months later the patient was asymptomatic but atrial arrhythmia persisted. She refused a pacemaker implant.

**Discussion:** In spite of the paucity of symptoms, the combination of ectopic rhythm with multifocal block and sinus node dysfunction suggested infiltrative cardiac disease. Echocardiography revealed additional clues towards undertaking focused work-up and establishing the diagnosis. However, the stress associated with minor thoracotomy was sufficient to trigger takotsubo cardiomyopathy.

**Conclusions:** 1. careful analysis of ECGs is mandatory in every patient, since it may contain clues of hidden diseases; 2. major cardiac complications may occur even after minor surgery. As an old Hebrew saying says: it is those "little foxes that spoil the vines".

**4522**

**Acute decompensated heart failure in young woman with lymphoproliferative disorder**

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**Introduction:** Large Granular Lymphocyte Leukemia (LGLL) is a chronic indolent leukemia. The heart leukemic infiltration is frequently diagnosed at biopsy, rarely clinically.

**Case report description:** A woman with history of LGLL was admitted to the Emergency Department for congestive heart failure. An ECG showed sinus tachycardia, Q5 morphology in anterior leads and interstitial negative T-waves. Repeat dosages of Troponin were negative. An echocardiography showed a severe biventricular dilatation, severe reduction of ejection fraction and a ground-glass concentric hypertrophy. The absence of myocardial edema and the presence of subendocardial delay enhancement (DE) on cardiac magnetic resonance (CMR) ruled out the diagnosis of myocarditis. Normal coronary arteries was found at coronarography. Endomyocardial biopsy showed the presence of mononuclear lymphocytes consistent with myocarditis or lymphoproliferative disease; no viral genome was detected. The patient was treated with steroids achieving a quick recovery in only a week. Serial CMR confirmed the presence of a persistent subendocardial DE, without edema and a complete normalization of kinetics.

**Discussion:** This case represents a challenging diagnosis: the absence of edema at CMR exclude an acute fibrinous or ischemic injury in consistent with the absence of troponin release. Ruling out myocarditis as the most likely diagnosis.

**4524**

**How sweet is the pacemaker?**

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**Introduction:** A 65-year-old gentleman with two previous myocardial infarctions, a dual chamber pacemaker, chronic obstructive pulmonary disease and bronchiectasis requiring home oxygen, chronic kidney disease stage 3, erythropoietin-dependent normocytic anaemia and recently biopsy-diagnosed Sweet’s syndrome was admitted from the community with increasing breathlessness. He had 11 admissions in the last 3 years with persistently raised inflammatory markers; no definitive diagnosis had been reached and was always allowed home following a course of antibiotics.

**Case report description**

Clinical examination was consistent with left lower zone pneumonia. His inflammatory markers were raised and both chest radiography and Computed Tomography confirmed left lower lobe pneumonia. He was treated with antibiotics and showed a good response. However, ten days later, he developed cardiac tamponade requiring emergency pericardiocentesis and transient hemofiltration. 28 days into the admission a rash was noted on his feet raising the possibility of vasculitis as a unifying diagnosis but all subsequent investigations excluded this possibility. He made a slow progress but 51 days into the admission he spiked a temperature and blood cultures grew coagulase negative staphylococci. Review of microbiology records over the last 2 years revealed 7 blood cultures growing coagulase negative staphylococci with the same sensitivities. A diagnosis of chronic pacemaker lead endocarditis was therefore considered. Transsthoracic and transesophageal echocardiography did not show any valvular pathology but the entire lead could not be visualised to rule out pacemaker wire infection. The pacemaker was explanted following 6 weeks of intravenous vancomycin and oral rifampicin. Two years later our patient remains well without any further hospital admissions. From the time of admission his renal function normalised and he does no longer require erythropoetin or oxygen.

**Discussion:** Pacemaker infection is common occurring in 1%-19% of pacemaker implantations and coagulase negative staphylococci (often considered a contaminant or apathogenic in patients without indwelling devices), Staphylococcus aureus and gram-negative bacilli are the most common pathogens. Implications to clinical practice: Sweet’s syndrome is a rare manifestation of infective endocarditis and endocarditis should be considered in all patients with Sweet’s syndrome and any evidence of infection, particularly in the presence of any foreign material, as the infection might only be cured once the foreign material is removed and lead to dramatic improvement as exemplified by our case.