PP-B-05

HYPOKALEMIA AS A NEGLECTED CAUSE OF METABOLIC BONE DISEASE: TWO CASE REPORTS

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BACKGROUND
Hypokalemia occurs secondary to several possible causes that ultimately lead to excessive potassium loss in the body. Long-standing hypokalemia via renal losses could lead to metabolic bone disease (MBD).

CASES
We report two cases of young patients who came in due to fractures. Both patients had a history of lower extremity paralysis. Biochemical analysis showed chronic hypokalemia and metabolic acidosis with normal anion gap, suggestive of renal tubular acidosis (RTA). Patient 1 had impaired renal function with a normal vitamin D level, while patient 2 had normal renal function but had low vitamin D. Genetic testing for RTA could not be performed due to resource constraints. Their MBD was confirmed by radiological assessment. Treatment of both patients involved correction of the acidosis and physical rehabilitation without the need for orthopedic intervention.

RTA is the most common cause of hypokalemia. It is characterized by a normal anion gap metabolic acidosis and renal potassium wasting. Chronic uncorrected acidosis could increase RANKL expression that will promote the differentiation of osteoclasts, leading to increased bone resorption. The most common skeletal manifestations of uncorrected RTA are rickets or osteomalacia, fracture, pseudofracture, secondary osteoporosis and sclerotic bone disease. Since our two patients came in with severe MBD, it would be difficult to reverse these changes and revert to optimal skeletal function.

CONCLUSION
In a patient with chronic hypokalemia and metabolic bone disease, RTA must always be considered as a cause. Increasing awareness regarding the causes of hypokalemia and its long-term impact on the body may facilitate early diagnosis and treatment, thereby preventing permanent sequelae such as MBD.

PP-B-06

ROLE OF BONE MINERAL DENSITY ADDED TO FRACTURE RISK ASSESSMENT TOOL IN THERAPEUTIC DECISION-MAKING FOR OSTEOPOROSIS IN A MALAYSIAN POPULATION

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OBJECTIVES
To examine the role of bone mineral density (BMD) added to Fracture Risk Assessment tool(FRAX) in therapeutic decision-making for osteoporosis in a Malaysian population.

METHODOLOGY
Data were collated from four centers in Malaysia. This study included individuals ages 40 to 90 years old who underwent routine BMD. Patients who had metabolic bone disease or were on anti-osteoporotic treatment were excluded. Ten-year probability of major osteoporotic fractures (MOF) and hip fractures (HF) was calculated using FRAX+BMD and FRAX-BMD. Treatment recommendations for FRAX+BMD and FRAX-BMD were compared and categorized as 'concordant' and 'discordant.'

RESULTS
A total of 1381 participants were included in the study, with the majority being female. There was strong correlation between FRAX+BMD and FRAX-BMD for both MOF (r = 0.889, p<0.001) and HF (r = 0.796, p<0.001). Concordance of 80.1% (p<0.001) was seen in treatment recommendation between FRAX+BMD and FRAX-BMD (treatment recommended n = 505; no treatment recommended, n= 601). Concordance was highest in the youngest and eldest age groups with 91.1% and 85.8%, respectively. Among the discordant, FRAX-BMD underestimated treatment recommendation in 147 (10.6%) and overestimated in 128 (9.2%) participants. Age was the sole important predictor of discordance in treatment recommendations comparing both groups. FRAX-BMD had the least underestimation of treatment among the 80 to 90-year-old group (0.9%) and least overestimation in the 40 to 69-year-old group (1.2%).

CONCLUSION
FRAX-BMD had a good correlation with FRAX+BMD in a Malaysian population and is an acceptable alternative for treatment decision-making in situations where BMD services are not readily available.

PP-B-07

DILEMMAS IN THE DIAGNOSIS AND MANAGEMENT OF OSTEOPOROSIS IN A PATIENT WITH ALKAPTONURIA: SUCCESSFUL TREATMENT WITH TERIPARATIDE

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BACKGROUND
Management of osteoporosis in patients with alkaptonuria can be challenging. We report a patient with alkaptonuria who was successfully treated with teriparatide.

CASE
Laboratory and DXA were completed at our hospital.

A 69-year-old female with a diagnosis of alkaptonuria came in for osteoporosis follow-up. Following the diagnosis of multiple joint arthritis, she underwent several joint replacement surgeries. She also sustained fragility fractures in the foot. Physical exam revealed bluish discoloration of the conjunctiva, normal S1, split S2 and IV/VI systolic murmur over the right parasternal border. She also had limited mobility of the thoracic and lumbar spines, wrists, ankles, knees and hip joints. Laboratory examination revealed the following results: serum PTH 33 pg/mL, 25-OH vitamin D 28 ng/mL, osteocalcin 12 mg/mL, C-telopeptide 318 pg/mL, tyrosine 79.1 umol/L, 24-hour urine homogentisic acid 4.2 gms. Genetic testing showed compound heterozygous mutation for the HGD C. 496T2T>C and HGD C.1102A < G (p.mev368Va) variants, consistent with a diagnosis of alkaptonuria. DXA scan done at the age of 56 years showed osteoporosis (T score of -2.7 over femoral neck, -2.5 over total hip). She was treated with alendronate for 5 years in addition to nitisinone. While on alendronate, she sustained fragility fractures of the right radius and left ankle. After 5 years of alendronate, the patient was transitioned to teriparatide 20 mcg subcutaneously daily for 2 years, followed by annual intravenous zoledronic acid. For the subsequent seven years, the patient led an active life with no fractures. Follow-up DXA showed improvement to osteopenia at the radius. The presence of degenerative arthritis made the other sites difficult to interpret.

CONCLUSION
In this subset of patients, bisphosphonates are not as effective in preventing fragility fractures. However, teriparatide has shown some promise as an alternative treatment.