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Case Report

Anaemia of Chronic Kidney Disease (CKD) in an 80 Year Old Male

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Abstract

An 80 year old gentleman was referred by his GP to Rapid Medical/Frailty Unit Blaenau and Gwent for ongoing SOB, anaemia, frailty and worsening mobility. The gentleman was known to have anaemia of CKD.

The patient explained that since his discharge from hospital on the 21/11/2019 following admission for Acute on chronic SDH and AKI on CKD his SOB and mobility had gradually deteriorated. His son who is his main carer reported that in the last few days his father has become frailer to the extent that now he can't stand up from the chair without assistance and he is constantly SOB now.

Keywords: PMH; PPM; Hiatus Hernia; SAH and SDH on Previous CT; Bowel Cancer; Epilepsy; High Cholesterol; CKD; Anaemia

Introduction

SH: Lives in house with son in ground floor bedroom has POCx2.

Current Medication:

LEVETIRACETAM 250 mg OD
LISNOPRIL 5 mg OD
OMEPRAZOLE 20 mg OD
SODIUM BICARBONATE 500 mg TDS
TAMSULOSIN HYDROCHLORIDE MR 400 MCG NOCTE
EPO Monthly

Vital Signs: Temp 37.6, HR 101, Sitting BP 82/45, Standing BP 76/43, Sats 99 % on RA, RR 20

Admission Bloods Tests: 10/01/2019, abnormal results only

Hb 66, Platelets 148, MCV 102, Ferritin 368, transferrin 1.8, TRS 11.1, Iron 5, CRP 33, Urea 27.1, Creatinine 284, eGFR 18

Physical Examination

Alert, Pale, No Cognitive Impairment

Dry Mucous Membranes

HS – I + II + O sinus tachycardia, mild peripheral oedema, JVP not raised GI- Abdo SNT, RS- good air entry with minor basal crackles bilaterally.

Impression; Severe Anaemia of CKD, Orthostatic Hypotension, Dehydration, SOB, Frailty, UTI

Management

Because of the patient's severe orthostatic hypotension and SOB a decision was made to transfer him to an acute hospital for RDC transfusion, IV fluids and further work up.

Discussion

This case represents a classic example of anaemia of chronic kidney disease (CKD) in a geriatric patient. This patient had CKD 4 which was presumably caused by ischaemic nephropathy. On this encounter with the Frailty service, the patient was referred by his GP for worsening SOB, anaemia and reduced mobility. Symptoms of anaemia include tiredness or lethargy, and in extreme cases shortness of breath and palpitations. When the patient was seen by our team in his house he had severe SOB barely keeping his head up in the chair he was sitting in. We found him to have severe orthostatic hypotension as indicated above. He also complained of urinary frequency. He also had dry mucous membranes indicating that the hypotension was likely due to poor hydration. When the patient was transferred to the acute hospital his Hb was found to be 66. In addition, his Urine MC&S tested positive Klebsiella oxytoca (KOXY) sensitive to Gentamicin, Nitrofurantoin, co-amoxiclav and trimethoprim. [1]

In this case what is illustrative is the decline in renal function was parallel to decline in Hb. Records indicated that on the 13/11/2018 the serum creatinine was 167, eGFR was 34 and Hb was 89. On the 10/01/2019 the serum creatinine has rose to 284, eGFR declined to 18 and Hb to 66. The 10/01/2018 is the day the patient became very symptomatic. In summary dehydration and a UTI contributed to the patient's worsening renal function and similarly decline in Hb and associated SOB.

In the pathogenesis anemia of CKD generally when the eGFR, which measures the rate at which blood is filtered by the kidney, falls below 60 ml/min/1.73 m^2; the prevalence and severity of the anemia increases. The cause of anaemia in CKD is multifactorial, but the two main contributors are erythropoietin deficiency and iron deficiency.

Erythropoietin is a hormone produced by the peritubular cells in the kidney to help make red blood cells. Iron deficiency may be functional (this particularly occurs in the presence of inflammation—when body iron stores may be normal or increased but there is a failure of iron delivery to the bone marrow), or absolute (when body iron stores are exhausted). Absolute iron deficiency occurs, in part, because of impaired absorption of iron from the gastrointestinal tract in uraemia and also partly due to increased iron losses as a result of platelet dysfunction associated with, for example, uraemia, or aspirin therapy.[2]

The other interesting aspect of this case is the controversy of relying on ferritin as the prime indicator of anaemia. In this case the ferritin level was 368 (15 -300), the transferrin 1.8 (2.0–4.0), Transferrin saturation was 11.1% (20 – 50), Iron level 5. According to NICE guidelines, it is not recommended to rely on ferritin level or transferrin saturation singularly to diagnose anaemia of CKD. In anaemia of CKD testing to diagnose iron deficiency and determine potential responsiveness to iron therapy and longterm iron requirements every 3 months should include percentage of hypochromic red blood cells (% HRC; more than 6%) if processing of blood sample is possible within 6 hours or reticulocyte haemoglobin (Hb) content (CHr; less than 29 pg).

Ferritin is an acute phase reactant that rises in inflammation and consequently a normal to high level doesn't rule out deficiency. In addition, ferritin levels tend to rise with age and hence in geriatric patients on its own, a normal or high level of ferritin may not reflect the accurate picture of iron deficiency. Usually in anaemia of chronic disease the

transferrin saturation (iron binding capacity) is normal but in this patient is low indicating the severity and multifactorial component of his anaemia.

In this patient relying on ferritin alone would have given an incomplete picture as it was above the normal range.

In people treated with iron, serum ferritin levels should not rise above 800 micrograms/litre. In order to prevent this, review the dose of iron when serum ferritin levels reach 500 micrograms/litre. Maintain the aspirational Hb range between 100 and 120 g/litre, do not wait until Hb levels are outside the aspirational range before adjusting treatment (for example, take action when Hb levels are within 5 g/litre of the range's limits.[3]

Iron therapy and erythropoietic stimulating agents (ESAs) to people with anaemia of CKD who are deficient in iron will benefit them in terms of quality of life and physical function.

Summary and Conclusion

Decline in renal function parallels the decline in Hb level in anaemia of CKD and severity of associated symptoms. In anaemia of CKD decline in patient function should prompt investigations that include identifying and treating the common acute causes of decline in renal function such as UTIs, dehydration, renal ischaemia e.t.c. Although ESA therapy is the mainstay of treating anaemia of CKD there are associated safety concerns for example increased strokes, venous thromboembolism, arterial thromboembolism and malignancy according to the CHOIR and TREAT trials.

In these patients in addition to regular ESA therapy, iron reserves have to be monitored regularly because ESA therapy alone doesn't preclude worsening of anaemia.

Maintain the aspirational Hb range between 100 and 120 g/litre in adults with anaemia of CKD. Laboratory investigations should include ferritin, transferrin, and transferrin saturation, the percentage of hypochromic red blood cells or reticulocyte haemoglobin (Hb) content.

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