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Idiopathic intracranial hypertension as rickets unusual manifestation: establishing diagnosis through a skeletal survey imaging

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Learning Objectives: To increase awareness of any various unusual clinical manifestation of rickets, and skeletal conventional imaging modality solely can establish the diagnosis of rickets.

Background: Rickets is the most common cause of bone mineralization disruption in children with vitamin D deficiency and malnutrition remains a major health problem in Indonesia. Prevalence of nutritional rickets placed among the 5 most common diseases in children in developing regions. Idiopathic intracranial hypertension is a rare syndrome characterized by unknown cause of elevated intracranial pressure with absence of ventriculomegaly, abnormality of brain parenchyma, normal cerebrospinal fluid cell count and protein count. Rickets is diagnosed based on classical clinical finding, in conjunction with laboratory and radiology result. Skeletal survey using X-Ray as the solely imaging modality.
Idiopathic Intracranial Hypertension As Rickets Unusual Manifestation: Establishing Diagnosis Through A Skeletal Survey Imaging

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Learning objectives

• To increase awareness of any various unusual clinical manifestation of rickets, such as idiopathic intracranial hypertension
• To describe the mechanism underlying rickets radiology findings
• To establish Rickets diagnosis through skeletal survey as the sole imaging modality

Background

Rickets is the most common cause of bone mineralization disruption in children with vitamin D deficiency. Vitamin D deficiency rickets is defined as decreased levels of 25-hydroxyvitamin D (25OHD) to less than 30 nmol/L, and an increase of parathyroid hormone (PTH) level. This laboratory finding is important to source up rickets aetiology, as different primary conditions show different laboratory findings. Elevation of PTH will induced more bone resorption, decreased urinary calcium excretion while promoting phosphate excretion in the kidney, which indicates a worse demineralization condition.¹

One of the most common cause of vitamin deficiency is low intake of calcium and vitamin D. Malabsorption, dark skin children/infants, exclusively breastfed children are the most common cause of nutritional rickets worldwide²,³ while in developing countries and low income populations, malnutrition remains a major health problem and rickets is placed among the 5 most common diseases in children in developing countries.

Manifestations:

Manifestation of rickets can be divided as skeletal and non-skeletal (Table 1.) Idiopathic intracranial hypertension (IICH) is a rare syndrome characterized by unknown cause of elevated intracranial pressure with absence of ventriculomegaly, abnormality of brain parenchyma, and normal cerebrospinal fluid cell counts and protein levels.⁴

Table 1. Skeletal & Non-skeletal features of Rickets²,5,6

<table>
<thead>
<tr>
<th>Skeletal features</th>
<th>Non-Skeletal features</th>
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- Slowing linear growth
- Growth plate widening
- Metaphyseal swelling at long-bone ends
- Rickety rosary
- Bowing deformity of long bones
- Frontal bossing
- Craniotabes
- Persistent anterior fontanel
- Harrison's sulci
- Greenstick fractures
- Pectus excavatum/ carinatum
- Frontal/parietal skull bossing
- Osteitis fibrosa cystica
- Secondary hyperparathyroidism
- Lordosis, scoliosis, kyphosis
- Bone pain
- Hypocalcaemia convulsions
- Hypocalcaemia cardiac failure
- Hypotonia
- Delayed motor milestones
- Carpopedal spasm
- Enamel hypoplasia
- Delayed dentition
- Failure to thrive
- Fractious, irritable child
- Laryngospasm
- Defective ventilation with respiratory obstruction
- Hypochromic anaemia
- Von Jaksch-Luzet syndrome
- Splenomegaly & hepatomegaly
- Idiopathic intracranial hypertension /pseudotumor cerebri

A diagnosis of rickets has to be considered when there are symptoms in growing age children with sign of increased intracranial pressure, such as vomiting, nausea, headache, and papilledema. Body contour analysis should be done when skeletal abnormalities where delayed global development features are seen upon examination, hence a metabolic origin such as rickets should be suspected. Brain computed tomography scan (CT scan) must be done to rule out any other underlying aetiology of increased intracranial pressure.

The underlying mechanism of IICH still remains unknown, but several hypotheses deduct that there is an increased of intracranial pressure secondary to either cerebral spinal fluid (CSF) hypersecretion caused by increased choroid plexus adenylate cyclase activity, and mechanism of axonal damage. Deficiency of Vitamin D will lead to hypocalcaemia thus causing a decrease in axonal transport which causing axonal swelling and increase CSF hypersecretion with normal pressure.

