

Microbial Compositions in Osteoporotic Patients

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DESCRIPTION

Osteoporosis is characterised by low bone mass and increased bone fragility. The deterioration of bone tissue design in osteoporosis is principally caused by imbalanced bone formation slower than bone resorption, which will increase the incidence of bone fractures. Osteoporotic fractures lead to a significant burden to health services worldwide, particularly increased morbidity and reduced survival in the elderly [1]. Conventional therapeutic interventions on osteoporosis embrace general life-style changing (e.g., diet and regular exercise), and intake of approved medicine (e.g., oestrogen and bisphosphonates). In recent years, a series of studies prompt that gut microbiota colonizing within the intestinal tract not only affected the nutrition metabolism, however additionally contributed to the incidence and progression of osteoporosis. Raisz reported there have been variations of gut microbiota composition between osteoporosis/osteopenia and controls with an Irish cohort of older adults. Drake, et al. analyzed the gut microbiota diversity during a relatively small sample with 5 primary osteoporosis patients, 5 osteopenia patients and 5 healthy controls [2]. However, the analysis findings were based on subjects while not considering sex and therefore the results were also inconsistent.

Osteoporosis is a typical disease of females. One key reason is that the abrupt decline of estrogen hormone production for women after the menopause. Therefore, hormone replacement therapy effectively decreased the risk of bone fracture in post-menopausal women. It indicated that the pathophysiology of osteoporosis may differ between women and men. This analysis aimed to research the alterations in gut microbiota of female osteoporotic patient's victimisation 16S rRNA high-throughput sequencing analysis. Conventional osteoporosis therapies centered on mitigating the loss of bone related to decreases of sex steroids. With increasing information concerning the pathology of osteoporosis, clinical analysis of latest targets for therapeutic intervention is directed to gut microbiome associated to bone resorption and formation. During this study, we tend to conducted 16S rRNA sequencing to match the composition of gut microbiota between osteoporotic patients and management subjects [3].

Our results showed important variations within the alpha diversity and therefore the abundance of specific taxa in gut microbiome related to osteoporosis. To date, there are many studies analyzing the alternations of gut microbiota in osteoporotic patients. Here we tend to centered on the gut dysbiosis of female subjects with osteoporotic condition. One amongst our most notable performance was a transparent distinction between osteoporotic cases and management subjects.

In our study, gut dysbiosis was characterised by a series of differential abundant genera in osteoporosis group. As an example, we tend to found an increased abundance of *Bacteroides* genus in osteoporosis group. As reported, *Bacteroidetes* may be concerned in bone formation and bone resorption by deposition and reaction of serine dipeptide lipids. Another noteworthy genus was *Roseburia* belonging to Firmicutes phylum [4]. As a crucial producer of varied Short-Chain Fatty Acids (SCFAs), *Roseburia* mediate the changes of IGF-1 expression and contributed to bone growth.

Circulating endotoxin, chiefly composed by LPS, was secreted from intestine, and therefore the intestinal permeability determined the secretory levels. Estrogen hormone deficiency will influence intestinal epithelial permeability through mediating estrogen-associated pathways. These findings along pointed to an attainable theory that the estrogen level and the abundance of varied microorganism taxa were altered in osteoporosis status. The combined effects of estrogen hormone deficiency and gut dysbiosis on intestinal permeability will mediate bone loss and osteoporosis via influencing the host metabolism, endocrine, and immunity, which might provide potential targets for the clinical treatment of post-menopausal osteoporosis.

On the opposite hand, many limitations of results should to be taken into thought. Supported the look of cross-sectional study, it absolutely was insufferable to clarify the causative relationship between gut microbiota and osteoporosis. After diagnosis, the changes of gut microbiota may be either a consequence of osteoporosis or the cause [5]. Described the altered profiles of gut microbiota in female osteoporotic patients, and provided evidence for the relationship between dysbiosis and osteoporosis.

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Our results pointed towards to early prevention and clinical management of osteoporosis by monitoring the homeostasis of gut microbiota.

REFERENCES

1. Raisz LG. Pathogenesis of osteoporosis: Concepts, conflicts, and prospects. *J Clin Invest.* 2005;115: 3318-3325.
2. Drake MT, Clarke BL, Lewiecki EM. The pathophysiology and treatment of osteoporosis. *Clin Ther.* 2015;37: 1837-1850.
3. Demontiero O, Vidal C, Duque G. Aging and bone loss: New insights for the clinician. *Ther Adv Musculoskelet Dis.* 2012;4: 61-76.
4. Feskanich D, Willett W, Colditz G. Walking and leisure-time activity and risk of hip fracture in postmenopausal women. *JAMA.* 2002;288: 2300-2306.
5. Hass MA, Nichol P, Lee L, Levin RM. Estrogen modulates permeability and prostaglandin levels in the rabbit urinary bladder. *Prostaglandins Leukot Essent Fatty Acids.* 2009;80: 125-129.