УДК 616.12-008.318-008.46:616.12-002.7 doi:10.25298/2221-8785-2025-23-6-655-660

THE ROLE OF DIAGNOSTICS OF ARRHYTHMIAS AND CONDUCTION DISTURBANCES, HEART FUALURE IN THE PROGNOSIS OF PATIENTS WITH CARDIAC SARCOIDOSIS

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Cardiac sarcoidosis is a chronic inflammatory disease characterized by non-caseating patchy granulomatous lesions of the heart with suspected immunopathological response to an unidentified antigenic trigger in a genetically susceptible individual. Cardiac sarcoidosis is known to be the second most common cause of death in sarcoidosis patients globally. Sarcoidosis often occurs as a systemic inflammation with multi-organ involvement, although, rarely it can occur in isolated forms.

The above-mentioned particular phenotype is difficult to diagnose as it requires exclusion of a number of other diagnoses and when left untreated can progress to fibrosis while cardiac dysfunction eventually leads to sudden cardiac death, which encourages the researchers and clinicians to modify a new framework of diagnostic modalities and management tactics. Depending on the prevalence and severity of a particular phenotype of the disease, the clinical picture can vary from an asymptomatic form to severe fatal cardiac manifestations such as decompensated heart failure, severe forms of ventricular and atrial malignant arrhythmias, conduction abnormalities and sudden cardiac death.

Therefore, early diagnosis and timely initiation of the treatment tactics including immuno-suppressive medicine remain crucial to combat this pathological condition.

***Keywords**: cardiac sarcoidosis, arrhythmias, conduction disturbances, heart failure, sudden cardiac death, diagnostics, management*

***For citation**: Snezhitskiy VA, Himershi Kawya MGH, Jahas Ahamed MJ. The role of diagnostics of arrhythmias and conduction disturbances, heart failure in prognosis of patients with cardiac sarcoidosis. Journal of the Grodno State Medical University. 2025;23(6):655-660. https://doi.org/10.25298/2221-8785-2025-23-6-655-660*

Goal of the article

This review was done to emphasize on the importance of the early diagnosis and timely management of Cardiac Sarcoidosis (CS) using new diagnostics modalities along with newly developed treatment protocols established to minimize the catastrophic outcomes from the disease progression.

Epidemiology

CS has a significant prevalence in North American, African-Americans and in Europe comparatively to low known cases in Asia and Hispanics. Furthermore, its frequency of occurrence depends on many contributing factors such as age, sex, ethnicity and geographical localization. CS is diagnostically proven to exist in 5% of all sarcoidosis patients [1]. In addition, 3.8 times increased risk in the afro-american groups in comparison with Caucasians and the average age of disease occurrence is 48 years old. Almost 70% of the patients are diagnosed between 25 to 60 years of age [2, 3]. Over the past few years, there has been an increase in number of cases due to increased awareness among clinicians and advanced diagnostic modalities.

Etiological factors

Etiological factors influencing on the development of the inflammatory process, are known to be combination of genetic predisposition, environmental triggers including insecticides, mold, mildew, birds, radiation and certain bacteria (Mycobacteria and Proprioni bacterium), lifestyle and occupational risk such as automobile factory staffs, and blue-collar workers [4].

Pathophysiology

Hallmark of sarcoidosis is the formation of noncaseating granulomas characterized by the presence of epithelioid histiocytes, macrophages, giant cells and lymphocytes, significantly T Helper cells 1 type (Th1) and subsequently Th17 cells too [5, 6, 7, 8]. Although, mostly CS is multifactorial, sometimes the development of inflammation may occur due to overlap between the monogenic cardiomyopathies and myocarditis. Thus, non-caseating granulomatous inflammation is the main pathophysiology of CS hence, it is not pathognomonic.

Histological complexities of CS is almost similar to giant cell myocarditis, which is known as non-necrotizing granulomatous myocarditis with macrophages and multinucleated giant cells surrounded by fibrosis and a lymphocytic infiltrate. Notably, smoking is thought to be a protective factor against sarcoidosis due to, it is inhibiting effect on the macrophagic-lymphocytic association of forming granulomas [9].

