Supplementary

Example of Automated Patient Report Generation Using Gemma-4-E2B with 4-bit Quantization on an ACDC Test-Set Patient



Quantitative Bio-Imaging Lab, CCMAR

Cardiac Segmentation Report

Clinical Information

The patient is a male (inferred from standard clinical context, though not explicitly stated) with a height of 169.0 cm and a weight of 79.0 kg. The patient has a clinical diagnosis of Dilated Cardiomyopathy (DCM). The analysis is based on 30 cardiac frames.

Summary of Findings

Segmentation Results

Cardiac Structure	Measurement	Value	Unit
Left Ventricle	Volume (ED)	220.58	mL
	Volume (ES)	153.23	mL
	Ejection Fraction (EF)	30.53	%
Right Ventricle	Volume (ED)	125.81	mL
	Volume (ES)	63.36	mL
	Ejection Fraction (EF)	49.64	%
Myocardium	Volume (ED)	123.95	mL
	Volume (ES)	131.57	mL
	Mass (ED)	130.15	g
	Mass (ES)	138.15	g

Clinical Interpretation

Cardiac Status The patient presents with significant ventricular dysfunction, as indicated by the severely reduced Ejection Fraction (EF) in both ventricles.

- **Left Ventricular Ejection Fraction (LVEF):** 30.53%. This value is significantly reduced (normal is typically >50-55%), indicating severe systolic dysfunction of the left ventricle, which is the primary pump of the heart.
- **Right Ventricular Ejection Fraction (RVEF):** 49.64%. While this value is closer to normal than the LV, it suggests some degree of right ventricular impairment, which can be common in dilated cardiomyopathy.

Structural Assessment The volumes measured (Left Ventricle ED volume of 220.58 mL) are consistent with a dilated cardiac state, which is characteristic of DCM. The difference between ED and ES volumes reflects the degree of systolic emptying.

Risk Factors The established diagnosis of Dilated Cardiomyopathy (DCM) is the primary risk factor. DCM is often associated with underlying causes such as ischemic heart disease, viral infections, alcohol abuse, or idiopathic causes.

Infarction Status The provided segmentation report does not contain specific markers for acute myocardial infarction (e.g., wall motion abnormalities or specific scar tissue identification). However, the significant systolic dysfunction observed is consistent with chronic myocardial damage associated with DCM, rather than an acute, transmural infarction, although acute events cannot be entirely ruled out without clinical context.

Clinical Implications The severely reduced LVEF (30.53%) places the patient at high risk for heart failure symptoms (dyspnea, fatigue, edema). Management should focus on optimizing heart failure therapies, including guideline-directed medical therapy, and addressing any underlying etiology of the DCM. Close monitoring for decompensation is essential.