

A 48-year-old patient presented with H/O worsening breathlessness and orthopnoea with cough and whitish sputum for two weeks without chest pain or syncope or fever Background history: ischemic heart disease, recurrent VT, LVSD, percutaneously placed CRT-D 2 months earlier, and CKD3. They had improved with CRT-D therapy; diuretics were being down-titrated A - patent and own **B** - laboured breathing, RR 36/min, SPO2 94% on 10L O2, bilateral crackles and systolic murmur on auscultation C – warm center and cool peripheries, CRT 3sec, BP 104/68 mm Hg, HR 101/min **D** - GCS E4 V5 M6, BL pupils 3mm and reactive Figure 1: Chest X ray report: florid pulmonary oedema **E** – temperature 36.6C, peripheral oedema bilaterally and small bilateral pleural effusions. Appearances are Point of care testing: most likely to represent heart failure/fluid overload. Concurrent infective changes is challenging to exclude **ECG** showed paced rhythm, unchanged from last admission Blood gas revealed T1RF **Chest XR** refer to Figure 1 Working Diagnosis: Decompensated heart failure ?CAP R/O COVID19/influenza Plan: Maintain SPO2 > 94% Send swabs for respiratory infection, FBC, U&E, CRP, blood cultures, troponin Sepsis bundles Figure 2 Figure 1 IV diuretic **CTPA report:** No pulmonary embolism. Features of heart failure **Diagnostic investigation reports:** as seen on recent radiograph. The left ventricle lead of the CRTD COVID-19 & respiratory viruses' swab – negative has retracted in comparison to post insertion radiograph (Figure **2**) and is overlying left atrium rather than left ventricle likely to No inflammatory makers be contributing to the presentation of heart failure. Suggest Normal range electrolytes cardiology review and pacing check. Unchanged U&E Troponin negative Soon after, respiratory failure worsened, however, examination remained unchanged. Consequent D-dimer was raised and CTPA was performed that revealed retracted lead of the CRTD (refer to the report above). Subsequent ECGs showed VT, lacked pacing spikes, and it was cardioverted chemically, following which the patient was admitted for LV lead re-implantation in acute coronary unit.

CAN MOVEMENT LEAD TO DECOMPENSATED HEART FAILURE? Dr Dolly CYadav, Dr Himanshu Gul Mirani **Queen's Medical Centre, Nottingham University Hospital NHS Trust**

CASE SUMMARY









KEY LEARNING POINTS

- In patients with severe refractory heart failure and intraventricular conduction disease, CRT (cardiac resynchronisation therapy) is an effective treatment
- **%** If with patients present decompensated heart failure after an initial improvement from CRT, then failed LV sensing or pacing, as a result of a shift in the lead tip, should be considered
- Use LV lead dislodgement as the main cause of loss of LV stimulation reportedly ranges in 2% to 12% of patients



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