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Contextualizing Microvascular Dysfunction: The Emerging Role of Artificial Intelligence

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Abstract

According to the recent report from the world heart federation(WHF), a staggering 20.5 million global deaths were attributed to cardiovascular disease in 2021, a notable increase from the 12.1 million reported in 1999.Nowadays, ischemia emerges as a prominent factor, representing a tradable main problem that poses a formidable challenge.Conventional cardiology treatments often encounter limitations, compelling a shift towards invasive strategies like heart transplants or percutaneous interventions such fas stinting, even in cases of stable chronic ischemia.To overcome the limitations of established cardiovascular treatments, cutting-edge technologies based on arti cial intelligence(AI) can play a signi cant role in supporting and enhancing these treatments.In this context, the main objective of this paper is to spotlight a spectrum of non-invasive strategies that have recently surfaced through the implementation of AI technologies.Our focus will center on capturing recent relevant studies and successful approaches of microvascular dysfunction detection clinical scenarios coupled with AI models and technologies to actively support and enhance our understanding of contexts related to stable ischemic heart disease(SIHD).This investigation aims to provide a comprehensive overview of the progress made in employing AI techniques to address the challenges posed by ischemia, including an exploration of suitable data types and their combination.Furthermore, the discussion will extend to newly duced approaches aimed at pro ciently reversing the impact of ischemia.

Keywords: artificial intelligence, cardiovascular disease, microvascular dysfunction

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1. Introduction

Recently, cardiovascular illness is categorized as the third most life-threatening disease as example, in the united states of america (USA) statistics and predictions highlight the ongoing growth of pathology prevalence in population over 65[1].Furthermore these investigations estimated that about 15.4 million persons older than 20 years in USA have ischemic heart disease(SHD). In canada heart disease is the second leading (3) cause of death after cancer.According to the canadian society chronic disease surveillance system(CCDSS) datasets, the rate in ischemic heart disease approve 2.9 times higher among adults age 20 and over with diagnosed heart disease, 4.6 times higher among adults age 20 and over who have had a heart attack versus those, 6.3 times higher among adults age 40 and over with diagnosed heart failure versus those without it.while in recent statistics of the people's republic of china, over 11.3 million patients diagnosed coronary artery disease with[2]this range of age presents a signi cant socio-economic[3]challenge due to the costs[4]associated with hospitalizations, treatments, revascularization procedures, re-perfusion therapy, clinic visits, and emergency services,this situation impacts society as a whole not just individuals, due to disparities in the distribution of healthcare resources in low-income countries combined with the escalating costs of cardiac interventions, which greatly increase the likelihood of exacerbating this issue.

To reduce these high-stakes risks mentioned above, several health organizations such as WHF make as 2030's strategy and objective reducing the cardiovascular-related death rate per year to achieve this goal the integration of diverse contributions from cardiologists, scienti c researchers, high-tech engineers and even civil society providing actual use case data to improve the system from lifestyle habits to e ective therapeutic processes.One of these key advanced strategies that can be integrated in this context is the adoption of arti cial intelligence as a highly e ective and e cient technology.Based on the huge availability of public worldwide clinical data sets and advanced recent sub elds of AI(ex. machine learning, deep learning, generative AI)that could be used to analyse, understand and gain insights toward more complex assessment cardiovascular disease minimizing the costs. The objective of this paper is to measure and list and compare clinical to arti cial intelligence scenarios that approach a coronary micro vascular dysfunction possibilities to endorse and limitations.The remainder of this investigation is structured as follows: section 1 encompass an introduction recent statistics about the prevalence in the world followed a description of the physiology and pathophysiology of micro-circulation and the symptoms or risk factors that could approach it, the section divided to two parts the rst one list the clinical scenarios and protocols to cover the pathology of micro-circulation in precise contexts while the second one was to seek the projection of those scenarios on arti cial intelligence eld how the latter to intervene to solve the problems de ned from the clinical point of view.articles selected were from selection goggle scholar using the following keywords: stable ischemia, microvascular dysfunction, arti cial intelligence. . . and logical operators, and the types of articles consulted were newspaper articles, journals, conferences, even editorials, thesis, dissertation and reports than books.

2. Coronary Pathology: Insights and Challenges

2.1. Stable ischemia in microcirculation area

Stable cardiac ischemia[5, 6]is considered as a hallmark of ischemic diseases[7]characterized by an imbalance between the supply and demand of oxygen to the myocardium, or circulation sanguine, it's referenced in the most cases to the atherosclerosis plaque composition in the coronary artery surface specially in the pericardia cells for both the obstructive or non obstructive coronary artery disease(CAD), the pathological process development of micro vascular dysfunction within stable ischemia[8]is unclear yet due too multiform[9]and factors wish contribute to the pathology[10]and it could take a long stable period stabilized atheromas with thickened brous caps and an increasing proportion of densely calci ed plaque transforming from stable to unstable at any time, typically in acute use cases atherothrombotic event are caused by plaque rupture and erosion, the remarkable proposed mechanisms that tried decipher stable ischemia varies[11]between physiology, anatomy(clumped leukocytes or platelets), morphology and properties of lesions.overcoming that sometimes to psychology and environment factors as a recent trends show[12].coronary artery spasm(CAS)had persist as hypothesis that play a signi cant role engendering a stable ischemia(angina pectoris)veri ed typically by coronary angiogram or acetylcholine(ACh)or sympathetic vasoconstriction response(CPT)test to distinguish between pericardia and microvascular spasm[13]discussed in a burden of sense as the relationship between myocarditis cells and coronary spasm characterised by a recurrent thrombosis events, potassium and calcium levels in myocardium cells.Generally in micro vascular aspect the light were shedding on making clari cation abnormal responses of the coronary microcirculation phenomena depending to severity and coexistence with atherosclerosis, pression aortique, diastolic, systolic function reactivity through fractional ow reserve[14], hyperaemic microvascular resistance.(hMr).In ischemia with non obstructive coronary arteries(INOCA)[15]microvascular spasm represents one of the key mechanisms tackled[16, 17]as transient episodes of myocardial hypoperfusion snapshots which may appear as angina or angina equivalent symptoms[18].Main aspects explored were related to endothelial dysfunction,rho-kinase activation caused by coronary vasomotion abnormalities, coronary vasospasm and distal coronary constriction typically in patient with vasospatic angina who have high microvascular resistance[19].coronary microvascular dysfunction(CMD) were mostly accompanied or discussed via in ammation events as a primordial aspect that contribute to cover ischemia process development counting in C-reactive protein(CRP) as a powerful to be both a marker and a mediator of atherosclerosis[20]promoting endothelial dysfunction and inhibits nitric oxide, but the question remain uncovered is which type of intervention is the most convenient revascularisation, coronary artery bypass graft(GABS), percateneous coronary intervention(PCI) of the culprit lesion as frequent solution that interventional cardiologist adopt to restore the normal functioning of the coronaries and avoid irreversible necrosis[21].

2.2. micro-circulation normal physiology

Cardiovascular system is complex well structured schema that's s mostly by di erent linked components where the damage of one imply the damage of the another functionality taking scope from sinoatria(SA)node activation to the myocardium demand granting playing a primordial role in oxygen transport from the red blood cells(RBC)in the capillaries to the parenchymal cells, regulation of solute exchange between the intravascular and tissular space responsible for the transport of all blood-borne hormones and nutrients to the tissue cells including mediating the functional activity of the immune system and haemostasis[22, 23].The micro-circulation as a terminal vascular element of the systemic circulation consists of those blood vessels too small to be seen with the naked eye(arterioles, capillaries, venules and terminal lymphatic vessels), a healthy transport of oxygen in the micro circulation is described by two mechanisms of red blood cells(RBC) ux or ow.Microvessels are distinguished by their connection to both endothelial(ECs) and pericytes(PCs)cells that contribute to angiogenesis and vascular remodeling comprehension[24]through complex signalling pathways.A normal microcircolulation depends on the capacity to brings dynamic changes in the function of the arterioles the precapillary sphincters, the muscular venules and veins as increasing tissue perfu sion, to the muscles during vigorous exercise dilatation of arterioles and precapillary sphincters increases the ow of blood into an expanded capillary bed[25]to maintain an diastolic blood pressure and redistribute blood ow to di erent vascular beds while the return of the blood to the heart facilitated by the negative intra thoracic pressure.

The gure below illustrates the hierarchical structure of the coronary vascular bed, encompassing epicardial vessels, pre-arterioles, arterioles, and capillaries, and highlights their respective anatomical and physiological characteristics.

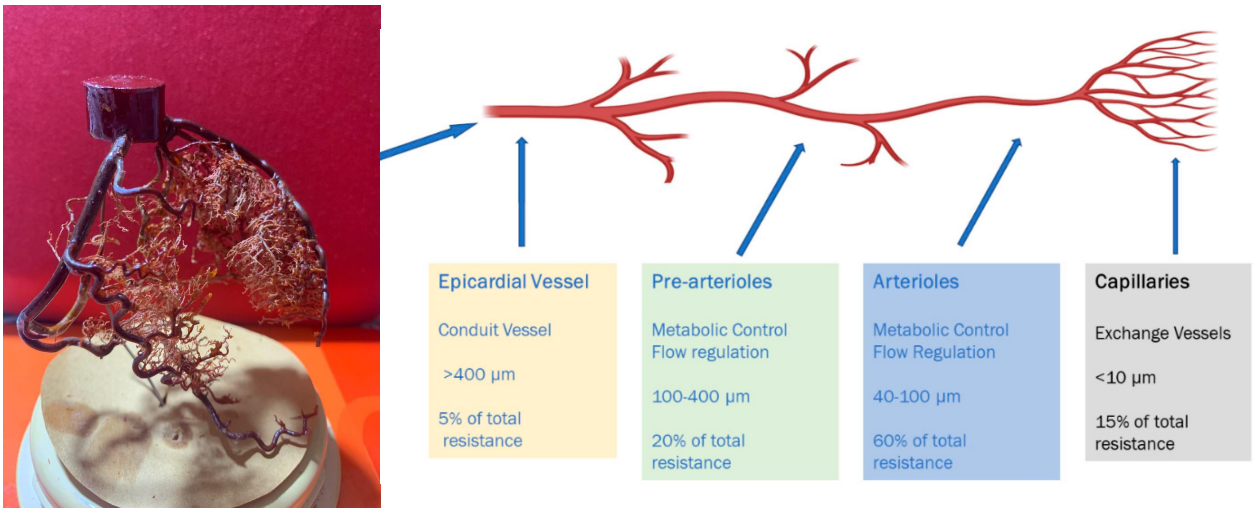

Figure 1: Anatomical and functional classi cation of the coronary macro and micro-arterial system[26].

2.3. Background

The problems associated with coronary pathology primarily arise in cases of chronic coronary ischemia, as the appropriate response to acute coronary ischemia is clear and immediate: emergency intervention is required.This category of patients are a ected directly to stent application or surgery uncertainty creating a persistent problem, in chronic ischemia the results of clinical test are not usually understood they depend on the patient coronary disease history, underwent surgery or stenting, or whether they underwent medical treatment and a radical change in lifestyle especially asymptomatic cases, In the results of the ISCHEMIA study, it remains unclear whether surgical intervention or stent placement yields better outcomes.

