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Alcohol Induced Cardiomyopathy in Non-Alcoholic Society: A Case Report of Determining the Etiology of Dilated Cardiomyopathy

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Abstract **Review Article**

Alcoholic cardiomyopathy (ACM) is a condition caused by excessive alcohol consumption, which causes a reduction in cardiac function and disturbance in heart pumping ability. Clinical manifestation ranges widely, from asymptomatic to acute fulminant heart failure, depending on the degree of deterioration of heart function. This is a case report of a 37-year-old man who presented to the emergency room, he was referred with the diagnosis of alcoholic cardiomyopathy. From echocardiography, we found reduced left ventricular function with ejection fraction Teich 41,8%, and left ventricular segmental analysis show global hypokinetic with eccentric left ventricular hypertrophy (LVH) which leads to diagnosis cardiomyopathy. Diagnosis of ACM is by exclusion. There is a two-step diagnosis process beginning with cardiac imaging and the second step involves laboratory tests To determining about the etiology of cardiomyophaty, we should depend on patient's history. The diagnosis of alcoholic cardiomyopathy is based on structured history related to alcohol abuse in the long period. Obtaining information about history of alcohol abuse is challange in a non-alcoholic culture and environment. It dipending on the ability of the physicians to explore in-depth information concerning personal problem. Good anamnesis and another supporting examination can help the physicians to making differential diagnosis and makes the right treatment plan for the patient.

Keywords: Cardiomyopathy; heart failure; diagnosis of alcoholic cadiomyopathy, determining the etiology of cardiomyopathy.

1. INTRODUCTION

Cardiomyopathy is a severe disorder of the heart muscle characterized by significant functional and/or electrical dysfunction of the myocardium. The most devastating complication is progressive heart failure with considerable morbidity and mortality. Cardiomyopathies can be classified as either primary or secondary. Primary cardiomyopathies are genetic in nature while secondary cardiomyopathies occur in the setting of a medical condition or due to environmental factors such as toxins or medications (Albakri, 2018).

Alcohol is one of the most frequently consumed beverages in various societies and cultures in the world. The burden caused by alcohol abuse creates losses in health, economic, and social fields. From a health perspective, the complications of excessive alcohol use are physichatric, gastrointestinal, neurological, and cardiological disturbance. Alcoholic cardiomyopathy is a form of cardiac abnormality caused by excessive alcohol consumption. As its name suggests; it mainly affects myocardial structure and function with associated cardiac dysfunction (Wikananda *et al.*, 2019).

The diagnosis of alcoholic cardiomyopathy is based on structured history related to alcohol abuse in the long period. Obtaining information about history of alcohol abuse is challange in a non-alcoholic culture and environment. It takes the ability to explore in-depth information concerning personal problem or is considered taboo by the family and people around the patient.

We present a case with alcoholic dilated cardiomyopathy (DCM) that has progressed to heart failure with reduced ejection fraction and LV enlargement that was come to emergency room in Petrokimia Gresik Hospital. The purpose of this case report is to consider all cardiomyopathy in determining diagnosis, therapy strategies and emphasizes the importance of educating patients about complication of alcohol abuse, recommendations for diagnosis strategies

and medical therapy. These things will help doctors and patients in determining the treatment to be taken next.

2. Case

A 37-year-old man came to the emergency room complaining of swelling in the both of legs. The patient had swollen legs since 2 month ago. Swelling is getting more widespread to the stomach. The stomach has been getting bigger since the last 1 month acocompanied by heavier weight. The patient also complained of abdominal discomfort and often felt tired in one week. He denied complaints of cough, fever, chest pain or dyspneu. The patient did not smoke and denied a family history of heart disease. Patient work as office workers and have a sedentary lifestyle. The patient was found to have a history of drinking alcohol in large quantities from last 5 years. He drinks around six bottles of beer third times in a week.

Vital sign examination shows; blood pressure was 165/103 mm Hg, pulse was 110 beats/min, and respiratory rate was 20 breaths/min. From the physical examination, the patient found increased JVP. Abdominal palpation revealed ascites, undulation test and ballotemen test is positive. Slight edema on both legs was found. On supporting examination; complete blood count (CBC) showed leukocytosis (10.58x103/μL) and decreased Hemoglobin level (10.7 g/dL). Hyponatremi (129 mmol/L) was found in electrolite serum and other laboratory values within normal limits. Posterior-anterior chest x-ray showed cardiomegaly (cardiothoracic ratio; 60%) and right pleural efussion. ECG examination showed sinus tachycardia 100x/min, normoaxis, with slow progression of R V1-V3 (Figure 1).

