



CVS EFFECTS WITH LOW VITAMIN D3 (REVIEW LITERATURE)

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ABSTRACT

Severe epidemiological studies suggested that there is a strong association between Low vitamin -D developing Cardio vascular diseases (CVD).

CVD considered to be the main cause of death in india. Low vitamin D is predictor for all cause cardio vascular mortality.

KEYWORDS : Hypertension , Diabetes , LVH , Heart strokes, Vitamin D

Modifiable Risk Factor

1. Low Vitamin D
2. Smoking & Alcohol
3. HTN & Diabetes
4. Hyperlipidemia
5. Obesity / Stress / Physical Inactivity

Non modifiable Risk Factor(1)

- Male sex
- Age
- Elderly Patient
- Family H/O CAD +ve

Definition:-

Vitamin-D Deficiency < 20 ng/ml
Vitamin-D Insufficiency 21-29 ng/ml.

Vitamin-D (2 In Active Precursors):-

1. Vitamin D3 (Cholecalciferol) Endogenous production
2. Vitamin D2 (Ergocholecalciferol) Exogenous production

Mechanism :- Low Vitamin D & Cardio Vascular Diseases

1. 1,25(OH)-D regulates R.A.Axis by direct suppression of Renin gene expression
2. Vascular smooth muscle & Endothelial cell – Express Vit D Receptor & ability to convert 25(OH) To 1,25(OH) Vitamin D
3. Vitamin D Deficiency Triggers Secondary Hypoparathyroidism

Functions Of Vitamin-D:-

1. Increase Intestinal absorption of calcium
2. Expand to find Vit D Receptor
3. Vit D Deficiency Promotes Hypertension leading to LVH
4. Vitamin D Deficiency considered as a marker of chronic non specific illness, rather than contributor to direct disease pathogenesis
5. Low vitamin-D causes LVH
6. Vitamin D increases Serum Calcium & Phosphate

Recommended Daily Doses:-

1. 600 IU Daily for 0-12 months age
2. 1000 IU Daily for 1-70 yrs
3. 800 IU Daily > 71 yrs.

Conclusion:-

1. LVH seen in patients with Absent Vitamin-D Receptor (2)
2. Vitamin D-Deficiency In Haemodialysis patients increases CVS Mortality(3)
3. Normal Vitamin-D Levels Decreases Fracture rates & maximal parathyroid suppression
4. Ultra violet rays are best natural source of vitamin D
5. Vitamin -D shows to have Immuno suppressive effects & have important role in atherogenesis

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