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Increased organ chlorine pesticides load in chronic kidney disease patients: Role of glomerular filtration rate and polymorphisms of xenobiotic metabolizing enzymes

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Until date there is no clear answer regarding whether high levels of organ chlorine pesticides (OCP) found associated with chronic kidney disease (CKD) cause kidney damage or they get accumulated due to falling glomerular function rate (GFR). We have observed that blood OCP levels as analyzed by gas chromatography, show significantly higher levels in CKD patients. Spearman's correlation analysis of OCP levels with eGFR exhibited significant negative correlation for most individual OCPs which persisted even after statistical adjustments for age, sex, BMI, total cholesterol and triglycerides. Another of our studies pointed out association of higher OCP loads in patients with genetic polymorphisms involving CYP1A1. Subjects carrying at least one mutant allele of CYP1A1*2A (TC, CC) and *2C (AG, GG) were found to have a modest rise of odds (1.4-2) of association with CKD. However, genotypic combinations of heterozygous/homozygous mutants were found to be significantly associated with CKD with odds ratios ranging from 1.8-3. Another of our studies, where we also analyzed genetic polymorphisms of GSTM1 and GSTT1, showed similar results. We observed that, presence of GSTM1 (-)/GSTT1 (-) genotype was associated with 1.8-fold higher odds of association with CKD compared to wild genotypes i.e., GSTM1 (+)/GSTT1(+). Logistic regression analysis by taking wild genotypes GSTM1 (+)/GSTT1 (+) as reference revealed that, in CKD patients several pesticides showed significant association with either null or both null genotypes. The above results suggest that decreasing GFR and genetic polymorphisms involving xenobiotic metabolizing genes both play a role in accumulation of OCPs in CKD patients.

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Cystatin isolation and characterization from kidney (CICK)

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Cystatin is virtually the most widely represented class of thiol proteinase inhibitor, ubiquitously distributed in plants and animals and devoted to regulating degradation of both intracellular and extracellular proteins. In our study, a thiol proteinase inhibitor was isolated from buffalo kidney making use of ammonium sulfate precipitation and gel filtration chromatography on Sephacryl S-100HR column. The basis behind selecting this source for cystatin isolation is the growing number of kidney disorders in the current era. Purified inhibitor is homogeneous as it displayed a single band in gel electrophoresis both under reducing and non-reducing environment and is of 65 kDa as revealed by gel filtration and SDS PAGE. Kinetic studies revealed the presence of reversible accompanied with competitive mode of inhibition; showing maximum efficacy against papain ($K_i=2.90 \times 10^{-4}$). It was maximally active at pH 8.0 and was stable for a period of 30, 60 and 90 days at 37, 4 and -20 °C respectively. Immunological studies confirmed its purity of epitopes as a single precipitin line is obtained in immunodiffusion. N-terminal analysis revealed that it shared a good homology with mouse kidney cystatin as well as with human Cys C and Cys E thereby advocating its use as a model for various human oriented studies which targets how the kidney cystatin level varies in accordance with various drugs that are currently being used as a target for variety of diseases. Further studies are taken into account in using this study as a model and treating various ailments related to kidney.

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