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Clinical Chemistry

CHRONIC RENAL FAILURE

CHRONIC RENAL FAILURE:

Chronic renal failure (CRF) is the progressive irreversible destruction of kidney tissue by disease which, if not treated by dialysis or transplant, will result in the death of the patient. the aetiology of CRF encompasses the spectrum of known of kidney disease. The end result of progressive renal damage is the same no matter what the cause of the disease may have been. The major effects of renal failure all occur because of the loss of functioning nephrons. It is a feature of CRF that patients may have few if any symptoms until the glomerular filtration rate falls below 15 ml/min (i.e. to 10% of normal function), and the disease is far advanced.

Sodium and water metabolism:

Most CRF patients retain the ability to reabsorb sodium ions, but the renal tubules may lose their ability to reabsorb water and effected on urine concentration. Polyuria, although present, may not be excessive because the GFR is so low. Because of their impaired ability to regulate water balance, patients in renal failure may become fluid overloaded or fluid depleted very easily.

Potassium metabolism:

Hyperkalaemia is a feature of advanced CRF and poses a threat to life. The ability to excrete potassium decreases as the GFR falls, but hyperkalaemia may not be a major problem in CRF until the GFR falls to very low levels. Then, a sudden deterioration of renal function may precipitate a rapid rise in serum potassium concentration.

Acid-base balance:

As CRF develops, the ability of kidneys to regenerate bicarbonate and excrete hydrogen ions in the urine becomes impaired. The retention of hydrogen ions causes a metabolic acidosis.

Calcium and phosphate metabolism:

The ability of the renal cells to make 1,25-dihydroxycholecalciferol falls as the renal tubular damage progresses. Calcium absorption is reduced and there is a tendency towards hypocalcaemia. Parathyroid hormone is stimulated in an attempt to restore plasma calcium to normal, and high circulating PTH may have adverse effects on bone if this is allowed to continue (Fig. 4). Secondary hyperparathyroidism cause the changes in
bone which are characteristic of renal osteodystrophy.

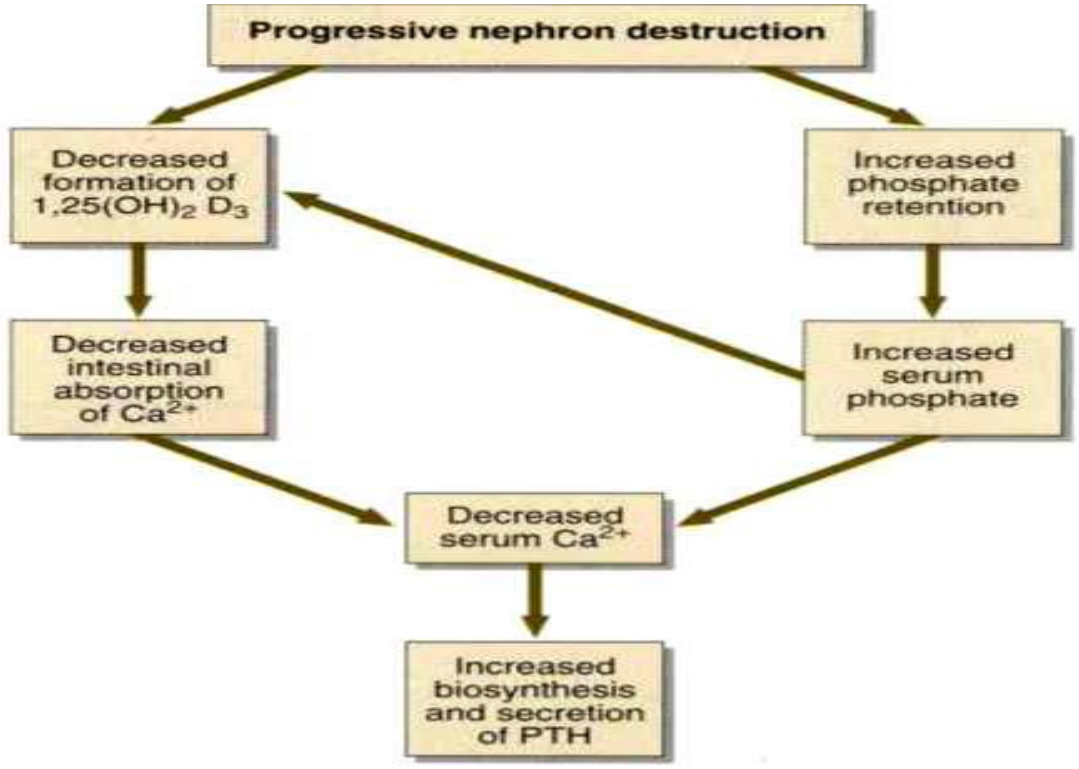

Fig. 4: How hypocalcaemia and secondary hyperparathyroidism develop in renal disease.

Erythropoietin synthesis:

Anemia is often associated with chronic renal disease. The normochromic normocytic anemia is due primarily to failure of erythropoietin production. Biosynthesized human erythropoietin may be used to treat the anemia of CRF.