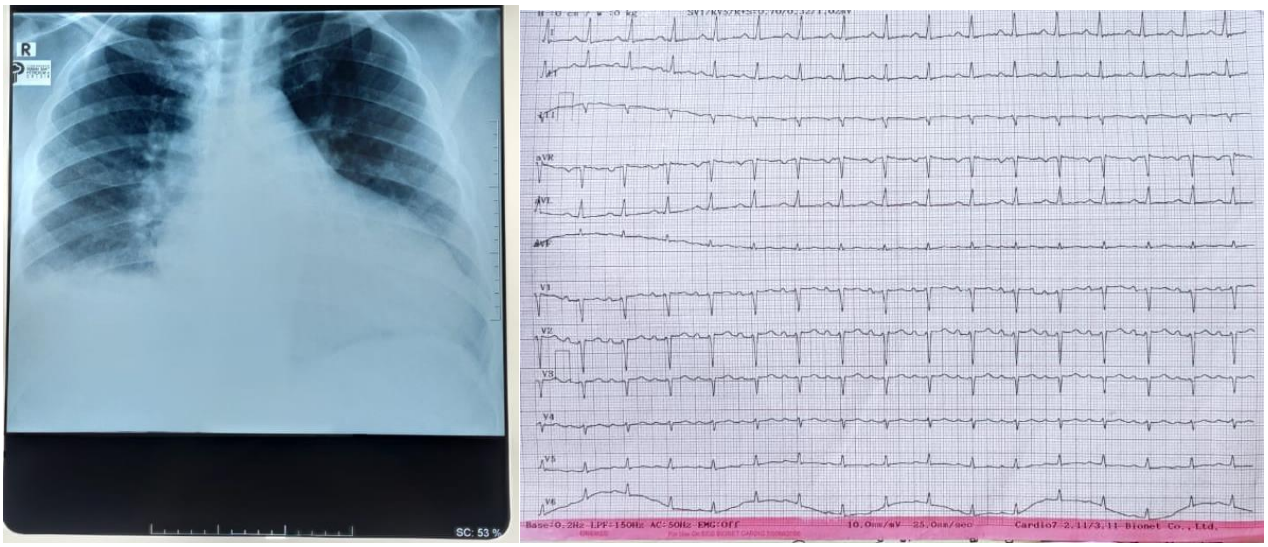

Figure 1: Patient chest x-ray (left) and patient ECG (right)

Echocardiography examination revealed reduced left ventricular (LV) function with EF Teich 41,8%, LV segmental analysis show global hypokinetic with eccentric left ventricular hypertrophy (LVH). Diastolic LV Function: E/A Fussion, Estimation right atrium pressure > 15 mmHg (IVC Exp 30,4 mm; IVC Collaps <50%) (Figure 2).

From the examination results, it was concluded that the patient was diagnosed with dilated cardiomyopathy with overload syndrom e.c Suspec Alcohol induced Cardiomyopathy and CHF FC III-IV.

Figure 2: Parasternal long-axis view showing LV wall motion global hypokinetik; Estimation RAP > 15 mmhg IVC Exp 30,4 mm; IVC Collaps <50%); LV EF Teich 41,8%; Eccentric LV hypertrophy; Abbreviations: LV: left ventrikel; RAP: right atrium pressure; IVC: inferior vena cava; EF: ejection fraction

The patient was admitted and given normal saline solution life line and then replaced with Nacl 3% 500cc/24h for the hyponatremia condition, furosemide bolus 4 ampule continued with furosemide pump 5 mg/h, lisinopril 1 x 2,5 mg as initial pharmacologic treatment. The interesting about this case, intially the patient was suspected of being a patient with liver problems because of the ascites, but after being examined by an internist and there didnt found liver problems, the patient was consulted to the cardiologist.

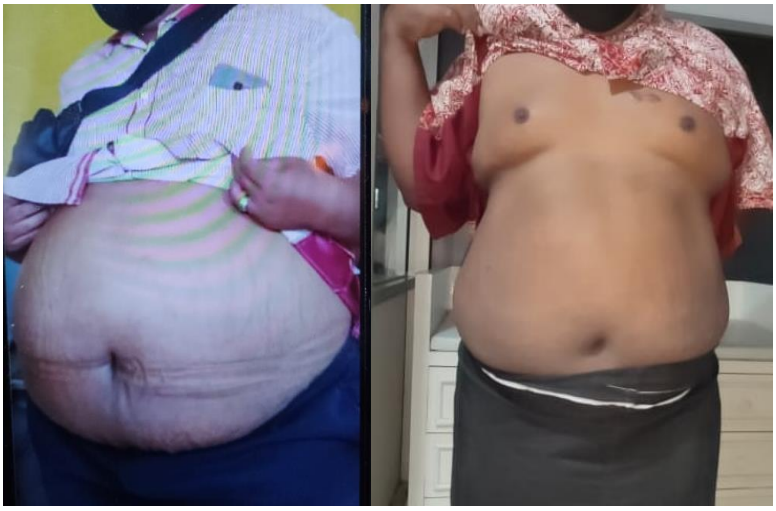

Figure 3: The clinical picture of patient with ascites. The pastient have not received theraphy (Left); (2) The patient during received theraphy (Right)

3. DISCUSSION

Excessive alcohol consumption induces several pathologic changes in the myocardial structur. The exact pathogenesis is still unclear, but it is believed to be related to direct toxic result of ethanol or its metabolites, which results in altered metabolism of fatty acid, disruption of cardiac calcium hemostasis, loss of contractile protein, decreased myocardial protein synthesis and impaired mitochondrial function. Histologically, it features myofibrillar necrosis and fibrosis. Various studies have also shown that alcohol exerts adverse inotropic effects on the myocardium. Ethanol may also cause oxidative stress through the generation of free radicals. It also increases circulating catecholamine. This condition is believed to play a role in generating myocardial damage, which can be proven by the increase of troponin release. Genetics factor is also assumed to contribute in developing alcohol cardiomyopathy.

