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**A cardiac arrest and a low haemoglobin.. a diagnostic conundrum**

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**Introduction**
A 62 year old male, a known case of thalassemia trait with no other co-morbidities was referred for urgent inpatient investigations for diarrhoea and worsening iron deficiency anaemia. Our patient also gave a longstanding history of chest pain for which he has had an invasive coronary angiogram which revealed normal coronaries. The patient also had a past isolated episode of atrial fibrillation for which he was DC-cardioverted. Upon examination, he also had marked lower limb oedema and evidence of peripheral neuropathy. A number of investigations were ordered in view of unexplained multi-organ involvement.

**Investigations**
Blood investigations revealed elevated cardiac enzymes (hs-cTnT 514ng/L), high NT-proBNP (2672 pg/mL) and a microcytic anemia(Hb 7.2g/dL, MCV 65fL). An unexplained deranged renal profile was noted with a creatinine of 165µmol/L. A CT trunk was performed in view of his anemia which showed thickening of the gastro-oesophageal junction, for which an OGD was done.

Early into his inpatient stay, the patient sustained a cardiac arrest after which he was transferred to Intensive Care. Electrocardiography before and after the incident did not reveal any ischemic changes. Small complexes in the limb leads were noted. Echocardiogram showed a preserved left ventricular systolic function and grade 2 diastolic dysfunction with raised filling pressures (E/Med e’37). A speckled myocardium with apical sparing was evident. No regional wall motion abnormalities were detected. A provisional diagnosis of amyloidosis was made. The patient was started on treatment for HFpEF. Electromyography confirmed a severe length-dependent sensory polyneuropathy. A positive monoclonal band, elevated serum free light-chains, (lambda 13990++ mg/L) strengthened the diagnosis and bone marrow trephine showed a hypercellular bone marrow which also confirmed the diagnosis of light chain amyloidosis (AL-CA). A histological diagnosis of infiltrative amyloidosis was also made through the gastric biopsies.

After discussion with haematologists, the patient was started on CyBorDex: cyclophosphamide, bortezomib and dexamethasone. Prophylactic enoxaparin was also started. Supportive treatment with repeated red cell concentrate transfusions and antibiotics was also administered.

**Problems Encountered**
The patient’s anemia could easily be dismissed in view of his history of thalassemia. However his unexplained lower limb oedema and peripheral neuropathy prompted further investigations. Unfortunately, the past history of chest pains with normal invasive cor-angiography gave a false sense of reassurance. The speckled pattern visualised on echocardiography helped in unraveling the diagnosis of the patient.

**Conclusion**
Amyloidosis complicating multiple myeloma may have different phenotypic presentations. Light chain
Amyloidosis (AL) requires a high index of suspicion for a timely diagnosis, prompt medical treatment and improved clinical outcome.