In recent years, greater attention has been paid to patients with chronic coronary insu - ciency of a certain type presented with pectoris angina, incomprehensible chest pain related to normal coronary angiogram test.in that context CMD rise as the most probable cause of irregular circulation[27], It would therefore be of particular interest to determine which speci c cases of chronic coronary insu ciency truly require revascularization through surgery or stenting to restore blood vessels.

The general schema that characterise the origin of CMD manifestation where each component of that latter is related to personalised context and di erent rule based criteria to insert under microcirculation dyseregulation is de ned as below subdividing to two principal categories functional and structural modi cation a ecting normal circulation process.

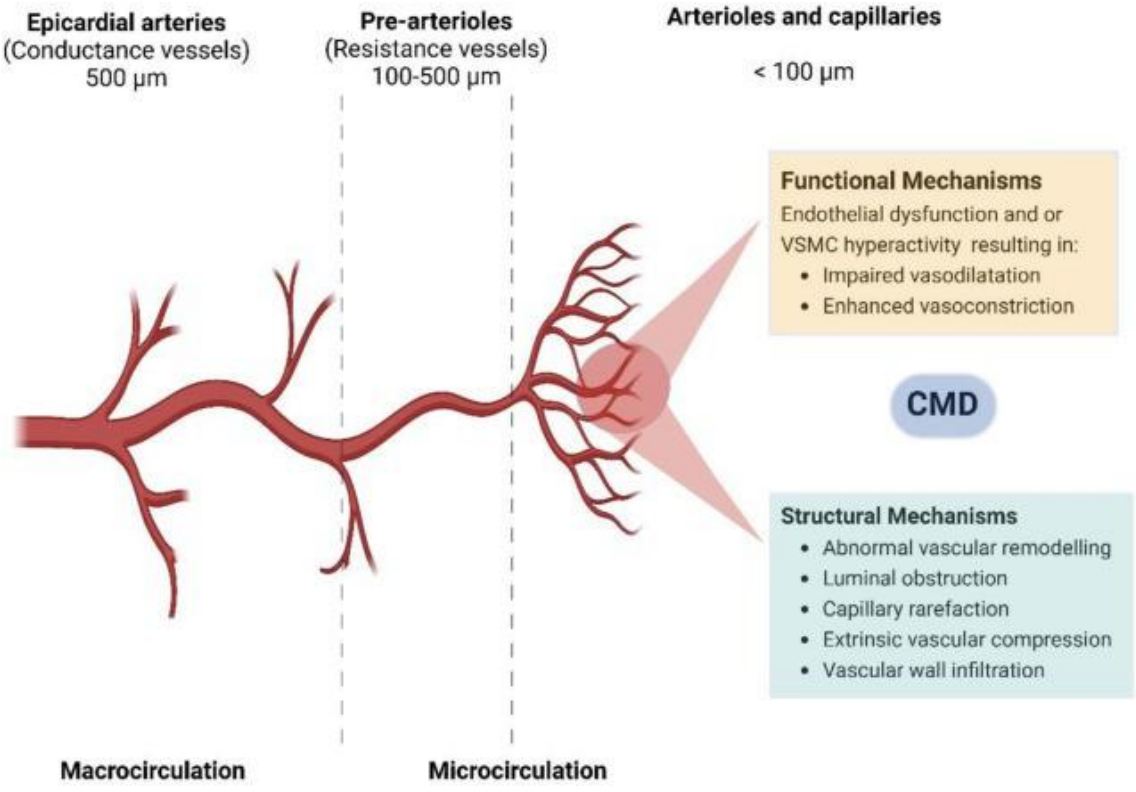

Figure 2: Coronary microvascular dysfunction pathophysiology[28]

It is therefore interesting for us to see what arti cial intelligence(AI)is capable to do switch context to adjust that dilemma stable ischemia revealed related microvascular damage, stage of disease and gravity, e cient strategies to adopt for better outcomes. the history of arti- cial intelligence in precise medicine had knew di erent stages that had began in the early of 2000's people's[29]making possible the processing and integration of large heterogeneous patient record[30]capturing complex patterns to enhance clinical decision making quality through augmented intelligence.Due to the multifaceted conditions that show up individu- ally or coexist to engender a pathological microcirculation, arti cial intelligence technology tried to dive deep and develop new markers to understand the process and mechanisms of CMD development as cause or e ect contributing to worse outcomes, sudden cardiac death(SCD)with or without ischemia signs translating clinical scenarios to real world prac- tical numerical simulations experiences in order to identify a practical clinical solutions and guidelines counting on non-invasive strategies and cardiac imaging less biased approaches to adhere cardiologists expectations and needs in order to match a clear view and triggers experimentally su cient and acceptable to recommend precise types of interventions regards pathological microcirculation in stable ischemia with normal coronary angiogram results.In this part we will focus in arti cial intelligence angle of view and recent advances released studies to tackle that issue in the microvascular aspect discussing some applications contexts that had tried to elucidate the role of arrhythmias, in ammation and hypertrophy to change normal circulation(structural and functional abnormalities)relationship to the activation of collateral circulation.the studies selected reviewed clearly outline the speci c objectives of each methodological approach and the corresponding data modalities utilized, correlation to CMD characterisation, shared and associated modi able index based CMD de nition, selected patients, accomplished tests for each study, we report the dataset type, eligibility criteria, and initiating hypothesis, aligning these elements with the prevailing operational de nitions CMD[31].

2.4. Structural and functional alterations associated with microvascular dysfunction via stable ischemia and risk factors

2.4.1. Arrhythmia

Atrial brillation(AF)as typical type of arrhythmia is in continuous arise[32, 33]inserting under the important challenging faces of the chronic coronary syndrome(CSS)[34]prognostic with primordial percent for microvascular dysfunction that arrives to 15% of prevalence changing atherosclerosis plaque morphology and stability leading to an increase in myocardium oxygen consumption creating a mismatch between supply and demand, the hypothesis those con rm that there's a causality between atrial brillation and microvascular dysfunction within stable ischemia were posed in the sense of irregular contraction incidence predominantly supported by thrombus formation and prothrombotic status description[35?]especially among patients undergone PCI.Inter-pathway crosstalk correlated with endothelial dysfunction in AF had earned a weak score and they were limited to epicardial endothelium dilation aspect[36]extended to be investigated mitochondrial dynamics area[37], frontal QRS-T angle[38], the biological studies involved were segmented to the localization of connexion between myocytes[39], calcium(Ca2+)current dynamics during systole, the characterization of outward ionic currents in myocardial cells, homocysteine level was also proposed as principal marker in contrast to b-type natriuretic peptide(BNP)and broblast growth factor(FGF), (FGF-23)[40], while T wave inversion[41, 42]in leads V2 and V3 of the electrocardiogram(ECG)associated to reversible function of LV systole[43, 44, 45]T-interval dispersion in ventricular repolarization distortion case formed with or without dystolic dysfunction context[46], QTc wave prolongation e ect on arrhythmogenic substrate reducing the repolarization process[47], QRS wave analysis induced microvascular diagnosis after a myocardial infraction(MI)had been explored in re-polarization phase of heart pumping in[48]related to coronary slow phenomena[49]tended to be longer in association with partly mediated CMD after adjustment of atherosclerosis[50], in that sense diver scenarios of repolarization alteration were captured to be classi ed as programmable paths giving rise to CMD as outcome[51], ventricular distortion repolarization[52]is inspected via an inhomogeneous ventricular electrical activity assessment based broblasts activation[53]expressed by dispersed and corrected QT intervals associated with re-patterning of gap-junction disruption morphology, extracellular vesicles(Evs)elevation characterise endothelial cells viability de ning di erent pro les of coronary ow reserve(CFR)in patients with heart failure in preserved fraction ejection(HFpEF)speci cally after MI, atrial remodelling characterisation versus microvascular resistance[54]stand a useful mechanism giving idea about ectopic or re-entrant activity using P-wave features, aortic pulse-wave velocity(aPWV)[55]analysis to left ventricular sti ness[56, 57].angiogenesis as a compensatory mechanism serve to microvascular augmentation through AF it contributes to play a role in microavscular rarefaction explaining ischemic injury and oxygen deprivation[58], mean scenarios de ned based synergistic role of angeogenesis to encounter CMD de nition were designed based elevated endothelial cell proliferation, migration and increased microvascular density with implications for exchange between circulating blood and surrounding tissues[59]revealing as result a rapid atria activation[58]and impaired vasoactive function slowing down action potential, focal necrosis[60].Variant strategies and techniques have been proposed and implemented to decipher AF or arrhythmia's relationship revealing or causing a CMD according to di erent clinical contexts and implicated factors quantifying abnormalities of CFR myocardial blood

ow(MBF), coronary blood ow(CBF) and coronary vascular resistance(CVR), fractional ow reserve(FFR) quanti cation[61]and correlation to untoward outcomes discussing the AI algorithms parameters, scienti c indicators based rule to de ne the key control of the groups de nition for classi cation problems, the main application of arti cial intelligence in that sense are oriented to an accurate vascular endothelial growth factor(VEGF)measurement[62, 63, 64, 65, 66], signalling pathway[67, 68]of angeogenic rarefraction description[69, 70], recurrent persistent AF morphological events had con rmed correlation to CMD occurrence modifying local ow condition in strain structure of the left atrial appendage(LAA)[71, 72], beside atrial arrhythmia the supraventricular disorder relationship to brosis replacement modi cation[73, 74]after late gadolinium enhancement(LGE)emerges as a plausible scenario for CMD detection, coronary sinus lling time[75]mark a sign of CMD also in that context[75, 76, 77]supraventricular arrhtythmia control partially relationship between epicardial fat and microvascular dysfunction[78]where CMD may be the consequence rather than the cause of AF[79, 80]extendible to left ventricular dysfunction[81]covered related to microvascular dysfunction obstruction[82, 83]in trials to understand the association-induced myocardial ischemia exercice, relationship of both supraventricular and ventricular arrhythmias in patient without epicardial coronary artery disease leading to channelopathie that may contribute to bradyarrhythmias and conduction abnormalities[74], the analysis of global longitudinal strain response stress echocardiography based CMD as hypothesis[84]were signi cant as response to dipyridamole[85]injecting test left ventricular(LV)contractile function at rest, the supraventricular arrhythmias is discussed frequently after MI with signs of ischemia on the designed clinical studies[86], P-wave surface dispersion and electromechanical delay combination with echocardiographic and clinical parameters regarding CMD development show better results[87, 88, 89].Ventricular arrhythmias had made intersection with CMD in the category of patients with Takotsubu syndrome persuant to infusion of ACh and dobutamine[90, 91]with signs of ischemia recentely magnetocardiography(MCG)features[92]combined with CT fractional ow reserve(CTFFR)diagnosis mark one of the best t techniques to guide CMD dysfunction[93]or discover it in early stage, arti cial intelligence contribution had found place on enhancing the quality of invasive test reading and interpreting as the angiography derived IMR(angioIMR)index[94], of microcirculatory resistance(caIMR) [95]for the assessment of microcirculatory resistance for STEMI patients[94] added to premature of atrial contraction(PACs)[96, 97]arrhythmogenic right ventricular cardiomyopathy[98].CMD and arrhythmias has link to impaired systolic function[99]within strain images analysis[100].Diabetes and cardiometabolic[101, 102]disease are known to result in CMD as progressive heart failure[103]in left ventricular remodeling process[104], recurrent lone AF[105]shown association to impaired atrial ow perfusion may be causal to CMD[106], brotic remodeling of epicardial adipose tissue in patients with AF[107]has even been postulated that HFpEF with identi ed heart rate in obese patients can be attributed to the presence of microvascular dysfunction and altered myocardial relaxation related in di erent degrees to myocardial brosis, dilatation and AF, and a pro-in ammatory state associated with increased EAT. arrhythmia is positioned to relate CMD to HFpEF linked to in ammation described by EAT justi ed by a correlation of EAT and both myocardial lipid content , interstitial brosis leading to in ltrated myocardium[108].CMD detection based arrhythmias relied on electrocardiograms are the ingenuity involved in this type of work lay in de ning a combination of factors capable of identifying microvascular dysfunction based on the patient's context and history.

