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EDITORIAL

BONE DISEASE IN RENAL FAILURE

It looks as though we may have to re-orientate our ideas concerning the type of bone disease produced by renal glomerular azotaemic failure. We have long been taught that 'renal rickets' was the wrong name to apply to the metaphyseal lesion seen in childhood renal failure—that the bone disease was osteitis fibrosa, caused either by the acidosis or by a secondary hyperparathyroidism.

At a meeting of the Renal Association,[1] both Stanbury and Dent agreed that, in fact, florid rickets and osteomalacia sometimes occur in renal failure, and further that they may appear with or without coincident osteitis fibrosa. Stanbury showed histological preparations of the metaphyses from uraemic children which were typical of true rickets—by definition, 'a failure of mineral deposition to keep pace with the endochondral growth of bone'. Both authorities agreed that the proximate cause of the osteomalacia was a defective intestinal absorption of calcium, rather than a wastage of minerals in the urine. In comparison with vitamin-D resistant rickets (or osteomalacia) this defect could be overcome by large doses of either calciferol or dihydrotachysterol (AT 10), with radiological evidence of quite rapid healing. There appears to be an acquired insensitivity to vitamin D to account for the metabolic defect, which cannot be blamed on the acidosis or the high serum phosphate. It is possible that some anti-vitamin-D factor circulates in renal failure, but little is known about this.

The osseous changes in the osteitis fibrosa of renal failure are indistinguishable from those of primary hyperparathyroidism. The earliest clinical changes may be seen in radiographs of the fingers, as erosions or 'scalloping' of the cortical surface of the phalanges. It seems extremely likely that a secondary hyperparathyroidism accounts for the osteitis, but the cause of this parathyroid change is not known with any certainty. Stanbury pointed out that rickets or osteomalacia was not a necessary precursor of the hyperparathyroidism. The Albright concept of 'a tendency to low serum calcium' acting as a stimulus to the parathyroids is not proven. As Stanbury further points out, even the development of a hypercalcaemia does not necessarily cause the osteitis to regress. Usually, however, the hyperparathyroidism as well as the osteomalacic changes heal rapidly on large doses of calciferol.

VAN DIE REDAKSIE

BEENSIEKTES BY NIERVERSAKING

Dit lyk asof ons ons idees van die soort beensiektes wat veroorsaak word deur asotemiese versaking van die nierbuisies ietwat moet verander. Dit word ons al lank voorgehou dat dit verkeerd is om die metafise-letsel van nierversaking by kinders ‚nier-rachitis' te noem—dat die beensiekte eintlik fibreuse beenverweking is wat veroorsaak word deur die suurvergiftiging òf deur 'n bykomende oormatige byskildklierwerking.

By 'n vergadering van die *Renal Association*[1] het beide Stanbury en Dent saamgestem dat vol-ontwikkelde rachitis en osteomalasie wel soms in aansluiting met nierversaking voorkom, en dat hulle ook met of sonder samevallende osteitis fibrosa kan voorkom. Stanbury het histologiese preparate van die metafises van ureniese kinders gedemonstreer wat kenmerkend was van egte rachitis—omskryfbaar as ‚'n kondisie waarby die minerale neerslag nie tred hou met die kraakbeengroei (van die been) nie.'

Hierdie twee gesaghebbendes stem saam dat 'n gebrekkige kalsiumopname uit die derm as die onmiddellike oorsaak van osteomalasie beskou moet word, eerder as 'n minerale-verlies in die urien. In teenstelling met rachitis vitamien-D-bestand (of osteomalasie) kan hierdie defek reggestel word met groot dosisse kalsiferol òf dihidrotachysterol (AT10), met radiologiese bewys dat die letsels heel spoedig genees. Daar is blykbaar 'n aangewende ongevoeligheid (of weerstand) vir Vitamien D wat die metaboliese defek verklaar—dit kan nie aan die suurvergiftiging of aan die hoë serum-fosfaatgehalte te wyte wees nie. Dit is moontlik dat die een of ander vitamien-D-bestrydende faktor in die bloed sirkuleer by 'n geval van nierversaking, maar ons kennis hieromtrent is maar beperk.

Die beenveranderings by die osteitis fibrosa van nierversaking kan nie onderskei word van dié wat by primêre oormatige byskildklierwerking voorkom nie. Die vroegste kliniese veranderings kan waargeneem word op X-straalplate van die vingers as wegvretings of ‚scalloping' van die skors oppervlaktes van die vingerbeentjies. Dit lyk baie moontlik dat die beenontsteking deur 'n sekondêre oormatige byskildklierwerking veroorsaak word, maar ons het nog geen duidelike kennis van hierdie byskildklierafwyking nie. Stanbury wys daarop dat rachitis of osteomalasie nie noodwendig 'n voorloper is van die oormatige byskildklierwerking nie. Die Albright-teorie dat ‚'n neiging tot 'n lae serumkalsiumgehalte' as prikkel tot die paratiroïede ageer, is nog nie bevestig nie. Soos Stanbury verder aantoon, lei selfs die ontwikkeling van 'n oormaat kalsium in die bloed nie noodwendig na 'n verbetering van die beenontsteking nie. Gewoonlik herstel die sieklike veranderings van oormatige byskildklierwerking sowel as dié van osteomalasie egter baie gou na groot dosisse kalsiferol.

Dent mentioned a third bone lesion which was occasionally seen in renal failure—osteosclerosis. He remarked that all 3 types of bone disease sometimes occur in steatorrhoea, and here also a resistance to the action of vitamin D could be found, even when it was given parentally.

So the old physicians who talked of renal rickets were not wrong after all, but the bone lesions which do occur in renal glomerular failure with azotaemia are certainly more complex than was formerly imagined. As usual, the more we learn about them, the more there seems to be to find out. One good feature from the clinical angle is that the same treatment—calciferol in high dosage—will cure both the usual types of bone disease even when the renal failure is actually progressing.

1. Stanbury, S. W. and Dent, C. E. (1957): Report of meeting of Renal Association of 24 January 1957. Lancet, **1**, 253.

Dent maak melding van 'n derde beenletsel wat soms by nierversaking gesien word—osteosklerose. Hy verklaar dat al 3 soorte beensiektes soms by steatorree voorkom, en ook by hierdie kondisie bied die liggaam weerstand teen vitamien D, selfs wanneer dit parentaal toegedien word.

Die ou geneeshere wat van nier-rachitis gepraat het was dus tog reg, maar die beenletsels wat by nierbuisversaking met asotemie voorkom is gewis baie meer ingewikkeld as wat eers vermoed was. Soos gewoonlik, hoe meer 'n mens daarvan te wete kom, hoe meer skyn daar wat nog geleer moet word. Een goeie ding, klinies gesproke, is dat dieselfde behandeling, nl. groot dosisse kalsiferol, albei die gewone soorte beensiektes sal genees, selfs al vererger die nierversaking.

1. Stanbury, S. W. en Dent, C. E. (1957): Verslag van 'n vergadering van die *Renal Association* op 24 Januarie 1957. Lancet, **1**., 253.

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