THESIS CONCLUSIONS

The thesis explored the CG, MDRD and MCQE equations for renal dysfunctions, but they have their known limitations. CG is influenced by weight and age, MDRD mostly derived from kidney disease patients and its accuracy in normal healthy individuals is not clear. MCQE is not showing low eGFR in normal healthy individuals. Finally, MCQE formula is acceptable for normal controls and MDRD and MCQE are both satisfactory for CKD patients.

The thesis hypothesised that there is a Dyslipidemia, increase in lipid peroxidation marker MDA and decrease in antioxidant enzymes like SOD. Based on the findings therapeutic measures for lipid profile and antioxidants may strengthen CKD patients against further complications especially in patients undergoing hemodialysis. Anemia is a risk factor for cardiovascular disease and our study demonstrated that there is significant alteration in erythrocyte indices in CKD. It has also shown decreased serum iron and TSAT% and increased TIBC and serum ferritin in CKD patients who are not under dialysis treatment. In hemodialysis group serum iron and TIBC are decreased and serum ferritin is raised which is associated with inflammatory status. Peripheral smear examination showed there is microcytic anemia and these alterations might reflect the burden of cardiovascular disease.

Cardiovascular disease can be considered as a cofounding factor in CKD as there is a need for early diagnosis and treatment of CVD in CKD patients. In hemodialysis patients different factors negatively show impact on cardiovascular system. In our study it was observed that there is raised value of IMA in CKD patients due to oxidative stress and raised NT-pro BNP due to left ventricular hypertrophy. In CKD patients there is muscle wasting which may cause raise in myoglobin and CK-MB. The rise in HFABP can be explained by the fact of uremia causing myocardial and skeletal muscle damage as well as decreased clearance. These values are still raised when compared between nondialysis and hemodialysis. However, our results suggest that cardiac troponin I (CTnI) raise in CKD is attributed to cardiac involvement this is because other factors will not influence the raise but in hemodialysis the presence of small vessel thrombosis is the cause for altered values.
Proteomic study in hemodialysis patients with LVH has revealed that there are 57 changes was observed in 2D-DIGE of which 3 unique spots in control and 28 unique spots were observed in cases. Not only this 22 over expressed spots and 4 under expressed spots were observed in the cases of these 57 spots we have selected 15 spots for further analysis by MALDI-TOF after analyzing the spots, they were identified to be plasma inflammatory proteins (such as Monoclonal antibody CH89 heavy chain (partial), Immunoglobin heavy chain variable region, IgM heavy chain VH1 region precursor), nuclear related proteins (such as zinc finger protein 224, lethal malignant brain tumor like protein, mitotic check point) and a number of the other identified serum proteins. The raise of inflammatory markers is due to the cardiovascular abnormalities.

**Further studies**

Proteome analysis has emerged as a new field of protein science offering the possibility of achieving unbiased identification, quantification and functional assessment of all proteins and peptides present in biological samples. The current research helped in envisaging the identification and characterization of some renal biomarkers from pooled serum of patients from Indian origin. Further studies of unique protein(s) that are expressed differently and differentially in various stages of chronic kidney disease helps in development of kits suitable as prognostic markers to differentiate various stages in CKD in a cost-efficient manner.