Table 1: Association between CMD and arrhythmias: summary of studies.

Authors	Category	Hypothesis \Question	Outcomes	Marker
A. Ahmad [109]	CMD	Endothelial independent, not associated to CAD Stenosis ≤ 40% $\Delta CBF \leq 50$ 50 CFR ≤ 2	Paroxysmal atrial brillation(PAF) detection based CMD	PAF
A. Ahmad[110]	Chest pain	Stenosis ≤ 40% Not associated to CAD CFR ≤ 2.5 $\Delta CBF \leq 50$	Silent AF in CMD cohort	Risk of AF
Xiaoye Zhao[111]	CMD patient	Not speci ed		Entropies derived from ECGs and vectorcar-diogram(VCGs)
Xiaoye Zhao[111]		Stenosis ≤ 40 Endothelial-independent		Distortion of ventricular repolarization, QT dispersion AR
Kalyan R.Chitturi[112]	ANOCA patient	CMD and its association with systemic endothelial dysfunction	Ventricular arrhythmias	association with classical(MACE)
Fares Alahdab[113]	Not speci ed	Associated to CAD	Major adverse cardiovascular events (MACEs)	Myocardial blood ow reserve(MFR)
Jaskanwal D. Sara[114]	with chest pain	Non obstructive CAD	Poor correlation to conventional cardiovascular risk factors	Microvascular abnormalities
Michel T. Corban[36]	With chest pain	Stenosis ≤ 40% endothelial dependent	Combined epicardial and microvascular dysfunction	Risk of developing AF
Hansen, Tine Willum [115]	Diabetes patient	General	Increased risk of cardiovascular disease	Quantitative measurement of MBF and MFR
Jaskanwal D. Sara[47]	underwent an invasive assessment of CMD and interpretable pre-procedural ECGs	T wave morphology could express signs of CMD	T wave repolarization parameters constitue a discriminant line between normal and abnormal CMD patient	T wave area in V6,T1 Y-center of gravity in lead II
J Ranjit Arnold [116]	HFpEF	brosis relationship to CMD	lack of correlation between CMD and brosis	not technically speci ed
Franco Cecchi[117]	HCM patient	CMD is associated with increased prevalence of ventricular arrhythmias in LGE	ventricular arrhythmias severe regional microvascular dysfunction	brosis replacement occurrence following microvascular ischemia and focal necrosis.
Abhiram Prasad MBBS, MD, MRCP[118]	HFpEF	coronary endothelial dysfunction is present in patients with asymptomatic left ventricular dysfunction	microvascular endothelial dysfunction is present in asymptomatic left ventricular dysfunction(ALVD)	Left ventricular ejection fraction(LVEF)

Table 1: Association between CMD and arrhythmias: summary of studies.

Authors	Category	Hypothesis \Question	Outcomes	Marker
Cevher Ozcan[119]	HFpEF	CMD is associated with AF and increases susceptibility to the co-existence of AF	Diabetes and angina were more common in patients with abnormal CFR	concomitant AF with HFpEF
Suvi Linna-Kuosmanen[120]	not speci ed	single-nucli RNA sequencing(snRNA-seq) and spatial transcriptomics of the gene expression changes in the human ex vivo right atrial tissue and pericardial uid d determine CMD pathology in ischemic heart disease	in ammatory microvascular dysfunction and changes in the right atrial tissue	Transcriptional patterns changes in atria
Shiko Okabe[121]	suspected CAD patients without obstructive CAD on Cardiac CT angiography(CCTA)	relationship between ECG abnormalities and the prevalence of coronary artery disease CAD	ECG abnormalities were linked to a 30% prevalence of microvascular dysfunction in matched patients without obstructive CAD, as assessed by CCTA.	ECG abnormalities
Jaskanwal D. Sara[122]	Diabetics with chest pain	association between endothelial-dependent and endothelial-independent coronary microvascular dysfunction and glycemic control	Poor glycemic control is associated with coronary microvascular dysfunction	higher glycated hemoglobin HbA1c
Ramazan Atak M.D[123]	chest pain	e ect of slow ow on Qt interval and dispersions	ventricular arrhythmias is subsequent to cardiac death in patients with slow coronary artery ow	presence of cigarette smoking, typical angina, and positive exercise test results
Karahan, MZ[124]	patient with coronary slow ow(CSF)	relationship between all these myocardial repolarization parameters and CSF	heterogeneity of ventricular repolarization in CSF	QTmax duration, QT dispersion, Tp-Te interval, and higher index of cardiac-electrophysiological balance (iCEB) score, wider frontal QRS-T angle
O Quesada , M Pico[125]	patients with suspected ischemia and(INOCA)	patients underwent both coronary functional angiography(CFA) and a non-invasive 36-channel MCG scan	MCG can be used with 94.8% accuracy to identify CMD among patients suspicious for INOCA	age matched controls

Table 1: Association between CMD and arrhythmias: summary of studies.

Authors	Category	Hypothesis \Question	Outcomes	Marker
Ercole Tagliamonte[84]	patients with CMD	speckle tracking echocardiography	global longitudinal strain(GLS) was signi cantly di erent contractile response to dipyridamole infusion, inverse correlation between CFR and ΔGLS, LV systolic dysfunction and lack of LV contractile reserve	value of GLS, lower in patients with CMD LV contractile function
A K Kahle[97]	stable angina and suspected CMD	the relationship between CMD and ventricular arrhythmias	maximum premature ventricular contractions(PVCs)per hour, patients with CMD present with more PVCs than those without CMD	ventricular and supraventricular arrhythmias occurrence based Holter electrocardiograms
Ae-Young Her[126]	chest pain	No underlined hypoyhesis	MCG variables has shown potential in in detection of myocardial ischemia in early stage	ST slope, ST shift, T peak amplitude, ST-T integral, and magnetic eld map(MFM)
Emmanuel I Skalidis[106]	lone recurrent atrial brillation(LRAF)	No underlined hypothesis	rst time isolated atrial myocardial perfusion combined to coronary ow reserve impairment may indicate CMD	lower values of coronary blood ow velocity(APV) h-APV and CFR of the left circum ex coronary artery(LACB)
Mustafa Y lmaz[127]	coronary syndrome X(CSX)	AF occurence in conjunction ventricular arrhythmia (VA)	patients with CSX have increased risks of AF and VA	Higher P wave dispersion(PWD), corrected QT interval dispersion(CQTD),Tp-e interval, corrected QT
Dantong Li[24]	angina	ECG stages and mental stress-induced myocardial ischemia(MSIMI)may identify patients with CMD	ECG stages and mental stress-induced myocardial ischemia(MSIMI), most ECG variables were correlated with both CFR and SDS	degree of correlation of ECG stages to CFR AND SDS to MSIMI)
H Onaka[128]	variant angina and normal coronary arteries	26 episodes of ST segment elevation (19%) were associated with arrhythmias with	Anginal attacks due to sequential and simultaneous multivessel spasm seem to be more dangerous than those involving single-vessel spasm or migratory multivessel spasm	not speci ed

Table 1: Association between CMD and arrhythmias: summary of studies.

Authors	Category	Hypothesis \Question	Outcomes	Marker
Junya Ako[129]	non-cardiac illness	Reversible left ventricular wall motion	Electrocardiography showed deep inverted T waves in precordial leads,and the echocardiography revealed di use akinesis of the apical region in the acute phase , non signi cant stenosis on angiography	Reversible left ventricular dysfunction
Huan Zhang[130]	chest pain underwent SPECT and MCG scans	The ability of MCG to predict impaired myocardial perfusion	MCG variables successfully predicted impaired myocardial perfusion	R and T-peak magnetic eld angle(RTA)
Nynne Dose MD[48]	with angina and NoCAD	CMD tended to be associated with prolonged QTc	ventricular repolarization alterations	T-wave morphology and QTc interval
Ilan Merdler[131]	CMD	suggested exercise-induced changes	/	

Table 1: Summary of studies on CMD, outcomes, and markers.

Table 2: Summury of arti cial intelligence approach based CMD detection

Reference	Data Set	Algorithm	Performance (Acc/AUC)	Marker
Supervised Learning	Machine Learning (ML)	Regression		
Henry Seligman[132]	Images extracted from coronary Doppler ow	Neural Network	/	Doppler Quality
Supervised Learning	Deep Learning (DL)	Regression		
Mingjun Tian[133]	Contrast-enhanced ultrasound	CNN	0.84 /	Microcirculation disorder
Farman Ali[62]	VEGF sequences	Residual convolution neural network(ERCNN)	83.45%	positive instances VEGF
Raed Alsini[63]	VEGF sequences	GRU and 2D CNN	95%	
Unsupervised Learning	Machine Learning (ML)			
Zhe Zhang[134]	Angio-based microvascular resistance images	Logistic Regression (LR)	0.648 / 0.694	Velocity hyp and QFR
		Random Forest (RF)	0.676 / 0.709	
		Extreme Gradient Boosting (XGBoost)	0.810 / 0.866	
		Decision Tree	0.771 / 0.772	
Zhe Zhang[134]	Angio-based microvascular resistance images	Support Vector Machine (SVM)	0.629 / 0.637	Velocityhyp and QFR
		K-Nearest Neighbor (KNN)	0.638 / 0.674	
		Naive Bayes (NB)	0.619 / 0.661	
Unsupervised Learning	Deep Learning (DL)			
Hui Ling Chua[135]	Ultrasonic waves	AlexNet LSTM	0.957 /	Microcirculation changes

Table 2 (continued)

Reference	Data Set	Algorithm	Performance (Acc/AUC)	Marker
Ana Carolina do A. H. Souza[136]	skeletal muscle(SM), subcutaneous adipose tissue(SAT), visceral adipose tissue (VAT) from abdominal CT	Not speci ed	/	Decreased (SM) but not increased SAT or VAT associated with CMD
Alejandro Recio-Mayoral[137]	CSX patients	In ammation in CSX de nition	/	Correlation between CRP levels and CFR, CFR level