**Diagnosing Rickets:**

The diagnosis of rickets is based on history taking, physical examination and biochemical testing. Plain radiographs are needed for further confirmation of diagnosis. Skeletal survey using X-Ray as the primary imaging modality used to achieve significant findings in diagnosing rickets.
Laboratory findings:

- Vitamin D deficiency is defined as 25OHD level below 30 nmol/L (1 ng/mL equals to 2.5 nmol/L) defined by Institute of Medicine (IOM), while 30-50 nmol/L is agreed as insufficient.\(^3,5\)
- Increase PTH level\(^3,5,10\)
- In stage I vitamin D deficient rickets radiograph may seem normal or little rachitic changes. The low calcium decreased in 1,25-dihydroxyvitamin D2 (1,25D2) can cause hypocalcaemia seizures in children. While in stage 2 PTH levels are increased leading to secondary hyperparathyroidism which stabilises calcium serum level, with hypophosphatemia as the consequences. Clinical manifestation and radiograph pathognomonic findings are prominent in this stage\(^11,12,13\)

![Diagram showing laboratory results in various aetiologies of rickets](image)

**Fig. 1:** Laboratory result in various aetiologies of rickets. When there is suspicion of rickets from history and examinations, laboratory analysis of PTH, 25OHD levels are needed to confirm rickets underlying etiology. Vitamin D deficiency rickets shows increased in PTH and low of 25OHD level, when PTH is not increased, stage 1 vitamin D deficiency should be considered. Increased alkaline phosphatase (ALP) level indicates a phosphate disorder, and low level of ALP should make one consider hypophosphatemia rickets. In situations of increased PTH without low level of 25OHD, vitamin D pathway defect should be confirmed through 1-25OH2D analyses.\(^[2,10,13]\)
References: A Tjan, Department of Radiology, Sanglah General Public Hospital- Udayana Faculty of Medicine, Denpasar-Bali, Indonesia

Herein we report an unusual presenting symptom of rickets on outpatient clinic. A 9 years old male presents with vomiting, nausea, and headache. Upon examination, abnormal figure and shorter height than the usual age were seen (Fig. 2 on page 5). On examination, he was hemodynamic stable, short stature, weight and head circumference below normal as well as global developmental delay. Funduscopy revealed papilledema. He had kyphosis and scoliosis, bowing extremity and genu valgus, with other physical examination within normal limits. CT Scan was ordered to rule out parenchymal abnormalities. Because of the body contour abnormality, a skeletal survey was done. Laboratory tests of renal function, liver function, thyroid hormone, FSH and LH were normal. Vitamin D 25-OH and phosphate were markedly decreased.

Images for this section:

Fig. 1: Laboratory result in various aetiologies of rickets. When there is suspicion of rickets from history and examinations, laboratory analysis of PTH, 25OHD levels are needed to confirm rickets underlying etiology. Vitamin D deficiency rickets shows increased in PTH and low of 25OHD level, when PTH is not increased, stage 1 vitamin D deficiency should be considered. Increased alkaline phosphatase (ALP) level indicates a phosphate disorder, and low level of ALP should make one consider hypophosphatemia rickets. In situations of increased PTH without low level of 25OHD, vitamin D pathway defect should be confirmed through 1-25OH2D analyses.[2,10,13]
Fig. 2: Body contour of a 9 years old male with rickets. Upon inspection we could see pectus carinatum, kyphosis, scoliosis, and genu valgum.
Imaging findings OR Procedure details

A computed tomography scan (CT-scan) was performed and turned out normal. Skeletal survey revealed global delayed bone development with generalized osteopenia with lots of long and flat bones deformities and joint dislocations. Skull x-ray showed widening of sella turcica, with craniotabes (Fig. 3 on page 27). Cupping sign, rachitic rosary, and pectus carinatum on the chest X-ray were found (Fig. 4 on page 28). Frying sign, widening of growth plate, and bowing deformity, were observed (Fig. 5 on page 28 & Fig. 6 on page 29). Acetabulum irregularities and slipped epiphysis of both femoral heads was detected with pelvic shape abnormality and coxa vara (Fig. 7 on page 30). Healed green stick fracture usually found on weight bearing bone (Fig. 8 on page 32), as well as compression, kyphosis and scoliosis of thoracolumbar were revealed (Fig. 9 on page 31). The bone age was matched to 4.5 years old boy.

Fig. 3: Skull lateral/AP projection radiograph. Widened sella turcica (short arrow), craniotabes (arrow) indicates there is softening of the skull.

References: Departement of Radiology, Sanglah Public General Hospital - Denpasar/ID
Fig. 4: Chest x-ray AP/lateral. Rachitic rosary (arrow) and increase antero-posterior diameter of the thoracic cavity (pectus carinatum).

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Fig. 5: Right and left humerus x-ray. Fraying with widening of growth plate (arrow), bowing deformity, as well as shoulder dislocation of both joints (short arrow).

