

Casual Comparative Analysis of Quality of Life in Congestive Heart Failure Patients Presenting With & Without 25-Hydroxy Vitamin D Deficiency

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ABSTRACT

Objective: It is only natural to assume that the course of disease is more aggressive in the face of 25-Hydroxy vitamin D deficiency the prognosis of patients presenting with congestive heart failure would be grim. More important so, their quality of life during the years that they live would be markedly reduced.

Study Design: Observational / descriptive study.

Place and Duration of Study: This study was conducted at the Department of Cardiology, Liaquat University of Medical & Health Sciences, Jamshoro during the month of May 2016.

Materials and Methods: Forty patients (chosen via purposive sampling) presenting with congestive heart failure were included in the study. Data was derived using a structured interview based questionnaire after taking written informed consent. Investigations were also carried out to determine the levels of 25-hydroxy vitamin D levels in the blood. The patient condition and the quality of life was investigated at the time of discharging the patient and comparison was drawn.

Results: Sixty percent of the patients were men, while the remaining forty percent were women. The ratio of the patients with or without 25-hydroxy vitamin D deficiency was 1:1 and the patients with 25-hydroxy vitamin D deficiency were experiencing markedly low levels of quality of life due to aggravated disease.

Conclusion: The deficient levels of 25-hydroxy vitamin D, as indicated in literature, alter the metabolism of minerals and lead to myocardial dysfunction in the congestive heart disease patients. It is thus an aggravating factor for pathogenesis of congestive heart failure and leads to marked decrease in the quality of life.

Keywords: Congestive Heart Failure, 25-Hydroxy vitamin D, Vitamin Deficiency

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INTRODUCTION

The cardiac anomaly named congestive heart failure is categorized as a disease in which the ejection fraction of the left ventricle is markedly lessened. It is also, more often than not, associated with retention of bodily salts and water. The 2 prime symptoms of the ailment are weakness of the muscles and early onset of fatigue and tiredness. Congestive heart failure is prevalent worldwide and the levels loom between one percent to three percent in the developed world and with the prevalence climbing particularly in the geriatric population.¹⁻⁴

The true reason behind such widespread prevalence has still not been unearthed and neither is the disease pathogenesis fully identified. However, we do know that the disease is aggravated once the mineral metabolism is disturbed by any factor, be it deficiency of 25-Hydroxy vitamin D levels or anything else. The said deficiency affects the intracellular management of calcium ions and disturbs the myocardial contractions⁵. In experiments conducted on extracted myocytes from individuals suffering from congestive heart failure, calcium ion transition during the contractile phase of the heart were lessened and the during the relaxation phase they were heightened. The decrease and the consequent increase both matched pathologic level⁶. The use of digitalis and beta-blockers is thus merited in such a condition and for secondary stoppage. These drugs effectively act to enhance the cardiac contraction and thus the ejection fraction⁷⁻⁸.

A review of recent literature brings to light a fresh research that concludes that vitamin D deficiency congestive heart failure occur together far too often for the occurrence to be coincidental⁹. It is important to note that multiple hypothesis exist that hope to explain

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how vitamin D deficiency steepens the course of this disease, some going as far as calling it catalytic¹⁰⁻¹². Research also indicate that individuals who are deficient in this crucial vitamin are more likely to have a poor prognosis and have a reduced life span.^{13,14}. Also, the effectiveness of the treatment of this disease is limited if the deficiency is not overcome¹⁵. Circulating levels of 25-Hydroxy vitamin D are thus important and need to be maintained if a good prognosis is to be achieved¹⁶.

MATERIALS AND METHODS

Forty patients (chosen via purposive sampling) presenting with congestive heart failure at Liaquat University Hospital, Department of Cardiology during the month of May 2016 were included in the study. Data was derived using a structured interview based questionnaire after taking written informed consent. Investigations were also carried out to determine the levels of 25-hydroxy vitamin D levels in the blood. The patient condition and the quality of life was investigated at the time of discharging the patient and comparison was drawn.

RESULTS

Sixty percent of the patients were men, while the remaining forty percent were women.