2.4.2. Hypertrophy

Hypertrophy[138, 139, 140, 141]de ned as a condition a ecting the left ventricle the main pumping chamber of the heart was strongly depends on stable ischemia and microvascular dysfunction in non-invasive search through di erent scenarios[142].Etiology of hypertrophied ventricular to small vessels dysfunction di er from a category and phenotypes of patient to other discussed frequently on patient with HFpEF[143, 144] with a percent of 70 80% having also CMD sharing hypertension, diabetes, smoking, obesity[145], and chronic in ammatory disorders as risk factors[146]characterising a di erent anatomical and hymodynamic alterations signi cantly expressed as impaired stress of myocardial blood ow and perfusion reserve suggestive of microvascular disease hypertrophic cardiomyopathy , the guidelines were moderately averaged on patients with diabetes as an impairment coronary arteriole vasomotion.According to many cohorts of clinical and cardiologist interventions, hypertrophy wasn't directly linked to microvascular vessels damage in category of patient with chest pain, left ventricular diastolic dysfunction and hypertrophy simultaneously justify CMD existence in certain contexts through echocardiographic parameters[147, 148]contributing to regional myocardial ischemia and metabolic changes[149], CMD concomitant to hypertrophy was explained though subendocardial ischemiaS[150]following dypiridamole injection predisposing ischemia on myocardium [151, 152, 153] stress echocardiograph[2]about contraction added to quantitative perfusion MRI integration[154, 155]with new methods , the control of regional myocardial blood ow during hyperaemia with PET give an early insight about sub-clinical abnormalities of arterioles[156]based perfusion of cardiac magnetic resonance imaging(CMRI)[157]linked to transient micro vessel density(MVD)changes[158, 159]to de ne cardiac structure injuries, e ect on myocardial scar[160]on patient with Anderson Fabry disease(AFD)[161], reduced capillary density extravascular compression during systole e ect on coronaries hymodynamics[162]progressive left ventricular remodeling development to heart failure(HF) smooth muscle cells dysfunction linked with abnormal air ow resistance at rest and after inhalation of methacholine[163], endothelial dysfunction, brosis were all deformation those occur with hypertrophic cardiomyopathy(HCM)mostly supported by abnormal ow arterioles revealed by a coronary ow deviation[164].Hypertrophy related to CMD was discussed also as result of capillary refraction[165]in category of obese patient[166, 167](associated insulin resistance and hypertension)through skeletal muscle performance changes and tissue tone volume dysregulation[168, 169]structural dilated vessel diameter and adverse remodelling of intramural coronary arterioles [170].The study of vessels dynamics as e ect of the pression of extravascular mechanical forces generated by hypertension[171, 172]de ne some ischemic patterns, left ventricular remodelling after and before AMI[173]realize a respectful aspect of CMD discussion in patient with MI after primary coronary angioplasty[174,

175]for physiological measurement of the coronary activity spatial heterogeneity and local MBF[176]though analysing micro vascular density with left ventricular brosis[177]hypertrophy during hyperaemia those patterns were listed as signs indicative structural dysfunction[178]severe microvascular abnormalities and hypercontractile stage of HCM myocyte disarray associated to and an inability to achieve complete relaxation[179].Among the con rmed bidirectional schemas of(CMD ,hypertrophy)exploration are introduced by inadequate blood supply via arti cial intelligence were the phenomena of left ventricular out ow tract obstruction[164]mostely characterised by mitral velocity using doppler imaging via atrial remodelling[179], myocardial scaring[180]description based the premature of ventricular contraction[97]during a testing exercice [181], the sequence of AF resulting in HCM performs regions of partial depolarization and slowed conduction underline a transient and unclear mechanism of suspected CMD[182].within hypertrophy CMD is mostly associated to HFpEF development[183, 184], transient events occurring during that transformation, common risk factors and modi able ones[185], relationship of risk factors to reduced ow reserve.Adjacent AI context formulation within hypertrophy had relied on microcirculatory indexes to predict left ventricular dysfunction in combination with demographic data, echocardiographic parameters[186]after MI, relationship to intramyocardial hemorrhage[187]and ischemia re-perfusion combining infarcts size[188], microvascular obstruction(MVO), coronary angiography-derived fractional ow reserve(caFFR)to product LVD overcoming CMR features related classical LVD de nition, local low shear stress relationship to endothelial microvascular damage surface[189, 190]undergoing plaque rupture based hypertrophy[191]related to cardiac troponin and electrocardiogram changes adopting as hypothesis that CMD reduce shear stress[192, 193]ischemia discussion with hypertrophy were related to recurrent episodes leading to brosis arrhythmic substrate[194], coronaries compression in early phase of diastolic[195], in that context AI precisely insert to estimate correctly CFR[196]based vascular deformation from multi-phase CTA[197], multi-contrast imaging and advanced vascular imaging including 4D ow[198, 199], motion analysis and correction[200, 185]depending on systolic and diastolic function, myocardial ow reserve velocity measurement at rest[201], brosis degree identi cation based MRI[202]phase contrast CT, reduced ow reserve quantitative exploration of CMR[203]characterise the correlation of atherscelorosis phenotype and gradient epicardial fat volume index(EFVI), microcirculation problems resulted from angiogenic response localize hypertrophy as important element that amplify the process heart failure[204] in that sens interaction between cardiomyocytes and non-cardiomyocytes contribution to myocardium development[205]enhanced CMR cine analysis[206]related to aortic stenosis.coronary steal phenomena impact on vasodilatory reserve of microcirculation [207], hypertrophy related lactate production[208].rise as a mechanism that contribute the energy of metabolism regulation in association to hypertrophy regional wall motion[209]detection from doppler echocardiograph[210]measuring coronary ow velocity, chronic coronary syndrome CMD-related ischaemia leading to HFpEF[211] suggest the study of relationship of troponin level and remodelling process, multidimensional(2D or 3D)GLS analysis correlation to syntax score(SS) and micro-infarct damage[212]. Hypertrophy related CMD description scenarios are discussed frequently counting on infraction properties following PCI[213, 143, 214]as the interval in patients with 3 artery cad vessels damage[215] averagely to be prolonged switch context[216], management of infraction under hyperglecimea as an important element of atherosclerosis status transformation[217] doppler tissue imaging with ST-segement elevation enhancing the understanding of LV mi-

crostructure [218].relationship of LV systolic indices to CMD is suggested to be studied within hypertrophy[219] intracoronary physiologic assessment[220, 221] the most AI models built approach target to nd correlation of cardiovascular events to CMD based regression models as logistic regression[171, 222], cox regression[223]linking cardiovascular events to CMD occurence.

the two tables below cite scenarios of cmd detection based hypertrophy as hypothesis or consequence.

Table 3: Summary of studies evaluating links between coronary microvascular dysfunction (CMD) and biomarkers across various patient populations.

Authors	Category	Hypothesis \Question	Outcomes	Marker
Alessandro[224]. Candreva	not speci ed	Intracoronary hemodynamics modeling may aid in the assessment of cellular atherogenesis		Computing ow dynamics(CFD)
James A. Coleman[142]	Hypertrophic car-diomyopathy(HCM)	Genetic cause of CMD	T wave pseudo-normalisation	T wave sensitivity to k^+ accumulation
Aish Sinha [225]	Stable angina	Subendocardial ischaemia, impaired lusitropy	HFpEF	Impaired CFR
Sanjiv J Shah[226]	HFpEF	microvascular dysfunction associated with systemic endothelial dysfunction	Smoking, atrial brillation	reactive hyperaemia index(RHI), urinary albumin-to-creatinine ratio(UACR)
S lvia Aguiar Rosa[227]	adult(HCM) patients without CAD	IMR≤ 22, CFR ≥ 2	Not speci ed	Tissue abnormalities on (CMR)
Paolo G Camici [228]	Left ventricular hypertrophy(LVH)	Di use remodelling of coronary arterioles	Heart Failure with Reduced Ejection Fraction(HFrEF)	Capillary rarefaction, medial wall thickening
A. Ito[229]	Suspected myocardial ischaemia (no epicardial stenosis)	Abnormal CFR& LVH coexistence	Higher risk of MACE, new-onset of HF	not speci ed
Giovanni Montino Pelagi[230]	not speci ed	Precise modeling of blood dynamics adds value to coronary microcirculation diagnostics	microvasculature hemodynamics that characterise CMD	Quanti cation of phasic ow patterns, diameter changes, regional and transmural heterogeneities in myocardial blood ow
Giovanni Montino Pelagi[231]	not speci ed	Predictive modeling of hyperemic coronary and MBF may have sens based time-varying microvascular resistance	regional and transmural heterogeneities	personalized pressure waveforms at the aortic root
Selma F. Mohammed [35]	HFpEF	Characterization of HFpEF with Hypertrophy give an idea about myocardial structural changes	not speci ed	coronary microvascular rarefaction

Table 3: Summary of studies evaluating links between coronary microvascular dysfunction (CMD) and biomarkers across various patient populations.

Authors	Category	Hypothesis \Question	Outcomes	Marker
Adri n I. L er MD[232]	HFpEF	relation between myocardial perfusion reserve (MPR) and di use myocardial brosis in patients with HFpEF induced CMD	reduced MPR	myocardial perfusion reserve (MPR) value
Xu, XL (Xu, Xiaolei)[153]	Patients With Hypertension	Relationship of subendocardial Perfusion to myocardial injury	MFRsubendo was associated with myocardial injury and with greater left ventricular wall thickness and volumes	subendocardial MFR(MFRsubendo).
N.W. Van Der Hoeven[233]	Stable angina pectoris	simultaneous assessment of epicardial- (CEPI) and microvascular conductance (CMICRO), might provide a more information about vascular damage	Median CEPI and CMICRO were 4.56 (IQR 2.18 8.64) and 1.28 (IQR 0.95 1.73) cm/s 1/mmHg 1 respectively	coronary pressure and ow velocity
Dai-Yin Lu MD, PhD [234]	hypertrophic car-diomyopathy(HCM)	Systolic blood pressure(SBP) ≤ 110 mm Hg is associated with greater severity of (CMD) and coronary microvascular ischemia	$SBP \leq 110$ mm Hg is associated with greater severity of CMD and coronary microvascular ischemia and higher incidence of ventricular arrhythmias in HCM.	volume of systolic blood pressure
Vincenzo Sucato[235]	patients with stable angina	Simultaneous assessment of myocardial perfusion and coronary blood ow using validated angiographic indices may help identify patients with CMD	HFpEF population has a greater involvement of microcirculation than patients without HFpEF.	Timi frame count(TFC), myocardial blush grade(MBG), and total myocardial blush score(TMBS) values
Tadashi Murai MD[236]	not speci ed	microvascular resistance a ects FFR after successful percutaneous coronary intervention PCI	Increased microvascular resistance may reduce coronary ow and increase FFR after successful elective PCI	IMR elevation
Maria Tafelmeier[237]	HCM	myocardial brosis and microvascular ischemia may cause a ventricular arrythmia	malignant VA	to be speci ed
SF Mohammed[35]	HFpEF	damage and LV diastolic dysfunction and impairment in cardiac reserve function	similarity in Heart weight, brosis and CMD group	impairment in cardiac reserve function

Table 3: Summary of studies evaluating links between coronary microvascular dysfunction (CMD) and biomarkers across various patient populations.

