An Ayurvedic Review On Phakka Roga W.S.R.To Rickets And Nutritional Deficiency In Children.

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Abstract:

AcharyaKashyap is considered as the father of Kaumarbhritya. The PHAKKA means walking inability in growing children.The childhood is dividedinto threestagesi.e .Garbh,Bal,kaumar which is accepted even today.Asthivah srotas dusti seen in Phakka Vyadhi.Phakka is a diseasecomplex characterized by a symptoms as the child is unable to walk even after the age of one year. Rickets may be vit D deficiency or non deficiency various metabolic disorder. Rickets is the disease of children caused by Vit D deficiency,characterized by imperfect calcification ,softning and distortion of bones typically results in bow legs.

Keywords: Ayurveda, Kaumarbhritya,Phakka ,Rickets, Nutritional deficiency.

Introduction.

Phakka disease is classified under kuposhanajanyavyadhi.A lack of adequate mineralization of growing bones results in rickets. The term Rickets is derived from english word wrick (twist) and Greek word rachitis means excess of osteoidtissue. Rickets is a condition that affects bone development in children.It causes the bones to become soft and weak which can lead to bone deformities .Rickets can cause bone pain, poor growth and deformity of the skeleton such as bowlegs , curvature of the spine and thickening of the ankle , wrists and knees.The incident is more in six month to 2 yearsage .More in poor socioeconomic condition with low vit D in diet.The area in which deprived of sunlight .Nutritional rickets usually presents in infancy or preschool age,usually as widened wrists or bowing of legs. Presentation in early infancy and finding of seizures or tetany suggest a defect in vitamin D metabolism.

PHAKKA. According to Ayurveda.

If a child after attaining age of one year does not walk on feet this is known as Phakka.

There are three types of Phakka Rog:KsheerajPhakka,GarbhajaPhakka,andVyadhijaPhakka.

1.KSHEERAJ PHAKKA:

It occurs due to kaphajstanya sevan by the child and child become krusha.

2.GARBHAJ PHAKKA:

If lactating mother becomes pregnant ~Quantity of milk secretion becomes less in that mother~their are less nutritional content in milk which is required for growth and development of child . Because most of the part of nutrition is used for growth of fetus in mother~ So child does not get sufficient nutrition from milk of mother ~ their is no proper growth and development in child.Then gradually child becomes krusha this is known as Garbhaj Phakka.

3.VYADHIJPHAKKA:

In this Vyadhija Phakka child suffer from nij and agantujjwaradi Rog, hence child suffers from ksheenata of mansa,bal,shifk,bahuanduru becomes emaciated,abdomen becomes protuberant,head and face becomes more dominant,wasting of muscles.

Management of phakka:

A)ABHYANTARCHIKITSA:

1) Orally use of Kalyanakghrita, Shatpalghrita, Amruta ghrita, Sanvardhanghrita.

2)Asthiposhak vati, Kukkutandatwak bhasma,Liq kumarikalpa.

B)BAHYACHIKITSA:

1)Sarvang Snehan:ByBalatail/Chandanbalalakshaditail/Rajtail.

2)Vedanashamak Tail: Narayantail, Dhanvantartail, Visgarbhatail, Dashamultail for Abhyang.

3)Sarvang Swedan:

1)Bashpa Swedan with Dashmulbharadchurna.

2)Nadi Swed with Vataghnadravya.

3)Swedan with shalishastikpindasweda.
C) Deepan and Pachandravya like; Rasana, Madhukasidhaghrita, Draksha, Punarnava, Milk for brahankarma.
D) PhysiotherapyanduseofPhakkaRath.

According To Modern Science:

Etiology Of Rickets :

1.Nutritional rickets.
2. Malabsorption states:
a) Cystic fibrosis
b) Biliaryatresia
c) chronic diarrhoea and vomiting
d)Liver disease
e)Excessive destruction of intestinal mucosa malabsorption
3.Refractory rickets:
a)Renal tubular dysfunction (hypophosphatemicVit D resistant rickets).
b) Renal tubular acidosis.
4)Prolonged anti convulsant therapy inducess rickets by interfering vit D metabolism.
5.Inadequate dietory intake of vit D and lack of sunlight.

