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Original Research Article

Prevalence of vitamin D deficiency in burn patients: an observational study

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ABSTRACT

Background: Burns are one of the most common and devastating forms of trauma in life. Patients with serious thermal injury require immediate specialized care in order to minimize morbidity and mortality. Thermal injury creates a breach in the surface of the skin. It is difficult to evaluate the immediate effects of burn injury on serum levels of vitamin D metabolites because the binding proteins for 25-hydroxyvitamin D (25(OH)D), the main circulating form of the vitamin, and for 1,25-dihydroxyvitamin D (1,25(OH)2D) are low.
Methods: This study is a cross-sectional study. It was carried out in burn ward of Government medical college Jammu during June to November 2020. 52 burn patients of both sexes and all age groups were enrolled in this study.
Results: Fifty-two patients were evaluated in this study including 36 males and 16 females. In second degree superficial burns the mean level of 25(OH)D was 13.60 ng/ml (SD=7.27) ,in second degree deep burns the mean level of 25(OH)D was 13.88 ng/ml (SD=7.03) and in third degree burns the mean level of 25(OH)D was 14.71 ng/ml (SD=7.08).
Conclusions: Based on the results of this study, we can conclude that 25-hydroxyvitamin D levels are low in patients after acute burns, and these patients should be given vitamin D supplementation.

Keywords: Burns, Vitamin D deficiency, Supplementation

INTRODUCTION

Burns are one of the most common and devastating forms of trauma in life. Patients with serious thermal injury require immediate specialized care in order to minimize morbidity and mortality.[1] Burn injury is very painful because of the multitude of pain receptors and nerves that traverse the skin layers. Beneath the skin lie the subcutaneous tissues, muscle, and bone. Several important physiological functions of the skin are altered by thermal injury.[1] The skin has immunological, neurosensory, and metabolic functions such as vitamin D synthesis. Skin is the main source of vitamin D for most vertebrates including humans. Vitamin D is not only necessary to regulate calcium and phosphorous metabolism but for maintaining human health in general. Vitamin D is an endogenous, naturally occurring, photochemically-produced steroidal molecule with essential functions in systemic homeostasis and physiology, including modulation of calcium metabolism, cell proliferation, cardiovascular dynamics, immune inflammatory balance, neurologic function, and genetic expression.[2] Vitamin D deficiency is a worldwide health problem that affects not only musculoskeletal health but can affect many chronic diseases such as osteoporosis, cardiovascular disease, hypertension, cancer, depression, epilepsy, type 1 diabetes, insulin resistance, autoimmune disease, migraine, polycystic ovary syndrome, and musculoskeletal pain.[3]

In the skin 7 dehydrocholesterol (provitamin D3), the immediate precursor in the cholesterol biosynthetic pathway, is produced in relatively large quantities. During exposure to sunlight, ultraviolet B (UVB) radiation (290–315 nm) penetrating the epidermis and dermis cleaves the B-ring of the precursor to form pre-cholecalciferol. Pre cholecalciferol is unstable and rapidly undergoes rearrangement of its double bonds to form cholecalciferol. Thereafter, assisted by vitamin D binding protein (DBP), it enters into the dermal capillary bed. Cholecalciferol from the skin or ingested by diet undergoes two obligate hydroxylations, the first in the liver to 25 hydroxyvitamin D (25(OH)D). 25(OH)D bound to its DBP enters the circulation and is transported to the kidney where the second hydroxylation takes place at the 1a-position. 1,25(OH)2D is the metabolite responsible for the specific vitamin D effects, the active D-hormone.[4] Currently a level of at least 30 ng/ml 25(OH)D is considered as sufficient, values between 29 and 20 ng/ml as insufficiency, levels less than 20 ng/ml as deficiency.[5]

