory myocardial infiltrate; the extent of which was to be determined by immunohistochemical examination* (WHF Classification and Consensus Conference; 1997).

This Consensus states that the diagnosis of myocarditis should be confirmed when at least 14 infiltrated leukocytes/mm² are detected; predominantly T lymphocytes (CD45RO) or activated T cells (e.g.; CD45RO). This total cell count could also include up to 4 macrophages and 7 cells/mm² or more CD3-positive T lymphocytes (WHF Classification and Consensus Conference; 1997).

It was also recommended that the amount and distribution of fibrosis be taken into account. The classification system employed in this study categorized fibrosis as follows: grade 0; no fibrosis; grade 1; mild fibrosis; grade 2; moderate fibrosis; and grade 3; severe fibrosis. The localization or formation of fibrosis was to be described as endocardial; replacement; or interstitial. The process was considered to be reparative if focal or diffuse leukocytes were localized in fibrotic areas (WHF Classification and Consensus Conference; 1997).

According to the results obtained from the initial biopsy; the following findings were made (WHF Classification and Consensus Conference; 1997):

1. Acute (active) myocarditis: A clear-cut infiltrate (diffuse; focal or confluent) of ≥ 14 leukocytes/mm² (preferably activated T-cells). The amount of the infiltrate should be quantitated by immunohistochemistry. Necrosis or degeneration are compulsory; fibrosis may be absent or present and should be graded.
2. Chronic myocarditis: An infiltrate of ≥ 14 leukocytes/mm² (diffuse; focal or confluent; preferably activated T-cells). Quantification should be made by immunohistochemistry. Necrosis or degeneration are usually not evident; fibrosis may be absent or present and should be graded.
3. No myocarditis: No infiltrating cells or < 14 leukocytes/mm².
4. The results of the subsequent (repeated) biopsy were determined:
5. Ongoing (persistent) myocarditis. Criteria as in 1 or 2 (features of an acute or chronic myocarditis).
6. Resolving (healing) myocarditis. Criteria as in 1 or 2 but the immunological process is sparser than in the first biopsy.
7. Resolved (healed) myocarditis. Corresponds to the “Dallas classification”.

The inflammatory infiltrate should be classified as lymphocytic; eosinophilic; neutrophilic; giant cell; granulomatous; or mixed. The distribution should be classified as focal; confluent; or diffuse; respectively.

The EMB method has evolved quite rapidly and; as a result of the integration of histological; immunohistochemical and molecular analysis; has been established as the "gold standard" for the definitive diagnosis of myocarditis (Thiene et al.; 2013).

Subsequent to the publication of the joint American Heart Association (AHA) and European Society of Cardiology (ESC) guidelines on the role of EMB in cardiovascular disease in 2007; this method has been employed in tertiary care centers worldwide (Cooper et al.; 2007; Cooper; 2024).

In 2013; the European Society of Cardiology Working Group on Myocardial and Pericardial Diseases published a Position Statement; which represented the first attempt to provide specific diagnostic criteria and recommendations for the treatment of myocarditis (Caforio et al.; 2013). The statement summarised current knowledge of the aetiology; pathogenesis; and diagnosis of myocarditis; and attempted to provide standardised definitions for use in future registries and clinical trials on the subject.

The Statement asserted that the EMB should be considered the "gold standard" for accurate diagnosis of myocarditis. The EMB is capable of verifying a diagnosis of myocarditis; in addition to establishing the fundamental cause of the inflammation and identifying the specific inflammation type (e.g.; giant cell; eosinophilic myocarditis; sarcoidosis). This information enables the determination of different treatment methods and the prediction of patient prognosis. Simultaneously; it was emphasized that this method is not a standard procedure. It was further noted that not all patients with suspected myocarditis should undergo EMB (Caforio et al.; 2013).

Over the past 10-15 years; there have been significant conceptual changes in the medical community's understanding of the etiopathogenesis; interpretation; classification; and approaches to the treatment of myocarditis. This paradigm change was the result of a substantial number of clinical and experimental studies; as well as the integration of a diverse array of immunological; molecular biological and non-invasive research methodologies into clinical practice. At the same time; the views on the role of EMB in diagnosing the disease also changed (Ammirati et al; 2020). This was “facilitated” by a significant number of false-negative EMB results due to tissue biopsy errors; especially in moderate and focal inflammatory infiltrates. Thus; A.J. Hauck et al. (1989) showed that usually 4 to 6 biopsy samples are taken during the diagnostic procedure. However; a thorough postmortem analysis of confirmed cases of myocarditis showed that for the correct diagnosis of myocarditis in more than 80% of cases; 17 samples or more are required. It is evident that this number of biopsies cannot be performed in clinical practice; thereby underscoring the deficiency in EMB sensitivity. Moreover; the ESC Task Force (2023) emphasized the necessity of employing experienced teams to perform EMB; and that diagnostic biopsy processing should be conducted exclusively by pathologists with experience working with cardiomyopathies (Arbelo et al.; 2023). Therefore; it was suggested to use (reserve) EMB with quantifying inflammatory cells using immunohistochemistry and the identification of viral genomes only for specific cases. This approach is recommended when the potential benefits of EMB outweigh any potential risks and it results can influence treatment (Arbelo et al.; 2023; Parrillo; 2001; Vidusa et al.; 2022; Dominguez et al.; 2016).