Authors	Category	Hypothesis \Question	Outcomes	Marker
Viviany R Taqueti[238]	HFpEF	coronary microvascular ischemia play an important role in HFpEF pathophysiology	Impaired CFR was independently associated with diastolic dysfunction and adverse events, The presence of both coronary microvascular and diastolic dysfunction was associated with a markedly increased risk of HFpEF events	troponin level,worse diastolic function
Kathryn Dryer[239]	HFpEF	CMD is common in these patients	the presence of four distinct coronary physiology groups in HFpEF	patients with HFpEF had more abnormalities of coronary ow and resistance
Francesco Pelliccia[194]	HCM	myocardial ischemia is CMD in the absence of epicardial coronary artery abnormalities	brosis, culminating in LV remodeling and HF	not technically speci ed
Ali Ahmad[240]	HFpEF	coronary microvascular function and exercise haemodynamics ,endothelium dependent, CMD was de ned as ≤ 50%	CMD is inversely associated with lling pressures	pulmonary arterial wedge pressure(PAWP)level
Joshua F Lee[241]	HFpEF	patients with HFpEF would demonstrate reduced vascular function HFpEF	reduced vascular function	ow-mediated dilation level
Erika Jones[242]	preserved LVEF, signs of ischemia	elevated resting left CMD related ischemia	lower CFR ventricular end diastolic pressure(LVEDP), CMD-related ischemia may be a precursor to HFpEF	 higher NT-proBNP initially associated with lower CFR
Rina Mauricio MD[243]	MINOCA patient	Abnormal perfusion may co-localize with ischemic LGE and T2weighted signal hyper intensity may capture CMD contributing to MIT^{+2}	Low MPRI, possibly indicating di use microvascular disease	value of myocardial perfusion reserve index(MPRI) co-localized with LGE and/or T^{+2}
Rob Krams, MD, PhD[244]	HCM patient	modi cation of the diastolic coronary could detect changes in the coronary microcirculation	Higher LV out ow tract gradient, septal wall thickness	decreased diastolic coronary vasodilator reserve(DCVR)

Table 3: Summary of studies evaluating links between coronary microvascular dysfunction (CMD) and biomarkers across various patient populations.

Authors	Category	Hypothesis \Question	Outcomes	Marker
Ahmet G l [245]	HCM patient	Left ventricular out ow tract gradient is associated with reduced capillary density	left ventricular out ow tract(LVOT) gradient is associated to reduced	capillary density
Leonarda Galiuto[246]	STEMI	LV hy may a ect microvascular dysfunction and LV remodelling during 6 months	Left ventricular hypertrophy has no e ect development of post acute myocardial infarction LV remodelling and microvascular dysfunction	end-diastolic volume(EDV) and end-diastolic volume(ESV)
Pasquale Paolisso, MD[247]	HFrEF	assess absolute coronary ow, absolute microvascular resistance, myocardial perfusion, coronary ow reserve	higher left ventricular and left anterior descending artery	myocardial mass
Thomas H.Schindler[248]	angina pectoris and dyspnea	yperemic vasodilation with subsequent derivation of MFR a ords the non-invasive detection and delineation of CMD	not reported	not reported
Onciul[249]	cardiac amyloidosis(CA)	coronary microcirculation alterations any setting of LVH	CMD in LVH manifests as a low coronary blood ow reserve during vasodilator stress	not reported
Kenichi Tsujita[250]	LVH, value of hMR	microcirculation evaluation based on hyperemic MV resistance(hMR), the relationship between the degree of CMD and several severity parameters of LVH on echocardiography	impaired MV function might be one of the underlying mechanisms of LV dysfunction, negative correlation between hMR and eGFR	age, worse glomerular ltration rate excretion(eGFR)
Petersen SE[251]	HCM	Microvascular dysfunction may create an ischemic substrate conducive to sudden death	hyperemic myocardial blood ow(hMBF) SCD, CMD and subsequent ischemia may be important components of the risk attributable to HCM	hMBF decreased with increasing end-diastolic wall thickness in the endocardial layer
Paul Knaapen[172]	HCM	the relative contribution of extravascular compressive forces to microvascular dysfunction in HCM	Hyperemic MBF was inversely correlated with LV out ow tract gradient(LVOTG)	hemodynamic LV loading conditions and LV mass

Table 3: Summary of studies evaluating links between coronary microvascular dysfunction (CMD) and biomarkers across various patient populations.

Authors	Category	Hypothesis \Question	Outcomes	Marker
B Schwartzkop [252]	Angina pectoris	structural alterations of the intramyocardial coronary vasculature contribute to an increased minimal coronary resistance	reduced coronary dilatory capacity	external arteriolar diameter,left ventricular mass index, left ventricular end-diastolic pressure
M J Ko ard[164]	HCM, eight heart transplant	haemodynamic, echocardiographic, and histological parameters contribution to decreased CFR	increased septal thickness , indexed LV mass , LV end diastolic pressure , LV out ow tract gradient and a decrease in arteriolar lumen size related to low CFR	LV remodeling and systolic dysfunction
Acopo Olivotto, MD [253]	HCM	LVD de ned as LVEF≤ 50%	CMD is a potent long-term predictor of LV remodeling and systolic dysfunction based on PET	dipyridamole MBF at rest
Seung Hun Lee[220]	stable patients, INOCA	Exploring incremental prognostic value of CMD with intracoronary physiologic assessment on top of non-invasive stress tests(NIT), fractional ow reserve≥ 0.80, CFR≤ 2.5	lower coronary ow reserve and higher hyperemic microvascular resistance	NIT positivity correlation to long term cardiovascular events
Shuxuan Qin, MD[254]	stable patients with HF	left atrioventricular coupling index (LACI) index on echocardiographic may	increased LACI associated to to high degree of cardiac dysfunction may be evident to con rm CMD existence	LACI index
Szekely, Anna[255]	suspected CCS patient elective to invasive coronary angiography(ICA) not technically declared	the mismatch between quantitative rst-pass perfusion(qFPP)CMR and dynamic cardiac, CMD de ned based microvascular resistance	lowest myocardial perfusion where there's a match between qFPP and ICA parameters	mean of myocardial perfusion reserve PET
Wen Zheng[186]	STEMI patients undergoing PCI	MVO and caFFR assessment	LV remodelling	CFD
Francesco Pelliccia[256]	MINOCA	LV systolic dysfunction	recurrent episodes of ischemia	not specied
Aino M nnist [257]	not speci ed	Combined angiogenic and hypertrophic gene	microvascular density impairment	VEGF B activation in endothelial cells
Franco Cecchi[117]	HCM patient	maximal hyperemia	severe myocardial hypoperfusion	underperfused myo cardial area

Table 3: Summary of studies evaluating links between coronary microvascular dysfunction (CMD) and biomarkers across various patient populations.

Authors	Category	Hypothesis \Question	Outcomes	Marker
Mauro Echavarria-Pinto, MD[258]	patient with normal CFR, vessels with FFR≤ 0.80 and normal(CFR)	combination of di use atherosclerotic narrowings and CMD	wide dispersion of IMR, the lowest IMR with	focal and di use coronary narrowings are associated to degrees of CMD

Table 4: Summary of Arti cial Intelligence Approaches for Cardiac Data Analysis

Reference	Data Set	Algorithm	Performance (Acc/AUC)	Marker
Supervised Learning	Machine Learning (ML)	Regression		
Henry Seligman[132]	Images extracted from coronary Doppler ow	Neural Network	/	Doppler Quality
M J Ko ard[164]	haemodynamic(LV end-diastolic pressure, LV out ow tract gradient), echocardiographic, and histological(luminal area of the arterioles) parameters	logistic regression		The relationship between those variables and decreased CFR values
Supervised Learning	Deep Learning (DL)	Classi cation		
Kenichi Tsujita[250]	interventricular septal thickness(IVST), left ventricular posterior wall thickness(LVPW)	logistic regression		LV mass, increased hMR
Supervised Learning	Deep Learning (DL)	Regression		
Mingjun Tian[133]	Contrast-enhanced ultrasound	CNN	0.84 /	Microcirculation disorder
Unsupervised Learning	Machine Learning (ML)			
Zhe Zhang[134]	Angio-based microvascular resistance images	Logistic Regression (LR)	0.648 / 0.694	Velocityhyp and QFR
		Random Forest (RF)	0.676 / 0.709	
		Extreme Gradient Boosting (XGBoost)	0.810 / 0.866	
		Decision Tree	0.771 / 0.772	
Zhe Zhang[134]	Angio-based microvascular resistance images	Support Vector Machine (SVM)	0.629 / 0.637	Velocityhyp and QFR
		K-Nearest Neighbor (KNN)	0.638 / 0.674	
		Naive Bayes (NB)	0.619 / 0.661	
Wen Zheng[186]	clinical and procedural	RF	0.77	
Wen Zheng[186]	clinical and procedural, CMR parameters	RF	0.84	
Wen Zheng[186]	clinical and procedural, CMR, echocardiogram plus (caFFR and caIMR)	RF	0.85	
Unsupervised Learning	Deep Learning (DL)			
Hui Ling Chua[135]	Ultrasonic waves	AlexNet LSTM	0.957 /	Microcirculation changes

Table 4 (continued)

Reference	Data Set	Algorithm	Performance (Acc/AUC)	Marker
Ana Carolina do A. H. Souza[136]	SM, SAT, VAT from abdominal CT	Not speci ed	/	Decreased (SM) but not increased SAT or VAT associated with CMD
Alejandro Recio-Mayoral[137]	CSX patients	In ammation in CSX de nition	/	correlation between CRP levels and CFR; CFR level

2.4.3. in ammation

In ammation as a response of coronary arteries were mostly related to category of patient with or undergoing revacularization operation explained clinically with endothelial cell injury pathways.In the context of invasively testing techniques to evaluate coronaries reactivity the response to adenosine and acetylcholine(Ach) are the classical strategies adopted, the most in ammation scenarios those represent a projection of non-invasive testing methods and were related to microvascular dysfunction had relied on positron emission tomography(PET)exploration, cardiac magnetic resonance imaging(CMR), and transthoracic doppler echocardiography(TTDE)[259, 260]pointing on atheroscelosis development as an entry point.Generally recent hypothesis those discuss CMD in the absence of obstructive CAD induced in ammation were de ned by a decrease in(CFR) as indirect indication[238]of CMD development under receptors over-activation.Exploration of how coronary ow reserve(CFR)is linked to the historical and symptomatic characteristics of ischemia show up as a ubiquitous prototype or baseline that orient the therapeutic guidelines and adjust the percent of micro circulation mechanisms understanding[261, 262]dysregulated angiogenesis[263].The principal elaborated studies within CMD and microvascular dysfunction dimension put as attended object one of those axes the analysing the impact of CMD on the endothelial function[264](TNF reproduction in relationship too the levels of glucose and cystatin C -α, IL-6, CRP, initric the production of leukostasis[265], increased permeability and leukocyte in ltration, pro-coagulant and pro-thrombotic state via the status of vasodilation vascular tone veri cation, and pro-thrombotic markers, metabolic defects modi cation,oxidative stress control of in ux in ammatory cells[266]where a regulate a connexion with cytokines and chemokines are the subject to characterize patient with MI and patient with heart failure with structural remodeling[267], the immune cell activation.for the rst line it a ects the small vessels across a reduced vasodilator response[268], increased vasoconstriction(endothelin-1[269]),CCL5), impaired shear stress adaptation promotion of microvascular spasm[13], epicardial fat volume deformation expressed the vasoreactivity of the coronary microcirculation[270]the second had made intersection with in ammation[271]as altered function of skeletal muscles[272]depending on a spatial-temporal communication of intracellular myocardium signalling leading two atherosclerosis plaque destabilization[273], cardiac perivascular brosis[274, 275], oxidative stress share aspect with hyperglycemia under CMD[276]such as activation of protein kinase C, polyol and hexosamine, advanced glycation end products production in type 2 diabetes mellitus patients[276, 266], the third were coupled to hypertension as risk factor induced CMD explored in the sense of vascular resistance determination that re ects the hymodynamics load and the last giving rise to platelets activation[277]associated to no-re ow phenomena occurrence[278, 279], autonomic cardiac function disorder for patient