Thermal injury creates a breach in the surface of the skin. It is difficult to evaluate the immediate effects of burn injury on serum levels of vitamin D metabolites because the binding proteins for 25-hydroxyvitamin D (25(OH)D), the main circulating form of the vitamin, and for 1,25-dihydroxyvitamin D (1,25(OH)2D) are low. Thus, vitamin D Binding Protein and albumin two constitutive plasma proteins, are reduced, presumably as a result of the inflammatory response to the burn.[6] Also, burn victims do not spend a great deal of time outside because sweat glands are destroyed by the injury and the patients develop heat intolerance. Also burn specialists think that direct exposure to sunlight will cause hyperpigmentation of the burn wound. These two factors in conjunction with the failure of burn specialists to provide routine supplementation of vitamin D upon hospital discharge could contribute to the development of hypovitaminosis D. The absolute amount of 7 DHC in the skin of the burn victims was significantly reduced compared to normal controls not only in the burn scar but also in the normal-appearing adjacent tissue.[7]

It is also important to recognize patient demographic factors that may be associated with vitamin D deficiency including age, ethnic group (skin pigmentation), obesity, medical history (such as malabsorption pathologies and liver/renal disease), season, latitude, and time of day.[5] Vitamin D levels following severe thermal injury can also be reduced secondary to extrinsic causes including prolonged in hospital stay (including ICU), prolonged immobilization, and lack of supplementation. Although critically ill burn patients receive oral or enteral feed supplements, current regimens have proved ineffective in replenishing vitamin D levels in the acute phase.[8]

Furthermore, current long-term burn management regimens involve scar management comprising mainly of sun avoidance and protection, as well as the use of pressure garments. These factors minimize sun exposure, hence reducing 25(OH)D levels. In addition, both burn scar and adjacent normal skin in burn patients exhibit subnormal conversion levels of 7-DHC to pre-D3 compared to healthy individuals. This further potentiates vitamin D deficiency, resulting in low levels of 25(OH)D and 1,25(OH)2D for many years.[7]

METHODS

This study is a cross-sectional study. It was carried out in burn ward of Government medical college Jammu during June to November 2020. 52burn patients of both sexes and all age groups were enrolled in this study. Patients with history of surgery, hospitalization, or major medical illness within the past one year were excluded from the study. Patients on hormone replacement therapy, glucocorticoids, biophosphonates, and other drugs affecting bone metabolism were excluded as well. Intake of conventional calcium/vitamin D supplements was not considered an exclusion criterion. All subjects were enrolled after taking a written informed voluntary consent. Blood samples were sent to the laboratory during the first week after the acute burn injury. The 25(OH)D concentrations were measured by competitive radioimmunoassay after acetonitril extraction (Diasorin, stillwater, MN; catalog no. 68100E). Theminimal detectable limit of the 25(OH)D assay is 1.5 ng/ml.

Serum 25(OH)D, total protein, albumin were measured. A checklist consisting of age, sex, degree of burn, and total body surface area (TBSA) affected by burn was also completed for each patient. This study was approved by the ethics committee of Government medical college Jammu and was therefore performed in accordance with the ethical standards laid down in the 1964 declaration of Helsinki and its later amendments.

RESULTS

Fifty-two patients were evaluated in this study including 36 males and 16 females. The socio-demographic characteristics are shown in (Table 1). There were 14 patients in <10 years age group, 9 patients in 10-24, 10 patients in 24-39 and 19 in >40 age group. Seven patients had history of substance abuse and 45 did not. The details of burn injury are given in (Table 2). Out of 52 patients, 22 were flame burns, 16 were scald burns, 13 were electrical burns and 1 was a case of chemical burns. The mode of injury was mostly accidental. The degree of burns was second degree superficial in 15 patients, second degree deep in 34 patients, one had third degree and another one had fourth degree burns. Less than 30% total body surface area was involved in 38 patients, 31-50% in 7 patients and >50% in 7 patients. The frequency of patients for burn degree and mean vitamin D3 level are given in (Table 3). In second degree superficial burns the mean level of 25(OH)D was 13.60 ng/ml (SD=7.27), in second degree deep burns the mean level of 25(OH)D

was 13.88 ng/ml (SD=7.03) and in third degree burns the mean level of 25(OH)D was 14.71 ng/ml (SD=7.08).

Table 1: Socio-demographic characteristics of patients.

