



Libyan International Medical University



Faculty of Basic Medical Science

The relation between Vitamin D deficiency and prostate cancer

Submitted by: Malik Ali Gargoum (1364), Third Year Medical Student,
Libyan International Medical University

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Abstract

Prostate cancer is a major cause of cancer deaths among males, prostate cancer has 3 distinct risk factors : age , race and geography. these factors all are associated with decreased synthesis of vitamin D. The first two studies revealed the relation between the low levels of serum Vitamin D and high risk of prostate cancer and show a differences according to the age and the race, And the last study show relation between the Vitamin D receptor gene polymorphism and prostate cancer risk.

Introduction

Prostate cancer is the second largest cause of cancer deaths among men. One man in 11 will develop prostate cancer during his lifetime . The epidemiology of prostate cancer has 3 distinct risk factors : age , race and geography. Prostate cancer shows a pronounced in incidence with age : over 80% of prostate cancers are diagnosed in men over the age of 65 , Race also is an important factor .The incidence rates for American blacks are twice those of Whites . Conversely the rates for the indigenous Japanese are 1/20 those of U.S Whites. This indicates that environmental factors influence the risk of developing prostate cancer. This report propose that Vitamin D deficiency increase the risk of clinical prostate cancer . Vitamin D is a group of fat-soluble secosteroids responsible for increasing intestinal absorption of calcium, magnesium, and phosphate, and multiple other biological effects. In humans, the most important compounds in this group are vitamin D₃ (also known as cholecalciferol) and vitamin D₂ (ergocalciferol).^[1] Cholecalciferol and ergocalciferol can be ingested from the diet and from supplements. Only a few foods contain vitamin D. The major natural source of the vitamin is synthesis of cholecalciferol in the skin from cholesterol through a chemical reaction that is dependent on sun exposure . Vitamin D from the diet or skin synthesis is biologically inactive; enzymatic conversion (hydroxylation) in the liver and kidney is required for activation. As vitamin D can be synthesized in adequate amounts by most mammals exposed to sufficient sunlight, it is not an essential dietary factor. Cholecalciferol is converted in the liver to calcifediol (25-hydroxycholecalciferol); ergocalciferol is converted to 25-hydroxyergocalciferol. These two vitamin D metabolites (called 25-hydroxyvitamin D or 25(OH)D) are measured in serum to determine a person's vitamin D status. Calcifediol is further hydroxylated by the kidneys to form calcitriol (also known as 1,25-dihydroxycholecalciferol), the biologically active form of vitamin D. Calcitriol circulates as a hormone in the blood, having a major role regulating the concentration of calcium and phosphate, and promoting the healthy growth and remodeling of bone. Vitamin D is a common element of the major risk factors for prostate cancer consider first the increase in morbidity and mortality with age , The elderly are D-deficient due to little UV exposure and declines the ability to synthesize vitamin D . And the Blacks are at high risk because the high melanin of skin inhibits the formation of Previtamin D. The low risk for Japanese due to the traditional diet which is rich in oily fish is protective .Vitamin D₃ inhibits the growth of cancer cells in vitro and vivo ,The mechanism is unknown ,but may related to the ability of inhibits the expression of the myc oncogene .And genetic variation in vitamin D binding protein also may be a risk⁽¹⁾ .

Discussion

Results from three (3) different studies have been gathered, and they were as follows:

1. 1st Study :

Case-control study on Nordic men (Norway, Finland and Sweden) studied serum 25(OH)-vitamin D levels of 622 prostate cancer cases and found that both low (≤ 19 nmol/l) and high (≥ 80 nmol/l) 25(OH)-vitamin D serum concentrations are associated with higher prostate cancer risk. The normal average serum concentration of 25(OH)-vitamin D (40–60 nmol/l) comprises the lowest risk of prostate cancer. low vitamin D serum concentration apparently leads to a low tissue concentration and to weakened mitotic control of target cells, whereas a high vitamin D level might lead to vitamin D resistance through increased inactivation⁽²⁾ .

2nd Study:

This study evaluates the risk of prostate cancer in relation to serum levels of the major vitamin D metabolites, 25-hydroxyvitamin D (25-D3) and 1,25-dihydroxyvitamin D (1,25-D). more than 250,000 serum samples were collected. Levels of 25-D and 1,25-D were measured in samples from 90 black and 91 white men diagnosed with prostate cancer and controls individually matched on age, race. Risk of prostate cancer decreased with higher levels of 1,25-D . The association of lower 1,25-D with prostate cancer was found in men above the median age of 57 years but not younger men and was similar in black and white men. In men \geq 57 years of age, 1,25-D was an important predictor of risk for palpable and anaplastic tumors⁽³⁾.

3rd Study :

This study tested the hypothesis that vitamin D receptor gene polymorphisms are associated with prostate cancer risk using a case-control study of 108 men undergoing radical prostatectomy and 170 male urology clinic controls with no history of cancer. Among the white control group, 22% were homozygous for the presence of a *TaqI* RFLP, but only 8% of cases had this genotype. A similar trend was seen among the small number of blacks in this study (13% for controls, 8% for cases), although the difference was not statistically significant. Race-adjusted combined analysis suggests that men who are homozygous for the *t* allele (shown to correlate with higher serum levels of the active form of vitamin D) have one-third the risk of developing prostate cancer requiring prostatectomy compared to men who are heterozygotes or homozygous for the *T* allele⁽⁴⁾.

Conclusions

We conclude that vitamin D deficiency may promote prostate cancer ,also may be an important determinant of prostate cancer risk.

Bibliography (References)

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