According to some experts; this method with the use immunohistochemical methods (IHC) and polymerase chain reaction

(PCR) can be considered the "gold standard" for final diagnosis in patients with fulminant course (i.e.; suspicion of giant cell or eosinophilic myocarditis; vasculitis or sarcoidosis) or lack of response to empiric treatment (Vidusa et al.; 2022; Dominguez et al.; 2016).

In the 2024 American College of Cardiology Report (ACC Expert Consensus Decision Pathway on Strategies and Criteria for the Diagnosis and Management of Myocarditis); the use of EMB is also recommended in individual cases to confirm specific types of myocarditis; including giant cell; eosinophilic; and sarcoidosis (Drazner et al.; 2024). It is emphasised that the evaluation of EMB samples should be performed using IHC counting of inflammatory cells and the use of PCR and reverse transcription of mRNA to DNA (PCR-RT). IHC and molecular biological analysis of EMB help identify the cause of myocarditis and determine which patients need specific therapy (Drazner et al.; 2024).

This is applicable to patients suffering from myocarditis complicated by; for example; decreased ventricular function; symptomatic heart failure; hemodynamic or electrical instability (Drazner et al.; 2024).

At the same time; the place of various methods for the detection of inflammation in the myocardium in routine clinical practice is still under discussion.

Among other non-invasive imaging techniques; cardiac MRI with contrast has become increasingly important in the diagnosis of myocarditis.

Magnetic resonance imaging (MRI) with gadolinium.

Since the late 1990s; MRI has been extensively used for the diagnosis of myocarditis. The initial publication by M.G. Gagliardi et al. (1991) on T2-weighted MRI results in children with myocarditis was published; and the first controlled clinical trial with contrast enhancement was conducted in 1998 (Friedrich et al.; 1998). Since that time; a significant number of studies have been carried out to demonstrate the diagnostic usefulness of contrast-free and contrast-enhanced MRI in patients with myocarditis (Laissy et al.; 2002; Abdel-Aty et al.; 2005; Gutberlet et al.; 2008; Roditi & Hartnell; 2000; Laissy et al.; 2005; De Cobelli et al.; 2006; Friedrich et al.; 2009).

Contrast-enhanced MRI provides significant insights into the analysis of myocardial morphology and function. This advancement can also be attributed to the replacement of the 2009 Lake Louise criteria with the updated 2018 criteria (Ferreira et al.; 2018; Joudar et al.; 2023).

These updated Lake Louise Criteria (2018) for non-ischemic myocardial inflammation use T1/T2 mapping (in order to obtain signal values of the myocardium itself) and extracellular volume (ECV) (Ferreira et al.; 2018). Myocardial fibrosis/edema may be assessed by T1 mapping (Class I recommendation; Level of Evidence: B); and myocardial edema may be evaluated using T2 mapping or T2-weighted imaging (Class I recommendation; Level of Evidence: B) (Nagai et al.; 2023). According to these Criteria (2018); the diagnosis of an inflammatory process requires the presence of at least one T2-based criterion (global or regional increased myocardial T2 relaxation time or increased signal intensity on T2-weighted images) in addition to one T1-based criterion (increased myocardial T1; LGE or ECV) (Ferreira et al.; 2018).

The use of the updated Lake Louise Criteria (2018) resulted in an enhancement of the method's sensitivity to 87.5% and its specificity to 96.2% (Joudar et al.; 2023). In the ACC Expert Consensus (2024); cardiac MRI using the updated Lake Louise criteria (2018) is classified as a Class I recommendation for stable patients; regardless of myocarditis etiology (Drazner et al.; 2024). MRI provides non-invasive multi-parametric characterisation of myocardial tissue and is the gold standard imaging test for the assessment of cardiac structure and function.

Myocardial changes observed in myocarditis include myocardial edema (intracellular and interstitial); hyperemia; increased vascular permeability or capillary leakage; myocyte injury and necrosis; and the formation of interstitial and/or focal fibrosis and pericardial inflammation or effusion (Drazner et al.; 2024).

This method is especially important for detecting inflammation in the epicardial and middle regions of the myocardium; which is characteristic of acute myocarditis and cannot be diagnosed by EMB (Cooper; 2024). Furthermore; the results of the MRI scan permitted the control of the prognosis (Tschöpe et al.; 2021).

Therefore; it's now believed that cardiac MRI with enhancement should be used not only to diagnose myocarditis in haemodynamically stable patients (class I recommendation; level of evidence A); but also to assess myocardial pathology; risk stratification (class I recommendation; level of evidence B) and evaluation of therapy effectiveness (Nagai et al.; 2023).

Cardiac MRI with enhancement has the highest sensitivity (>85%) and efficacy when performed within the first 1-2 weeks of symptom onset; when edema/inflammation are present (Joudar et al.; 2023; Nagai et al.; 2023; Tschöpe et al.; 2021; Luetkens et al.; 2019; Pan et al.; 2018).