Variable	N
Gender	
Male	36
Female	16
Age (years)	
<10	14
10-24	9
24-39	10
>=40	19
Ethinicity	
Hindu	41
Muslim	10
Sikh	1
Others	0
Marital status	
Married	30
Unmarried	22
Socioeconomic status	
Lower	38
Middle	7
Upper	7
H/o substance abuse	
Yes	7
No	45

Table 2: Details of burn injury.

Parameters	N
Type of burn	
Flame	22
Scald	16
Electrical	13
Chemical	1
Mode of Injury	
Suicidal	1
Homicidal	1
Accidental	50
Degree of burns	
Second degree superficial	15
Second degree deep	34
Third degree	2
Fourth degree	1
Total body surface area involved (%)	
1-30	38
31-50	7
>50	7
Any operative procedure done	
Yes	8
No	44

Table 3: Frequency of patients for burn degree and mean vitamin D3 level.

Degree of burn	N	Vitamin D3 levels (mean±SD)
2nd degree superficial	15	13.60±7.27
2nd degree deep	34	13.88±7.03
3rd degree and above	3	14.71±7.08

DISCUSSION

The normal mean level of 25(OH) D is considered to be 30ng/ml. In our study, 2 patients had 25(OH)D levels of >30 ng/ml. In addition, the serum level of 25(OH)D was <20ng/ml in 40 patients, indicating vitamin D deficiency. The 25(OH)D levels were 20-30 ng/ml (insufficient) in 10 patients. Our study concluded that vitamin d levels are chronically low in burn injury patients and thus they need supplementation. Other studies like Klein et al, Klein et al also concluded that vitamin D levels are chronically low in burn patients and need to be supplemented. The main site for endogenous synthesis of 25(OH)D is the skin, which provides the majority of vitamin D precursor, with a small amount being absorbed through dietary intake.[10] In burn patients, this source is impaired; however, other factors also contribute to 25(OH) D deficiency in these patients, including electrolyte disturbances, malabsorption, reduced albumin levels, post-burn hyper-metabolism, and immobilization.[11]

Klein et al in his study standard multivitamin supplementation does not improve vitamin D insufficiency after burns stated that children suffering severe burns develop progressive vitamin D deficiency because of inability of burned skin to produce normal quantities of vitamin D3 and lack of vitamin D supplementation on discharge and concluded that Supplementation of burned children with a standard multivitamin tablet stated to contain 400 IU of vitamin D2 failed to correct the vitamin D insufficiency.[12] Klein et al in his study burns stated that the skin of the burned patient, both scarred area and normal-appearing adjacent skin, convert 7 dehydrocholesterol to pre-vitamin D3 at a rate that is 20–25% of normal skin and circulating levels of 25-hydroxyvitamin D are chronically low and burn injury gives rise to calcium wasting, failure of bone to take up excessive calcium, and vitamin D insufficiency to frank deficiency.[13] Sobouti et al in their study serum 25-hydroxyvitamin D levels in pediatric burn patients concluded that 25-hydroxyvitamin D levels are low in children after acute burns and these children should be given vitamin-D supplementation at 2-3 times the physiologic range (800-1200 IU/day).[14]

Chen et al conducted a study on additional vitamin and mineral support for patients with severe burns. They concluded that the supplementation of multiple vitamins, calcium, and magnesium reduced the risk of wound infection and sepsis and shortened hospitalization time.[15] Also, serum albumin concentrations are reduced

following thermal damage, and can remain low for a long period of time.[16] A significant correlation was observed between the 25(OH)D levels and albumin in the present study, which is in concordance with this finding. We began administering vitamin D to our burn patients as a part of their treatment protocol. Whatever the results indicate, it is obvious that patients with acute burn injuries will need essential vitamins and minerals more than normal individuals will.

CONCLUSION

Based on the results of this study, we can conclude that 25-hydroxyvitamin D levels are low in patients after acute burns, and these patients should be given vitamin D supplementation. The present study also showed a higher frequency of vitamin D deficiency; vitamin D levels were lower than recommended levels in 96.15% of patients, while 76.92% had deficiencies and 19.2% had insufficiencies.

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