CHAPTER 7

Metabolic, Endocrine and Toxic Disorders

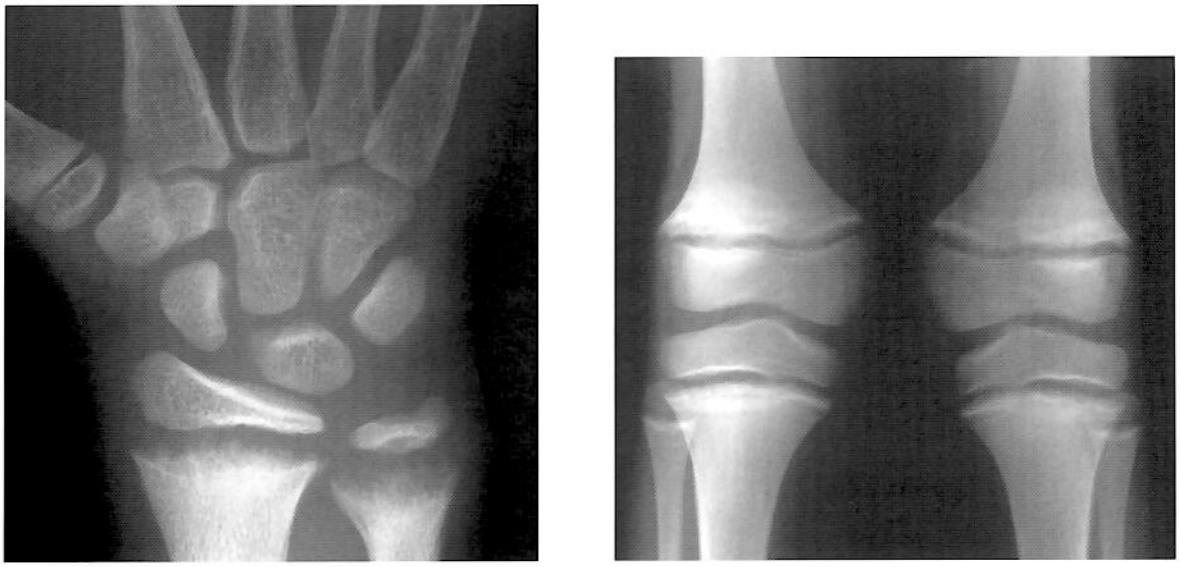
7.1 Metabolic Disturbances

7.1.1 Osteoporosis

- ***osteoporosis*** is the most common metabolic bone disease and is causing a decrease in bone mass. The microstructure of bone is normal, but the quantity of bone is reduced.
- the loss of bone density increases the risk of fractures, particularly the neck of femur, spine (wedge compression fractures, fig 4.141), and distal radius.
- there are numerous causes of osteoporosis including;
  - — deficiency states and malnutrition
  - — steroid therapy and Cushing's disease
  - — chronic liver disease
- the radiographic features of osteoporosis are:
  - — reduced bone density (at least 40–50% reduction must occur before it can be recognized on the radiograph)
  - — loss of trabeculae (remaining trabeculae appear more prominent)
  - — cortical thinning
  - — fractures (vertebral wedge compression fractures)
- osteoporosis is a specific term referring to loss of bone mass and is not synonymous with osteopenia or de-ossification. Osteopenia and demineralization are general descriptive terms referring to reduced bone density, irrespective of the cause.

7.1.2 Rickets and osteomalacia

- ***rickets and osteomalacia*** are the same condition, occurring in children and adults respectively. There is lack of mineralization of normal osteoid.
- caused by ***vitamin D deficiency*** which may be due to;
  - — dietary deficiency
  - — gastrointestinal malabsorption
  - — liver disease
  - — anticonvulsant therapy
  - — renal osteodystrophy
    (combination of osteomalacia and secondary hyperparathyroidism)
  - — lack of sunlight (ultraviolet light)
- radiographic features common to both rickets and osteomalacia include;
  - — generalized loss of bone density (osteopenia)
  - — loss of corticomedullar differentiation
    (cortex and medulla of bone no longer seen as separate structures)
  - — in severe cases bone softening with development of deformity
- the radiographic features of rickets are as above plus (figs 7.1);
  - — splayed frayed metaphyses with widening of the growth plate
  - — in less severe cases lucent metaphyseal band will be seen
- radiographic features of osteomalacia as above plus ***pseudofractures*** (demineralized zones) (fig 7.2);
  - — a narrow lucent zone running perpendicular to cortex
  - — this is due to insufficiency stress fractures repaired by non-ossified osteoid
  - — frequently bilateral and symmetrical
  - — common sites include pubic rami, proximal femur, scapula, ribs and ulna
  - — uncommon in rickets



(a) (b)

Figure 7.1
PA of the wrist (a) and knees (b) in a child with dietary rickets. There is generalized reduced bone density with widening of the growth plates and splaying of the metaphyses.