Genetic Predisposition of CS

Interestingly, a proposition evolving the genetic susceptibility of CS has been made that reveals, there is a fourfold higher prevalence within a particular ethnic group (northern European and African American descendants) in homozygotic twins and first-degree relatives [10, 11, 12]. CS consists of a complicated genetic formula that includes Human Leukocytic Antigen gene, Annexin A11, NOTCH4 and X-linked inhibitor of apoptosis associated factor 1 gene and DQB10601 and DRB1*0803 alleles specially in cardiac phenotypes of sarcoidosis, while DRB1*0101 and DQB1*0501 known to be the protective alleles against a predominant pulmonary sarcoidosis [13].

Clinical manifestations

According to the clinical picture, the disease can be recognized as following various pathological subtypes' thus subclinical, active, quiescent and burnt-out disease. The clinical manifestations of CS mostly denotes a broad spectrum of features due to its progressive development of inflammation leading to fibrosis and scarring in the cardiac tissues. After an intense study, it has been brought to the light that the specific clinical signs and symptoms of CS can be accommodated into following groups:

1. Unexplained mobitz type II or third degree atrioventricular (AV) block in young patients

2. Sustained monomorphic Ventricular Tachycardia (VT), Ventricular Fibrillation and premature ventricular contractions of unknown etiology

3. Arrythmogenic Right Ventricular Cardiomyopathy (ARVC)

ARVC and CS may have overlapping clinical features making difficult to diagnose. Phillips et al. described 15 patients who met task force criteria for a diagnosis of ARVC but were subsequently found to have CS [14].

4. Idiopathic Heart Failure

Patient may have edema, cough and dyspnea. Reduced cardiac output resulting in oliguria, malaise, and neurological sign such as syncope, confusion and decreased level of consciousness. Core left ventricular (LV) biopsies during LV assist device implantation identified previously undiagnosed CS in 6 of 177 mixed cardiomyopathy patients (3.4%) [15]. Examination of previously implanted hearts found that among 130 heart transplants with a clinical diagnosis of non-ischaemic cardiomyopathy, 8 (6.2%) had undiagnosed CS [16].

Diagnosis

Intensive diagnosing techniques should be focused with respect to cardiac sarcoidosis, as it plays a vital role in minimizing the life threatening complications or even sudden cardiac death (SCD). Initially, a detailed clinical anamnesis of the patient along with genetic counselling is recommended as a part of diagnostic plan of genotype-specific aspects for probable CS. Patients displays features of the classical triad of cardiac sarcoidosis which are 1) compatible clinical and/or radiological picture; 2) histological evidence of non-caseating granulomas and 3) exclusion of other diseases [17, 18]. The diagnostic strategy for patients with known extra cardiac sarcoidosis should be more extensive, initiating with thorough clinical anamnesis, biomarkers, electrocardiography, Holter monitoring and echocardiography.

Electrocardiography (ECG) and 24-hour Holter monitoring. Sensitivity of ECG in detecting CS has been improved from 69% to 81% in patients with cardiac manifestations. The most frequent abnormal findings of ECG in CS includes cardiac conduction abnormalities progressing towards complete AV heart block Mobitz type-II and third degree (complete), premature ventricular contraction and ventricular tachy-arrhythmia, complete or partial right bundle branch block, furthermore QRS complex fragmentation, pathological Q waves (mimicking myocardial infarction), ST segment changes and epsilon waves. Holter monitoring is a useful diagnostic tool for the assessment of the therapeutic prognosis in treatment with immune-suppressive in CS complicated with arrhythmogenic abnormalities can be a predictor of CS with a sensitivity of 89% and can be used to monitor the response to treatment with immuno-suppressive in arrhythmological abnormalities in CS.

