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A review of the literature on coronary micro vascular dysfunction

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SUMMARY

Patients with Coronary Micro vascular Dysfunction (CMVD) are a common occurrence, and despite the fair prognosis, many of them have a poor quality of life due to angina symptoms that limit their daily activities. The most common clinical presentation pictures, such as stable and unstable micro vascular angina, are summarized in this article. The main risk factors are discussed, followed by the most recent information on the topic, including pathogenic hypotheses, diagnostics, and treatment options. Micro vascular abnormalities that aren't fully understood, such as slow flow and no reflow are explored, as well as prognosis and the disease's influence on quality of life. Angina without Coronary Artery Disease (CAD) is associated with significant morbidity and is found in 10% to 30% of individuals who undergo angiography. In 50 percent to 65 percent of these patients, Coronary Micro vascular Dysfunction (CMD) is evident. The best treatment for this group is unknown.

Key words: COVID -19; Taste; Pregnant

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INTRODUCTION

In the absence of CAD, we conducted a systematic review to assess therapeutic options for objectively diagnosed CMD. We included studies using positron emission tomography, cardiac magnetic resonance imaging, dilution methods, or intracoronary Doppler in human subjects with angina and a coronary flow reserve or myocardial perfusion reserve of 2.5 in the absence of coronary artery stenosis of 50% or structural heart disease. Only eight papers satisfied the stringent standards for inclusion. Different treatments, outcomes, and definitions of CMD were used in the publications. With an average of 11 patients per investigation, the small sample sizes substantially restrict the power of this research studies looking at the effects of sildenafil, quinapril, oestrogen and transcutaneous electrical nerve stimulation all showed positive results. A considerable number of these patients exhibit Coronary Micro vascular Dysfunction (CMD), which is defined as an increased sensitivity to vasoconstrictor stimuli and a reduced capacity for micro vascular vasodilation. In the last two decades, the coronary microvasculature has been investigated more thoroughly thanks to the development of non-invasive and invasive procedures.

In addition to classic atherosclerotic disease and vasospastic illness, CMD has been recognized as a cause of cardiac ischemia. CMD can arise on its own or in conjunction with obstructive CAD. CMD and macro vascular CAD have many risk factors in common. The attenuation of coronary blood flow in response to vasodilator drugs is used to diagnose the condition. Traditional intracoronary vaso-reactivity testing has not yet been totally superseded by imaging modalities such as cardiovascular magnetic resonance, positron emission tomography, and transthoracic Doppler echocardiography. CMD treatment begins with a change in lifestyle and the management of risk factors. Traditional antinational, anti-atherosclerotic drugs, as well as some novel therapies, may be useful; nevertheless, clinical trials are required to determine the efficacy of pharmacologic and no pharmacologic therapy approaches. Furthermore, longer-term studies are required to assess the prognostic benefits of these medicines. The epidemiology, prognosis, pathophysiology, diagnosis, risk factors, and current treatments for CMD are all discussed.

The widespread scope of this remodeling, which affects coronary micro vessels across the left ventricle, is a key characteristic of these phenomena. Heart Failure with Preserved Ejection Fraction (HFpEF) and Heart Failure with Reduced Ejection Fraction (HFrEF) can occur in patients with LVH caused by arterial hypertension (HFrEF). These patients can develop HFrEF by a 'direct pathway' with or without an interval myocardial infarction. Patients with HFpEF, on the other hand,

can proceed to HFrEF with or without interval myocardial infarction. Patients with hypertrophic cardiomyopathy, the most common hereditary cardiomyopathy with a pattern marked by large LVH, can experience a similar progression to LV dysfunction and both HFpEF and HFrEF. We will examine both experimental and clinical findings that explain the mechanisms behind CMD in LVH, as well as data linking CMD to HFpEF and HFrEF, in this review paper. Angina has long been assumed to be caused by a variety of factors. When coronary angiography is performed on a large number of individuals with angina, it is discovered that they do not have obstructive CAD [1-5].

CONCLUSION

L-arginine, doxazosin, pravastatin, and diltiazem showed no benefit. Our systematic review found that there is little evidence to support CMD therapies. We evaluate material that meets stringent inclusion requirements and examine previously published data that is related but not included. We also go over the next stages in closing this research gap, which include a consistent definition of CMD, routine CMD assessment in studies of chest pain without obstructive CAD, and specific therapy assessment in the population with confirmed CMD. At this moment, we don't know enough about the relevance, processes, and consequences of coronary micro vascular dysfunction linked with diabetes mellitus. Endothelial dysfunction induced by diabetes in various arterial beds is well known for contributing to a wide range of problems and having negative effects on microcirculatory control. The coronary microcirculation is controlled by a series of interrelated physiological processes that aim to adapt local blood flow to myocardial metabolic needs. This network's deregulation could have varied degrees of pathogenic repercussions.

CONFLICTS OF INTEREST

The authors declare no competing interests.

All authors declare that the material has not been published elsewhere, or has not been submitted to another publisher.

DATA AVAILABILITY

Authors declare that all related data are available concerning researchers by the corresponding author's email.

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