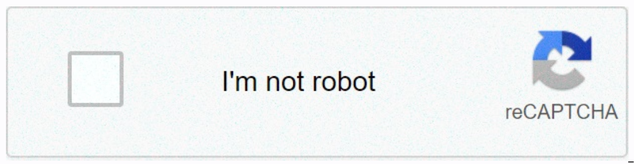

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Protein catabolic rate

Skip the navigation destination background. Spontaneous reduction of dietary protein intake is a recognized feature of severe renal insufficiency, and previous studies have shown that it can occur in the early stages of renal function deterioration. Methods. We examined the effect of progressive renal failure on normalized protein catabolism rate (nPCR) in 1,282 patients (mean age 55.8±15.5 years; 60.4% male) within 7 years. All nPCR values (n = 5082) obtained prior to dialysis were included. A total of 361 (28.2%) patients subsequently developed at the end stage of renal failure and dialysis was initiated. Results. Cross-sectional analysis showed that nPCR is significantly lower when creatinine clearance is lower. The mean nPCR in clearance >50±±04±0.27 was 1.17, 0.93, 0.93±21 at 10-25 and 0.74±0.18 at <10 ml/ min. The mean nPCr in each clearance group was different from all other groups (P<0.001 in all cases). When nPCR was studied lengthwise relative to the start time of dialysis, the nPCR drop became significant only within 3 months prior to initiation of treatment. The curve adjustment showed an exponential association of two phases between nPCR and renal function, mild decrease in NPCR in mild to moderate renal insufficiency, which resulted in a dramatic decrease in crcl reached 15 ml/min and weekly Kt/Vurea 2.5 nPCR dialysis resulted in a life-long dialysis, even if corrected for age, diabetes and co-administration of co-administration. However, this was no longer relevant if the remaining renal function was included in the model. The group initiated dialysis with normal NPCR maintained it for the first 3 years on dialysis, while the group in which low NPCR was initiated, although initially improved, had significantly lower NPCR levels throughout the observation period than normal nPCR counterparts. Conclusion. In advanced renal failure, nPCR is significantly reduced and dialysis may be predicted. nPCR levels <0.8 at initiation of therapy predict low future nPCR levels and dialysis mortality. The correlation between nPCR and CrCl in early renal failure may be partially artifactal. chronic renal failure, dialysis, nPCR, nutrition, protein intake, survival Spontaneous reduction in dietary protein intake is a recognised feature of severe chronic renal failure (CKD) [1,2] and is common in dialysis populations [3-6]. There is some evidence that a decrease in protein intake begins at an early stage of renal functional deterioration [7]. Malnutrition is a predictor of morbidity [8,9] and mortality [5,9,10] . Therefore, the prevention of malnutrition is a key theme of the guidelines (DOQI and others) on the initiation of dialysis [11,12]. The assessment of the nutritional situation is fraught with difficulty [13]. Previously accepted markers, such as serum albumin, are known to be highly affected by Normalized protein catabolism rate (nPCR) is widely used as a protein intake marker [3,15,16]. Total nitrogen-looking protein equivalent (nPNA), similar to nPCR, is also used, but also takes into account protein loss in urine. Although both of these measures are widely used, their inherent assumptions are often not realised. These include (i) a non-anabolic/non-catabolic state; (ii) normal liver function; and (iii) stable urea in the blood. We studied nPCR changes in renal function in a large number of predialysis patients with advanced CRF. In a subset of patients who then started dialysis, we studied the relationship between nPCR at the beginning of dialysis and subsequent dialysis outcome. The purpose of these studies was to try to define the usefulness of this protein content marker as an aid when deciding on the optimal time of starting dialysis. Patients and methods Patients with nephrogytics Patients with renal impairment