An interesting case of type 2 myocardial infarction

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Declaration of Interest:

I have nothing to declare
55-year-old female presented to the Emergency Department with light-headedness

Woke during the night with palpitations and was found collapsed on cold bathroom floor by partner.

Complaining of palpitations earlier that day

History of chest palpitations since aged 10 and Reynaud's syndrome

Regular medication was SSRI

Initial Observations
- Respiration rate: 18 bpm
- Oxygen saturations: 100% on air
- B.P.: 80/60mmHg
- Heart rate: 150 bpm
- Temperature: 33.4 °C
- GCS 13/15
- Normal blood sugar
**Admission blood results**

Abnormal results included raised creatinine (123 μmol/L).

High-sensitivity cardiac troponin concentration was raised 478 ng/L and her peak troponin at 12 hours was 22,845 ng/L (normal range 1-16ng/L).

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**Echocardiogram**

Normal left ventricular size and function. No significant valvular wall abnormalities or regional wall motion abnormalities.

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**CT Pulmonary Angiogram**

No evidence of pulmonary embolus.
Warmed & IV fluids

Cardioverted to sinus rhythm without intervention

Admission

Diagnosis of type 1 Myocardial Infarction

Day 2: intermittent episode of non-sustained ventricular tachycardia

Invasive coronary angiogram showed normal arteries. Type 2 Myocardial Infarction diagnosis

Discharged home

Electrophysiology study identified AV nodal re-entry tachycardia (AVNRT) & successfully managed with catheter ablation

Criteria for type 2 MI

Detection of a rise and/or fall of cTn values with at least one value above the 99th percentile URL, and evidence of an imbalance between myocardial oxygen supply and demand unrelated to acute coronary athero-thrombosis, requiring at least one of the following:

- Symptoms of acute myocardial ischaemia;
- New ischaemic ECG changes;
- Development of pathological Q waves;
- Imaging evidence of new loss of viable myocardium or new regional wall motion abnormality in a pattern consistent with an ischaemic aetiology.
DEMAND-MI Research Study investigating type 2 myocardial infarction

Cardiac Magnetic Resonance Imaging (MRI) scan

diffuse subendocardial late gadolinium

It is likely that this was caused by global ischaemia as a result of her haemodynamic compromise and her arrhythmia

Treatment of type 2 myocardial infarction = identifying & managing supply and demand imbalance

Advised not to drive for 1 week and was followed up by local cardiology team
High-sensitivity cardiac troponin assays have improved the diagnosis of myocardial infarction & identify patients with previous myocardial injury.

Making timely and accurate definitions in the clinical settings can be challenging due to overlap of diagnostic criteria. A coronary angiogram is not always clinically indicated or required.

No compelling data for optimal treatment due to heterogeneity in responsible mechanisms.

Manage underlying aetiology of supply and demand imbalance.

Chapman et al 2017 Long-Term Outcomes in Patients with Type 2 Myocardial Infarction and Myocardial Injury. Circulation 137 pp.1236-1245