PHA6020Y – Exam 2 – Formative

JH is attending their routine out-patient clinic appointment – they are reviewed by their consultant who makes the following record in their notes:

Patient:	JH	
Hospital number:	788993	
DoB:	24/04/1956	
Gender:	F	
Address:	13 Clover Hill, Flatplace	
PMH:	Type 1 DM (since childhood) ESRF (secondary to diabetic nephropathy) – on haemodialysis (HD) 3 x a week Hypertension 15 years	
DH:	Atorvastatin 20mg ON Amlodipine 10mg OM Calcium acetate 1-2 tablets with each meal Erythropoietin injection (Eprex®) 4000IU IV whilst on HD Lantus® Solostar® insulin 15IU ON NovoRapid® Flexpen® insulin – variable dose TDS with meals NKDA	
SH:	Retired teacher	
Alcohol:	Nil	
Smoking Status:	Non-smoker	
OE:	BP	129/80 mmHg
	Temp	36.2 degrees Celsius
	Pulse	66 BPM
	Weight	62kg
Clinic blood tests:	HbA1c	46.4mmol/mol / 6.8%
	eGFR	9 ml/min/1.73m^2
	Phosphate	1.33 mmol/L (Ref: 0.8-1.45)
	Corrected calcium	1.75 mmol/L (Ref: 2.1-2.6)
	Hb	13.5g/dL (Ref: 13.0-18.0)
	Ferritin	243 µg/L (Target 200-500)

a) Critique JH's current drug therapy. Describe and explain any interventions you would like to make regarding their treatment.

[60%]

b) Describe the underlying pathophysiology and potential clinical consequences of the condition(s) you have recommended intervention(s) for in part (a)

[40%]

a)

Atorvastatin 20mg ON – appropriate according to EBM for primary prevention of CVD (Type 1DM and RF)

Amlodipine 10mg OM – appropriate choice (where ACEI had be stopped in context of CKD – originally first-lien choice as Type 1 DM) – target of <130/80 achieved

Calcium acetate – appropriate for hyperphosphataemia in ESRF – phosphate levels controlled

Erythropoietin injection (Eprex®) 4000IU IV whilst on HD – appropriate for anaemia in ESRF (Hb in range + also ferritin – no IV iron needed)

Lantus + NovoRapid – appropriate basal/bolus regime – HBA1c controlled – target 48-59 mmol/mol (6.5-7.5%)

JH has hypocalcaemia requiring treatment with activated/partially activated form of Vitamin D: E.g: Alfacalcidol [1α-hydroxycholecalciferol] – partially activated form of Vitamin D – 500ng-1mcg od or Calcitriol (1,25-dihydroxycholecalciferol) – fully activated form of Vitamin D- 250ng od

b)

- Vit D usually found in the skin from sunlight and gut from food
- Inactive form requires activation, through hydroxylation in the 25 position in the liver and 1 position in the kidney. In ESRF this cannot be done
- This leads to a decrease in active vitamin D to carry out its usual role
- Vitamin D is required for the absorption of calcium from the gut, if this is not able to occur due to decreased vit D it leads to hypocalcaemia
- Reduced vitamin D, subsequent reduced calcium can cause renal bone disease due to decreased bone mineralisation (osteomalacia)
- Hypocalcaemia may be exacerbated by hyperphosphatemia (not in patient JH), due to the kidneys inability to excrete phosphate. Phosphate ions are able to sequester calcium in the form of calcium phosphate in the bones
- Decreased calcium => release of PTH. Kidneys are unable to respond to PTH in their normal way to increase renal absorption of calcium as pt ESRF, this leads to secondary hyperparathyroidism (causing hyperplasia of parathyroid gland) and disturbs bone architecture leading to bone hardening (osteosclerosis)