Oral Manifestations of Malnutrition II. The Effect of Minerals

Pages with reference to book, From 193 To 196

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About 4 percent of the body weight, is made up of minerals. Calcium and Phosphorus account for three-fourths of the mineral elements in the body, and several others constitute the rest. Some of these elements are present in such a minute quantity that they are generally referred to as "trace elements" or micronutrients. Some of the trace elements are essential for body functions; others may be present as contaminants.

Deficiency of any mineral may occur whenever the demand in the body exceeds the supply in food, or assimilation from food in un-usually poor or when extra large amounts are lost from the body. Considerable mineral losses may occur through vomiting, bleeding, excessive perspiration diarrhoea, or in the urine under certain conditions. Mineral deficiencies are more in the urine under certain conditions. Mineral deficiencies are more serious and show up more quickly during the growth period or during pregnancy and lactation, when the demands increase. Even in normal adults, small amounts of mineral elements are constantly excreted from the body, chiefly as salts in the urine, and these losses must be made up by the mineral elements taken in food if the body is not to become depleted.

If mineral deficiencies occur during the growth period, growth will be stunted; if they occur during pregnancy, the offspring may be still born, too weak to survive for long, or born with certain deformities. Tissues that have a special need for certain elements naturally are the first to show the effects of an insufficient supply of the particular elements...for example, bones and teeth suffer first from the lack of calcium or phosphorus, the red blood cells (hemoglobin) from lack of iron, and the thyroid gland from a lack of iodine. Prolonged shortage or excessive drainage from the body of any mineral elements may result in serious symptoms, as contraction of muscles or disturbances of acid base equilibrium.

Calcium and Phosphorus

There is two to three times as much calcium and phosphorus in the body as all the rest of the mineral elements combined, as calcium and phosphorus make up a large proportion of the bones, and the structural frame work of the body. Because they are so closely associated in the formation and upkeep of the bones and teeth, these two elements may conveniently be considered together.

Calcium is the most abundant and phosphorus the second in abundance of the mineral elements in the body. Ninety-nine percent of the calcium and about 80% of the phosphorus is found in bones and teeth. Although amounts of calcium and phosphorus in the blood are small by comparison with those contained in the body structures, each element has a vital role to play. Calcium in blood plasma is one of the essential factors for blood clotting, maintains muscle tone and irritability, and is essential for normal nerve impulse transmission. Phosphorus performs an important role in combining with calcium in the formation and strengthening of bone tissues. Phosphoric acid is indispensable for the oxidation of carbohydrates by which the energy for body processes is obtained. It is a part of several enzymes and co-enzymes that are essential for this oxidation. The action of adenosine tri and di-phosphate provides quick release of energy in muscular contraction.

In children or young animals, who need calcium and phosphorus in relatively large amounts for building bones and teeth, an insufficient supply of either element or both produces effects that are readily seen or otherwise demonstrated. The effect of such deficiencies during the growth period may be manifested by stunting of growth, poor quality of bones and teeth or by malformation of bones.

The teeth are largely formed during the latter part of fetal life and during infancy. Any lack of calcium or phosphorus during this period is likely to result in malformed teeth and jaws, or in poor quality teeth that are more subject to decay in later life. Many instances of unhealthy teeth or of teeth crowded too

closely in a narrow jaw may be attributable to the mothers receiving an insufficient supply of calcium, phosphorus or other dietary essentials during pregnancy or to the child receiving an insufficient supply of these dietary essentials during its first year of life (Sherman and Lanford, 1957).

A low calcium intake may induce a state of gradual demineralization of bony tissue known as osteoporosis and is characterized by porosity, thinness and fragility of bones, including that of the jaws. Dietary calcium deficiency accentuates bone resorption because it induces a secondary hyperparathyroidism (Harnikson, 1968) and often the first sign of hyperparathyroidism is some major oral Pathology such as a tumor in the jaw or marked loosening and spreading of the teeth or overgrowth of the mandible. Osteitis fibrosa cystica (Von Recklinghausen's Disease) and Osteitis Deformans (Paget's Disease) are the common findings in calcium deficiency. Excess of calcium results in a decrease in caries development but at the same time leads to complete obliteration of marrow cavities, the bones becoming nearly solid and fragile (Orent et al., 1934).

According to Colman et al. (1953) with a deficiency in phosphorus the formation and mineralization of dentine is severely retarded and the predentine is abnormally wide. The alveolar bone in the molar areas consists of a large amount of osteoid and in the condyle head the cartilage fails to mineralize and osteoid is formed. Excessive resorption of the alveolar bone has also been shown by Becks and Simmonds (1935). Irving (1950) has demonstrated that if rats are fed on a low phosphorus rachitic diet and then partially starved or given extra phosphorus in the diet, and subsequently returned to the low rachitic diet, enamel matrix formation stops with subsequent hypoplasia. Carbonate levels of the tooth is increased in phosphorus deficiency and since a tooth with a high carbonate level is relatively acid soluble, it is thought that teeth may become more susceptible to dental decay (Sobel et al., 1960). High levels of phosphorus decrease magnesium absorption and increase magnesium requirements thereby aceentuating the symptoms of magnesium deficiency (O'Dell et al., 1957; O'Dell et al., 1960).

Magnesium

In magnesium deficiency, the ameloblasts show various stages of localized degeneration with subsequent development of enamel hypoplasia or complete cessation of enamel formation. Calcification of dentine is disturbed so that its formation is retarded, particulaily in the coronal part of the teeth (Becks and Furata, 1939; Beeks and Furata, 1941; Klein et al., 1935). The pulp shows degenerative changes in odontoblasts and other cells (Watchhorn and McCane, 1937).