Trans-Thoracic Echocardiography (TTE). The modern tactic of using echocardiography together with ambulatory ECG has improved combined sensitivity to 63% in diagnosing CS as per the American Thoracic Society records [19]. In cases of cardiac sarcoidosis, echocardiography reveals cardiac-wall anomalies, interventricular septal thinning, LV hypertrophy or hypertrophic cardiomyopathy, segmental post-systolic contractions, delayed reduced ejection fraction and aneurysms [20]. Also it noteworthy that regional wall motion anomalies are displayed even in non-coronary distribution. If patients who are clinically insignificant shows any of the following signs: LV enlargement, dysfunction, global hypokinesis, aneurysms, diastolic dysfunctions, pericardial effusion and increased echogenicity it points out towards the presence of cardiac sarcoidosis. New modalities such as strain rate has been incorporated to TTE in order to improve sensitivity of echocardiography and Speckle tracking echocardiography has showed promising results in detecting CS.

Endomyocardial Biopsy. The specificity of endomyocardial biopsy depends on the following factors mentioned; 1) In the biopsy there is non-caseating granulomas and increased lymphatic vessels 2) In immune-histology there is increased dendritic cells with decreased M2 type-macrophages in non-granulomatous areas of the cardiac biopsy. Advancements have been made to increase the sensitivity of procedure, which includes electrophysiological (electro-anatomic mapping), and imaging (PET/MRI) guided biopsy hence there has been a significant rise of 50% of positive biopsy rates as per the consensus guidelines [21].

Cardiac Magnetic Resonance Imaging (CMR). The gold standard diagnostic technique is the use of CMR imaging simultaneously with 18-fluorodeoxyglucose positron emission tomography (18F-FDG PET CT). Cardiac MRI illustrates multifocal late gadolinium enhancement (LGE) along with the involvement of mainly the basal segments: the interventricular septum, left lateral wall, right ventricle. Commonly displayed in the epicardium, mid myocardium and a possibility of sub-endocardial coronary artery disease like pattern [22]. LGE has a key feature of prognosticating the fatal future complications such as VT, AV block, heart failure and even SCD. Interestingly CMR also recognizes small areas of myocardial damage in-patient with preserved LV systolic function, which detects silent cardiac sarcoidosis. In order to improve the imaging diagnostics, novel CMR techniques that utilizes the T1 & T2 mapping allows

to detect the myocardial inflammation that helps in monitoring the patient's response to the treatment [23], maintain the high specificity and sensitivity of CMR.

F18-Flurodeoxyglucose PET-CT. In cases of a strong suspicion of cardiac sarcoidosis PET Scan is performed together with the CMR in order to increase the diagnostic specificity as it allows to get the imaging of both stages (inflammation and fibrosis/scarring) simultaneously. F18-FDG PET scan displays focal, focal on diffuse FDG uptake patterns that indicates active cardiac sarcoidosis [24]. The anomalies and perfusion defects are often associated with increased possibility of having ventricular tachy-arrhythmias or death therefore it has a significant role in early diagnosis along with directing the necessary treatment plans with a follow up done every 6 months. Myocardial perfusion imaging with rubidium-82 or ammonia N-13 is done together with PET scan to enhance the presence and characterize the type of CS. Notably PET scan depicts regional myocardial abnormalities that maybe due to cardiac sarcoidosis or atherosclerotic coronary artery disease therefore concurrently coronary angiography must be performed to exclude pathologies related to coronary arterial disease. To assess the extra-cardiac association along with aiding to find biopsy targets it is necessary to perform whole body FDG–PET scanning. Many preparatory guidelines have been established to elevate the specificity of scan results which include:

Prolonged fasting of a minimum of 12 hours and with respect to the day prior the testing, the diet must be rich in fat and low in carbohydrates as the inadequate preparation can eventually lead to false positive PET scans.

Bio Markers. Sarcoidosis specific and cardiac specific are the two major groups of biochemical markers play the pivotal role in terms of diagnosing CS, in which B-type natriuretic peptide (BNP), N-terminal pro-B-type natriuretic peptide (NT-proBNP) and High-sensitivity troponin are the typical cardiac specific biomarkers used for both screening and risk stratification of CS. Among various sarcoidosis specific bio markers, Serum Angiotensin-Converting Enzyme is being the most useful for the diagnosis and monitoring purposes as it's sensitivity varying from 41% to 100% and the specificity ranging between 83-99% [25]. Furthermore, according to many intense studies have been done recently regarding to biological markers in the diagnosis of CS reveal that, neopterin and soluble interleukin-2 Receptor (sIL-2R) levels are high in patients with active CS disease, marking its sensitivity and specificity 88% and 85% respectively [26]. Also, there are other several bio markers such as chitotriosidase, chemokines (CXCL9–11), lysozyme, KL-6, vitamin D dysregulation, and serum amyloid A which can be used in the assessment of CS.

