EuroSciCon

11th International Conference on

Endocrinology and Diabetology

August 09-10, 2018
Madrid, Spain

Yasuo Imanishi, J Clin Mol Endocrinol 2018, Volume 3
DOI: 10.21767/2572-5432-C2-004

HYPOPHOSPHATEMIC RICKETS AND OSTEOMALACIA: PATHOGENESIS, DIAGNOSIS AND TREATMENTS

Yasuo Imanishi

Osaka City University, Japan

Chronic hypophosphatemia develops rickets and osteomalacia characterized by impaired mineralization of bone matrix. Rickets, such as X-linked hypophsohatemic rickets (XLH), occurs in childhood before the closure of epiphyseal plate and results in growth retardation and bone deformities. On the other hand, osteomalacia occurs in adulthood with severe muscle weakness and bone pain. Tumor-induced osteomalacia (TIO) is an underrecognized paraneoplastic syndrome presenting with hypophosphatemic osteomalacia, which is one of causes of adult onset osteomalacia. Recently elevated circulated levels of fibroblast growth factor 23 (FGF23) was identified as a cause of hypophosphatemic rickets/osteomalacia such as XLH and TIO. FGF23 is a bone-derived hormone, which regulate phosphate and vitamin D metabolism. Chronic elevation of circulated FGF23 causes hypophosphatemia by urinary phosphate wasting and attenuate activation of vitamin D, resulted in rickets and osteomalacia. Circulating FGF23 measurement is required to obtain proper diagnosis in rickets and osteomalacia. In familial rickets, genetic testing such as PHEX gene is recommended. In TIO, it is difficult to identify the causative tumor, which secrete FGF23. Recently, somatostatin receptor scintigraphy was reported to be useful for the diagnosis and localization of causative tumors. Conventional therapies for these FGF23-induced rickets and osteomalacia are active vitamin D and/or inorganic phosphate, however, the clinical efficacy of these therapies are limited. Burosumab, a monoclonal antibody that targets FGF-23, improved renal tubular phosphate reabsorption, serum phosphate levels, linear growth, physical function and reduced pain in children with XLH. This new antibody is promising treatments not only for XLH but also for TIO.

Biography

Yasuo Imanishi graduated from Kagawa Medical School (MD), Japan. He has completed his PhD from Osaka City University, and Postdoctoral Fellowships from Massachusetts General Hospital in Harvard Medical School and University of Connecticut Health Center. He is an Associate Professor of Osaka City University Graduate School of Medicine. His major interests are calcium and phosphate homeostasis in the clinical filed of osteoporosis, rickets & osteomalacia, and chronic kidney disease-mineral and bone disorder (CKD-MBD). He has been working as an Endocrinologist/Nephrologist at Osaka City University Graduate School of Medicine from 2000.

imanishi@med.osaka-cu.ac.jp