DISCUSSION AND SUMMARY

Rickets is a systemic disease of infancy and childhood in which the calcification of growing skeletal elements is deficient. It is equivalent of osteomalacia in mature skeleton. Prematurity is a predisposing factor.

Clinically there are several classifications of different types of rickets according to the aetiology and the age of occurance but vit. D deficiency rickets is a main clinical problem in Egypt rather than any other types of rickets.

Infantile vit. D. deficiency rickets usually develops between 6th month and one year of age and resulting from deficiency of vit. D in the diet or from lack of exposure to ultra-violet rays. The human skin contains pro-vitamin D3 (7-dehydrocholecalciferol) which is activated by ultra-violet rays. This vitamin is not active, but is transported to the liver where it is hydroxylated and from there to the kidney where it is hydroxlated again and becomes dihydroxy-vit. D. which is the most active form of the vitamin. It acts as a hormone. Liver and kidney disease may cause rickets of hydroxylation cannot take place.

Rickets is characterised by defect of bone growth due to lack of normal mineralisation. Cartilage cells of the epiphyseal plate grow and reproduce normally, but fails to

calcify and degenerate as normal. These cells continue to proliferate but with disturbance of the columnar configuration causing patchy widening of the epiphyseal plate seen roentgengraphically as a radiolucent area, so that the end of bones are frayed and the epiphyseal line is irregular.

Mineralisation of the osseus tissue and cartilage matrix fails, the zone of preparatory calcification does not form and malleable non rigid tissue is produced instead. It become compressed, cupped and flared.

Changes in bone shafts are caused by failure of osteoid mineralisation and a shell of sub-periosteal osteoid tissue surrounds the shaft. The osteoid continues to heap, the underlying cortical bone is resorbed and replaced by un-mineralised osteoid, thus the shaft loss rigidity and molding and fractures occures.

By administeration of vit. D. healing occurs and the zone of preparatory calcification becomes identifiable. Cartilage cells then degenerate normally and invasion of blood vessels and osteoblasts allows normal epiphyseal growth. The skeleton is rapidly mineralised and the sub-periosteal osteoid collections are mineralised and resorbed and bone outline returns.

The serum calcium level in vit. D. deficiency rickets ranges from normal to slightly lowered and the serum phosphate concentration is slightly lowered. The serum alkaline phosphatase level is elevated but not specific.

Clinically, softening of skull bones or craniotabes may be presented initially followed by frontal and parietal bossing in later stages of the disease. The wrists and ankles swell. Also the ribs show enlargement of the costo-chondral junction "Rachitic rosary". Increased irritability and sweating also occurs.

In advanced rickets, calvarial sutures are open and soft with box-like appearance skull, denition is delayed, the sides of the thorax are flattend and develops pigeon breat deformity and curvature of the long bones also occurs.

Roentgenographically, the characteristic changes occur in the ends of the long bones. These changes are similar for rickets of all aetiologies **(Greenfield, 1975)**. Epiphyseal widening and ill definition of zone of provisional calcification followed by metaphyseal cupping and widening owing to the pull of muscular and ligamentous attachment. In the shafts of long bones uncalcified sub-periosteal osteoid is present, causing loss of sharp cortical outline and allowing development of bowing deformities particularly in tibia.

Bone texture is also coarsened. Green-stick fractures are also common Pseudofractures are rarely present. The appearance of ossification centers in the epiphysis of small bones are delayed because of the lack calcification.

With healing, mineralisation of the zone of provisional calcification appears as a dense line in the epiphyseal cartilage, separated from the metaphysis. The cupping increases. Remineralization of the sub-periosteal osteoid appears as periosteal new bone formation which may be solid and laminated.

Calcification of the ossification centers occurs with a marginal ring shadow that gradually fuses with the center.

Although complete healing usually occurs, residual deformities may persist.

The rachitic skull in active phase of the disease characterised by bossings at frontal and parietal emineences that are devoid of mineral contents and so, are not well visualised roentgenographically with the onset of healing the calcium contents of bosses increases and the healed hyperostosis become more evident radiologically **(Caffey, 1972)**.

This rachitic bossing persist in adult life and giving the head a "Caput quadratum" deformity.

Due to bone softening in rachitic skull especially skull base, basilar invagination occurs, which can be demonstrated radiologically in postero - anterior view by cephalic angulation of a line drawn along the superior margins of petrous ridges (normally this line directed caudally). And in lateral view by increase in the basal angle more than 150° (normally between 125-142°), and by protrusion of the odontoid process ½ cm. or more above the Mc-Gregor's line.

The rachitic vertebrae are characterised by rarefaction either diffuse or in the form of globular areas of rarefaction. The heights of the vertebral bodies are reduced with consequent widening of the intervertebral spaces.

The spinal deformity in the form of dorso-lumbar Kyphosis occurs in several cases of rachitic children who sit-up.

The radiological changes in rachitic chest appear early in the anterior ends of the ribs in the form of splaying and irregularity and impaired endochondral ossification.

Gross demineralization of the thoracic cage associated with formation of Harrison sulcus at the site of costal origin of the diaphragm.

The rachitic deformities in the thoracic cage usually associated with chest infection as bronchitis and broncho-pneumonia.

These roentgenographic findings of rickets simulate other many skeletal disorders and the radiological differentiating signs should be considered.

Hypophosphatasia is similar radiologically to rickets but with more pronounced demineralisation and marked liability to fractures.

In scurvey the deficiency is mainly in bone matrix not in its mineralisation. So, the zone of provisional calcifications more dense with sharp definition and marked periosteal reaction is noticed due to sub-peri-osteal haematoma. Scurvey is also characterised by Pencil-thin epiphyseal cortex.

The widened metaphysis which is seen in rickets should be differentiated from other causes of metaphyseal widening as cretinism, leukaemia, hypervitaminosis D, osteopetrosis and congenital syphilis.

Basilar invagination in rachitic skull due to softening of bones of skull base. This radiological sign is also seen in other conditions leading to bone softening as Paget's disease, fibrous dysplasia, osteogensis imperfecta Achondroplasia and cleidocranial dysplasia.

The double contour of the long bones seen in rickets is not fairly due to periosteal elevation and periosteal reaction but mainly due to non-mineralised sub-cortical osteoid tissue formation and falsly termed periosteal reaction and so must be differentiated from other causes of periosteal elevation as congenital syphilis, scurvey, prematurity and Caffey's disease.

It is to be noted that the radiation risks for a paediatric patients include genetic, leukogenic carcinogenic factors and cataret production. And although, theoretically there is really no safe dose but the risk will be greater if a large portion of the body irradiated or if a higher dosage is used and also, the sensitivity of the irradiated area should be considered.

A proper communication between the radiologist and paediatrician is essential for delivery of a good radiologic care.

Also, a good communication between the radiologist and technologist is essential for the maintenance of a good quality films.

The rooms for paediatric radiology should have the biggest x-ray generators possible. High milliamperage is essential so that short times can be used.

Accurate callimators with light localization is essential.

Pleasant and safe surroundings in the radiologic room is essential to gain a sympathetic attitude toward the child to ensure a good examination.