This patient clinical finding matched with alcoholic cardiomyopathy. From anamnesis, the patient complained of swelling in the both of legs and getting more widespread to the stomach. Physical examination revealed increasing JVP, ascites and slight edema on both legs. From this examination, we can suspect that the patient was in overload fluid condition that can be caused by acute state of heart failure (HF) or chronic liver disease. After the internist had examined of ALT, AST and other laboratory investigation but did not find any problems with the liver function, therefore the internist consults with cardiologist to look possible cardiac disfunction. Our cardiologist does echocardiography examination and then he confirmed that dilated cardiomyopathy was the underlying mechanism for patient complaints. It is a very useful supporting examination to help exclude other non-ischemic cardiomyopathies (Wikananda *et al*., 2019). The echocardiography of this patient only informed us about the morphological and functional of the disease. Actually to distinguish dilated cardiomyopathy (DCM) from common differential diagnostic considerations, research has proven that cardiac magnetic resonance imaging (CMR) is very useful (Sammami *et al*, 2021), but in this case CMR not performed because unavailable of that modalities.

In this case, to confirm the diagnosis we did was in accordance with the guideline. It is in line with theory that diagnosis of ACM is by exclusion. there is a two-step diagnosis process beginning with cardiac imaging to characterize myocardial abnormalities. The second step involves laboratory tests involving carbohydrate-deficient transferrin (CDT) tests, liver tests, and ECG abnormalities aimed to detect underlying cardiac and systemic disease that may cause myocardial dysfunction. According to the PERKI 2020 guideline about diagnosis of acute heart failure, if there is no NT-pro BNP examination tool, an echocardiography can be carried out directly without looking at the NT-proBNP result. In this case the patient not checked NT-proBNP and laboratory test CDT because the examination was not available at our hospital. To seek about the etiology of cardiomyophaty, we should depend on patient's history.

At the first interview the patient hardly to confess about the consumption of alcohol. The reason because he worried about his parent perception because he lived in religious and non-alcoholic society. So our

cardiologist try to make private anamnesis with the patient, so the patient can let us know about his truth of alcohol comsumption. Reduced human resources in the medical system and increased crowding in the interview setting, such as the emergency room and outpatient clinics, have strengthened the need for high quality and efficient interviews that fulfils the three goals of the interview. Good anamnesis can help the physicians to making differential diagnosis of patient's disease. During the medical interview the physician should be sensitive to social and cultural perceptions employed by the patient. (4) The interview also should be adjusted to patient's personality and emotional needs. In such patient and physician circumstances, can be the basis for patient's trust, and help the physician to making decision for proper treatment.

Supporting examination with x-ray showed cardiomegaly, which is usually a sign of another pathologic condition and reinforces our suspicion to HF. Blood pressure was 165/103 mm Hg and pulse rate was 110 beats/min this condition occours because alcohol may lead increased blood pressure. it can be happen probably mediated by the atrial natriuretic peptide (ANP), but also short and long term pressor effects mediated by the renin–aldosterone system and plasma vasopressin.

3.1 Definition and clasification of Dilated Cardiomyopathy (DCM)

Dilated cardiomyopathy (DCM) constitutes an anatomic description of abnormal LV morphology and function in the absence of common pathophysiologic conditions (i.e., coronary artery disease or abnormal loading conditions). As such, it may be a final common pathway to many disease entities where outcome is strongly influenced by aetiology [7]. One of its first descriptions may be found in a case series by William Evans in 1948, describing "Familial Cardiomegaly" after excluding valvular, hypertensive, and congenital heart disease as causes of cardiac enlargement [20]. An autopsy in a subsequent family of two young sisters with "idiopathic cardiomegaly" also revealed dilatation of the LV, but the distinction between hypertrophic and dilated phenotypes was not yet as distinct as it is nowadays [21] (Sammami *et al.*, 2021).