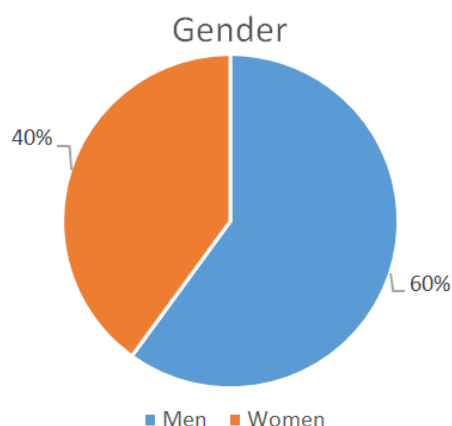
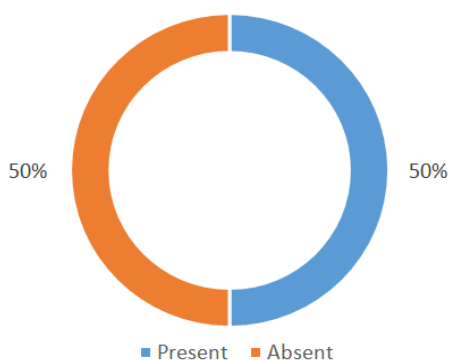


Figure No.1: Congestive heart Failure



FigureNo.2: Patients Ratio

Congestive heart Failure was more common in men and less common in women. The cardio-protective effect of the hormone in women prior to menopause may be the reason behind the disease being less common in women. While aggravating factors like smoking, which are more common in men, may explain the high levels of the disease in men. (Figure 1)

The ratio of the patients with or without 25-hydroxy vitamin D deficiency was 1:1.

The patients with 25-hydroxy vitamin D deficiency were experiencing markedly low levels of quality of life due to aggravated disease

It was important that the ratio be kept same to remove all bias that may have altered the result credibility and validity. (Figure 2).

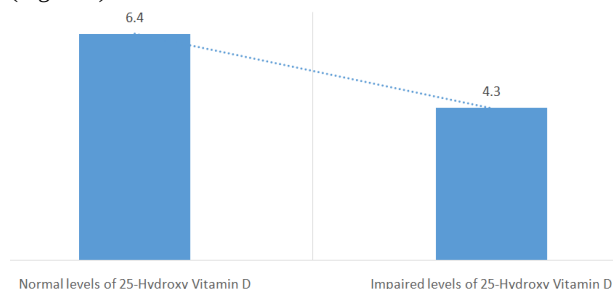


Figure No.3: Impaired Quality of Life

DISCUSSION

The vitamin 25-Hydroxy D casts multiple individual effects on muscle cells. It influences the calcium ion transfer to intracellular region and it is proved by culture studies where the mechanism was studied on a petri dish.¹⁷ The impact that this crucial vitamin casts on cardiac cells is somewhat lower than what it does to the regular muscle cells but still its significant in every aspect.¹⁸⁻²⁰ In diseased individuals, the levels of this vitamin are thus of particular importance.

In the western world, the levels of 25-Hydroxy vitamin D are mostly dependent on the amount of time that an individual spends in broad day light and the area of exposure on body of ultraviolet spectrum of light (a.k.a UV B light).²¹ In normal circumstances, a low level of exposure does not cause the levels of the said vitamin to drop but in diseased individuals who are often bedridden, the exposure level and duration drop to nearly a zero. The diet too of such individuals is not very supportive and so in the face of heightened demand and poor supply, the condition worsens and the disease is aggravated.²²⁻²⁵

The dropped levels of the vitamin, when they aggravate the disease and worsen the adverse symptoms, cause distress to the patients and reduce their quality of life to a large extent.^{26,27} It is noted in our study that individuals suffering from the disease benefit from the use of medication as the use of digitalis and beta-blockers acts to enhance the cardiac contraction and thus the ejection fraction⁷⁻⁸, but it is still not enough to counter the negative effects cast by 25-Hydroxy vitamin D deficiency.

CONCLUSION

The deficient levels of 25-hydroxy vitamin D, as indicated in literature, alter the metabolism of minerals and lead to myocardial dysfunction in the congestive heart disease patients. It is thus an aggravating factor for pathogenesis of congestive heart failure and leads to marked decrease in the quality of life.

Conflict of Interest: The study has no conflict of interest to declare by any author.

