Table S1: Overview of the potential plasma biomarkers for genetic cardiomyopathies. HF = heart failure, HCM = hypertrophic cardiomyopathy, DCM = dilated cardiomyopathy, ACM = arrhythmogenic cardiomyopathy, NPs = natriuretic peptides (ANP, BNP, NT-proBNP), cTns = cardiac specific troponins (cTnI, cTnT), Gal-3 = Galectin-3, GDF-15 = Growth differentiation factor 15, sST2 = soluble suppression of tumorigenesis-2, β1 = G-protein coupled β1 receptor, M2 = Muscarin-2- receptor, DSG2 = desmoglein-2, RV = right ventricle, LV = left ventricle, LGE = Late Gadolinium Enhancement, VT/VF = Ventricular Tachycardia/ Ventricular Fibrillation, LVAD = left ventricular assist device, BrS = Brugada syndrome, SCD = sudden cardiac death.

Name	Biomarker type	Biological function	CM type	Evidence	References
NPs	Cardiac specific Plasma protein	Vasodilatation, Diuresis	HCM	Elevated plasma levels in patients in contrast to subclinical carriers. Levels are increased by exercise and are associated with fibrosis. In patients with structural heart disease and/or clinical symptoms elevated levels are associated with higher risk of cardiovascular events, HF and death.	[1–10]
			DCM	Already part of the HF (DCM) guidelines.	[11]
			ACM	Levels are associated with RV dilatation and dysfunction. In patients with structural heart disease and/or clinical symptoms elevated levels are associated with higher risk of cardiovascular events, HF and death.	[3,6–10,12,13]
cTns	Cardiac specific Plasma protein	Sarcomere function	HCM	Levels are elevated in patients, in contrast to the levels in subclinical carriers. Levels are predictive for myocardial fibrosis in non-high risk patients. In patients with structural heart disease and/or clinical symptoms elevated levels are associated with higher risk of cardiovascular events, HF and death.	[1–3,6–10]
			DCM	In patients with structural heart disease and/or clinical symptoms elevated levels are associated with higher risk of cardiovascular events, HF and death.	[3,6–10]
			ACM	In patients with structural heart disease and/or clinical symptoms elevated levels are associated with higher risk of cardiovascular events, HF and death.	[3,6–10]
Gal-3	Non-cardiac specific Plasma protein	Fibrosis, Inflammation	HCM	Levels are elevated in patients and are related to disease severity. No elevation and no association with LV hypertrophy in patients with mild symptoms. Also no correlation with LGE detected fibrosis.	[1,5,14–16]
			DCM	Levels are elevated in patients and are also associated with cardiac fibrosis. Levels were predictive for prognosis	[16,17]
			ACM	Elevated levels in patients and higher levels in the presence of VT/VF. Levels were predictive for ventricular arrhythmias in patients with implantable defibrillators.	[18]
GDF-15	Non-cardiac specific Plasma protein	Inflammation, Remodeling, Cell death and growth	HCM	Levels are associated with disease severity. There was no correlation with LGE detected fibrosis.	[5,19]
			DCM	Levels are associated with increased risk of arrhythmic death. Levels are strongly elevated in end-stage patients and correlate with myocardial fibrosis and kidney function. Strong level decline within one month after LVAD.	[20,21]

			ACM	Elevated levels in patients with biventricular involvement.	[22]
sST2	Non-cardiac specific Plasma protein	Inflammation	HCM	Elevated levels in patients and associated with NYHA class.	[14,23]
			DCM	Levels are not predictive for arrhythmic death. Levels are associated with all-cause mortality	[20,24]
			ACM	Levels are elevated in patients with biventricular involvement and are associated with RV global strain and LV function. Higher levels are detected if ventricular arrhythmias are present.	[22,25]
miR-208	Cardiac specific Noncoding RNA	Cardiac development, Stress response	HCM	Levels are not associated with HCM	[26]
			DCM & ACM	Studies will be needed	
miR-499	Cardiac specific Noncoding RNA	Cardiac development, Stress response	HCM	Levels are not associated with HCM	[26]
			DCM & ACM	Studies will be needed	
miR-29a	Non-cardiac specific Noncoding RNA	Proliferation, Apoptosis, Differentiation, Fibrosis	HCM	Levels are associated with both hypertrophy and fibrosis	[26]
			DCM & ACM	Studies will be needed	
miR-133a-3p	Non-cardiac specific Noncoding RNA	Proliferation, Differentiation	HCM	Not differentially expressed in one study, more studies will be needed	[27]
			DCM	Differentially expressed in one study, more studies will be needed	[27]
			ACM	Differentially expressed in patients and non-affected family members	[27]
α-cTnI	Cardiac specific cAAbs	Inflammation	DCM	Independent predictor of disease development within 5 years follow up. Included in diagnostic criteria for DCM relatives	[28–30]
			HCM & ACM	Studies will be needed	
α-aggregate	Cardiac specific cAAbs	Inflammation	ACM	AHAs against cardiac α-actins, keratin-24, and connexin-43 in detected in BrS, while absent in healthy and HCM, DCM and ACM controls. Additional studies are required .	[31,32]
			HCM&DCM	Studies will be needed	
β1-AAb	Non-cardiac specific AAbs	Inflammation	HCM	Elevated levels in patients	[33]
			DCM & ACM	Studies will be needed	
M2-Aab	Non-cardiac specific AAbs	Inflammation	HCM	Elevated levels in patients. Concentration is higher in patients with a family history of SCD or atrial fibrillation.	[34,35]
			DCM & ACM	Studies will be needed	
α-DSG2	Non-cardiac specific AAbs	Inflammation	ACM	Sensitivity and specificity shown by one study (in which cardiac disease control groups lacked). Additional studies will be needed.	[36]
			HCM & DCM	Studies will be needed	

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