In the last decades, both European and American professional societies have proposed classifications of cardiomyopathic disorders (Figure 3). In 2006, the American Heart Association (AHA) published a seminal document describing the genetic basis of cardiomyopathies [22]. Subsequently in 2008, the European Society of Cardiology (ESC) emphasised that morphofunctional phenotype is the basis for cardiomyopathy classification and recognised extra-cardiac manifestations such as skeletal myopathy in cardiomyopathy patients [23]. The MOGE(S) classification was next proposed in 2013, which subclassified each of the cardiomyopathies into genetic forms and emphasised the necessity to further subdivide the DCM phenotype as it may affect prognosis and treatment. the MOGE(S) nosology system was developed, which incorporates the morphofunctional phenotype (M), organ(s) involvement (O), genetic inheritance pattern (G), etiologic annotation (E) including genetic defect or underlying disease/substrate, and the functional status (S) of the disease using both the ACC/AHA HF stages and New York Heart Association (NYHA) functional class. This nomenclature is endorsed by the World Heart Federation, is supported by an Internet-assisted application, and assists in the description of cardiomyopathy in symptomatic or asymptomatic patients and family members in the context of genetic testing (AHA, 2016).

After that the ESC Working Group on Myocardial and Pericardial Disease proposed a revised definition including "hypokinetic non-dilated cardiomyopathy" (HNDC) as a marker of early or preclinical DCM in 2016 [5]. As per this framework, DCM (and HNDC) may be caused by genetic (±30%) and non-genetic (±70%) causes, of which the latter includes toxic substances (medication (antineoplastic, psychiatric antiretroviral), alcohol, cocaine, amphetamines, ecstasy, iron overload, nutritional deficiency, endrocrinologic causes, tachycardiomyopathy, peri-partum cardiomyopathy, infection, adn autoimune disorder (Sammami *et al.*, 2021).

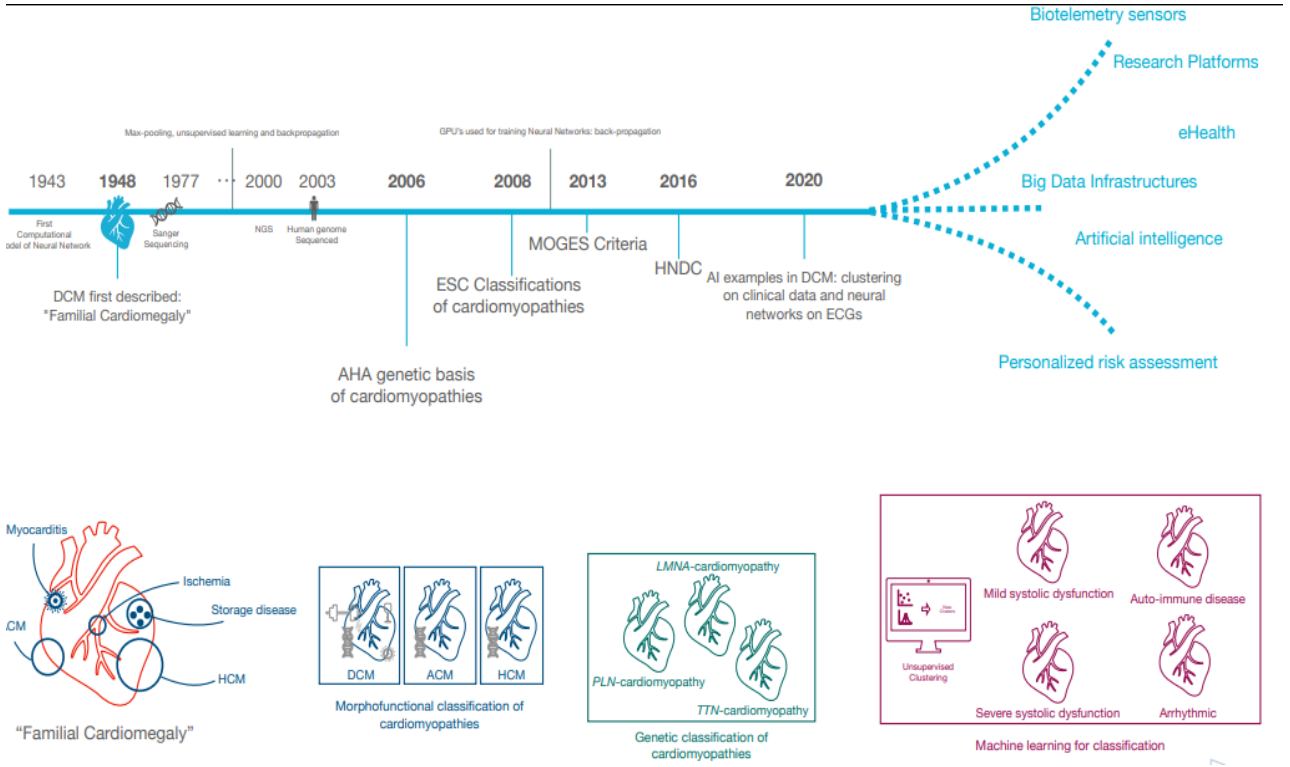

Figure 4: Historical milestones in the classification of dilated cardiomyopathy (DCM). A non-exhaustive list of historical milestones and future prospects are summarised. Additionally, the nomenclature from "familial cardiomegaly" to more specified disease is illustrated. Abbreviations: AHA (American Heart Association), ACM (arrhythmogenic cardiomyopathy), DCM (dilated cardiomyopathy), ESC (European Society of Cardiology), HCM (hypertrophic cardiomyopathy) (Sammami *et al,* 2021)

