Changing Reality: Resurgence of Old Disease in New Circumstances. Severe Vitamin D Deficiency Presenting as Hypocalcemic Seizures and Hyperphosphatemia in a Healthy Adolescent after a Prolonged COVID-19 lockdown

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INTRODUCTION

Nutritional rickets (NR), which is caused by a vitamin D or calcium deficiency or both, remains a major health problem worldwide.
Its manifestation is usually associated with a combination of risk factors such as skin pigmentation, lack of sunlight, malnutrition, malabsorption and food restriction.

In the 20th century, the incidence and prevalence of NR re-emerged as a global problem in developed countries, especially in risk ethnic minority groups (1).

The COVID-19 pandemic has caused huge changes in people's health, social relationships and lifestyle. The extreme indoor isolation during lockdowns had an enormous impact on children and adolescents, resulting in much less sleep and physical activity, an increase in screen time and general media usage and changes in eating habits such as increased in the consumption of frozen products, coffee and candy(2).

OBJECTIVE

1. To draw attention to the rise in severe vitamin D deficiency as a result of significant life style changes during the COVID-19 pandemic.
2. To increase the awareness to pseudohypoparathyroidism-like clinical picture at presentation of severe vitamin D deficiency.

CASE REPORT

The patient is a 12.5 year old healthy white-skinned teenager from a high income family in a sunny country. During the family's first outdoor weekend after nearly one year of indoor isolation, the boy experienced agitation, paresthesia, breathing difficulties, myalgia, perioral and carpal spasms. These symptoms resolved by themselves several hours later and were considered to be a panic attack by his parents. A physical examination the next day was unremarkable with a Tanner three pubertal stage at a growth rate of 8 cm/yr.

Biochemical evaluation (Table X) revealed **hypocalcemia, hyperphosphatemia, undetectable vitamin 25(OH)D, increased PTH and alkaline phosphatase.** Urinary calcium was undetectable. The magnesium, albumin, B12, folic acid, thyroid functions and renal function tests were normal. The electrocardiogram was normal. An X-ray of wrist revealed no signs of rickets.

An additional interview with the patient revealed that during the long lockdown he spent most of the time at his computer, in his room with the shutters closed, to avoid glare on the screen. In addition, his dietary preferences were limited to meat products and carbohydrates and excluded dairy products, fish, eggs, and vegetables.

This clinical and biochemical picture corresponds to a diagnosis of NR in a healthy adolescent boy at mid- puberty with growth acceleration, complicated by hypocalcemic seizures. This is a case of severe vitamin D deficiency against the backdrop of prolonged indoor isolation, without an opportunity for vitamin D skin synthesis, accompanied by a restricted diet due to pubertal behavioral habits.

MANAGEMENT

Vitamin D therapy was initiated with calcium supplementation, followed by quick clinical and biochemical improvement.

Lab tests (Table X) returned to normal range within 3 months.

	Normal range	1st Exam	1 mo later	3 mo later
Calcium , mg/dl	8.8-10.4	6.8	8.8	9.2
Phosphorus, mg/dl	3.0-5.9	7.4	6.7	5.6
Alk Phos, U/L	80-405	850	738	520
25(OH)D, ng/ml	>30	UD*	9.5	20
PTH, pg/ml	6.5-36.8	94	74.1	46

UD*= undetectable

DISCUSSION

NR is fully preventable and can be cured simply by vitamin D and calcium supplementation.

Severe vitamin D and Ca deficiency can lead to hypocalcaemic complications (seizures, tetany and cardiomyopathy) and late hypophosphatemic complications (muscle weakness, osteomalacia) (3).

The incidence of hypocalcaemic seizures, as a result of NR, that peaks in the spring/summer months (after a "vitamin D winter") is significantly greater in males than in females and in children from high-risk ethnic groups. NR is more frequent in two specific periods: early childhood and adolescence, possibly due to the higher metabolic demand for calcium during these periods of rapid bone growth (4).

Calcium deprivation leads to increased release of PTH that stimulates osteoclastic bone resorption. PTH also conserves renal calcium by reducing calcium loss and, at the same time, increases phosphate excretion, which can lead to hypophosphatemia.

In this case the phosphate level was higher rather than lower, as expected in vitamin D deficiency and PTH elevation. Similar cases have been reported previously (1,4,5,6). The pathophysiology of hyperphosphatemia in severe vitamin D deficiency could result from end organ (kidney, bone) resistance to PTH due to down-regulation of it's receptors, mimicking pseudohypoparathyroidism type Ib, leading to hyperphosphatemia which resolves after therapy with calcium and vitamin D (6).

CONCLUSIONS

- Global changes resulting from the COVID-19 pandemic have taught us to be alert to unexpected manifestations of old and known diseases.
- Hyperphosphatemia accompanying hypocalcemia can be a clue to severe vitamin D deficiency.
- As early as the mid-17th century, Glisson noted that rickets was more frequent in children from rich families that preferred to keep their children indoors and fed them mostly meat and bread, which made them highly vulnerable to vitamin D deficiency.
  Thus, there is nothing new under the sun.

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