undergoing PCI and coronary slow ow[280], the correlation between the corrected thrombolysis in myocardial infarction(TIMI) frame count(CTFC) and the index of microcirculatory resistance(IMR)[281], cellular reactive oxygen species(ROS)overproduction and accumulation via the epigenetic mechanisms.Tissue damage understanding within via perivascular adipose[282]relationship to left atrial and ventricular function parameters[283]exert signi cant e ect on cardiac remodelling and dysfunction[284]through functional sharing of a spatial micro vascular view[285]relating thrombosis status to micro circulation pathology, Crosstalk between the macrocirculation and microcirculation [286, 287]is mediated by paracrine signaling[286, 273]to the vascular wall, which regulates renal function via apelin binding to its G-protein coupled receptor(APLNR)[288, 289, 290].Furthermore blood ow dynamics exhibited a distinctive pattern characterize the examination of red blood cells(RBC)[291]aggregation behaviour within the capillary microcirculation and intravascular activated mononuclear cells [292, 293, 293]a ecting the endothelial function associated to an heteregeneous blood ow resistance. CMD within platelets are related vascular endothelial cells activation[277]reacting as e ector and target cells garanting in ammation[277], In sense of early stage of myocardial ischemia based CMD signs as pre-arterioles, arterioles and capillaries concentration change of metabolites analysed based on coronary ow reserve velocity[294, 250], the development of diastolic dysfunction depending on the extent of myocardial in ammation is examined alongside with spasm alteration stages via structural remodelling of the arterioles, left ventricular geometry parameters integrity and function during end systolic and diastolic coronary ow[295].AI algorithms had tried to approach CMD assessment based the measurement of biomarkers variation[296, 297], cardiac remodelling, the thickness of arteriole walls specifying the mechanism of the metabolism e ect on degree of microvascular ow integration[298]through intravascular imaging, CMD screening based local in ammation were more e cient[299]related to left ventricular end-systolic volume(LVMI)measurement correlated to high pericoronary adipose tissue attenuation(PCATA)[300, 301]on echocardiographic parameters[299]caused by long-standing chronic in mmation with diabetes.adipose tissue(AT) accumulation may reduce regional LV sys tolic function through cytokine-mediated coronary microvascular dysfunction to result in LV sub-endocardial perfusion[302], adipose tissue volume on CMR related to left ventricular dysfunction[303]quantify MBF thanks to its high temporal and spatial resolution[304, 305]. The ultra structural integrity of microvessels were studied within plaque morphology of atherosclerosis[291] exprinming an incomplete endothelial junction explaining microvascular leakage[291], diastolic function association to CMD of left ventricular in patient at the end stage of chronic kideney disease[306]measured by left ventricular lling pressure(LVFP) [307] in that context applications of AI for CMD description had focus on epecardial fat volume index on CTA with or without angiogram test[308]switch the history of the coronaries disease[309, 310], the relationship between that latter to CMD in unobstructed vessels is investigated[311], the association between(EAT)thickness and silent myocardial ischemia[312, 313, 314], microvessels complications based on combined use of pet perfusion and CCTA[315, 316], endothelial cell dysfunction related to in ammation had been covered via extracellular matrix(Ecs)sti ness[317]parameters understanding trying to imitate the biomechanics mechanisms, perivascular fat improve radiotranscriptomic signature capturing via microvascular PVAT remodelling[318], CMR-based assessment of pericardial adipose tissue in patients with ischemia and INOCA supports mechanisms of disrupted cellular integrity, including endothelial apoptosis, degradation of protective brous structures,

and enhanced platelet endothelial interactions[319], conversely some studies test the usefulness of epecardial fat density obtained from CT imaging as element overcoming the volume for intermediate lesions supporting the process of dynamic microvascular remodelling and complexity of microvascular network generation[320, 321], local interaction of pericoronary adipose tissue(PCAT)with the a ected blood vessel.in that sense microvascular angina correlation to acute ischemia nd place to be discussed based[322].

the two tables below resemue di erent contexts where CMD were ainsi the illustrated AI designed based on trials proposal or cardiologist context de nitions

Table 5: Association between CMD and in ammation: summary of studies.

Authors	Category	Hypothesis \Question	Outcomes	Marker
Eva Prescott MD, DMSc [323]	Angina & (NoCAD)	(CVFR) correlation to classical protein biomarkers	Sixty-one biomarkers were correlated with (CFVR)	Interleukin 6
Waiel Abusnina[324]	General population	In ammation may contribute to CMD	Epicardial fat volume (EFV), a signi cant risk factor, is associated with (CMD)	(EFV) index
Tetsuya Yamamoto[325]	chronic coronary syndrome	periprocedural myocardial injury(PMI) detection based microcirculatory dysfunction	microcirculatory dysfunction is important in PMI development, particularly in the presence of lesions with high PCAT attenuation	not speci ed
B nd icte Gaborit[270]	Healthy persons	epicradial fat (EF) could early in uence endothelial function, CMD de nition based MBF by measuring coronary sinus ow with velocity	EF is associated with a lower response in coronary endothelium-dependent vasoreactivity in healthy subjects	A high(EF) amount is associated with a lower coronary microvascular response
Erdo an Ya ar[326]	CSX with normal coronary arteries	Systemic immune-in ammation (SII) is related to microvascular dysfunction	SII levels were higher in CSX patients	High SII
Hong-Yang[327]	Ischemia with No Obstructive Coronary Arteries	AISI is related to the slow coronary ow phenomenon (SCFP)	Higher AISI scores are associated with increased SCFP risk	SCFP
Yan Lin .	Myocardial ischemia patients	CMD is linked to elevated D-dimer levels	Patients with CMD had older age and higher D-dimer levels	D-dimer elevation
Constantijn Franssen[328]	HFpEF	Low-grade in ammation contributes to diastolic dysfunction	HFpEF is linked to endothelial activation and oxidative stress	Endothelial cardiomyocyte signaling, NO reduction
Marcelo F. Di Carli[103, 329]	Diabetes patients	The role of hyperglycemia in CMD	Reduced and similar endothelium-dependent and -independent coronary vasodilator function in subjects with both type 1 and type 2 DM	Not speci ed

Table 5: Association between CMD and in ammation: summary of studies.

Authors	Category	Hypothesis \Question	Outcomes	Marker
Martin-Wortham[293]	Microcirculation	Red blood cell(RBC) aggregation a ects microvascular dysfunction	Altered aggregation may impair perfusion	rate of RBC aggregation
Hugo Rodriguez-Zanella[330]	INOCA	altered cardiac mechanics using two-dimensional speckle-tracking echocardiogra-phy(2DSTE) during SE	Coronary microvascular dysfunction is accompanied by an impairment of global and layer-speci c deformation indices during stress	lower global longitudinal at SE peak strain
Yousef Rasmi[331]	Patients with (CSX)	Higher plasma(IL-6) and (TNF-α) level	cytotoxin-associated gene A positive(CagA+)	The (CagA+) strain H. pylori, can not only be a trigger, of H. pylori, can not only be a trigger,and may also have a role via chronic in ammation in the pathogenesis of CSX
Zhuoya Yao[332]	(AMI) patients underwent PCI	The diagnosis of (CMD) is based on ndings from cardiac magnetic resonance (CMR)	sex, neutrophil to lymphocyte ratio (NLR), Gensini score, and diabetes mellitus were identi ed as independent predictors for the development of CMD	CMR analysis relationship to risk factors
Louise E.J. Thomson, MBChB[333]	asymptomatic patient susceptible to have CMD	relationship between coronary reactivity testing(CRT) ndings and MPRI may determine CMD prevalence	An MPRI threshold of 1.84 predicted CRT abnormality with sensitivity 73% and speci city 74%.	MPRI
Yang Yang[334]	patients with type 2 diabetes mellitus (T2DM) nd heart failure	assessing the relationship between perfusion and brosis under e ect of CMD	MPR was associated with markers of myocardial injury and brosis and was predictive of adverse clinical outcomes	Myocardial perfusion reserve
Bernt J. von Scholten[335]	Diabetes 2 patients	the prevalence of reduced CFR and high CAC scores	type 2 diabetic patients who were free of overt cardiovascular disease had a high prevalence of coronary microvascular dysfunction	albuminuria quantity
Jeong Hoon Yang[336]	HFpEF	Coronary microvascular in ammation is hypothesized to play a fundamental role in the pathophysiology of heart failure with preserved ejection fraction (HFpEF)	CMD is common in patients with HFpEF and is caused equally by endothelium-dependent and independent mechanisms	worse diastolic dysfunction and outcomes.

Table 5: Association between CMD and in ammation: summary of studies.

Authors	Category	Hypothesis \Question	Outcomes	Marker
Carr, Elizabeth[337]	not speci ed	Thermal sensing for direct intravascular ow measurements	Optical time-of- ight measurements of coronary ow may represent a promising advancement for assessing both blood ow	temperature level
Zhao, Yanglu.[338]	diabetes mellitus	Hypertriglyceridemia as common condition to microvascular complication	Not clearly de ned in technical terms.	increase of oxidative stress
Xiaoqi Cai MD[339]	hypertensive patients	Relationship between lactate dehydrogenase and albuminuria	glomerular endothelial damage	microalbuminuria
Margot Tragin[340]	Juvenile Dermatomyositis (JDM)	microvascular dysfunction and I Interferons(IFN-I) I upregulation	still under investigation	not speci ed
Osvaldo Masoli[341]	INOCA patients	Cadmium-Zinc-Telluride Single Photon Emission Computed Tomography(CZT-SPECT) may improve for the diagnosis of	The use of CZT-SPECT with both stress tests allowed the evaluation of di erent possible pathophysiological mechanisms of (CMD)	Dynamic myocardial perfusion
Jinxuan Zhao[342]	STEMI patients	correlation between EAT accumulation and MVO formation ischemia	excessive accumulation of EAT promoted MVO formation by promoting the polarization state of cardiac macrophages towards an in ammatory phenotype	EAT mass index associated to increased levels of Dipeptidyl peptidase-4 (DPP4) and high (DPP4)
Gerd Heusch[343]	angina and the absence of major epicardial coronary obstruction	the atherothrombotic debris is washed into the coronary microcirculation	Coronary microembolization contributes to microvascular obstruction after reperfused acute myocardial infarction	contractile function in the microembolized coronary artery perfusion status
Judith C Sluimer[291]	not speci ed	Microvessels in atherosclerotic plaques are an entry point for in ammatory and red blood cells	structural integrity of microvascular endothelium may explain the leakage responsible for intraplaque hemorrhage in advanced human coronary atherosclerosis	MVD
Zhihao Chen[344]	MI patients	the MI can lead to persistent microvascular obstruction (PMO)	persistant microvascular dysfunction	volume of normal and pathological tissues

Table 5: Association between CMD and in ammation: summary of studies.