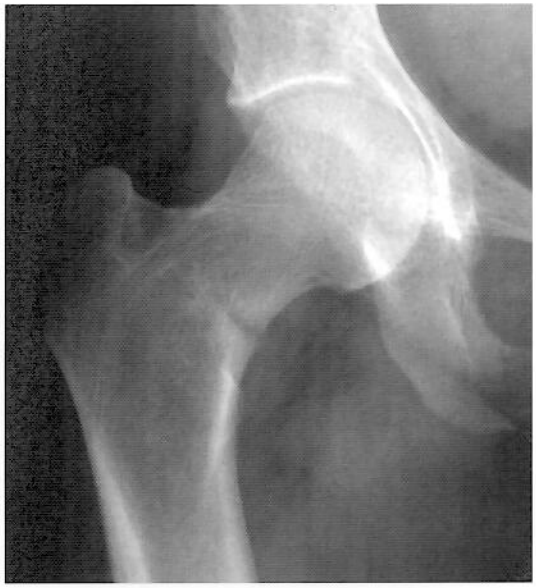


Figure 7.2
AP of the hip of an adult with osteomalacia. There is generalized reduced bone density and loss of corticomedullary differentiation of the femoral head. The lucent line traversing the medial cortex of the femoral neck is the typical appearance and site for a pseudofracture.

7.1.3 **Scurvy**

- ***scurvy*** is due to ***vitamin C deficiency*** resulting in abnormal collagen formation.
- the radiographic abnormalities in adults are nonspecific generalized loss of bone density (osteopenia).
- scurvy is rare under 6 months of age
- the radiographic features in children include (fig 7.3):
  - — generalized loss of bone density (osteopenia)
  - — subperiosteal haemorrhage leading to subperiosteal new bone formation
  - — small sharply marginated epiphyses
  - — dense metaphyseal line
  - — metaphyseal corner fractures

- the radiographic findings of scurvy must be distinguished from those caused by child abuse (the battered child syndrome) which in some communities is more common than scurvy.

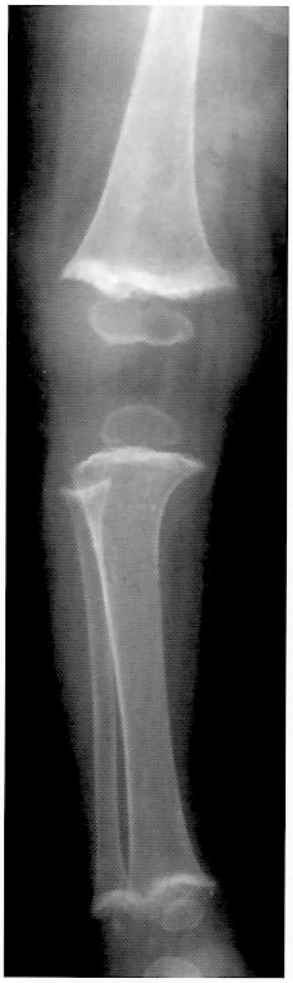


Figure 7.3
Lower leg in a child with scurvy. There is generalized reduced bone density, sharply defined epiphyses with sclerotic metaphyseal margins.

7.2 **Endocrine Disturbances**

7.2.1 **Acromegaly**

- skeletal overgrowth is known as ***gigantism*** in the immature skeleton, and ***acromegaly*** in the adult. Both result from excessive growth hormone production by a pituitary gland adenoma.
- the radiographic features include:
  - — skull: pituitary fossa enlargement
    frontal bossing (thickening of the frontal bones)
    enlarged paranasal sinuses
  - — spine: enlarged vertebral bodies
    posterior scalloping (concave edges)
  - — hand: large hands with spade-like terminal phalanges (fig 7.4)
    widening of joint space due to thick cartilage
    prominent bony projections at tendon attachments

