Cardiac and Non-cardiac Determinants of Exercise Capacity in Asymptomatic Non-Diabetic Chronic Kidney Disease Patients

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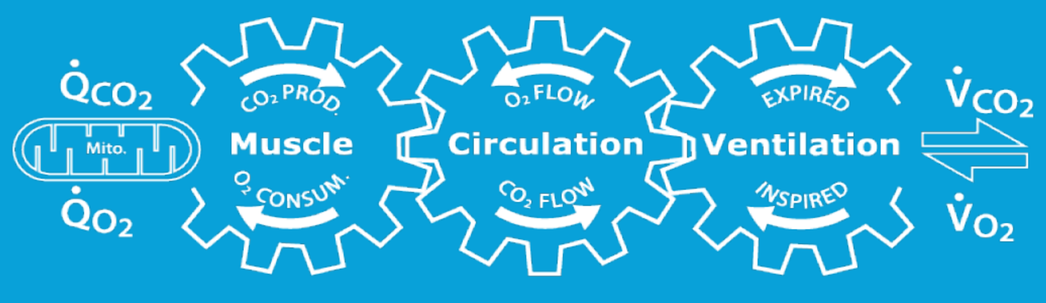

INTRODUCTION

As there is growing interest in utilising the measures of exercise capacity such as peak O_2 consumption (VO_{2max}) as markers of cardiac dysfunction in CKD, it is necessary to understand the differential role of cardiac output (CO) and peripheral O_2 extraction in determining exercise capacity in CKD. After all VO_{2max} is a product of CO and $C(a\text{-}v)O_2$.

Hypothesis: In the present study we tested the hypothesis that reduced peripheral O_2 extraction contributes significantly to impaired exercise capacity in CKD.

METHODS

- A cross sectional study of 60 asymptomatic male non-diabetic CKD patients [CKD stages 2-5 (pre dialysis)] without primary cardiac disease.
- Historical data from age matched healthy male volunteers (n=101) was used as controls.
- Data from heart failure (HF) patients of NYHA class II&III (n=39) was used as positive controls.
- Specialised CPX test with CO_2 rebreathing technique was utilised to measure peak cardiac output non-invasively.
- CKD related biochemical parameters were also measured. Results are presented as mean±SD. $P<0.05$ is considered as significant.

RESULTS

- The VO_{2max} of the study groups were: Controls: 2.98±0.9, CKD2-3: 2.74±0.5, CKD4: 2.54±0.5, CKD5: 2.18±0.4 & HF: 1.57±0.4 l/min.
- Fig 1 shows the correlations of Peak CO and $C(a\text{-}v)O_2$ with VO_{2max} and Fig 2 shows the relative differences in these parameters amongst the study groups (Healthy controls, CKD patients and Heart failure patients).
- Table 1 shows the significant correlates of VO_{2max} on univariate analysis (all $P<0.05$). No correlation was demonstrated with Ca, PO_4, PTH, total cholesterol or urine protein creatinine ratio.
- Multivariate analysis showed that PkCO (β=0.387), Hb (β=0.314), peak heart rate (β=0.236), BMI (β=0.22) and age (β =-0.194) were independent predictors of VO_{2max} in CKD together accounting for >75% of its variability.

