PP-51

ALCOHOLIC LIVER CIRRHOSIS AND WEAK BONES: A FORGOTTEN CAUSE OF FRAGILITY FRACTURE

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INTRODUCTION
There is little awareness on the effects of chronic alcoholism and liver cirrhosis on skeletal health. We present a case of fragility fracture and reduced bone density in a man with chronic alcoholic liver cirrhosis.

RESULTS
A 50-year-old male with Child-Pugh B liver cirrhosis due to alcoholic liver disease sustained a fragility fracture of the tibia after slipping and falling at home. He reported alcohol intake of more than 5 units/ day for 20 years. On presentation, he had been on spironolactone, propranolol and thiamine for a year. He had no history of glucocorticoid intake or family history of fractures. He had a BMI of 29.7 kg/m^2, with sparse axillary and pubic hair. His testes were 20 ml bilaterally and soft. He had normal serum corrected calcium (2.56 mmol/L), phosphate (1.3 mmol/L) and ALP (98 U/L) with vitamin D deficiency (34 nmol/L). Ultrasound established cirrhosis of the liver. Labs confirmed primary hypogonadism (AM testosterone- 0.7 nmol/L; LH -7.3 IU/L; FSH -18.3 IU/L). His bone density showed a T-score of -2.8 at the femoral neck and -2.0 at the spine. His vitamin D deficiency was corrected and he was commenced on intravenous zoledronic acid with vitamin D and calcium supplementation. Bone health is significantly compromised in liver cirrhosis due to impaired absorption and hydroxylation of vitamin D and vitamin K leading to increased bone resorption. Ethanol has a dose-dependent direct toxic effect on bone via increased cytokines IL-1, IL-6 and TNF-α leading to activation of RANKL and increased osteoclastic activity. Hormonal dysregulation with low IGF-1 and hypogonadism further augments bone loss in alcoholic liver cirrhosis.

CONCLUSION
This case illustrates the importance of screening for and treating osteoporosis in individuals with chronic alcoholism and liver cirrhosis in order to prevent detrimental effects of fragility fractures which contribute to morbidity and mortality.

PP-52

MC-CUNE ALBRIGHT SYNDROME AND PRE-PUBERTAL SPONTANEOUS IMPROVEMENT IN FRACTURE RISK: A CASE REPORT

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INTRODUCTION
Mc-Cune Albright Syndrome (MAS) is a rare disorder characterized by skeletal lesions, skin hyperpigmentation and hyperfunctioning endocrinopathies. We report a case of MAS with polyostotic fibrous dysplasia (FD) and highlight the natural history where the incidence of fracture starts to dwindle down once they reach adolescence.

RESULTS
A 34 year-old male presented with multiple spontaneous long limb fractures since the age of 2 years old. In addition, he has multiple café au lait spots and endocrinopathies namely, hyperthyroidism and normocalcemic hyperparathyroidism which was complicated by bilateral nephrolithiasis. The hyperthyroidism was treated with radioiodine which rendered him hypothyroid requiring thyroxine replacement. For his hyperparathyroidism, he refused any surgical intervention. With regard to his polyostotic FD, he had a total of 19 fractures over a 9-year period from the age of 2 to 11 years old. Subsequently, his fracture rate reduced markedly occurring 1to 2 times every 6 years. At the age of 15 to 17 years, he received multiple cycles of IV bisphosphonate. Thereafter, he only required intermittent IV bisphosphonates. His last fracture was at the age of 24 years old. At present, he is on a yearly IV zoledronic acid therapy. As he refuses parathyroid surgery, the plan is to give him cinacalcet in order to control his hyperparathyroidism.

CONCLUSION
This case illustrates vividly how the risk of fracture in MAS starts to improve markedly once affected individuals enter the second decade of life. However, the endocrinopathies associated with MAS tend to continue with the risk of new endocrinopathy occurring as they grow older.