



BONE METABOLISM BY PROF JOHN ATINGA

- CASE PRESENTATION

- 50-year-old lady with 1 ½ year history of back pain which became worse in the last few weeks. Pain was worsened with coughing and sneezing in the lumber region.
- She was found to be tender along the spine particularly in the lumber region.
- There are no any other neurological deficit.
- The rest of her examination were normal.
- Blood parameters were normal except for an elevated CRP. and ESR

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- ▶ THE FUNCTIONS OF THE BONE IN THE BODY;
 - ▶ PROTECTION OF THE VISCERA
 - ▶ RESERVOIR FOR MINERALS[CALCIUM]
 - ▶ LEVERAGE IN MOBILITY



IMPACT LOADING DICTATES THE FOLLOWING

CHANGES IN LENGTH WIDTH AND SHAPES

ENERGY IS ABSORBED

BONE DISPLAYS ELASTIC/PLASTIC PROPERTIES

ASCENARIO- BONE MUST BE IN A PERMANENT DYNAMIC STATE OF ;

MODELING/REMODELING

THESE OCCUR AGAINST FIXED STRUCTURALDESIGN(FIXED BY GENETICS)

MATERIAL COMPOSITION VS FUCTIONAL DEMAND

ENVIROMENTAL INTERPLAY



BMU[BASIC METABOLIC UNIT]

THIS CONSTITUTES VEHICLE BONE CELLULAR RESPONSE

OSTEOCYTE

OSTEOBLAST

OSTEOCLAST



BONE MODELING

CONSTRUCTION[ANABOLIC PROCESS]

AN OSTEOLASTIC PROCESS –CONCERNED WITH GROWTH/ BONE MASS ACCRURAL
GROWTH FACTORS/HORMONES/NUTRIENTS/MINERALS /VITAMINS

CALCIUM

THE MAIN MINERAL IN BONE HYDROXYAPATITE CRYSTALLINE COMPONENT OF BONE IS ABSORBED UNDER THE INFLUENCE OF VITAMIN D3 IN THE DUODENUM AND JEJUNUM

AN ADULT NEEDS ONE KG/DAY

VITAMIN D3

A VITAMIN MANUFACTURED BY THE BODY FROM THE SUN BY ULTRAVIOLET RAYS ON THE SKIN. CHOLECALCIFEROL IS TAKEN TO THE LIVER, WHERE 25-HYDROXYLATION TO D2 IS DONE.

THE ACTIVE FORM IS D3 WHICH IS HYDXYLATED AT ONE POSITION IN THE KIDNEY.

D3 FUNCTIONS MAIN FUNCTIONAL UNIT FOR BONE HEALTH IT IS ALSO FUNCTION AS ANTICANCER IN PUSHING CELL MATURATION CELL CYCLE OF DEVELOPMENT.

AMONG OTHER DIVERSE FUNCTIONS INCLUDE IMMUNITY BOOST ; MUSCLE DEVELOPMENT.

HORMONE



PRODUCED IN THE PARATHYROID GLAND

MOBILIZES CALCIUM FROM THE BONE STORES

LOW CALCIUM LEVELS IS SENSED CALCIUM SENSORS ON RECEPTORS ON CELL SURFACES WHICH THEN CAUSES THE GLAND TO PRODUCE PTH

THE GOAL IS LEVEL OF IONIZED CALCIUM MUST BE MAINTAINED IN THE BLOOD



BONE CALCIUM MOBILISATION


THE NET EFFECT OF PTH ACTION IS BONE RESORPTION

PTH WILL ONLY WORK THROUGH BMU

IT MUST STIMULATE CATABOLIC PATHWAY –OSTEOCLASTIC CELL MECHANISM OF BONE LYSIS

SUSTAINED RELEASE OF PTH CAUSES BONE LYSIS BUT IF RELEASED CYCLICALLY THEN IT HAS BEEN SHOWN TO BE ONE OF THE MOST POTENT BONE ANABOLIC FACTORS

BMU [BONE MULTICELLULAR UNIT]



THIS IS MADE UP OSTEOBLAST- OSTEOCLAST CELL UNIT

FROST THEORIZED THAT THESE CELL ACT IN COUPLED MANNER

THE SENSORY INPUT IS PICKED BY OSTEOCYTE PATHWAYS IN THE CANALICULI

BMU OPERATES AT THREE LEVELS

ENDOSTEAL COMPARTMENT

INTRACORTICAL COMPARTMENT

TRABECULAR COMPARTMENT

ADAPTATION

BONE IS A COMPLEX STRUCTURE OF MINERALS [HYDROXYAPATITE] AND MATRIX OF PROTEINS AND TYPE 1 COLLAGEN.