3.2 Diagnosis of DCM (non-genetic) and Differential Diagnostic Considerations

DCM is considered in the presence of (1) left ventricular dilatation (indexed left ventricular end-diastolic diameter (LVEDd) >117% for age and sex, or the LV end-diastolic volume (LVEDV) 2 standard deviations from normal according to normograms), and (2) left ventricular systolic dysfunction (LV ejection fraction (LVEF) <45% and/or LV fractional shortening <25%).

A full diagnostic work-up for DCM typically includes a focused history, laboratory evaluation, electrocardiography (ECG), Holter monitoring, echocardiography, CMR (with late gadolinium enhancement (LGE)), and genetic testing. In addition, differential diagnosis should be ruled out (e.g., ischemia detection to exclude coronary artery disease) (Sammami *et al.*, 2021).

Given that LV dilatation and dysfunction are the final common pathways in many heart diseases, other cardiomyopathies (arrhythmogenic cardiomyopathy (ACM), hypertrophic cardiomyopathy (HCM), non-compaction cardiomyopathy (NCCM), and restrictive cardiomyopathy (RCM)) may mimic the DCM phenotype [25]. For instance, end-stage HCM may show overlapping clinical characteristics (LV dilatation and reduced LVEF), and ACM may present with a biventricular or left-dominant phenotype. Additionally, Chagas disease, which is caused by a parasite (Trypanosoma cruzi), may lead to cardiomyopathy in 30–40% of affected individuals [29]. To distinguish DCM from common differential diagnostic considerations, research has proven that cardiac magnetic resonance imaging (CMR) is very useful in recent years as it provides a good visualisation of not only the LV but also the right ventricular (RV) myocardium [9]. In addition, LGE patterns on CMR may assist in determining the aetiology—while not mutually exclusive, LV midwall LGE may be seen in genetic forms of DCM or myocarditis, whereas subepicardial LGE may be caused by myocarditis, sarcoidosis, or chemotherapy [30]. Myocarditis may also cause DCM by a complex interplay of inflammation and (auto)-immunologic response [25]. A definite diagnosis of myocarditis, however, cannot be made without endomyocardial biopsy and should focus on immuno-histochemistry ("Dallas criteria") and detection of DNA or RNA of the infectious agent (Sammami *et al.*, 2021).

The clinical presentation of DCM is generally unrelated to the underlying aetiology and ranges from dyspnea, swollen legs, ankles and stomach, fatigue and chest pain caused by reduced oxygen levels reaching the heart to arrhythmia, acute decompensation or cardiogenic shock. The signs and symptoms of DCM mainly relate to the degree of LV or biventricular systolic dysfunction leading to pump failure; heart

failure signs and symptoms may be fulminant, acute, subacute or chronic. In addition, atypical chest pain and palpitation may be present (Heinz *et al.*, 2019).

DCM is typically diagnosed between 20 and 50 years of age. Dilated chambers are readily identified using echocardiography; the diagnostic criteria are LV end-diastolic volumes or diameters >2 s.d. from normal according to normograms (*z*- scores >2 s.d.) corrected for age and body surface area and ejection fraction <50%. Cardiac MRI may assist with imaging dilatation and is used to determine the presence of oedema and/or fibrosis, which are suggestive of inflammation. Electrocardiography (ECG) in patients with DCM may be remarkably normal, but abnormalities ranging from isolated T wave changes and left bundle branch block to prolongation of atrioventricular conduction can occur.Sinus tachycardia and supraventricular arrhythmias are common; ~20–30% of patients have non- sustained ventricular tachycardia (Heinz *et al.*, 2019).

In individuals who are diagnosed with idiopathic DCM, a genetic aetiology should be considered. Thus, the basic evaluation of these patients should include a detailed family history as well as clinical screening of first-degree relatives. Thus, assessment of familial DCM would benefit from an improved understanding of the influence of environmental and epigenetic factors (Heinz *et al.*, 2019).

3.2 Chemical and Toxin Exposure cause DCM

Chronic alcohol abuse is an important cause of DCM, occurring most often in men of 30–55 years of age who have been heavy consumers of alcohol for at least 10 years110. The proportion of alcoholic cardiomyopathy among all cardiomyopathy deaths was estimated at 6.9% globally111, and alcoholic DCM occurred more often in men (8.9%) than in women (2.9%). Studies in animals have demonstrated that acute and chronic ethanol administration impairs cardiac contractility112 and decreases the contractile protein α-myosin heavy chain110. The metabolite of alcohol, acetildehyde, can alter cellular calcium, magnesium and phosphate homeostasis and impair mitochondrial respiration, for example113. Damage to the myocardium from alcohol also recruits inflammatory cells to the heart (shultezz *et al.*, 2019).

