FAQ for Lifelong Bone health webinar

We asked Prof Harvey, Dr Curtis and Dr Nedungayil to respond to the questions that remained unanswered at the webinar on 24th June: their answers are summarised below.

| Question | Response |
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| Where a patient with osteoporosis has very suggestive bone pain symptoms, what kind of additional radiology imaging is used in combination with a DXA scan to detect vertebral micro-fractures that don't show up on X-rays? | The differential for bone pain is wide of course, so care needs to be taken to consider a range of possibilities. In general MRI is good for detecting bone marrow oedema and thus evidence of subtle fracture changes or inflammatory processes. CT scan in is better to look for subtle cortical discontinuity for example and the approach should be discussed with the musculoskeletal radiology team and is ideally managed in the metabolic bone clinic |
| Please could you discuss any evidence for increased osteoporosis and fracture risks in people with long term conditions like CVA, PD, MS, CP. | There is evidence for increased fracture risk in all these conditions with contributions from bone loss and increased falls risk. This evidence most frequently comes from large database studies such as the Clinical Research Practice Datalink, with fracture outcomes, and smaller studies in which BMD has been measured. Clinical Practice Research Datalink \| CPRD |
| Is it possible to reverse osteoporosis in premenopausal women whose reason for having poor BMD has been removed? | There is evidence for reversal of BMD loss in premenopausal women with conditions such as anorexia (reversed by recovery of BMI and recommencement of periods) and disorders such as Coeliac disease, though the time taken for recovery is long (years) and varies with severity of the underlying cause - e.g. gluten free diet in coeliac disease leads to partial recovery by 1 year, full recovery by 5 years (Farfaglia et al, Nutrients 2015 10.3390/nu7053347). |
| From the case study presented, do vegans in general have higher risk of having low bone mass? | Yes, people consuming plant based diets rather than omnivorous diets have been shown to have lower BMD (see this meta-analysis by Li et al published in 2021 in Archives of Osteoporosis 10.1007/s11657-021-00955-0 ). In some studies this difference is explained by BMI differences (lower in vegans) but in others these differences persist after adjustment for BMI. |
| Do you have advice on osteoporosis management in older adults in the 85+ age groups and 90 + year olds? Often these patients are high risk and need treatment, but guidance doesn't appear to be clear. | There is no evidence currently on treatment benefits beyond 10 years and assuming that the over 80s have been on bone sparing agents for a long time, management needs to be individualised. While they are at higher risk of fracture and falls and there are studies showing benefit on continuing bone spring agents, this decision has to be balanced with the risks posed by other co-morbidities, particularly renal function, polypharmacy, gastrointestinal motility issues and the ability to adhere to taking the medications in the correct way. |
| When a patient has been commenced on anti-resorptive medication due to long term glucocorticoid, should these be automatically discontinued once the steroids have been weaned or should a FRAX +/- DXA be done after discontinuation of steroids to re-assess if they are still above the NOGG treatment threshold? | The decision to stop the anti-resorptive depends upon the patient's risk of fracture when reassessed following cessation of the corticosteroid. If the risk is below the intervention threshold at this point then I would still continue the bisphosphonate for 6 months or so after cessation of the steroid to cover the residual effect on fracture risk. |
| Local trust guidelines suggest P1NP levels should be assessed before | Agreed. Bone turnover markers will be raised in the months following a fracture and thus may not be reliable. |

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| initiating treatment with bisphosphonates and after 6 months on treatment in primary care to assess treatment response. Since, PINP increases following a fracture, would this still be appropriate when bisphosphonates are started as secondary prevention treatment following a fragility fracture. Would the P1NP levels taken at this point this be representative? | |
| Is having a history of upper GI bleed/ oesophageal ulcer/ stomach ulcer contraindicates using oral Bisphosphonates? | While abnormalities of the oesophagus- Strictures, achalasia, dysmotility – are contraindications for oral Bisphosphonates, caution needs to be exercised when prescribing in the presence of upper GI disorders. This includes Active gastro-intestinal bleeding; duodenitis; dysphagia gastritis; history (within 1 year) of ulcers; surgery of the upper gastro-intestinal tract; symptomatic oesophageal disease; ulcers; upper gastro-intestinal disorders. https://bnf.nice.org.uk/drug/alendronic-acid.html#contraIndications |
| Could you please explain how GI adverse effects should be managed in patients taking bisphosphonates? I have seen a lot of de-prescribing of bisphosphonates due to symptoms such as mild dyspepsia / mild reflux in secondary care. Should alendronate be changed to binosto / switched to risedronate (as some evidence of possible better GI side effect profile) / should concurrent PPI be trialled or would referral for parenteral treatment be appropriate? | Please see the narrative above regarding the 'cautions' for oral bisphosphonates. In clinical practice clinicians tend to err on the side of caution and hence the tendency to deprescribe. In my local area the preference is for IV Zoledronate or Denosumab decided on an individual basis.<br>There are studies which do indicate risedronate may have a slightly better GI profile, but the risks still exist. Co-prescribing PPI with Bisphosphonates in presence of upper GI disorders is not advisable. I do not have experience with the effervescent preparations as its not part of the local formulary. |