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Case Report

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A Case of Vitamin D Deficiency in a Young Athlete Diagnosed by Cardiopulmonary Exercise Test

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ABSTRACT

In this case report, we present a 16-year-old athlete woman with the history of dyspnea, especially during exercise with vitamin D deficiency that was diagnosed by cardiopulmonary exercise test.

Introduction

Vitamin D is a fat-soluble vitamin that has an essential role in maintaining a healthy musculoskeletal system. Vitamin D is naturally present in a few foods such as fish and liver. Research studies have documented the importance and versatility of vitamin D. Vitamin D has many roles in the body, including modulation of cell growth, neuromuscular, and immune function; and reduction of inflammation [1-4]. Serum concentration of 25(OH)D (25-Hydroxyvitamin D) is the best indicator for vitamin D status. Less than 12 ng/mL of 25(OH)D is associated with vitamin D deficiency, leading to osteomalacia in adults and rickets in children [5].

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Because athletes and sports medicine physicians are primarily concerned with performance, the risk of vitamin D insufficiency among athletes has received growing interest. The research available to support vitamin D influence on performance is quite limited. In one study, higher 25(OH)D levels were associated with an increased VO_2max, compared to those with lower vitamin D level [6]. Therefore, optimum treatment of vitamin D deficiency particularly in athletes for improving health status and performance is critical.

Case Presentation

In March 2017, a 16-year-old athlete woman referred to the pulmonary clinic of Imam Khomeini Hospital of Tehran University of Medical Sciences, with dyspnea on exercise. She reported 5 months history of dyspnea and her complain got worse, particularly on exercise. She was a professional tennis player. She had no history of smoking, cough and chest pain. On examination, she was not agitated and there was no respiratory distress. Her vital signs were as follows: blood pressure: 110/70 mm Hg, pulse rate: 70/min, respiratory rate: 12/min, oral temperature: 37°C, arterial saturation O_2 in room air: 97%, BMI: 25 kg/m^2; chest examination was intact showed within normal limit. All other examination results were unremarkable.

Complete blood count, thyroid function test and electrolytes were all within the normal range. Chest X-ray radiography was normal. She underwent pulmonary function tests (Table 1).

Due to dyspnea on exercise, Eucapnic Voluntary Hyperventilation (EVH) was done instead which was normal. As there was not any decrease in successive FEV1 after EVH test, exercise asthma would be ruled out. She underwent Cardiopulmonary Exercise Testing (CPET) with

Table 1. Selected respiratory function data

Measurement	Predicted	Measured
Age, y		16
Sex		Female
Height, kg		160
Weight, kg	62	64
FEV1	3.3	3.1(94%)
FVC	2.7	2.55(93%)
FEV1/FVC		

Table 2. Selected exercise data

Measurement	Predicted	Measurement Air (%)
Peak VO_2 (L/min)	2.1	1.48(68%)
Peak VO_2 (mL/kg/min)		23.1
AT (L/min)	2.1	1.16(53%)
O_2 Pulse	11.7	8.2(70%)
Breathing reserve (L/min)	>15	7(49%)
SpO_2 (end of test)		97%
VD/VT (max ex rest)		0.23
VE/VCO_2 (AT)		28

Table 3. Selected exercise data

Measured	Before Treatment	After Treatment
Peak VO_2	0.48(68%)	1.95(89%)
Peak VO_2 (mL/kg/min)	23.1	30.3
AT (L/min)	1.16	1.12(51%)
O_2 Pulse	8.2	10.5(89%)

bicycle. She started without loading for three minutes and 60 rmp. Then load was added 17 w/min. she terminated test due to dyspnea (7/10 scale score) (Table 2).

Without any changes on ECG due to low peak VO_2 and normal other parameters, myopathy was suspected. Further evaluations were done. ABG test, CPK, LDH, EMG, NCV PImax, and Pemex were within normal range, but 25(OH)D level was 7 mg/mL. She received 50000 IU supplemental vitamin D every week for 8 weeks. After treatment with supplemental vitamin D, her 25(OH)D level was 31 mg/mL. CEPT was repeated in May 2017. The finding was as follows (Table 3).