3.4 Alcoholic Cardiomyopathy (ACM)

Chronic and excessive consumption of alcohol could result into progressive cardiac dysfunction and heart failure (HF). Chronic alcohol consumption or abuse is defined as the daily intake of more than 80 g of alcohol for a period of at least five years. This degree of exposure to alcohol results in reduced contractility of cardiomyocytes, ventricular dilation, fibrosis, and ultimately HF. Excessive consumption of alcohol is the primary cause of ACM and ACM related HF. ACM potentially leads to increased left ventricular mass, ventricular dilation, wall thinning, and ventricular dysfunction [7]. ACM patients may also exhibit symptoms of arrhythmias and electrocardiographic abnormalities.

Heavy drinking could lead to hypertension that alcohol may cause an acute but transient vasodilation, which may lead to an initial fall in blood pressure probably mediated by the atrial natriuretic peptide (ANP). But also short and long term pressor effects mediated by the renin–aldosterone system and plasma vasopressin have been described. The long-term hypertensive effect of alcohol hasbeen confirmed in manystudies. Remarkably, alcohol also interacts with brain stem receptors and exerts thereby central hypertensive effects (Maisch, 2016).

Pathophysiology refers to the biological and physical manifestations of the ACM as they correlate with the underlying abnormalities and physiological disturbances. As have been noted in the subsequent sections of this paper, ACM is a specific heart muscle condition, which is common in individuals with a history of prolonged chronic alcohol abuse. Piano and Phillips literature review study finds ACM occasions a range of adverse histological, structural, and cellular changes in the myocardium. They find the main pathological elements of ACM are accelerated protein catabolism, generation of oxidative stress, derangements in fatty acid metabolism and transport, apoptotic cell death, and impaired mitochondrial biogenetics/stress.

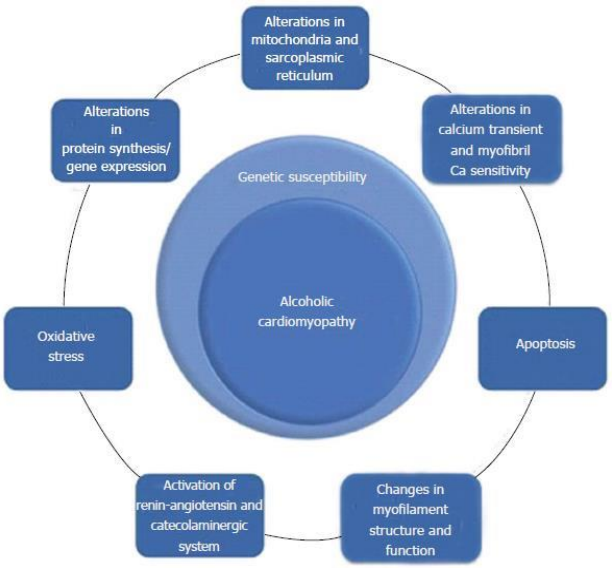

Figure 5: The Pathophysiology of Alcoholic Cardiomyopathy Common pathophysiologic mechanisms for ACM are oxidative stress, apoptosis, alteration on calcium hemostasis, impaired mitochondrial stress, altered protein synthesis and changes in myofilament structure and function. Adapted from Guzzo-Merello *et al*., 2014

The diagnosis of ACM is often one of exclusion of DCM patients with a long history of chronic and excessive consumption of alcohol [3]. Most studies report a daily consumption of more than 80 g of alcohol for at least five consecutive years is the threshold for establishing a diagnosis of ACM [6,11-14]. However, the current definition of ACM – chronic and excessive exposure to more than 80 g/day of alcohol for at least five years – lacks sufficient epidemiological or experimental evidence.

Taking diagnosis of ACM is primarily by exclusion of other probable or suspected etiologies. Despite the diagnostic challenges, the present analysis reveals diagnosis of alcoholic cardiomyopathy is complemented using laboratory tests. The cornerstone of laboratory test is the assessment of carbohydratedeficient transferrin (CDT) assessed by 73% of studies included in the present literature-based meta-analysis. The specificity, sensitivity and accuracy of CDT is supplemented by the assessment of other important biomarkers including liver function tests (alanine transaminase (ALT), aspartate aminotransferase (AST), and alkaline phosphatase (ALP). In addition to CDT, the ratio of SGOT/SGPT (2.85 +/- 0.2) and AST/ALT ratio < 1.0 strongly suggests the presence of ACM.