REFERENCES

- McKee PA, Castelli WP, McNamara PM, Kannel WB. The natural history of congestive heart failure. the Framingham study. *N Engl J Med* 1971;144:144-146.
- McDonald TF, Pelzer S, Trautwein W, Pelzer DJ. Regulation and modulation of calcium channels in cardiac, skeletal, and smooth muscle cells. *Physiol Rev* 1994; 74:365-507.
- Dargie HJ, McMurray JJ. Diagnosis and management of heart failure. *Br Med J* 1994; 308: 321-328.
- Statistisches Bundesamt; Statistisches Jahrbuch für die Bundesrepublik Deutschland. Metzler Poeschel Verlag Stuttgart 2000:415-427.
- Krüger C, Erdmann E, Nabauer M, Beuckelmann DJ. Intracellular calcium handling in isolated ventricular myocytes from cardiomyopathic hamsters (strain BIO 14.6) with congestive heart failure. *Cell Calcium* 1994; 16:500-508.
- Beuckelmann DJ, Näbauer M, Erdmann E; Intracellular calcium handling in isolated ventricular myocardium. *Circulation* 1992;85: 1046-1055.
- Colucci WS, Wright RF, Braunwald E. New positive inotropic agents in the treatment of congestive heart failure. Mechanisms of action and recent clinical developments. *N Engl J Med* 1986; 314:290-299.
- Kubo H, Margulies KB, Piacentino V, Gaughan JP, Houser SR. Patients with end-stage congestive heart failure treated with beta-adrenergic receptor antagonists have improved ventricular myocyte calcium regulatory protein abundance. *Circulation*. 2001; 104:1012-1018.
- Shane E, Mancini D, Aaronson K. Bone mass, vitamin D deficiency, and hyperparathyroidism in congestive heart failure. *Am J Med* 1997; 103: 197-207.
- Thomasset M, Parkes C.O, Cuisinier-Gleizes P. Rat calcium-binding proteins. distribution, development, and vitamin D dependence. *Am J Physiol* 1982; 243:E483-E488.
- Simpson RU, Weishaar RE. Involvement of 1,25-dihydroxyvitamin D3 in regulating myocardial calcium metabolism. physiological and pathological actions. *Cell Calcium* 1988;9:285-292.
- De Boland AR, Boland RL. Non-genomic signal transduction pathway of vitamin D in muscle. *Cell Signal* 1994; 6:717-724.
- Weishaar RE, Simpson RU. Involvement of vitamin D3 with cardiovascular function. II. Direct and indirect effects. *Am J Physiol* 1987; 253: E675-E683.
- Hochhauser E, Barak J, Kushnir T. Mechanical, biochemical, and structural effects of vitamin D deficiency on the chick heart. *Angiol* 1989; 40: 300-308.
- Wu J, Garami M, Cao L, Li Q, Gardner DG. 1,25(OH)2D3 suppresses expression and secretion of atrial natriuretic peptide from cardiac myocytes. *Am J Physiol* 1995; 268:E1108-E1113.
- Lerman A, Gibbons RJ, Rodeheffer RJ. Circulating N-terminal atrial natriuretic peptide as a marker for symptomless left-ventricular dysfunction. *Lancet* 1993; 341:1105-1109.
- Zittermann A, Scheld K, Stehle P. Seasonal variations in vitamin D status and calcium absorption do not influence bone turnover in young women. *Eur J Clin Nutr* 1998; 52:501-506.
- Haug CJ, Aukrust P, Haug E, Morkrid L, Muller F, Froland SS. Severe deficiency of 1,25-dihydroxyvitamin D3 in human immunodeficiency virus infection. Association with immunological hyperactivity and only minor changes in calcium homeostasis. *J Clin Endocrinol Metab* 1998; 83: 3832-3838.
- Selles J, Bellido T, Boland R. Modulation of calcium uptake in cultured cardiac muscle cells by 1,25-dihydroxyvitamin D3. *J Mol Cell Cardiol* 1994; 26:1593-1599.
- Penpargkul S, Bhan A, Scheuer J; Studies of subcellular control factors in hearts of uremic rats. *J Lab Clin Med* 1976; 88:563-570.
- Birge SJ, Haddad JG. 25-Hydroxycholecalciferol stimulation of muscle metabolism. *J Clin Invest* 1975; 56:1100-1107.
- Brunvand L, Haga P, Tangsrud SE, Haug E. Congestive heart failure caused by vitamin D deficiency. *Acta Paediatr* 1995;84:106-108.
- Gillor A, Groneck P, Kaiser J, Schmitz-Stolbrink A. Congestive heart failure in rickets caused by vitamin D deficiency. *Monatsschr Kinderheilk* 1989; 137:108-110.
- Avery PG, Arnold IR, Hubner PJ, Iqbal SJ. Cardiac failure secondary to hypocalcaemia of nutritional osteomalacia. *Eur Heart J* 1992; 13:426-427.
- McKenna M. Differences in vitamin D status between countries in young adults and elderly. *Am J Med* 1992; 93:69-77.
- MacLaughlin J, Holick MF. Aging decreases the capacity of human skin to produce vitamin D3. *J Clin Invest* 1985; 76:1536-1538.
- Heaney RP. Vitamin D. How much do we need, and how much is too much. *Osteoporos Int* 2000; 11:553-555.