Functions Of Vitmin 'D':

1) Vitamin D is required for normal growth in Bone's is related to its role in calcium and phosphorus absorption which is needed for bone development.
2) Vitamin D increases calcium and phosphorus absorption in intestine.
3) Vitamin D increases the reabsorption of phosphate by renal tubular cells and rise the level of phosphate in the blood.
4) In normal growth of bones,the bone forming cell's appears as cartilege cells which degenerate anddisappear and calcium and phosphorus are deposited in vitamin D deficiency cartilage cell do not degenerate but continue the grow.

Metabolic Changes In Rickets:

Deficiency of vit D
⇩
Decreased calcium and phosphate absorption from intestine
⇩
Decreased level of calcium and phosphate
⇩
Compensatory mechanism by parat hormone
⇩
a) Reduced calcium excretion by kidney
b) Calcium released from bones
c) Decreased Renal tubular reabsorption of phosphate
⇩
Sr calcium return to normal but phosphate level fallsProlonged vit D deficiency
⇩
Even parat hormone can not sustained it's action
⇩
Sr calcium and phosphate both level decreased
⇩
Interfere with calcification of osteoid tissue
⇩
Cartilage cells of bone can not be disappears
⇩
Increase osteoblastic activity

Clinical Features Of Rickets:

Rickets commonly present at 6 months to 2 years of age with bony deformities and hypotonia of supporting ligaments / muscles. Important changes in Rickets are:

A) Craniofacial changes:
1) Craniotabes : thinning softening of skull bones with pin-pong ball like resilience on pressure over parietal bones.
2) Frontal bossing : Prominence of frontal bones.
3) Delayed closure of anterior fontanel.
4) Delayed dentition.

B) Thoracic Changes:
1) Rachitic rosary:
Round ,non tender bending due to widening of costochondral junction's.
2) Harrison sulcus:
A groove / depression along the lower costal margins.
3)Sternal Deformities like pectus excavatum i.e.depression of sternum .Pigeon -Chest deformity.

C) Limb Deformities:
1)Widening of wrist ankles due to widened epiphysis and metaphysis
2) Gait abnormalities e.g.Knock-Knee ,Bow legs and Coxa -Vera.
3) Green stick pathological tactures of long bones.

D) Spinal Deformities:
1) Kyphosis or scoliosis due to lax ligament.
2)Short stature due to deformed spinal curvature.

E) Generalized hypotonia with.
1) Pot -belly due to abdominal muscle hypotonia.
2) Visceroptosis due to ligamental laxity.
3) Hyper- extensible joints (acrobatic rickets).
F)Other manifestation
1) Excessive sweating over forehead, recurrent respiratory infection.

Evaluation.:

1. Radiologic changes are characteristically seen at the metaphysis.
2. 2.The first change is loss of normal zone of provisional calcification adjacent to the metaphysis.
3. This begins as an indistinctness of the metaphyseal marg in progressing to afrayed appearance with widened growth plate due to lack of calcification of metaphyseal bone.
4. Weight bearing and stress on uncalcified bone gives rise to splaying and cupping of metaphysis.
5. Eventually ageneralized reduction in bonedensity is seen.
6. Labor atorydiagnos is of vitamin Ddeficiency is based on low circulating level of 25(OH)D3.
7. Values below 10ug/ml are indicative of deficiency.
8. An increased plas male vel of 1,25(OH)2D3 indicates deficientin take of calcium or phosphorus.
9. Blood levels of alkaline phosphate are elevated; calcium and phosphate level may be normal or low.

Treatment Of Rickets (According to modern science):

A)Vit D is administered orally either in a single dose of 600,000 IU or over 10 days
(60,000IU daily for 10 days) followed by a maintenance dose of 400-800IU/day and oral calcium supplement 30 -75 mg/kg/day for 2 months.

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