Authors	Category	Hypothesis \Question	Outcomes	Marker
Denise Cristiana Faro[345]	Anderson-Fabry disease(AFD)	The accumulation of globotriaosylce-ramide(Gb3) in arterial walls triggers upregulation of adhesion molecules	vascular complication	globotriaosylceramide(GB3) accumulation in arterial walls
AhmeD Tas[346]	with chest pain and unobstructed coronary arteries	Blunted microvascular resistance reserve(MRR)(≤ 3.0)	no relationship found between echocardiographic indices of left ventricular diastolic function and coronary microvascular function at rest	intrabeat pulsatility characteristics, septal mitral annular e velocity
F. Tona[347]	obese patient patients	coronary microvascular dysfunction as an early marker of atherosclerosis	CFR is often reduced in obeses	coronary multislice computed tomography
Junzhen Zhan[348]	patients with angina	link between stenosis of epicardial coronary artery and myocardial ischemia	CMD	MCE derived MPR
Jakob Schrode[349]	angina pectoris	associations between cardiovascular protein biomarkers and non endothelium dependent CMD	Correlation MBFR Gal4, GDF15, tPA and vWF to of	Gal4, GDF15, tPA and vWF.
Carlos Kaski, MD, DSc[3]	stable angina	prevalence of epicardial and microvascular coronary spasm in patients with anginal symptoms	abnormal coronary vasomotion plays a pathogenic role to identify patients with cardiac symptoms	The ACH test results
Ihab Mahmoud[350]	HFpEF	picardial fat modulation of local in ammation	EAT thickness is independently associated with CMD and can di erentiate between patients with and without CMD especially in older age groups	EAT index
Ana Carolina do A H Souza [351]	normal perfusion and preserved left ventricular ejection fraction	The relationship between SM quality, CMD	Increased intermuscular fat is associated with CMD	intermuscular adipose tissue value
Mohammed S. Alam MBBS[352, 353]	NoCAD	the association between EAT with coronary microvascular dysfunction	Increased EAT appears to be associated with impaired MFR	Increased EAT appears to be associated with impaired MFR

Table 5: Association between CMD and in ammation: summary of studies.

Authors	Category	Hypothesis \Question	Outcomes	Marker
Ilan Merdler[131]	non-obstructive coronary arteries	relationship between CMD using common in ammatory markers derived from complete blood count(CBC) analysis.	worsen endothelial dysfunction	neutrophil-to-lymphocyte ratio (NLR), eosinophil-to-monocyte ratio (EMR) and monocyte-to-high-density lipoprotein ratio(MHR)
Nikos Papamichail MD[354]	hemodialysis patients at end-stage Chronic kidney disease (CKD)	the associations of CFR with echocardiographic indices of systolic and diastolic cardiac function	impaired coronary vascular function	posterior wall thickness and dipyridamole-induced changes in Tei index
Mihaela Mocan[355]	chronic kidney disease(CKD)	myocardial microvascular dysfunction based left ventricular diastolic dysfunction(LVDD)	metabolic and systemic abnormalities in circulating factor	activation of (CRP, TNFα, IL 6, sST2, and pentraxin 3)
H E Suhrs[356]	angina but no ow limiting coronary artery stenosis	the e ect of in ammation on diastolic function is partly mediated by CMD	poorest diastolic	Correlation of each biomarker with CFVR and E/e'
P Rezaeian[357]	HFpEF	Arterial sti ness assessment in coronary microvascular dysfunction	greater arterial sti ness and lower myocardial perfusion reserve, with lower LVEF	Higher aortic pulse-wave velocity(aPWV)
Mengyu Chen[358]	patients with suspected coronary artery disease	the relationship between the attenuation of peri-coronary adipose tissue (PCAT) and the assessment of coronary vascular function	FAI was higher in the presence of reduced CFR, FAI can help reveal microcirculatory damage in patients who do not exhibit epicardial artery stenosis	FAI was higher
P Rezaeian[357]	HFpEF	Arterial sti ness assessment in coronary microvascular dysfunction	greater arterial sti ness and lower myocardial perfusion reserve, with lower LVEF	Higher aortic pulse-wave velocity(aPWV)
Hugo Rodriguez-Zanella[359]	Ischemia with INOCA	CMD is accompanied by an impairment of global and layer-speci c deformation indices during stress	similar 2DSTE values at rest. At peak SE, lower global longitudinal strain, higher mechanical dispersion, lower endocardial and epicardial layer speci c strain	GLS value
Jia Ling Diau[360]	MINOCA	study based localized changes in the attenuation values of PCAT and EAT	Assessing PCAT and EAT in ammation via CCTA and AI-driven risk algorithms enable precise risk prediction of MINOCA	not speci ed

Table 6: Summary of Arti cial Intelligence Approaches for Cardiac Data Analysis

John B. Gavin[361]	Dilated cardiomyopa-thy(DCM)	relationship between LGE and CMD	CMD evaluated using angio-IMR was associated with LGE	Angio-IMR was signi cantly higher in patients with LGE
Karen Y. Stoke[362]	not speci ed	the capacity for cross-talk between platelets and other cells (endothelial cells, leucocytes)contribute to an in ammatory response within the microvasculature	generation of PAF and other arachidonic acid metabolites , the generation of adenosine diphosphate	the degranulation of dense granules
Daniel Faria MD[363]	stable angina	the prevalence of BEW intensity and DMVC below the 25th percentile increased with age (25.0% vs. 52.0% vs. 72.7 %)	Ageing is independently associated with structural microcirculatory remodeling that is re ected in backward expansion wave(BEW) intensity andcalculated diastolic microvascular conductance(DMVC) measurements, and with an increased prevalence of structural CMD	not speci ed
Haseeb Rahman, BMBCh, PhD[364]	angina and NoCAD	CMD de ned based coronary ow reserve ≤ 2.5 and controls if coronary ow reserve ≥ 2.5	diminished CFR induced ischemia , 2 endotype identi ed based of vascular system response to exercise	lower MR associated to functional CMD, higher SPB associated to structural CMD
WJ Paulus[365]	HFpEF	progression of diastolic dysfunction	arterial hypertension (HTN)	interstitial brosis and cardiac hypertrophy

Reference	Data Set	Algorithm	Performance (Acc/AUC)	Marker
Supervised Learning	Machine Learning (ML)	Classi cation		
Louise E.J. Thomson, MBChB. B n dicte Gaborit[270]	Vasodilator stress cardiac MRI MRI images	Logistic Regression Multiple Logistic Regression	not speci ed / not speci ed not speci ed / not speci ed	Myocardial perfusion reserve index (MPRI) High EF associated with lower coronary microvascular response
Zhihao Chen[344]	Delayed Enhancement MRI(DE-MRI)		/	Volume of normal and pathological tissues
Supervised Learning	Deep Learning (DL)	Classi cation		
Supervised Learning	Deep Learning (DL)	Regression		
Carr, Elizabeth[337]	Not speci ed	Thermal sensing for direct intravascular ow measurements	/	

Table 6: Summary of Arti cial Intelligence Approaches for Cardiac Data Analysis

2.5. Generalized contexts

Authors	Category	Hypothesis \Question	Outcomes	Marker
Floor Groepenho [366]	not speci ed	CMD may evaluated based on sex	impaired myocardial perfusion within women's	myocardial perfusion fractional ow reserve index of microcirculatory resistance coronary ow reserve(CFR)
Joo Myung Lee[367]	MI patient	Intermediate coronary stenosis	patient-oriented composite outcome(POCO) of any death or revascularization	CFR and IMR correlation
Ziyu Zhou[222]	acute myocardial infarction (AMI)patients undergoing PCI	early prediction of microvascular obstruction prior to percutaneous coronary intervention	MVO appeared as a depicted in black within the infarct core(depicted in white with high signal intensity on LGE images on CMR	non-signal or Troponin T Fibrinogen and LVEF Neutrophil Count,Creatine Kinase-MB
Halvorsr d[368]	NSTEMI	value of the echocardiograph echocardiograph could precise the capability of strain-based measurements, particularly post systolic shortening, to identify viable myocardium	myocardial deformation measurements, such as strain by speckle tracking echocardiography, can identify both acute coronary occlusion and viable myocardium.	mechanical wave velocities
Masanao Naya[369]	symptomatic patients with normal myocardial perfusion imaging(MPI)	the interrelation of atherosclerotic ton vascular function	global CFR but not CAC provides signi cant incremental risk strati cation over clinical risk score	CAC score

Authors	Category	Hypothesis \Question	Outcomes	Marker
Jucheng Zhang[370]	patients with suspicion of CAD and normal epicardial lesions	myocardial contrast echocardiogra- phy(MCE) may be used to diagnose CMD	CMD was present in 23(28%) patients	MBF
Gaetano A. Lanza, MD [371]	Patients with cardiac syndrome X	StressInduced Myocardial Perfusion Defects on CMR has a relationship to CMD	CFR to adenosine signi cantly correlated with a CMR perfusion defect score	CMR perfusion defect score based response to adenosine
Scott M. Gaignard[372]	suspected to have myocardial ischemia	Epicardial coronary vascular dysfunction and coronary microvascular dysfunction (CMD) are prevalent in a majority of INOCA patients	epicardial coronary vascular dysfunction and coronary microvascular dysfunction (CMD) are prevalent in a majority of INOCA patients	not speci ed
Shigeo Godo[373]	chest pain	coronary microvascular endothelial dysfunction (CMED) is associated with epicardial coronary atherosclerosis	CMED was associated with vulnerable plaque characteristics in patients with non-obstructive CAD	not technically speci ed
Shigeo Godo[16]	INOCA	Coronary microvascular spasm is one of the key mechanisms responsible for myocardial ischaemia	not founded	not founded
Steven E Reis[374]	chest pain and no obstructive coronary disease	Chest pain with No CAD is common in women and may be caused by caused by coronary microvascular	Age and number of years past menopause correlated with ow velocity reserve	ow velocity reserve
Nico H.J. Pijls[375]	patients scheduled for PCI of an intermediate stenosis	PCI utility in intermediate stenosis $FFR \geq 0.75$	PCI of an intermediate coronary stenosis based on FFR ≥ 0.75 is excellent	not technically speci ed
Heinrich Wieneke[376]	typical angina	Endothelial dysfunction might be the reason for the observed perfusion defects regional endothelial dysfunction may cause hypoperfusion in myocardial perfusion inhomogeneous perfusion pattern is caused by microvascular dysfunction.	regional myocardial hypoperfusion interpreting pathological scintigraphic results	slow- ow phenomenon re ecting decreased resting ow velocity

Authors	Category	Hypothesis \Question	Outcomes	Marker
Chauhan [377]	atypical chest pain, negative exercise test, and normal coronary	endothelium-dependent and endothelium-independent vasodilatation in syndrome X	both endothelium-dependent and endothelium-independent dilatation of the coronary microvasculature is impaired in syndrome X	coronary blood ow level
Linlin Sun [378]	microvascular angina	hemodynamic association between epicardial coronary arteries and myocardium	The abnormally decreased V/M value may serve as a potential biomarker of Primary microvascular angina(PMVA)	myocardial mass and lower average
Daria Frestad Bechsgaard[379]	angina	women with angina, CMD and no obstructive CAD have reduced exercise capacity compared with asymptomatic sex-matched controls	reduced exercise capacity , impaired heart rate response and heart rate recovery	cardiopulmonary exercise testing(CPET)-derived variables
Wang Qian[101]	Diabete type 2(T2DM) and (CMD)	uctuations in fasting blood glucose(FBG) levels on left ventricular function	the association between FBG-CV levels and the E/e' ratio	BG-CV levels and the E/e' ratio
Chen-Yan Min, Yue Gao[380]	Metabolic syndrome	myocardial energetic e ciency impairment on left ventricular function	microvascular dysfunction	signi cant increase in the proportion of HTN, obesity and T2DM
Julien La Mela[381]	non-obstructed coronary arteries	The relationship between CMD and left atrial strain(LASr)	reduced LASr compared	LASr value.
Beltrame, JF[382]	chest pain with ST elevation	spontaneous ST elevation in patients with angiographic normal coronary arteries and implicated microvascular dysfunction as the cause of the transmural ischemia	microvascular constriction may occur based ST-segement elevation	biphasic T wave after restoration of ST-segment. episodes persistence duration,episodes persistence duration
Rebecca K. Hughes, MBBS[383]	patients with ApHCM,	apical perfusion defects would be common in Apical hypertrophic cardiomyopa-thy(ApHCM)	ECG changes and apical ischemia could lead to e ective therapies, targeting either microvascular disease or contraction	combination of tall ECG R waves with ischemic-looking deep T-wave inversion and Apical perfusion
Tien Vuong Tran[384]	patients with a con rmed diagnosis of HCM	CMD could detect HCM patients with more impaired cardiac function by LACI	CMD is applicable to HCM patients and is associated with more impaired cardiac function	left LACI value