Vitamin D deficiency in this athlete youth patient caused dyspnea on exercise and low peak VO_2 on CEPT. After treatment, her complaint was relieved and CPET parameters become within normal rage.

Discussion

Vitamin D is prohormone that is produced by skin exposed to sunlight or obtained from dietary sources. Inadequate sun exposure or limited oral intake from poor diet or impaired intestinal absorption could induce vitamin D deficiency. Vitamin D consists of two bioequivalent forms. Vitamin D2 or ergocalciferol is obtained from oral supplements and Vitamin D3 or cholecalciferol is obtained from UVB. Commonly recommended daily intakes of Vitamin D are known to be insufficient if sunlight exposure is limited [7].

Measurement of the total 25(OH)D level is the best way to assess body storage of Vitamin D. Whereas quantification of 25(OH)D2 and 25(OH)D3 fractions may facilitate treatment monitoring. A wide optimal range for 25(OH)D is reported (25-80 ng/mL); geographic and seasonal variability are affected by vitamin D levels [8].

Vitamin D deficiency is often defined as <20 ng/mL and insufficient defined as 20-30 ng/mL and optimal level are >40 ng/mL [9]. Apart from inadequate intake and loss of sun exposure, gastrointestinal and renal diseases, severe liver disease, some antiepileptic medications, and aging are clinical risk factors for vitamin D deficiency. Musculoskeletal symptoms such as myalgia, bone pain, generalized fatigue, and weakness are related to Vitamin D deficiency and these symptoms might be misdiagnosed as fibromyalgia chronic fatigue syndrome, age-related weakness and depression [10].

Laboratory and radiographic findings that suggest Vitamin D deficiency are low urine calcium excretion, high PTH level, high alkaline phosphatase level, low serum calcium or phosphorus level, osteopenia on X-ray, nontraumatic bone fracture and skeletal pseudofracture. Early European researchers suggested that significant improvement in time trials, cardiovascular fitness, and strength performance are observed at >32 ng/mL level of vitamin D in athletes [11]. Due to storage of vitamin D in the muscle and fat for future use [12], higher 25(OH)D levels may improve aerobic performance [13] and 25(OH)D goal of 40 ng/mL is recommended for athletes. In the deficient state of vitamin D, the athlete may be at an increased risk for potential problems such as stress fractures, respiratory infections, and muscle injuries [14].

For the treatment of Vitamin D deficiency, a supplement of vitamin D is safe and inexpensive [15]. Both Vitamin D2 and Vitamin D3 appear to be effective for increasing the total 25(OH)D level [16] but oral Vitamin D3 (Cholecalciferol) is the treatment of choice in Vitamin D deficiency [17].

Treatment dosage included 50000 IU capsules, 1 per week for 6 weeks, 20000 IU capsules, 2 per week, for 7 weeks, or 800 IU capsules, 5 per day for lo weeks. After treatment, checking the serum 25(OH)D level is generally unnecessary, but may be appropriate in patients with symptomatic Vitamin D deficiency or malabsorption [18].

In our case, she was an athlete and complained from dyspnea during exercise and this finding is an unusual symptom in vitamin D deficiency. For evaluation of dyspnea, pulmonary function test and cardiopulmonary exercise test were done. On CEPT, her peak VO_2 was low due to low consumption of oxygen by muscles, due to Vitamin D deficiency.

After correction of Vitamin D deficiency, another CEPT was done which showed her peak VO_2 was improved. To the best our knowledge, dyspnea is not prevalent in Vitamin D deficiency and diagnosis of Vitamin D deficiency myopathy by CPET is not usual.

Ethical Considerations

Compliance with ethical guidelines

There were no ethical considerations to be considered in this research.

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Conflict of interest

The authors declared no conflict of interest.

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