In addition; American experts have recommended a new staging classification and step-by-step algorithm for the diagnosis of acute myocarditis. The proposed staging of myocarditis includes (Drazner et al.; 2024):

- Stage A (at-risk for myocarditis) - patients with risk factors for myocarditis (history of previous viral infection (upper respiratory or gastrointestinal); previous myocarditis or autoimmune disease; family history of cardiomyopathy or sudden death; exposure to known cardiotoxic factors; and further development of any of these three symptoms should raise suspicion of myocarditis).
- Stage B - asymptomatic patients but with signs of myocarditis;
- Stage C - patients with "symptomatic" myocarditis;
- Stage D - patients with progressive myocarditis (with haemodynamic or electrical instability requiring intervention).

For general clinical practice; distinguishing between stages A and B is particularly important. Stage A not only reminds doctors to take preventive measures against this disease; but also encourages them to monitor patients who have recovered from myocarditis.

Stage B (asymptomatic myocarditis); on the other hand; allows diagnosis and treatment in the early stages of the disease; increasing the chances that the myocarditis will be uncomplicated and speeding up the recovery of such patients.

Thus; taking into account the conceptual changes in the view of myocarditis and the role of instrumental diagnostic methods of this disease; some revision of the definition and classification of myocarditis is relevant. We propose to discuss the possibility of using the following definition of myocarditis.

Myocarditis is a group of multifactorial inflammatory diseases of the heart muscle characterized by the presence of inflammation and/or non-ischemic damage to heart muscle cells (degeneration and necrosis of adjacent heart muscle cells).

The term "non-ischemic" cardiomyocyte damage is used because these manifestations are one of the MRI criteria for the diagnosis of acute myocarditis and are very common in infarct-like; giant cell and eosinophilic types of myocarditis.

We also propose the following classification of myocarditis. Myocarditis may be:

- primary - isolated myocardial damage;

- secondary - as one of the manifestations (complications) of general diseases (e.g. borreliosis; systemic connective tissue diseases; chronic viral hepatitis; etc.).

I. According to aetiology:

- infectious (viral; bacterial; fungal; parasitic; protozoan; rickettsial and spirochetal);
- non-infectious (immune-mediated (allergic and toxic-allergic reactions; autoimmune diseases; burn disease and conditions after organ transplantation); associated with cancer treatment with immune checkpoint inhibitors (immune checkpoint inhibitor-related myocarditis) and toxic (exposure to drugs and various cardiotoxic compounds).

II. By the extent of the inflammatory process (according to MRI and/or EMB):

- focal;
- diffuse;

III. The morphological characteristics of the infiltrate (according to the data from the EMB):

- lymphocytic;
- eosinophilic
- Giant cellular;
- granulomatous.

IV. According to the course:

- Risk of myocarditis (stage A according to the American College of Cardiology report) - patients with risk factors for myocarditis (history of previous viral infection (upper respiratory or gastrointestinal); previous myocarditis or autoimmune disease; family history of cardiomyopathy or sudden death; exposure to known cardiotoxic factors; and further development of any of these three symptoms should raise suspicion of myocarditis).
- Acute myocarditis - myocardial inflammation with symptoms of recent onset; usually ≤1 month; elevated high-sensitivity Tn levels and evidence of edema; inflammation and/or damage to the CMV on cardiac MRI or positive cardiac FDG-PET (increased glucose metabolism detected by focal FDG uptake):
  - stage B (according to the American College of Cardiology Report) - asymptomatic patients; but with signs of myocarditis;
  - stage C (according to the American College of Cardiology Report) - patients with "symptomatic" myocarditis.
- Subacute myocarditis - where symptoms have been present for more than 1 month. It is characterized by signs of interstitial edema and mixed inflammation and the development of healing - granulation tissue consisting of proliferating fibroblasts and new vessels; and initiation of deposition of immature collagen in areas of previous previous cardiomyocyte injury (no fibrosis).
- Chronic myocarditis - persistent active myocardial inflammation with a duration of symptoms (>1 month) and the presence of healing manifestations - areas of focal or diffuse interstitial; replacement and/or perivascular myocardial fibrosis.
- Healed (resolved) myocarditis (myocardial fibrosis).

V.According to the severity of the symptoms and the type of disease:

- complicated - myocarditis with one or more of the following signs (LVEF <50% at first echocardiogram; persistent VT; high degree of heart block; low cardiac output syndrome; cardiogenic shock or haemodynamic instability on admission; thromboembolism; etc.).) Complicated course includes fulminant myocarditis (working diagnosis) - acute myocardial inflammation with sudden; distinct onset and rapid progression of HF symptoms with haemodynamic disturbances; including the cardiogenic shock or low cardiac output syndrome.
- uncomplicated - myocarditis characterised by stable patient condition; preserved LVEF and absence of life-threatening rhythm or conduction disturbances) (i.e. patients hospitalised with suspected acute myocarditis):

We believe that such changes in the definition and classification of the "myocarditis" disease group will not only help to improve diagnostic approaches; but also allow for more differentiated treatment of such patients.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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