When rats are fed on a diet deficient in magnesium, there is almost complete obliteration of the marrow cavities, the bone becoming nearly solid and at the same time fragile (Orent et al., 1934). The rate of growth of the alveolar bone is retarded, there is gingival hypertrophy and the eruption of teeth is considerably delayed (Gagnon et al., 1942). Other changes include altered alveolar bone architecture, increased resorption, calculus formation, widening of the periodontal ligments and loosening of the teeth (Klein et al., 1935; Becks, 1942).

In magnesium deficient pigs, the symptoms include sIow growth, soft tissues calcification, over growth of the molars, exostosis of the mandible, erosions, softening and decay of the incisor teeth (O'Dell et al., 1960) .Excess of magnesium has been reported to inhibit mineralization (McCollum et al., 1923) and seems to increase dental caries development.

Fluorine

The protective effect of a proper intake of fluoride against dental caries is well established. The literature contains numerous reports of investigations demonstrating the value of fluorides in caries prevention, and the safety with which they may be used. The fluorine content of the diet and of specific food stuffs in particular has been investigated. Varying amounts of fluoride are found in a good many plant substances, depending to some extent upon the flouride content of the soil in which they are grown. Since the dietary fluoride is relatively unimportant compared to fluoride in the drinking water because of its metabolic unavailability (Shafer et al., 1974) there have been little attempts to study the dietary fluoride in relation to dental caries as has been done for the fluoride content of drinking water. Fluoride in food is usually measured as milligram (mg) and in water is measured as parts per million

(ppm).
Experimental fluorine deficiency in rats has been reported to cause dental caries (Anderson, 1971). The reports of other investigators (Dean, 1938; Dean et al., 1941; Dean et al., 1942) show that the incidence of caries decreases as fluoride concentrations increase from 0.5 to 1.5 ppm. The average number of dental caries encountered was seven DMF (Decayed, missing, filled) teeth for children living in cities where the water supply contained 0. 5 or less ppm fluoride, whereas children of comparable ages who drank water with a fluoride concentration of 1.0 to 1.4 ppm had a caries incidence of only three DMF teeth showing a decrease of about 50%.
Ingestion of excessive quantities of fluorine by children result in the production of mottled enamel or hypoplasia, characterized clinically as a discoloration and roughening of the enamel surface which appears as a horizontal striation or pits accompanied by chalky white patches that may later acquire a brownish stain. Mottled enamel occurs in children who have consumed drinking water, containing 1.5 ppm fluoride or more during the time when tooth enamel is being deposited in the developing unerupted teeth (Anderson, 1971).
Excessive amounts of fluorine also produce skeletal defects; the bones may become dense and sclerotic or they may show excessive porosity and abnormal depositi on of calcium.

Lron

Since the integrity of epithelium is dependent upon adequate scrum iron levels, changes in the oral mucosa are a fairly frequent feature of iron deficiency anemia. The presenting oral symptoms are glossitis and fissures at the corners of the mouth. Stomatitis, ulceration, and petechial hemorrhages in the oral mucosa occur in some cases (Derby, 1946; Nizel, 1972; Shafer et al., 1974; Dolby,'1975). Changes in the tongue are first manifested as a burning sensation accompanied by a reddening at the tip and around the margins. The papillae, first the filiform and then the fungiform atrophy in a patchy or diffuse pattern, thus giving the tongue a smooth, shiny red appearance. In more advanced cases the epithelium may loose the keratohyaline granules, becoming parakeratotic or non-Keratinized, thin and eventually erodes, leaving shallow ulcers which may become the seat of secondary fungal and bacterial infections. Small vesicles have also been described on the lingual and buccal mucosa, which on rupturing result in aphthous like ulcers (Jasosilewsky et al., 1970; Kalinin, 1970). Further more traumatic ulceration is more commonly seen on an atrophic tongue which may in some cases result in the development of leukoplakia or even carcinoma (Savilanti, 1946; Ahlbom, 1936).

Zinc

Zinc plays an important role in metabolism of tissues and blood. Zinc deficiency in animals will produce thickening of the skin, loss of hair and thickening of the oral epithelium together with an increase in the rate of cell division. In the tongue the epithelium of the anterior dorsum becomes thick, parakeratotic and exhibits hyperchromatism and the underlying muscles become atrophic.
Zinc gets concentrated in healing tissues indicating an increased demand for tissue production during healing. Recently it has been reported that Zinc sulphate supplements will decrease wound healing time significantly (Pories et al., 1967). Zinc peroxide has also been seen to quicken wound healing (Nizel and Rubin, 1943). Excess dietary zinc fed to rats will produce hypochromic anemia and subnormal growth (Nizel, 1972).

Iodine

Iodine is essential for the synthesis of thyroid hormone thyroxine, which is necessary for the development and maintenance of the hard and soft tissues of the oral cavity. Deficiency of Iodine therefore, leads to a diminution or absence of thyroid secretion which in turn results in goitrous cretinism in infancy (Anderson, 1971). The orofacial changes occurring in cretinism include maldevelopment of jaw bones, retarded tooth eruption (Nizel, 1972) and predisposition to root resorption (Becks, 1942).

Copper

Copper plays on important role in kera-tinization (Dolby, 1975). The importance of copper in normal

skeletal development has been demonstrated both in field experiments and in histologic examinations of copper deficient animals (Asling and Hurley, 1963; Lahey et al., 1952; Levene, 1967) and it has been concluded that lack of dietary copper causes impairment of osteogenesis. The condyle of the temporomandibular joint exhibits widening with irregularly arranged cartilagenous layers and a decrease in vascularity and trabeculae. Osteoporosis of the shaft is also evident (Furstman and Rothman, 1972).

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