visit our predialysis clinics at a frequency determined by clinical need. A number of regular studies are carried out at each clinic visit in addition to clinical evaluation. These often include an assessment of creatinine clearance based on 24-hour urine collection. Since January 1992, urea clearance, renal kt/V and nPCR have been calculated regularly for all patients required for creatinine clearance. Our clinical protocols do not include prescription dietary protein restrictions. Indications for dialysis Dialysis were initiated in response to uremic symptoms including general malaise, anorexia, nausea and vomiting, or fluid overload, non-reactive agents against diuretics. Dialysis patients Haemodialysis program All patients were treated only with high-flow synthetic membranes, mainly polysulfonium. Dialisers were reused using peracetic acid. Bicarbonate was used only as a buffer. Ultrapure water was used for all dialysis procedures. Dialysis was administered and controlled using a two-basin kinetic model to provide 1.1-1.2 (per dialysis) weekly for dialysis. Urea clearance and nPCR were controlled 1-3 times monthly. CAPD program Continuous outpatient peritoneal dialysis (CAPD) prescription was individualized to try to deliver daily total Kt/ V (kidney remaining part plus dialysis) of at least 0.25. To achieve this exchange rate increased as the residual renal function decreased. Urea clearance, renal Kt/V and nPCR were measured at least three times a month. Data storage All information was recorded in a computerised database [DiProton, CCL, London (until 1999); Renal Plus, CHI, UK (since 1999)]. Study groups Three patient groups were studied. (i) Group 1 included all patients who attended nephrogic clinics from 1 January 1992 to 31 June 1999. In this group, we examined the relationship between nPCR and renal function in several cross-sectional analyses. (ii) Group 2 included all patients in Group 1 on whom dialysis was initiated before 31 June 1999. In this group, we examined the change in nPCR longitudinally relative to the time before the start of dialysis. (iii) Group 3 included all group 2 patients with nPCR estimates obtained both 3 months prior to on-site dialysis and 3 months after initiation of treatment. In this group, the effect of nPCR on the treatment of dialysis outcome measures was analysed. Demographic and clinical data Age, gender, weight, height, diabetes status and Carnofska performance indicator were noted. The number of co-morbidity scores was determined as indicated above [17]. In short, the dysfunction of each main system (heart disease, peripheral vascular disease, cerebrovascular disease and respiratory disease) was evaluated from 0 (normal function) to 4 (severe dysfunction). Cirrhosis was classified as 4. The cancer was classified from 1 to 4 under a matrix determined by its aggressive nature and its current state of operation. Myeloma was classified as 3. Scores from each system were combined to form the total number of co-morbidity scores. Batch biochemical data for predialysis urea clearance and creatinine clearance were derived from harmonised blood and 24 h urine samples obtained in batch clinics prior to onc line. Protein excretion was also determined from the same 24 h collections. The following parameters were obtained in all patients studied: Total body water (V) was obtained from watson formula [18]: \[\mathrm{V}\ {=}\ 1000\\left(2,447\ {-}\ 0.09516 \\mathrm{A}\ (+}\ 0,1074\mathrm{H}\ {+}\ 0,3362\mathrm{W}\right)\ (men)}\] \[\mathrm{V}\ {=}\ 1000\ \left({-}\ 2097\ (+}\ 0.1069\mathrm{H}\ \\{+}\ 0.2466\mathrm{W}\right)\,\}\mathrm{ml\(females)}}]] where A = age (years), H = height (cm), W = weight (kg). Normalized urea clearance (Kt/V) where K = KRU (ml/min), t = min/day (min) and V = total number of water (ml). NPCR [19] \[\mathrm{nPCR}\left(\mathrm{g{/}kg{/}day}\right)\ {=}149.7\ {\times \mathrm{G{/}V}\ {+}\ 0,17\] where G/V = Uv × Uc/1440V [Uv = 24 hour urine collection volume; Etc = urea concentration during 24-hour urine collection]. Serial data in dialysis patients In haemodialysis (HD) patients, serial nPCR levels were recorded 1 to 3 times per month based on blood urea calculations and estimates of urea concentrations in interdialytic urine collections. The following formula was used: \[\mathrm{nPCR}\\left(\mathrm{g{/}kg{/}day}\right)\ {=}\ 149.7\ {\times}\\\ mathrm{G{/}V}\ {+}\ 0.17\], where G/V = [P2(V+W)/v − P1 + (Uv × Uc/V]/tid; P1 = blood urea concentration after dialysis 1 (mmol/l); P2 = P2 urea blood urea 2 (mmol/l) W = interdialytic weight gain (grams); Uv = volume of interdialytic urine collection (ml); Uc = urea concentration in interdialytic urine collection (mmol/l); tid = interdialytic time interval (minutes). In CAPD patients, serial nPCR levels were recorded 3 once a month based on estimates of urea concentrations in the blood, 24-hour urine collection and concurrent collection of dialysis. The following formula was used: \[\mathrm{nPCR}\\left(\mathrm{g{/}kg{/}day}\right)\ {=}\ 149.7\ {\times}\\\mathrm{G {/}V}\ {+}\ 0,17 \] where G/V = [(Uv × Etc) + (MS × Dc)]/1440V [Uv = 24 hour urine collection volume; Uc = urea concentration in 24-hour urine collection; DV = collection rate of a dialysis used for 24 h; Dc = urea concentration in the 24-hour spent dialison collection. Biochemical measurements Standard autoanalyser methods were used. Urea was tested with urease reaction using glutamate dehydrogenase. Creatin was tasted by jafe kinetic method. Data analysis One-way variance analysis (ANOVA) with post hoc Bonferroni test or Student's t-test was used to compare groups relative to continuous data. Categorically, the data were compared between groups using a ×2 test with Yates correction and Fisher's accurate assay as needed. Correlations were tested using pearson's correlation coefficient. We analyzed all predialysis nPCR data to investigate the relationship between nPCR and renal function. In patients who later started dialysis, we tested a continuous relationship between nPCR and survival using the Kaplan-Meier schedule and log rank test. Cox regression analysis was performed to investigate the effect of nPCR on age-adjusted survival, diabetes mellitus, other co-morbidity (8-point scale as described above [17]), functional capacity (Karnofsky performance scale) and renal Kt/Vurea at baseline. All of these statistical tests were conducted using SPSS v11.0 (SPSS Inc., Chicago, IL). We also used nonlinear regression analysis to test the relationship between nPCR and various renal performance indicators (Prism 4, Graphpad Software Inc., USA). Demographic data 5,082 nPCR measurements were performed in 1,282 patients during the study [Group 1, mean age 55.1 years (SD 17.05); 778 (60.7%) men]. Two hundred sixteen (16.8%) proteinuria. Three hundred and sixty-one (28.2%) end-stage renal failure and dialysis was initiated (group 2, mean age 59.3 years, SD 14.69; 64.5% male). A total of 2,244 predialysis measurements of nPCR were obtained in these patients. In 3018-eighteen patients, nPCR measurements were to be performed both 3 months before and 3 months after on-the-slide and were included in an additional analysis of the effects of nPCR at initiation of (3. grupa, vidējais vecums 59,1 gads, SD 14,96; 64,8% vīriešu). Korelācija ar nPCR vidējo nPCR pirmsdialīzes fāzē bija 0, 97 (SD 0, 27). Bija korelācija starp nPCR un kreatinīna klīrensu/1,73 m2 (r = 0,472, P <0.001), urea= clearance= (r=0.481,></0.001),> <0.001) and= kt/vurea= (r=0.504,></0.001)> <0.001)]. npcr= correlated= poorly= with= serum= albumin= (r=0.113,></0.001)].> <0.001) and= weakly= and= inversely= with= age= (r=−0.130,></0.001)> <0.001). the= difference= between= mean= npcr= in= patients= with= and= those= without= nephrotic= range= proteinuria= was= marginal= and= non-significant.