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QUALITY OF LIFE (QOL) DURING THE COURSE OF CKD: A LONGITUDINAL STUDY

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Background and Aims: Measuring QoL in CKD patients is fundamental to estimate the human cost of chronic diseases and to assess the effect of treatments. Non-medical factors play an important role in CKD progression and QoL. However, there is very limited information on the evolution of QoL over time in CKD patients and it remains unclear whether traditional and CKD specific risk factors are implicated QoL in CKD patients over CKD progression.

Method: We studied a cohort of 582 stage 2-5 CKD patients (age: 61 ± 12 years; M: 60%, diabetics: 33%) and measured QoL by the short form of the Rand corporation questionnaire (SF36), an instrument which measures eight domains of QoL (physical functioning, role physical health, energy fatigue, pain, role emotional problem, emotional well-being, social function, and general health) and two summary scores, (the physical component score and the mental component score) which are calculated by a well validated algorithm (Taft C et al, 2001, Quality of life research). In all patients the SF36 was administered at enrolment and after one (489 patients), two (n=434) and three (n=287) years. The evolution of SF36 dimensions over-time and the predictors of SF36 changes were analyzed by the Linear Mixed Model (LMM).

Results: At baseline the median value of the Physical Component Score (PCS) was 43.7 (Interquartile range: 34.0-50.3) and the Mental Component Score (MCS) was 46.3 (37-52.9) and on average did not change over the 3 years follow up [median PCS at the 3rd year: 46.3 (35.7-52.0), median MCS at the 3rd year 43 (33.6-50.6)]. On average the GFR at baseline was 36 ± 13 ml/min/1.73 m² and declined to 34 ± 17 ml/min/1.73m² at the 3rd year. On detailed longitudinal analysis by the LMM the PCS associated with the evolution of the GFR over time (beta=0.10; 95% CI from 0.06 to 0.13; P<0.001).

Adjustment for time (0,1,2,3 years), age and gender did not materially modify such an association (beta=0.09; 95%CI from 0.06 to 0.13, p<0.001) while further adjustment for traditional (Systolic BP, diabetes, smoking, cholesterol), BMI, CV comorbidities and CKD specific (hemoglobin, albumin, calcium, phosphate) risk factors attenuated but did not cancel out the PCS-GFR link (beta=0.05, 95%CI 0.006 to 0.093, P=0.03). This finding suggests that the PCS-GFR link is either largely confounded or mediated by these risk factors but that the same risk factors do not explain in full the same link. The MCS – GFR association was weaker (beta=0.05, 95%CI from 0.008 to 0.09; P=0.02) than the PCS-GFR relationship, became non significant after simple adjustment for time, age and gender (beta=0.04; 95%CI -0.003 to 0.08; P=0.07) and was nullified after full adjustment (beta=-0.01; 95%CI -0.07 to 0.04; P=0.59) for the same risk factors.

Conclusion: The PCS and the MCS remain stable over the course of CKD but appear associated with the evolution of the GFR over time. Traditional and CKD specific risk factors substantially confound and/ or mediate these associations.