Authors	Category	Hypothesis \Question	Outcomes	Marker
Giampaolo Niccoli[385]	angina pectoris	MVO is caused by a combination of 4 pathogenic components: ischemic injury, reperfusion injury, distal embolization, and individual susceptibility to reperfusion injury	coronary microvascular obstruction	not speci ed
Teresa Salvatore[386]	Diabetes	altered vasomotion and long-term structural change to coronary arterioles	impaired regulation of blood ow in response to changing cardiomyocyte oxygen requirements	antidiabetic drugs e ect on the myocardial microvascular compartment
Jianjun Wu[387]	diabetes	relationship altered fatty acid metabolism and microcirculatory impairments	elevated levels of saturated fatty acids(SFA) lipid peroxidation products (LPPs) fatty acid peroxidation products (FAPPs)	fatty acid peroxidation products(Fapps) generation and 4-HNE's e ects
Bojer[388]	diabetes patients	changes with microvascular dysfunction and interstitial brosis	3D GLS could be a potential screening tool for myocardial microvascular dysfunction	GLS
Olga Hudlicka[389]	chronic heart failure(CHF) patients	capillary supply and change in the microcirculation during contraction	increased skele tal muscle fatigue	capillary velocity of red blood cell (Vrbc)
Stanislav Dil[390]	patients with refractory no-re ow post-PCI	MVO and increased left ventricular end-diastolic pressure (LVEDP) are implicated in the pathogenesis,	LVEDP was signi cantly correlated with indexed MVO	global relative ow increase(gRFI)
Jeng Hwan Lam MBBS[391]	chest pain and unobstructed coronary arteries	association between coronary microvascular function and echocardiographic indices of left ventricular diastolic function at rest	no relationship found between echocardiographic indices of left ventricular diastolic function and coronary microvascular function at rest	Indices of diastolic function, septal mitral annular e', septal mitral annular E/e'
Beltrame[7]	cardiac syndrome X	myocardial ischaemia secondary to coronary microvascular dysfunction	exertional angina, electrocardiographic evidence of ischaemia on stress	not speci ed
Muhammet Salih Ate [392]	INOCA	Ischemia with non-obstructive coronary arteries(INOCA) represents is often related to coronary microvascular dysfunction(CMD)	P wave peak time in leads DII and V1 was signi cantly longer	Electrocardiographic P Wave Peak Time

Authors	Category	Hypothesis \Question	Outcomes	Marker
Philipp A Kaufmann, MD[393]	asymptomatic subjects with hypercholesterolemia	the impact of total cholesterol(TC) and its subfractions on coronary ow reserve (CFR)	low density lipoprotein(LDL) cholesterol levels, high density lipoprotein (HDL)cholesterol levels and triglyceride levels a signi cant but weak correlation was found between CFR and HDL a signi cant inverse correlation between LDL and CFR	total cholesterol
Yarong Yu[394]	diabetic patients	microvascular myocardial ischemia relationship to angina , CMD de ned MBF ≤ of100 mL/min/100 mL	Dynamic CT MPI + CCTA revealed a high incidence of microvascular myocardial ischemia, microvascular myocardial ischemia is strongly associated with angina	extracellular volume
Masahiro Hoshino[395]	intermediate stenosis in the left anterior descending artery (LAD) who underwent CCTA and invasive physiological mesearement	CMD was de ned by $IMR \geq 25$	the prevalence of CMD was about one-third in patients with intermediate stenosis in LAD regardless of the presence or absence of functional stenosis signi cance	greater lumen volume
Christopher J. Lockhart[396]	uncomplicated Type 1 diabetes mellitus	in uence on shear stress and microvascular reactivity Flow mediated dialation(FMD) response in relation to evoked Dioastolic shear stress(DSS)	changes in doppler ow velocity waveforms change in distal microcirculatory haemodynamics	ow-mediated dilatation(FMD)
Maria C. Ziadi, MD[397]	patients assesed for ischemia	the prognostic value of MFR based rubidium-82 PET	di erent values of (SSS) between groups de ned MFR ≥ 2 and those with MFR≤ 2	summed stress score(SSS) value
Benedetta Tomberli[398]	AFD	investigate the presence of CMD by positron emission tomography(PET) in AFD patients of both genders, with and without evidence of LVH	CMD may represent the only sign of cardiac involvement in AFD patients	regional heterogeneity of MBF, prevalent hypopkalierfusion of the apical region
Shah, JS [399]	AFD	enzyme replacement therapy(ERT) e ect on microvascular development	abnormal coronary microvascular function	no relationship of ERT to CMD)

Authors	Category	Hypothesis \Question	Outcomes	Marker
Ioannis Skalidis, MD, PhD[400]	ANOCA	CMD de ned based abnormal CFR, Frame count measurement *leq*2 ,the possibility to asses CMD based TIMI frame count at hyperemia(TFCchy)	signi cant timi frame cout(TFC) at rest	TFCchy
Naja Dam Mygind[401]	women with angina and NoCAD	the association between CMD and CMR rst-pass perfusion	moderate correlation of CMR myocardial perfusion reserve from contrast upslope($CMR_MPRupslope$) to myocardial blood ow reserve by positron emission tomogra- phy(PETMBFR)	($CMR_MPRupslope$) association to PETMBFR may be a measure of CMD
Tess E. Allan MD a[402]	myocardial bridge(MB)and chest pain	CMD was de ned as $CFR \leq 2.0$ or $CFR \leq 25$, CMD prevalence and epicardial spasm	Epicardial spasm, microvascular spasm, and endothelium-independent CMD are prevalent in patients presenting with known MB and chest pain independently to hemodynamic conditions	not speci ed
Ahmed AlBadri[204]	patients with signs of ischemic and normal angiography FFR≥ 0.8	mechanistic relationship between epicardial atherosclerosis and CMD	higher minimum, median, and maximum plaque burden(PB), a higher percentage of IVUS, similar FFR	plaque burden value
Martijn W. Smulders[188]	AMI	pericardia surface region infraction relationship to MVO	epicardial surface area(EpiSA) was the strongest predictor of MVO	EpiSA of infarction
Louis Potier[403]	Diabetes	the relationship between CMD and microvascular complications	unresolved	Albuminuria
Ercole Tagliamonte[404]	sinus rhythm in stable CAD	bisoprolol and ivabradine e ect on CVFR	signi cant decreased coronary ow velocity on ivabradine and bisoprolol group	doppler-derived Coronary Flow Velocity Reserve
Zhaoxue Sheng[405]	ANOCA	CMD de nition based as CFR≤ 2.5 or IMR≥ 25 o	TFChyp correlated more strongly with IMR than TFCrest	TFChyp correlation to with IMR
Ankur Gupta[406]	known or suspected stable CAD	value of maximal MBF integrated to CFR e ect on cardiovascular death	CFR is a stronger predictor of cardiovascular mortality than maximal MBF of CFR	CFR values
Ta-Chuan Hung[171]	HFpEF	CMD and heart failure are associated with unfavorable cardiac mechanics speci cally impact on MBF, CMD de ned as	CMD by dynamic SPECT is frequently observed among patients with HFpEF and correlated with more impaired overall cardiac mechanics	worse longitudinal strain indices, lower post-stress MBF, higher global rest MBF , and lower global MFR

Authors	Category	Hypothesis \Question	Outcomes	Marker
Vincenzo Sucato[100]	INOCA and MVA	GLS analysis correlation to TFC	CMD may be described by TFC	signi cant reduction in GLS associated to LVEF≥ 50%

Table 7: Summary of relevant studies evaluating microvascular function in general contexts.

2.6. Discussion

Patients with chest pain and non-obstructive CAD have a high prevalence of coronary microvascular abnormalities.These abnormalities correlate poorly with conventional cardiovascular risk factors and are dissociated from classical ndings of non-invasive functional testing[407, 367, 1], partly due to the lower microvascular arterial compliance characteristic of these patients[408, 409, 410, 10, 411, 412]after a deep research in articles it seems like that CMD signature it hard to capture due too multifaceted factors those intervenes and contribute to CMD development mechanisms[413, 414], Beyond the relevance of temporal effects commonly investigated in relation to atherosclerotic progression and plaque morphology both before and after PCI the most salient observation in this area of cardiovascular disease research is the considerable disparity between the clinical scenarios proposed and the current capacity to adhere them with analytical or numerical models within realistic, AI-assisted simulation environments. due too rarity of micro-coronary databases and personalized contexts de nition[415],CMD progression had overcome formal and familiar organic techniques of diagnostic and prognosis to be projected in the psychiatry area and mental health[131, 416], sex di erentiation[8][417, 418, 419, 375, 168, 178]switch international statistics women's are the most a ected by that syndrome related to postmenopausal period, microvascular dysfunction syndrome interacts in a complex often bidirectional or multidirectional manner with various cardiovascular risk factors. In some studies, it is hypothesized as a central mechanism the evaluation invasive indices such as(CBF, and FFR)concatenated to cardiologist expertise.Alternatively, it may also be considered as a predictive element inferred from apparent clinical risk factors [420, 421].Establishing a clear and standardized framework for the study of CMD remains a signi cant challenge, largely due to the multidisciplinary nature of the eld.E ective representation of the problem requires contributions from diverse domains such as physics, computational modelling and arti cial intelligence[115] Although restoring epicardial blood ow after AMI represents the cornerstone of therapeutic strategies[422], CMD continues to exert a decisive in uence, particularly in clinical entities such as INOCA and ANOCA[423].The interplay between anatomical and functional characteristics of coronary stenosis as a key element[424]highlighted in studies on coronary microembolization[425] [426, 427]rheumatoid diseases, epicardial vascular dysfunction[428, 372, 248]CMD discovering has been related to the justi cation of revascularization as choice to regulate blood ow[429].A major determinant of outcome in patients with MVA and NoCAD seems instead related to non-critical atherosclerotic disease suggesting a more aggressive management of cardiovascular risk factors and preventive management[430]multimodality approach complexity controlling, coronary microcirculatory pathophysiology can we a ord it to remain a black box axel pries that persist as he principal dielemma[431, 432]relationship of macrocirculation to microcirculation[433]PCI utility after folow up and some medication e ects[372]as β blockers on stable angina development to ischemia[434, 21, 435] recommandation[395] , this

article was an attempt to de ne the contexts for the onset of microvascular dysfunction, but there is as yet no well-de ned formula for detecting the development of CMD with normal coronary arteries.

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