= npcr= and= renal= function= (cross-sectional= analysis)= we= examined= the= relationship= between= npcr= and= creatinine= clearance/1.73 m2= performing= a= series= of= cross-sectional= analyses.initially= we= compared= mean= npcr= values= in= groups= of= patients= with= varying= degrees= of= renal= impairment= after= the= fashion= of= ikizler= et= al.= [7]= when= viewed= in= this= fashion,= npcr= appeared to= fall= steadily= and= significantly=></0.001).> <0.001 by= one-way= anova)= as= creatinine= clearance/1.73 m2= declined= (figure= 1),= falling= from= 1.17±0.31= in= those= with= clearances=>50 ml/min līdz 1,04±0,27 tiem, kuru klīrenss bija 25–50 ml/min, līdz 0,93±0,21 pie klīrensa 10–25 ml/min un līdz 0,74±0,18 , kad klīrenss <10 ml in.= the= mean= npcr= level= in= each= of= these= clearance= groups= was= significantly= different= from= mean= levels= in= all= other= groups=></10 ml> <0.001 by= post= hoc= bonferroni= test).= the= same= relationship= persisted= if= only= group= 2= patients= were= considered= (figure= 1= inset)= except= that= the= number= of= patients= with= values= of= creatinine= clearance=>bija samazinājies līdz 50, bija ļoti mazs. Atveriet jaunu cilniLejupielādēt slaiduNosūtīt nPCR vērtības 1282 pacientiem pirms dialīzes (5082 paraugi) ar dažādas pakāpes nieru darbības traucējumiem. (šķērsgriezuma dati). nPCR bija ievērojami <0.001 by= one-way= anova)= at= lower= creatinine= clearance/1.73 m2= bands.= the= inset= shows= the= same= data= in= the= 361= patients= who= started= on= dialysis= during= the= course= of= the= study.in= spite= of= the= significant= linear= correlations= between= npcr= and= markers= of= renal= function= described= above,= inspection= of= scatterplots= suggested= that= these= relationships= are= non-linear.= non-linear= regression= analysis= demonstrated= that= the= best-fit= curves= took= the= form= of= a= two-phase= exponential= association.= figure= 2= demonstrates= a= similar= relationship= between= npcr= and= both= creatinine= and= kt/vurea= (r2=0.332). in= both= cases,= there= appears= to= be= a= gentle= decline= of= npcr= as= renal= function= falls,= culminating= in= a= dramatic= increase= in= the= rate= of= decline= the= creatinine= clearance= has= reached= ~15 ml/min= and= the= weekly= kt/v= 2.5.= from= figure= 2,= it= might= be= predicted= that= npcr= would= fall= to=></0.001> <0.8 at= a= creatinine= clearance/1.73 m2= of=></0.8> <9 ml in= and= a= weekly= kt/vurea=></9 ml> <1.6. Open in new tabDownload slideA total of 5082 nPCR values in 1282 pre-dialysis patients plotted against matched level of renal open= in= new= tabdownload= slidea= total= of= 5082= npcr= values= in= 1282= pre-dialysis= patients= plotted= against= matched= level= of= renal=></1.6. Open in new tabDownload slideA total of 5082 nPCR values in 1282 pre-dialysis patients plotted against matched level of renal > zemāks (P</0.001> </0.001> </0.001> creatinine clearance/1.73 m2 in the upper panel and Kt/Vurea in the lower panel. The best fit curves and equations shown were calculated by non-linear regression analysis. Time changes in nPCR We examined longitudinal changes in nPCR for the time prior to the onsolysis in patients whose renal function decreased to this point during the study (Group 2). The mean nPCR remained stable until a sudden drop in the 3 months immediately prior to the on-going dialysis (Figure 3). The time intervals compared were 3 months per year prior to the on-going dialysis, 6 months in the penultimate year prior to initiation of therapy, all values being combined after 2 years. This was necessary in order to obtain comparable group sizes. The only mean nPCR that differed from the previous values was the level in the 3 months prior to the start of dialysis, which was significantly lower than all previous values. The severity levels are shown in Figure 3. Open a