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Biomechanics in Pathological Fractures

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Editorial

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Abbreviations: FE: Finite Element; CTRA: Computed Tomography Based Rigidity Analysis; CT: Computed Tomography Scan.

Editorial

In the setting of a benign or malignant lesion the altered physiology and mechanics may lead to pathological fractures. Patient outcome depends on proper diagnosis, staging and treatment of pathological fractures. On conventional X rays or computed tomography scan (CT) images it becomes extremely difficult to assess the clinical fracture risks. Accurate predication can't be made by experienced clinicians [1].

In common cancer types such as thyroid, kidney, lung, prostate and breast cancer the primary cell tumor cell seeding will occur in bone [2-4]. Tumor cells invades the fertile environment for seeding in bone marrow of the axial skeleton of long bones, skeletal parts of skull, ribs and spine [3,5,6]. The physical status and expected survival of patient with pathological fractures which require complex surgical procedures are weighed by the operating surgeons. Preventive surgery for pathological lesion with an impending fracture is better than surgical treatment of actual pathological fractures which is less complex and is having better survival rates [3,6,7]. The lesions which don't jeopardize the bone mechanical integrity is treated with standalone or combination of biosphospates, hormonal therapy, chemotherapy, analgesics, radiation therapy conservatively to relive the pain and prevent further seeding of primary cell tumor [8].

Patient-specific finite element (FE) and computed tomography based rigidity analysis (CTRA) are the mechanical models for the fracture risk assessment [9]. These are extensively studied in past two decades. The outcome of the studies is positive and they are now in clinical practice.

We will further focus on bone and muscle metabolic interaction which are interconnected both anatomically and physiologically. Bone and muscle releases secretory factors with paracrine-endocrine cross talk which influence nearby tissues, distant organs, muscle to bone and bone to muscle interaction [10] (Table 1).

As the days advance the latest research in treating the pathological fractures are improving with combination of monoclonal antibodies, targeted immunotherapy etc. for reduction of pathological lesion and prevention in loss of bone density, osteopenia, osteoporosis and mechanical strength. The early diagnosis and proper intervention will reduces the occurrence of pathological fractures which increases the quality of life.

Myokines	Secretion Stimulants	Bone Metabolism
Growth Factors		
IGF-1	Resistance Exercise	Stimulates Formation
FGF-2	Eccentric Muscle Contraction	Stimulates Formation
GDF-8	Muscle Damage / Atrophy	Supresses Healing / Formation
TGF-β1	Muscle Damage / Atrophy	Supresses Healing / Formation

Matrix Molecules		
SPARC	Resistance Exercise	Promotes Mineralisation
MMP-2	Resistance Exercise	Promotes Healing / Remodelling
BMP-1	Blast trauma to Muscle	Procollagen Cleaving / Bone Formation
Inflammatory Factors		
IL-6	Muscle Contraction	Bone Resorption / Turnover
IL-7	Muscle Contraction	Bone Resorption
IL-15	Resistance Exercise	Increase Bone / Decrease Adiposity

Table 1: Myokines (peptides) secreted by muscle to influence bone, the mechanisms which stimulate release, and the bone metabolism outcomes.

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