Laboratory marker	Indicative for	Time to normalize	Monitor abstinence
Alcohol concentration	In acute alcohol intoxication	Hours	Yes
Mean corpuscular volume of red blood cells (MCV)	Increased	3 months	No
GGT, GOT, GPT, GOT/GPT ratio	Liver disease in patients with alcohol abuse	4 weeks	No
CDT (carbohydrate-deficient transferrin)	Chronic alcohol abuse	4 weeks	No
Ethyl glucuronide and ethyl sulphate	High-risk drinkers	2 days	Yes
Phosphatidyl ethanol	High-risk drinkers	4 weeks	No
NT-proBNP	Heart failure, helpful in follow-ups	Several weeks	No
Troponins, CKMB	Acute myocyte destruction	1–3 days	No

MCV mean corpuscular volume, *GGT* gamma-glutamytransferase, *GOT* glutamic oxalacetic transaminase, *GPT* glutamic pyruvic transaminase, *CDT* carbohydrate-deficient transferrin, *NT-proBNP* n-terminal pro brain natriuretic peptide, *CKMB* creatinin kinase, muscle, brain subunit

Figure 6: Markers of alcoholismand cardiac involvement (Maisch, 2016)

Laboratory testing are useful to provide complementary diagnostic clues for confirming the presence of ACM. The history of the patient is another key diagnostic procedure valuable for excluding other potential etiologies such as anti-cancer medication or ischemic heart disease strengthening diagnosis. Imaging modalities are the initial diagnostic methods to characterize and detect underlying alcohol induced myocardial dysfunction. Cardiac functional parameters such as echocardiographic abnormalities including increased left atrial dimension, increased LV wall thickness and decreased fractional shortening precede onset of clinical symptoms in individuals with a history of alcohol abuse, suggest the presence of ACM but are not definitive [29,97]. Other echocardiographic abnormalities are four chamber dilatation, low cardiac output and normal/decreased LV wall thickness [29]. Tests such as electrocardiography abnormalities such as a third heart sound and elevated jugular venous pulse and cardiomegaly with or without rales are common in decompensated state.

Diagnosis of ACM is by exclusion. It is a two-step diagnosis process beginning with cardiac imaging to characterize myocardial abnormalities. The second step involves laboratory tests involving carbohydrate-deficient transferrin (CDT) tests, liver tests (ALT, AST and ALP) and ECG abnormalities aimed to detect excessive alcohol consumption and to exclude other potential underlying cardiac and systemic disease that may cause myocardial dysfunction. Clinical management of ACM is challenging due to the lack of standard clinical guidelines. However, absolute abstinence from alcohol is the principal treatment option efficacious in relieving symptoms and reversing myocardial damage. For patients with severely depressed LV function and symptomatic heart failure, conventional therapy for heart failure mostly pharmacotherapy and device therapy (ICD) are recommended. Heart transplantation is the last line of treatment recommended for ACM patients with end-stage heart failure or refractory to optimal medical therapy and ICD (Albakri *et al*., 2018).