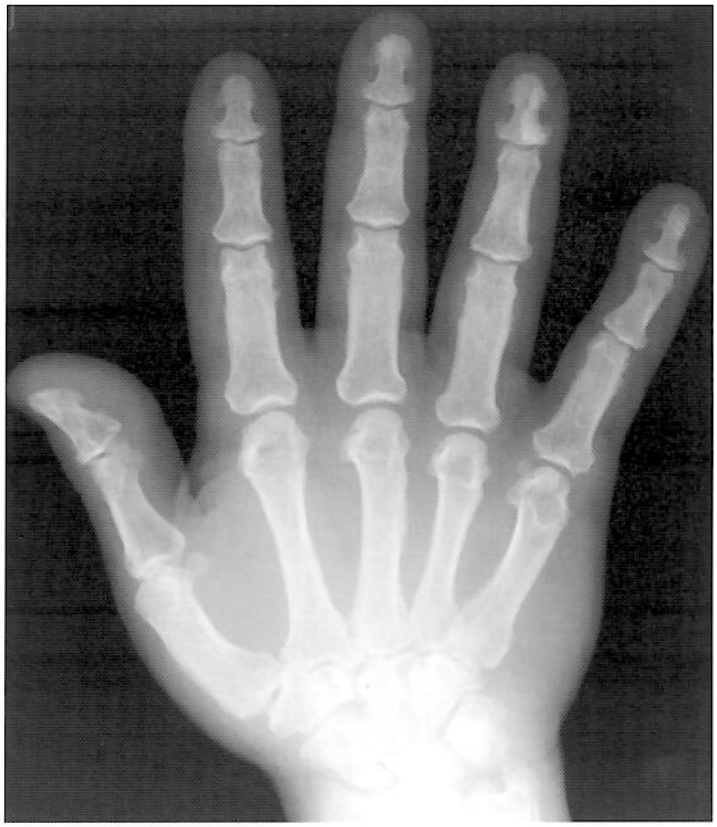


Figure 7.4
Acromegaly. Overall enlargement of the hand with spade-like terminal phalanges, wide joint spaces and hook-like appearance to the distal metacarpals.

7.2.2 **Hypothyroidism**

- it is due to insufficient production of thyroxine.
- in children (cretinism) there is delayed skeletal maturation and growth retardation. The epiphyses, particularly the proximal femur, appear irregular and fragmented (fig 7.5).

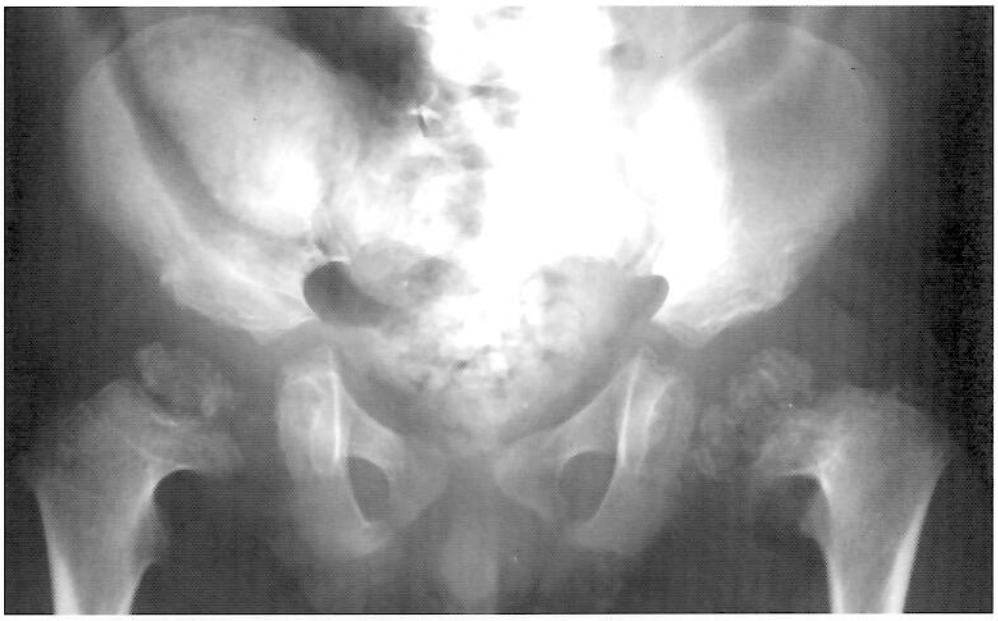


Figure 7.5
AP of the pelvis in a child with hypothyroidism (cretinism). There is irregular fragmentation of the *proximal femoral epiphyses mimicking a congenital* epiphyseal dysplasia. Note the severe constipation which is another feature of this condition.

7.2.3 **Hyperparathyroidism (HPT)**

- there are three forms of HPT; primary, secondary and tertiary.

  Primary HPT is due to excessive production of parathormone as a result of parathyroid adenoma (75%), hyperplasia or carcinoma.

  Secondary HPT is due to parathyroid hyperplasia in response to persistent hypocalcaemia (low blood calcium) and is seen in rickets, osteomalacia and chronic renal failure.