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Fig. 6: Wrist x-ray AP projection. Fraying (short arrow) of metaphysis with widening of growth plate in distal radial and ulna, small epiphysis, flaring of both ends of metaphysis giving an appearance of champagne glass (arrow). Cupping of all metaphyseal base of phalanx and metacarpals bone with bristle brush appearance as fraying appearance (short arrow). Rachitic metaphysis increase distance between epiphysis and metaphysis. Prominent trabeculae (osteopenia). Hand bone age similar to 4.5 years old boy

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Fig. 7: Pelvic x-ray AP projection. The lateral metaphyseal cortex did not bisect either right or left femoral epiphysis, giving a slipped capital femoral epiphysis (dash line). Also noted there is irregularity of both acetabulum (arrow). Triradiate pelvis with separation of pubic symphysis (open arrow), coxa vara, flat rachitic pelvis, under development of both sacral wings (short arrow). On this image the zone provisional of calcification (ZPC) is visualised in disorganised pattern with sclerotic line on epiphysis, which indicates recovering rickets due to vitamin D supplementation.

References: Departement of Radiology, Sanglah Public General Hospital - Denpasar/ID
Fig. 8: Femur AP projection. Fraying of both distal femur and widening of growth plate (arrow). There is increased thickness of the proximal third of right and left lateral side of femur periosteal (dash line), and uninterrupted periosteal new bone formation (arrow) due to an unidentified old healed green stick fracture.
**Fig. 9:** Thoracolumbar x-ray AP/lateral. Kyphosis, scoliosis of vertebrae (dash line), with multiple sclerotic and compressed end plates due to soft bony (osteopenia) vertebrae (arrow).

**References:** Departement of Radiology, Sanglah Public General Hospital - Denpasar/ID

**Let's Back To The Basic of Normal Bone VS Rickets**

Ossification is the normal process of bone formation and mineralization. The growth plate is composed of cartilage and chondrocytes of osteoid matrix secreted from osteoblast. Calcium and phosphate will eventually mineralized the osteoid matrix therefore induced
apoptosis of chondrocytes, and new bone deposition replaces the dead cells through the action of osteoclast. Low calcium and phosphate will disrupt this mechanism, thus causing development of axial growth in long bones (rickets).\textsuperscript{13}

The affected bones first reveal fast growing plates such as costochondral junction of middle ribs, distal femur, proximal humerus, both ends of tibia, distal ulna and radius.\textsuperscript{13}

\begin{center}
\textbf{Bone mineralization}
\end{center}

\begin{figure}
\centering
\includegraphics[width=\textwidth]{bone_mineralization.png}
\caption{Bone mineralization, normal (A) and unmineralized in Rickets (B).}
\end{figure}

\textbf{References:} A Tjan, Department of Radiology, Sanglah General Public Hospital-Udayana Faculty of Medicine, Denpasar-Bali, Indonesia
Fig. 11: Normal bone formation and maturation. Osteoblast secretes osteoid, osteocalcin, and collagen to form a scaffold matrix, resulting in spongy bone, woven bone, and lamellar bone through mineralization and resorption process.

References: A Tjan, Department of Radiology, Sanglah General Public Hospital-Udayana Faculty of Medicine, Denpasar-Bali, Indonesia

Fig. 12: Bone formation and maturation disruption due to vitamin D deficiency. As the previous figure explained, low vitamin D levels will lead to decreased calcium and phosphate therefore causing disturbance of bone mineralization.

References: A Tjan, Department of Radiology, Sanglah General Public Hospital-Udayana Faculty of Medicine, Denpasar-Bali, Indonesia

<table>
<thead>
<tr>
<th>Region</th>
<th>Radiology Signs</th>
<th>Mechanism</th>
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<tr>
<td>Long Bones</td>
<td>Epiphysis</td>
<td>Slipped capital femoral epiphysis (SCFE)</td>
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<td></td>
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<td>• SCFE is an idiopathic Salter</td>
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Harris type 1 fracture that can occur on rickets. The loose trabecular zone in combine with gravity beard by the lower limbs lead to bowing deformity, are the mechanism underlying this insufficiency fracture. 12,15

**Small epiphysis**

- Due to increased unmineralized osteoid deposition and cartilage growth which appear lucent on radiograph

**Increase distance of epiphysis and epiphyseal end of long bone**

- Cartilage and osteoid still expanding under inadequate
mineralization condition and they appear radiolucent on X-ray thus resulting in increase distance between epiphysis and epiphyseal end of long bone.\textsuperscript{13}

Growth plate

Widening

• The earliest and specific radiological changes, due to increase in cartilaginous cell mass and disorganization in chondrocytes maturation.\textsuperscript{5}

Zone of maturation is affected

• Increase number of disorganised cartilaginous cells without mineralization\textsuperscript{2}

• Increase in length
and width of growth plate, which appear lucent on radiograph

• Zone of provisional calcification shows deficient mineralization

• Increased uncalcified osteoid and decreased of calcified osteoid

• Mixture of calcified and non-calcified ZPC causing a patchy irregular margin, unlike the normal ZPC, which is uniform.

