

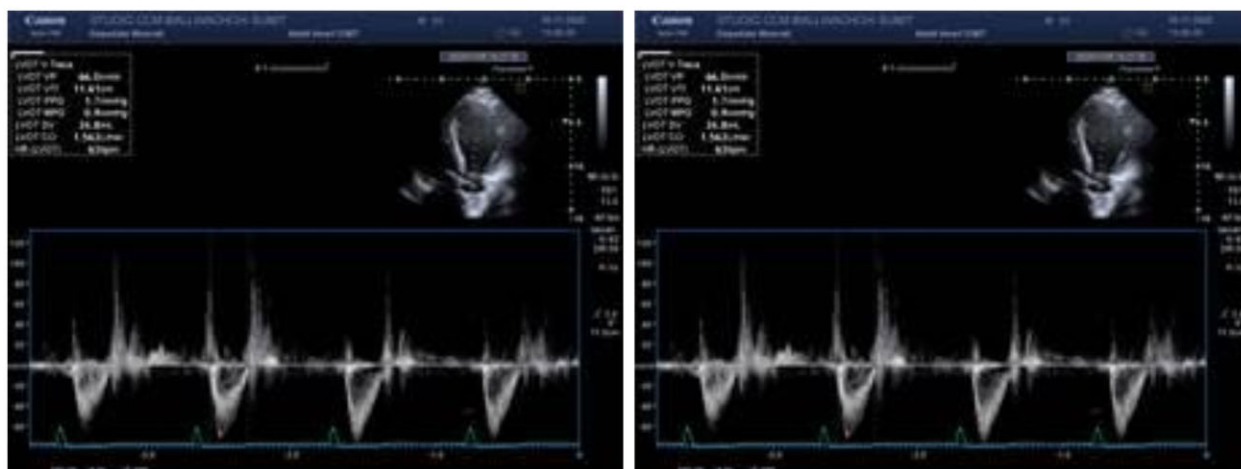
765 Is acute response to calcium sensitizers drugs predictive of response to cardiac contractility modulation in NYHA IV patients?

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A 53-years old man presented to our institution with a diagnosis of decompensated heart failure NYHA Class IV. He had a history of ischaemic heart disease with severe biventricular dysfunction, diabetes, hypertension, dyslipidaemia, advanced chronic kidney disease, previous explanation of dual-chamber implantable electronic device (ICD) due to endocarditis and subsequent implantation of subcutaneous ICD in primary prevention. Home therapy included uptitrated angiotensin-converting enzyme inhibitor, β -blocker, loop-diuretic, spironolactone, acetylsalicylic acid, and oral hypoglycemics. Clinical examination showed signs and symptoms of systemic and pulmonary congestion with pleural effusion and ascites. Echocardiography revealed diffuse left ventricular (LV) hypokinesis with an ejection fraction (EF) of 25%, severe right ventricular dysfunction and increased filling pressures. He was treated with high dose of i.v. diuretics with mild improvement of dyspnoea. However, haemodynamic stability was labile with worsening of symptoms as soon as mild down-titration of iv diuretics was attempted. Levosimendan, a calcium-sensitizer inodilator, indicated for short-term treatment of acutely decompensated severe chronic heart failure (HF), was administered with good clinical response. Thus, we thought that the patient could have benefited from contractility modulation therapy (CCM) which acts on intramyocardial calcium handling. CCM is a novel therapeutic option for patients with classes III-IV HF with $EF \geq 25\%$ to $\leq 45\%$ and narrow QRS complex that acts on intramyocardial calcium-handling. CCM proved effective in alleviating symptoms, improving exercise tolerance and quality of life, and reducing hospitalization rates in HF. It improves myocardial contractility, reverses the foetal myocyte gene program associated with HF and facilitates cardiac reverse remodelling. Therefore, an Optimizer Smart System (Impulse Dynamics) was implanted. Two pacing

electrodes were placed on the interventricular septum in apical and mid-septal position, respectively. The leads were connected to a pulse-generator in a right pectoral pocket. In the following days, we observed a progressive improvement in clinical status, with gradual resolution of peripheral oedema, dyspnoea and fatigue and significant weight loss. Six-month echocardiography showed a stable value of EF and significant improvement in stroke volume (35.2 ml from 24.8 ml at baseline). The patient did not undergo further hospitalization for decompensated HF and was in stable ambulatory NYHA Class IV. We believe CCM is an option in patients with advanced HF in which avoiding recurrent hospitalizations, with their overt increase mortality, is often a challenging therapeutic goal.



765 Figure