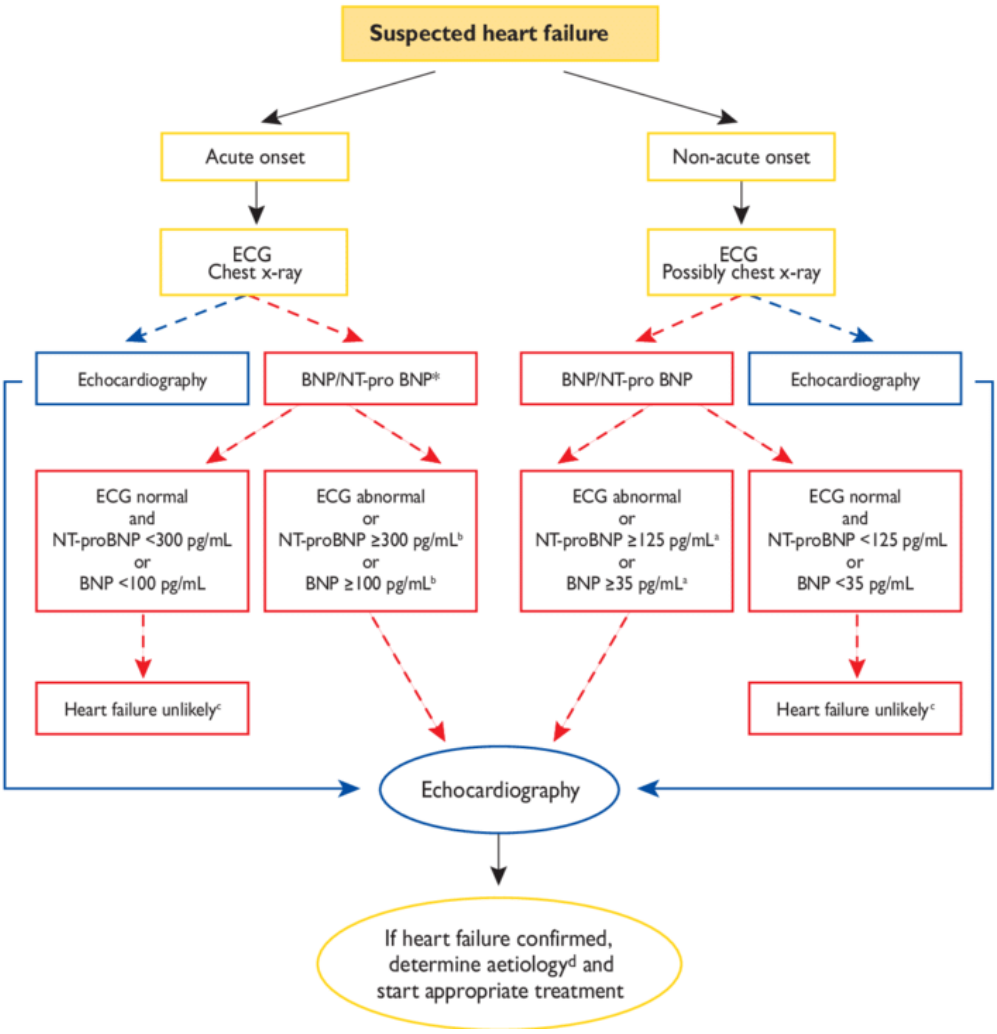

Figure 7: Diagnostic scheme for uspected heart failure (PERKI, 2020)

Prognosis in individuals with low or moderate consumption up to one or two drinks per day in men and one drink in women is not different from people who do not drink at all. In patients with chronic alcohol use disorders and severe heart failure prognosis is poor, since continued alcohol abuse results in refractory congestive heart failure. Death might also be sudden due to arrhythmias, heart conduction block, and systemic or pulmonary embolism.In these patients, only early and absolute abstinence of alcohol can reverse myocardial dysfunction (Maisch, 2016).

To maintain abstinence, recent investigations suggest the benefits of adjuvant medications, e. g., naltrexone, which is an opiate receptor antagonist that blocks endogenous opioid reward and reduces alcohol-cue-conditioned reinforcement signals; acamprosate, an agent that exerts action through excitatory amino acids; by disulfiram, an aldehyde dehydrogenase inhibitor, which causes in alcohol use acetaldehyde accumulation and symptoms such as nausea, flushing, sweating, and tachycardia or by selective serotonin re-uptake inhibitors (SSRI). To treat the alcohol problem, a combined approach comprising pharmacologic and psychosocial therapy involving self-help groups or Alcoholics Anonymous is essential.

Treatment of alcoholic cardiomyopathy follows the usual regimen for therapy of heart failure, including ACE inhibitors, betablockers, diuretics including spironolactone or eplerinone, and digitalis in atrial fibrillation for rate control together with anticoagulation, whenever appropriate (Figure 8) (Maisch, 2016).

Medication	Treatment goal	Dosage	Adverse reaction	Evidence
Pharmacological for maintaining abstinence				
Naltrexone	Abstinence	50–100 mg/day (oral) 380 mg i. m. per month	Nausea, headache, dizziness, joint and muscle pain	High
Acamprosate	Abstinence	666 mg three times daily	Diarhea, pruritus, rash, altered libido	High
Disulfiram	Abstinence	200 mg/day (oral)	Dizziness, rash, headache, polyneuritis, impotence, hepatotoxicity	Mixed, needs supervision
Nalmefene	Reduced drinking or abstinence	18 mg/day (oral)	Dizziness, rash, headache, nausea, vomiting	Moderate
Diazepam	Avoid delirium	As needed	Dizziness, sleepiness	Only symptomatic
Pharmacological for heart failure (HF)				
ACE inhibitors	HF+ prognosis	As tolerated	–	High in HF
Betablockers	HF+ prognosis	As tolerated	–	High in HF
Diuretics	HF+ prognosis	As needed	–	High in HF
Digitalis	Rate control	According to digoxin or digitoxin level	Avoid overdosage	Moderate in atrial fibrillation (AF)
Anticoagulants	Avoid stroke	INR 1.8–2.2 in AF	Bleeding	High in AF

Figure 8: Treatment of alcoholism and alcoholic cardiomyopathy (Maisch, 2016)

4. CONCLUSION

Alcoholic cardiomyopathy is a condition caused by excessive alcohol consumption, which causes a reduction in cardiac function and disturbance in heart pumping ability. Clinical manifestation ranges widely, from asymptomatic to acute fulminant heart failure, depending on the degree of deterioration of heart function. Diagnosis of ACM is by exclusion. It is a two-step diagnosis process beginning with cardiac imaging to characterize myocardial abnormalities. The second step involves laboratory tests involving carbohydrate-deficient transferrin (CDT) tests, liver tests (ALT, AST and ALP) and ECG abnormalities aimed to detect excessive alcohol consumption and to exclude other potential underlying cardiac and systemic disease that may cause myocardial dysfunction. To seek about the etiology in order to treat patient, we should depend on patient's history, so good anamnesis can help the physicians to making differential diagnosis and making therapy plan based on patient condition. Early diagnosis, total alcohol abstinence and early management with drugs used in systolic heart failure are the mainstay of treatment to prevent disease progression, relieve symptoms, and prevent complications and reversal of the disease if possible.

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