• Severe rickets and early onset age

Absent/ poor definition of zone provisional of calcification (ZPC)
rickets can have an absent ZPC\textsuperscript{12}

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<th>Metaphysis Fraying</th>
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<td>Flaring</td>
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- Due to irregular metaphyseal margin, disorganization of spongy bone in metaphyseal region.\textsuperscript{1}
- Protrusion of bulky mass of cartilaginous cell in the zone of hypertrophy into the poorly mineralized metaphysis.\textsuperscript{12,16}
- Flaring, due to axial growth and cupping mechanism giving an appearance similar to champagne glass.\textsuperscript{16}

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<th>Cupping</th>
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- Enlarge mass and
disorganized cartilage expand in multiple direction in additional to axial widening, it expand on metaphysis transversely, thus causing enlargement of bone ends and collapsed of poorly mineralized trabecular bone. 1,12

- Rim bone surrounding the enlarge lucent physis 12,16

Diaphysis Bowing

- Gravitational pushing and weight bearing in children who begins to stand with
Rickets will lead to bowing deformity particularly anterior and lateral bowing on long bones such as tibia and femoral bones (genu varum) and kyphotic, or lordotic with scoliosis of vertebrae. 2,5,6,12

Greenstick fracture

• Prominent trabecular, with poor density will result in bending and causing incomplete fracture of the long bones. 1,12,16
Lifting periosteal reaction

- Mimicking periosteal reaction in rickets because the excess of non-calcified subperiosteal osteoid
- Or as the complication of increase PTH, which induced subperiosteal bone resorption or osteoblastic effect of PTH causing periosteal new bone formation.

Skull

Craniotabes
- Softening of the skull due to failure of bone maturation and mineralization, with increase

Delayed anterior fontanels closure

Frontal Bossing
osteoid deposition lead to craniotabes, delayed anterior fontanels closure as well as flattening skull bones. 2,6,16

- Delay of closure and flattening of occipital and parietal bones, will bossed up the frontal bones. 16

• Hypertrophy of the costochondral junction with increased osteoid deposition giving the beading and classic rachitic rosary and may

Thorax

Rachitic rosary & thoracic deformity
progress into protrusion of the ribs and sternum, thus abnormal thoracic contour (pigeon chest) 12,16

- Muscle traction on the softened ribs causing thoracic asymmetry and widening of the thoracic base
- Rachitic changes of the ribs as well as protrusion of the sternum will deform thoracic shape in pigeon chest form 6

Harrison's sulci

- Caused by drawing or flaring
Pelvis

Triradiate pelvis

- The under development of both sacral wings with flat rachitic pelvis which reduce AP diameter of pelvic inlet.
- Coxa vara and increased distance between pubic symphysis combined with lumbar scoliosis, which caused the deformity of pelvis.

Recovering Rickets
After several months of treatment (approximately 2-3 months) the ZPC is detected in the epiphysis. There is irregular dense areas due to mineralization (Fig. 7 on page 30). However deformities can persist for quite a long time. \[12\]

Fig. 13: Normal bone growth VS Rickets, and the mechanism behind it's radiographic findings. Normal growth plate will show mineralization of bone matrix and normal apoptosis of chondrocytes, which will be replaced by new bone. Zone of provisional calcification will be prominent as the opaque density on radiograph due to bone maturation in hypertrophic zone. In contrast with lack of supplementation for mineralization (calcium and phosphate) the chondrocyte become disorganized, loss of its columnar and linear uniform sequence, as well as uncontrolled cells enlargement, without proper mineralization, thus absent or poor provisional calcification zone. Lack of bony growth leads to axial widening. Metaphyseal collar, which is the region of metaphysis adjacent to physis, should appear denser and closer to epiphysis because of normal calcified osteoid replaced with new bone and formed denser bone, while in rickets uncalcified osteoid still growing in numbers and disorganized, giving more distance due to its lucent appearance in radiograph and smaller epiphysis. The softening of the bone will result in bowing deformity.
Fig. 14: Normal VS rickets skull ossification. In normal intramembranous ossification, osteoblast secretes osteoid which then becomes mineralized and comprises the woven and compact bone, while in rickets lack of mineralization with increased of uncalcified osteoid, will result in widening and delay closure of sutures lead to skull abnormal appearance (bossing of frontal or parietal bone). Poor trabecular or soft skull may appear as thumb printing phenomenon (craniotabes).

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Conclusion

An awareness of any unusual clinical manifestations of rickets should raise suspicions of this condition. Understanding the basic mechanism behind rickets presenting radiograph are important. X-ray is used as the sole imaging modality, which can be used to confirm the diagnosis. Additional examination such as CT scan is used to rule out other underlying aetiologies in patients presenting with increased intracranial pressure.

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