Treatment

Due to the multimodal distribution of the disease, the treatment strategies of CS is mostly multifarious with the combination of multi-disciplinary team approach, Immuno-suppressant, Anti-arrhythmic medications, Heart failure medications, Anti-inflammatory medications, Implantable Cardiac devices (ICDs - cardioverter defibrillator) and Ablation. The main therapeutic aims of the treatment is to slowdown the ongoing active inflammation, recurrent relapses, minimize the myocardial damage and prevent development of complications.

Steroids and Immuno-suppressants: Early administration of steroid therapy in high doses could help in the reversal of severe form of AV block leading to keep the patient away from the Right ventricular pacing and the need for a pacemaker [27, 28].

Recommended initial dose for the prednisolone is 30-40 mg/day and according to the assessment of the treatment response slowly the initial dose can be tapered off. The follow-up for the initial treatment response should be done in 1-3 months with the dose adjustment as needed. If there is, a good response observed the initial dose could be gradually reduced to 5-15 mg /day and continued for 9-12 months. The follow-up should be done for 3-4 years after completing the full course of treatment due to the possible alarming risk of relapse. There are other immune-suppressive have been identified as second line steroid sparing agents in the treatment sarcoidosis such as Methotrexate, Azathioprine, Mycophenolate-mofetil, Leflunomide, Cyclophospamide, Infliximab and Adalimumab.

Conduction abnormalities. Most common presentations of cardiac conduction abnormalities in patients with CS are known to be AV block and ventricular arrhythmias. Patients with significant risk of ventricular arrhythmias are highly suggested to be managed with the implantation of ICD. The patients with complete AV block are well treated with the implantation of permanent pacemaker. Furthermore, anti-arrhythmic medications and catheter ablations are also effective treatment modalities in refractory Ventricular arrhythmias to other medications. There are some well-known anti-arrhythmics used in the treatment of CS associated conduction disorders such as, amiodarone, sotalol, mexiletine, quinidine, dofetilide, or a combination of these. In patients with pulmonary sarcoidosis and CS, amiodarone should be avoided due to the pulmonary toxicity and Class-I anti-arrhythmics due to possibility of inducing pro-arrhythmias. Additionally, there has been an underlying arrhythmogenic effect observed in a well-reputed cohort study denoting that, in patients with CS who underwent implantable cardiac defibrillator in-situ, treatment with steroids resulted a considerable amount of reduction of myocardial inflammation on FDG-PET, though simultaneously the rate of premature ventricular contractions and non-sustained ventricular tachycardia has been increased. As well as, CS can lead to autonomic instability or dysfunction [29].

Management of Heart Failure (HF). Development of extensive myocardial fibrosis is the main cause of HF associations in CS. Complete AV block requires ICD implantation and resultant pacing can lead to cardiac dyssynchrony progressing to HF. CS

patients with reduced ejection fraction and elevated levels of BNP or NT-proBNP are known to have bad prognosis. Restrictive cardiomyopathy with inflammation is treated by administering immune-suppressive medications and diuretics to control the volume status, and improve symptoms while dilated cardiomyopathy is treated with neuro-hormonal blockade along with immune-suppression drugs [30].

Sudden cardiac death. SCD is the leading cause of death in CS and often occurs due to life-threatening arrhythmias. According to the last five years of cohort studies on CS, that the incidence of SCD and sustained VT in CS was reported to be 24.6% [31].

Implantable cardiac electronic devices therapy. As per the recommendation of the American Heart Association, American College of Cardiology and Heart Rhythm Society comprehensive guideline for the management of patients with ventricular tachy-arrhythmias and prevention of SCD emphasizes implantable cardioverter defibrillators with Class-I antiarrhythmics for the secondary prevention and LV ejection fraction ≤35%. It is also recommended as a possible treatment method for the Class-IIa patients with bradycardia despite a LV ejection fraction >35% apart from the use of pacemaker in such cases [32].

