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Editorial Note

Role of Vitamin D Receptor (VDR) Gene in Rheumatoid Arthritis

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EDITORS WORDS

Rheumatoid Joint Pain (RA) is generally connected with diminished Bone Mineral Thickness (BMD) because of various variables. BsmI polymorphism of the nutrient D receptor (VDR) quality has been involved in the pathogenesis of osteoporosis. Nutrient D has a few immunomodulatory impacts and along these lines may assume a part throughout joint pain. Nonetheless, little information is accessible on the conceivable connection among RA and VDR quality polymorphisms. In this examination, the recurrence of BsmI polymorphism genotypes were contrasted and that found in different nations.

In this examination, 64 RA patients and 40 sound controls were tried for VDR quality BsmI polymorphism genotypes. Frequencies of B and b alleles were related with markers of bone digestion and RA. Among control subjects, the recurrence of the BB genotype is generally high (27.5%). In RA with auxiliary osteopenia/osteoporosis the BB genotype was more uncommon, the bb was more normal than in charge subjects. Markers of bone digestion were related with the B allele. RA patients conveying the B allele had lower BMD and expanded bone misfortune more than 1 year.

The B allele was additionally related with expanded osteoclast and osteoblast work, as controlled by the evaluation of biochemical markers of bone digestion. Rheumatoid factor titer, which is a free marker for sickness movement in RA, was higher in bb patients. Our information recommend, that the awkwardness in B and b allele articulation might be engaged with the pathogenesis of RA-related osteoporosis. The conceivable association of nutrient D and VDR quality polymorphisms in the turn of events and movement of RA needs further clarification.

Vitamin D Receptor (VDR) gene is key to bone formation and hence it might have good contribution in RA. Therefore, in this study association of VDR SNP rs1544410 was evaluated in 500 individuals including 250 RA cases and 250 matching controls. VDR gene was genotyped using ARMS-PCR method and the product was resolved on 2% agarose gel. Through co-dominant model analysis we found that homozygous "AA" genotype was 24.80% (n= 62) in cases and 28.8% (n=72) in controls. Similarly, heterozygous "AG" was 39.20% (n=98) in cases and 35.20% (n=88) in controls. While the homozygous GG was 36.0% (n=90) and 36.0% (n=90) in cases and controls, respectively ($\chi 2$=1.284; P=0.526).

Through additive model analysis we determined that 44.4% (n=222) cases and 46.4% (n=232) control individuals had allele "A". While 55.6% (n=278) of cases and 53.6% (n=268) of control individuals had allele "G" (OR=0.922(0.719-1.183); RR=0.96(0.847-1.088); P=0.567). Effect of major allele on association of VDR to RA was checked through homozygous dominant model analysis. Homozygous "GG" was found in 36.0% (n=90) of cases and 36.0% (n=90) in controls. Similarly, "AG+AA" was found similar in both cases and controls that is 64.0% (n=160). The distribution was insignificant at VDR rs1544410 (OR=1.0(0.694-1.44); RR=1.0(0.833-1.20); P=1.000).

Similarly, effect of minor allele on the association of VDR to RA was assisted through recessive model analysis. Homozygous "AA" was present in 24.80% (n=62) cases and 28.8% (n=72) controls. Whereas, "AG+GG" was 75.20% (n=188) in cases and 71.20% (n=178) in controls (OR=0.815(0.548-1.212); RR=0.9008(0.731-1.109); P=0.363. This data suggests that there is no association between VDR and RA in Pakistani patients. Although, we did this study on a sample set of 500 individuals, so, a study on large sample set will be needed to confirm its role in RA.

All in all, high BMI and positive maternal and fatherly family ancestry are huge elements in the beginning of RA just as OA. Also, nutrient D level isn't fundamentally deficient however VDR quality polymorphism is a critical danger factor of RA just as OA beginning in Pakistani populace.

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