Letters to the Editor

Parkinson’s Disease and Bone Health

SIR—We were very interested in the paper by Abou-Raya et al. [1] regarding bone health and Parkinson’s disease. In our rural community of Ceredigion, West Wales, we carried out a similar study in 2007. We were prompted by a spate of hip fractures amongst our patients attending the movement disorder clinic. We looked at 50 patients (31 males) and enrolled their carers as controls for vitamin D levels. We excluded those with other causes for osteoporosis and whilst we did not perform spinal radiology to look for occult vertebral fracture, we did collect data on known fractures in the patient group. Our data were presented in poster form at the National Osteoporosis Society meeting in 2007. Five of our patient group had a prevalent fragility fracture (four females). Four of the group (three males) had osteoporosis at the femoral neck (T score ≤−2.5) and six (four males) had vertebral osteoporosis. Forty patients (29 males) had insufficient vitamin D as defined by a vitamin D level of <30 ng/ml. We found a weak correlation between bone health and Hoehn and Yahr score (r = −0.32, P = 0.06).

We also found that 32 of 41 carers had vitamin D levels <30 ng/ml.

As a consequence of our findings and published data relating to bone health in movement disorder patients, we now perform dual energy X-ray absorptiometry scan at diagnosis and at two yearly intervals thereafter. Experience tells us that patients with movement disorder fare poorly after major fragility fracture and we need to do all we can to reduce this risk whilst there is time to do so.

Time will tell if we have been able to reduce the risk of fragility fracture in this vulnerable population, and we are gratified to find other centres recommending the same approach!

Conflicts of interest

None declared.

Reference


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Hypothesis: intestinal barrier permeability may contribute to cognitive dysfunction and dementia

SIR—I read the article by Umegaki [1] with interest, with reference to cognitive changes in older adults with type 2 diabetes. Although vascular risk factors and amyloid load may contribute to understanding of loss of cognitive function, other factors may play a role as well.

Powerful pancreatic digestive enzymes may cause destruction of body tissues if their compartmentalisation in the lumen of the intestine and pancreas is compromised. If such enzymes damage the intestinal wall or if the tight junctions making up the intestinal mucosal barrier are disrupted, uncontrolled degrading activity in the plasma would cause proteolytic cleavage of the extracellular domain of membrane receptors and loss of associated cellular functions. Proteolytic cleavage of the insulin receptor would cause inability of insulin to signal glucose across the cellular membrane [2].

Dysfunction of brain insulin receptors has been seen in Alzheimer disease, with extensive abnormalities in insulin and insulin-like growth factor signalling mechanisms in CNS neurons resulting in reduced glucose utilisation and energy metabolism early in the course of the disease [3].

Beta-amyloid and hyperphosphorylated tau deposits have been seen in the pancreas of patients with type 2 diabetes [4].

If the intestinal mucosal barrier becomes hyperpermeable to intestinal contents such as pancreatic enzymes or beta-amyloid from pancreatic juice, pancreatic enzymes and beta-amyloid may cross the intestinal mucosal barrier into the circulation where they could interact with the brain causing disruption of neuronal function and development of Alzheimer disease and impaired cognition.

Conflicts of interest

None declared.