  Tertiary HPT applies to cases of secondary HPT which gives rise to autonomous HPT (i.e. HPT exists irrespective of the initial low blood calcium).

- the radiographic appearances of HPT are due to bone resorption which results in loss of bone density (osteopenia) and include (figs 7.6 and 7.7);
  - — subperiosteal erosion (particularly along the radial aspect of phalanges in the hand)
  - — subchondral resorption (erosions of the distal clavicle, pubic symphysis and SI joints)
  - — Brown tumours (see below)
- ***Brown tumours***, called brown because of their macroscopic blood stained appearance, are lytic expansive lesions resulting from intense localized osteoclastic (bone-resorbing cells) activity. They are quite well defined and can mimic true tumours. They are less common in secondary HPT.
- in secondary HPT, the radiographic appearances are as in primary HPT with features of the underlying cause i.e. rickets, osteomalacia or chronic renal failure. Calcification of arteries and soft tissues occurs in secondary HPT particularly in cases with chronic renal failure.
- ***renal osteodystrophy*** is the term applied to the bone changes associated with chronic renal failure. These include secondary HPT, osteomalacia and osteosclerosis.

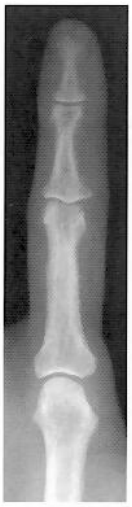


Figure 7.6
Primary hyperparathyroidism. Generalised osteopenia, terminal phalangeal and subperiosteal resorption.

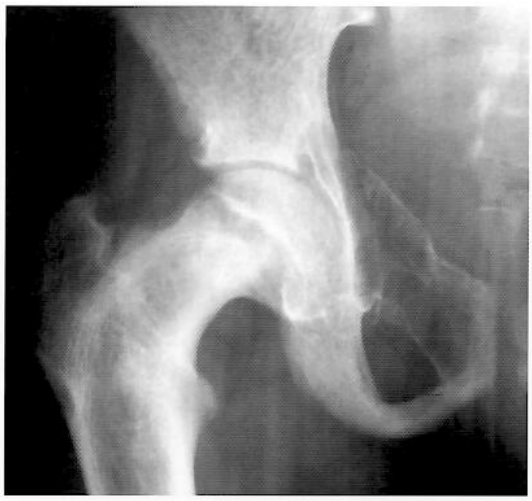


Figure 7.7
Secondary hyperparathyroidism due to chronic renal failure. There is a Brown tumour in the pubis and evidence of rickets with widening of the proximal femoral growth plate.

7.3 **Toxic Reactions**

7.3.1 **Lead poisoning**

- lead poisoning occurs in children who ingest lead-containing paints or water delivered by lead pipes. The typical radiographic appearance is a transverse band of metaphyseal sclerosis (fig 7.8).
- a similar appearance may be seen with Bismuth poisoning.

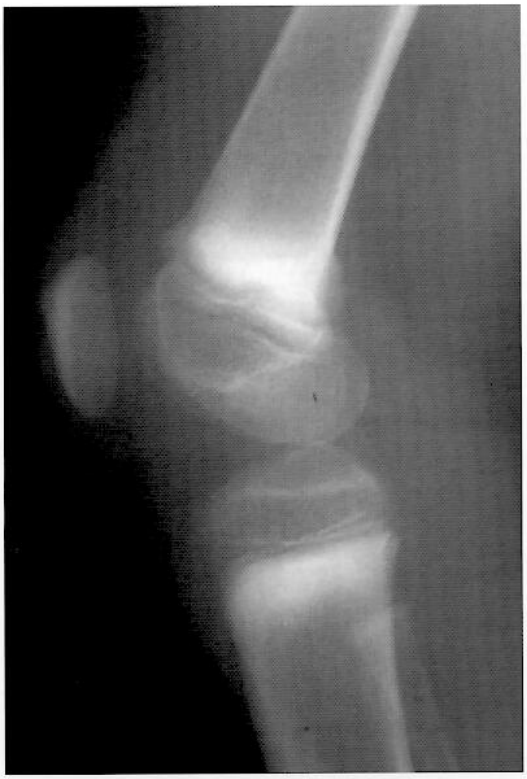


Figure 7.8
Child with lead poisoning. The diagnosis is indicated by the presence of a sclerotic metaphyseal bands.

7.3.2 **Fluorosis**

- fluorosis occurs due to chronic fluoride poisoning. It is endemic in some parts of the world.
- there is a generalized increase in bone density with prominence of ligamentous and musculotendinous attachments. It needs to be distinguished from other causes of diffuse increased bone density such as prostatic metastases and myelofibrosis.