BONE HAS MECHANICAL SURMATED CHARACTERISTICS

IT RESPONDS TO LOADING BY LAYING MORE BONE TO MEET THE FUNTIONAL NEED

BUT OVERALL STRUCTURAL DESIGN NEVER CHANGES

SUBJECTED TO FREQUENT LOADS WILL CAUSE INCREASE OF CROSS-SECTIONAL AREA BUT LIGHTNESS CANT BE COMPROMISED SO THE RADIUS MAY INCREASE BUT CORRESPONDING ENDOSTEAL RESORPTION WILL OCCUR

THE INCREASE OF THE RADIUS [D]] IS PROPORTIONATE BENDING RESISTANCE TO 4TH POWER

THE WHOLE METABOLIC STUDIES IS DEALING WITH WHAT HAPPENS TO FIFTY PER CENNT ACCOUTED FOR BY MATERIAL [MATRIX] OF THE BONE

BONE MATURITY IS ATTAINED AT 30YRS

BONE CELLS



OSTEOCYTES

THEY CONSTITUTE 90% OF THE BONE CELLS.

CELL CONCENTRATION IS 10,000/CC OF BONE.

EACH CELL HAS GOT 50 PROCESSES /CELL

THE CELL IS ASSOCIATED WITH HARVESIAN SYSTEM AND VOLKMAN CANALS AND LACUNA SYSTEM

IT IS THE OCHESTRA OF BONE METABOLISM .

IT DICTATES THE PROCESSES OF MODELLING AND REMODELLING.

CRACKS WILL AFFECT IT.

LOAD WILL BE SENSED BY IT.

SOME IMPORTANT CYTOKINES PRODUCTIONS

- ▶ FIBROBLAST GROWTH FACTORS I.E FG23
- ▶ SCLEROSTIN –WNT INHIBITOR
- ▶ OSTEOCACIN
- ▶ OSTEOPONTIN
- ▶ OSTEONECTIN

OSTEOBLAST

BONE PRODUCING CELL

BONE MATRIX

BONE COLLAGEN PRODUCTION

IT ORIGINATES FROM MESENCHYMAL STEM CELLS .

OSTEOCLAST

IT ORIGINATES FROM MONOCYTE /MACROPHAGE CELL LINE.

IT RESIDES IN HOWSHIP LACUNAE

IT'S THE AGENT OF BONE RESORPTION .

DISEASES

THESE ARE BASICALLY DISEASES THAT AFFECT THE INTEGRITY OF THE BONE.

THE COMMONEST KNOWN CONDITION ARE;

1.OSTEOPOROSIS.

IT'S A BONE DEFICIENCY CONDITION COMMONLY SEEN IN WOMEN.

IT MAY LEAD TO BONE FRACTURE.

2.TUMORS

MOST OF THE TUMORS INVOLVING THE BONE CAUSE BONE WEAKNESS

EXAMPLE;- MULTIPLE MYELOMA

3.HORMONAL DISEASES

THESE LEAD TO BONE WEAKNESSES

EXAMPLE;- HYPERPARATHYROIDISM

-THYROTOXICOSIS

INVESTIGATIONS OF METABOLIC BONE

DISEASE

1. SIMPLE X-RAYS WILL PICK UP BONE CHANGES WHEN MORE THAN 30% OF BONE MATERIAL IS LOST. ITS NOT VERY ACCURATE .

2. CT SCAN-2D SCANS VS 3D SCANS ARE MUCH MORE ACCURATE IN PICKING SMALL BONE LOSSES .IT HAS BEEN USED TO DETERMINE THE BONE DENSITY WHICH IS ONE MAJOR CRITERIA FOR DETERMINING THE BONE DENSITY.THE BONE DENSITY IS USED TO CLASSIFY THE BONE LOSS STATUS – DXA /QCT DENSITOMETRY.

3. BLOOD TESTS - CALCIUM LEVELS

- ▶ -HORMONAL LEVELS
- ▶ -BONE FORMATION AND BONE LOSS MARKERS –AKL
- ▶ -TELOPEPTIDES

4. URINE TESTS-HYDROXYPROLINE LEVELS

- N-TELOPEPTIDES
- AND OTHERS

MANAGEMENT

GENERAL PRINCIPLES OF METABOLIC BONE DISEASE DEPENDS ON THE DIAGNOSTIC FINDINGS.
REPLACEMENT THERAPY PARTICULARLY THE HORMONAL AND NUTRITIONAL CAUSES.

IMMOBILIZATION

SPLINTAGE

BISPHOSPHONATES

VITAMIN D3 AND CALCIUM REPLACEMENT