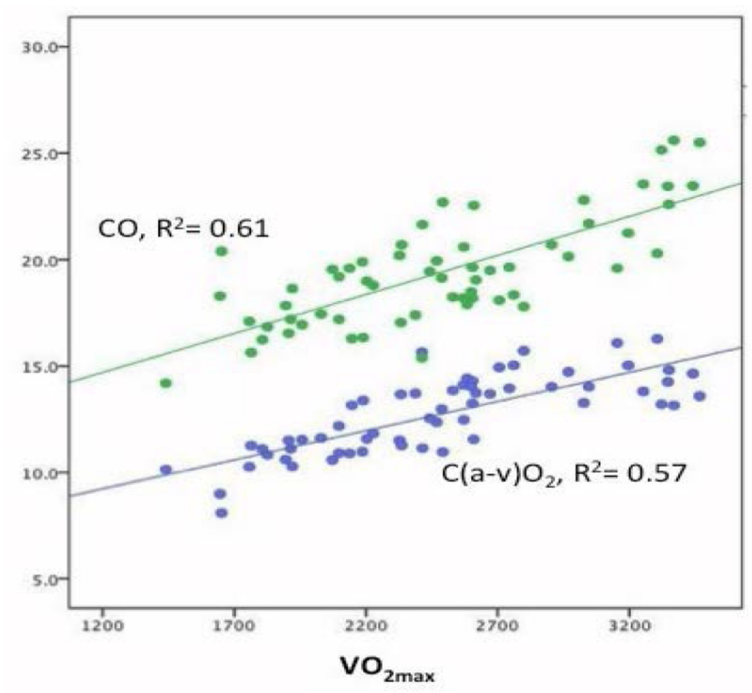

Fig 1: Correlation of peak O2 consumption with Peak cardiac output and arterio-venous O_2 difference in CKD patients.

Table 1

Parameters	Correlation Coefficient (r)
Pk heart rate	0.48
Age	0.50
BMI	0.30
eGFR	0.51
Hb	0.60

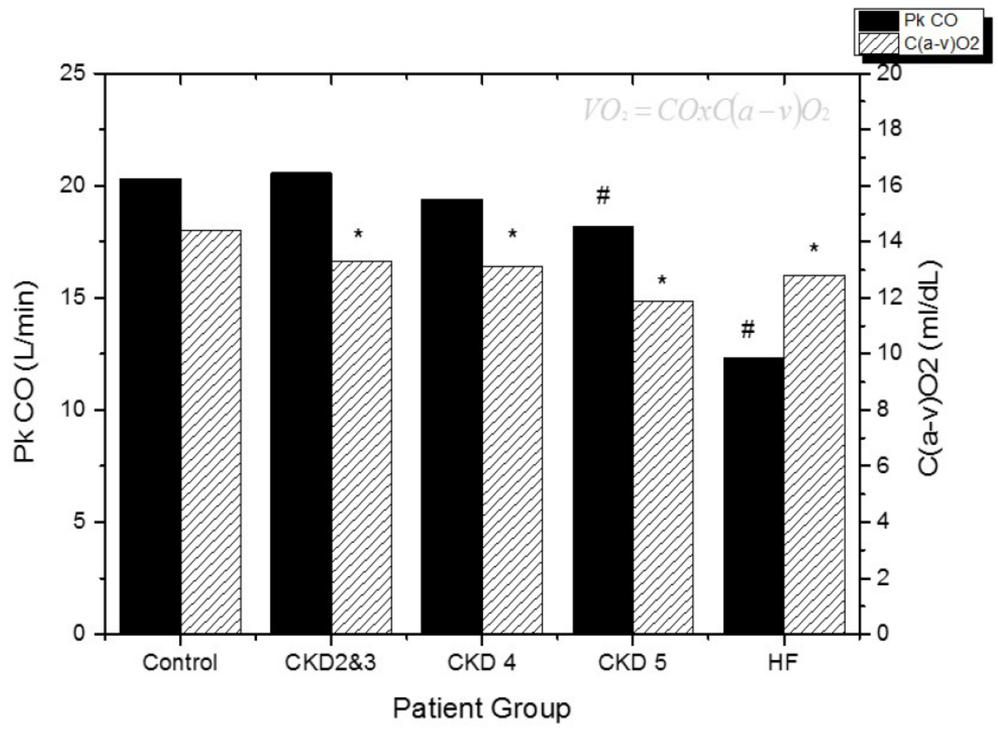

Fig 2: Peak cardiac output and arterio-venous O_2 difference across the study categories. The reduction in VO_{2max} in CKD patients is influenced by greater reduction in $C(a\text{-}v)O_2$ than Pk CO in contrast to that of HF patients. # $p<0.05$ vs Controls for Pk CO. *$p<0.05$ vs Controls for $C(a\text{-}v)O_2$.

CONCLUSION

- This study demonstrates the relative contributions of cardiac and non-cardiac determinants of exercise capacity in CKD.
- It highlights the significant role of peripheral O_2 extraction and hence the limitation in utilising VO_{2max} as a surrogate of cardiac dysfunction.

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