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The HPCSA and the Med Tech Society have confirmed that this clinical case study, plus your routine review of your EQA reports from Thistle QA, should be documented as a "Journal Club" activity. This means that you must record those attending for CEU purposes. Thistle will **not** issue a certificate to cover these activities, nor send out "correct" answers to the CEU questions at the end of this case study.

The Thistle QA CEU No is: **MT00025**.

Each attendee should claim **THREE** CEU points for completing this Quality Control Journal Club exercise, and retain a copy of the relevant Thistle QA Participation Certificate as proof of registration on a Thistle QA EQA.

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Gamma-glutamyltransferase (GGT)

A 27-year-old man, on long-term anticonculsant therapy (phenytoin sodium and phenobarbitone), was admitted to hospital for reassessment. His clinical and physical examinations were unremarkable but his liver function tests revealed the abnormalities shown below.

Plasma

TProt	74 g/L	(62-82)
Alb	43 g/L	(30-50)
Bili	12 µmol/L	(<20)
ALP	195 U/L	(30-120)
ALT	26 U/L	(<35)
GGT	140 U/L	(<45)

Investigation of a raised GGT

Although GGT has a wide distribution throughout the body tissues, raised plasma levels probably only originate from the liver. However, a raised level does not always indicate liver pathology as induction of hepatic GGT by alcohol and a number of drugs can also cause increased plasma activity.

The plasma GGT level should be interpreted with care as it may sometimes be too sensitive an indicator of liver disease, due to its ready induction by various factors.

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Case discussion:

Further biochemical investigations on this patient showed disordered calcium metabolism, as reflected by alterations in his plasma Ca, PO₄, parathyroid hormone (PTH) and 25-hydroxycholecalciferol (25-OHCC) levels.

Plasma

Ca	1.85 mmol/L	(2.15-2.55)
PO ₄	0.55 mmol/L	(0.65-1.25)
PTH	10 U/L	(2-6)
25-OHCC	33 mmol/L	(40-160)

Final diagnosis

This patient shows two of the complications of long-term anticonvulsant therapy: increased plasma GGT level (enzyme induction) and hypocalcaemia. The disordered calcium metabolism reflects vitamin D deficiency due to anticonvulsant-induced conversion of cholecalciferol and 25-OHCC to inactive metabolites. The deficiency of vitamin D activity results in hypocalcaemia (decreased gut absorption) which in turn stimulates PTH secretion. The increased PTH activity stimulates bone resorption (increased ALP activity) and decreases renal reabsorption of phosphate (hypophosphataemia).

N.B. Not all patients with drug-induced vitamin D metabolism abnormalities will have a low plasma 25-OHCC level. In some patients the value of this analyte may be normal and the disorder is only recognized by demonstrating a low plasma level of 1,25-dihydroxycholecalciferol, the biologically active metabolite of vitamin D.

CPD Questions:

1. Why is this patient's ALP elevated?
2. List the items that can result in hepatic enzyme induction and an elevated GGT.

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