Catheter Ablation. Ablation plays a vital role in the management of cases where immunosuppression and use of anti-arrhythmic drugs had little or no response. It is useful in the treatment of recurrent ventricular arrhythmias without any active disease. In the right ventricle usually the scar areas are widely distributed, confluent and transmural therefore, they are approached in combination of endo/epicardial technique. Secondly in scars of the LV, are normally appeared to be patchy and can be located across the ventricle, commonly in the septum and anterior wall where the conventional endo/epicardial mapping is done. Catheter ablation is not performed as a primary therapy in the cases with a positive 18F-FDG PET scan implying existing active inflammation because arrhythmia substrate changes and mechanism is altered. It is contraindicated to be used in the presence of edema due to the poor penetration of thermal energy and higher recurrence [33].

Prognosis. To assess the prognosis of the disease during the follow-ups since there are no specific biomarkers, single imaging techniques or clinical parameters to indicate the longterm therapy regimen [34, 35, 36]. PET-CT based follow-up regimen can be used if initially the abnormality has been found.

1. PET-CT baseline and after 3 months of treatment.
2. If no abnormal uptake: PET-CT 3 months after stopping treatment.
3. If no relapse: evaluation at 6, 12, 24, 36 months – clinical, ECG and Echocardiogram
4. If relapse occurs: intensification of treatment/ more frequent PET-CT [34].

Conclusion

In fact, early diagnosis of arrhythmias, conduction disturbances and heart failure play a crucial role in prognosis of patients with cardiac sarcoidosis.

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РОЛЬ ДИАГНОСТИКИ АРИТМИЙ И НАРУШЕНИЙ ПРОВОДИМОСТИ, СЕРДЕЧНОЙ НЕДОСТАТОЧНОСТИ В ПРОГНОЗЕ ПАЦИЕНТОВ С САРКОИДОЗОМ СЕРДЦА

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Саркоидоз сердца – это хроническое воспалительное заболевание, проявляющееся неказеозными очаговыми гранулематозными поражениями сердца с подозрением на иммунопатологический ответ на неустановленный антигенный фактор у человека с генетической предрасположенностью. Известно, что саркоидоз сердца является второй по распространенности причиной смерти пациентов с саркоидозом во всем мире. Саркоидоз часто протекает как системное воспаление с поражением многих органов, хотя редко он может проявляться в изолированных формах.

Вышеупомянутый специфический фенотип трудно диагностировать, поскольку он требует ряда других исключений, и при отсутствии лечения может прогрессировать до фиброза, а сердечная дисфункция в конечном итоге приводит к внезапной сердечной смерти, что побуждает исследователей и клиницистов модифицировать новые методы диагностики и тактику ведения. В зависимости от распространения и тяжести конкретного фенотипа заболевания, клиническая картина может варьироваться от бессимптомной формы до тяжелых фатальных сердечных проявлений, таких как декомпенсированная сердечная недостаточность, тяжелые формы желудочковой и предсердной аритмий, нарушения проводимости и внезапной смерти.

Поэтому ранняя диагностика и своевременное начало лечебной тактики, включая иммуносупрессивные препараты, по-прежнему имеют решающее значение для борьбы с этим патологическим состоянием.

Ключевые слова: *саркоидоз сердца, аритмии, нарушения проводимости, сердечная недостаточность, внезапная сердечная смерть, диагностика, лечение*

Для цитирования: *Снежицикий, В. А. Роль диагностики аритмий и нарушений проводимости, сердечной недостаточности в прогнозе пациентов с саркоидозом сердца / В. А. Снежицикий, Г. Х. М. Химерши Кавья, М. Д. Джахас Ахамед // Журнал Гродненского государственного медицинского университета. 2025. Т. 23, № 6. С. 655-660. https://doi.org/10.25298/2221-8785-2025-23-6-655-660*

Конфликт интересов. Авторы заявляют об отсутствии конфликта интересов.
Conflict of interest. The authors declare no conflict of interest.

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Поступила / Received: 07.10.2025 *Принята к публикации / Accepted for publication: 25.11.2025*