new tabDownload slideFromwart nPCR (upper panel) and nPNA (bottom panel) displayed lengthwise relative to the time before the start of dialysis 361 patients whose renal function decreased to this point during the study (group 2) (longitudinal data). To obtain comparable group sizes, the time intervals compared were 3 months per year before the start of dialysis, 6 months in the penultimate year prior to initiation of therapy, combining all values at 2 years. The only mean nPCR value that differed from the previous values was that which was in the 3 months prior to the start of dialysis, which was significantly lower than all previous values. The severity levels shown above the error bars are associated with the difference between this level and the level during the 3 months prior to the onso the on-line start. nPCR groups Since DOQI and other authorities consider <0.8 nPCR (or nPNA) [11] as a marker of malnutrition, we divided patients into two groups depending on whether their nPCR at baseline was above or below this threshold at baseline. The last predialysis nPCR was ≥0.8 171 (53.8%) and <0.8 147 (46.2%). Patients with nPCR <0.8 were slightly older (P<0.054), more dependent on significantly lower Karnofsky performance (P<0.001) and more uremic with significantly lower Weekly Kt/ V (P<0.001). In group 3 patients, post-dialysis patients had a significant improvement in nPCR in the low baseline NPCR arm, which was peaked during the first 15 months of treatment. All average 3 monthly nPCR values obtained during this period were significantly higher than predialysis values (P<0.001 in all cases). Thereafter, the improvement in this group was that the majority of the three-month average nPCR levels were not significantly different after 18 months (Figure 4). In high nPCR group 3, monthly mean nPCR levels remained throughout the 36-month follow-up period prior to dialysis. None of these mean three-month levels and mean predialysis values showed significant differences. In addition, in all 3-month periods from pre-dialysis to 36 months after initiation of therapy, mean NPCR levels were significantly higher in the baseline NPCR group than in the low baseline nPCR group (Figure 4). Open a new tabDownloadNosalineNodums 3 monthly nPCR level during the first 36 months of dialysis 318 patients divided into two groups according to nPCR at the beginning of dialysis: nPCR ≥0. At the beginning of dialysis (high nPCR groups: black symbols and error bars) and patients with nPCR <0.8 at baseline (low nPCR group: gray symbols and error bars). *P<001; **P<0.01 between the low and high nPCR groups relative to the average 3-month nPCR. †P<001; ‡‡P<0.005; ‡P<0.05 between the 3-month mean nPCR level and the mean predialysis nPCR in the low nPCR group. Characteristics of the group of patients with normal (≥0.8) and low (<0.8) nPCR at baseline. nPCR group. . Meaning. . ≥0.8 (171) . <0.8 (147) | . Agea 57.6 (14.68) 60.9 (15.14) 0.054 Maleb 115 (67.2%) 91 (61.9%) NS Diabetic b 44 (25.7%) 40 (27.2%) NS Co-morbidity score 1.9 (2.16) 2.0 (2.14) NS KPS at 78. (15.56) 70.(20.21) < 0.001 Weekly Kt/Va 1.65 (0.36) 1.24 (0.36) <0.001 HD: PD (% per HD) 91:79 (53.5%) 91:53 (63.2%) 0.053 Survival Single-pack analysis. During follow-up, there were 31 to 120 months during which 152 patients (47.8%) Died. The median survival across the group was 64 months. As expected [17], age, co-morbidity, functional capacity and diabetes were important predictors of survival (Table 2). The last predialysis albumin levels in serum <35 g/l, nPCR <0.8 and weekly Kt/Vurea <1.4 also predicted poor survival (Table 2). There were smaller but insignificant differences in survival at higher levels of nPCR and Kt/Vurea. Median survival was 54.5 months in the creatinine clearance group (at 1.73 m2) <7.5 ml/min at baseline, compared to 79.9 months in the ≥7.5 ml/min group (P = 0.01). The median survival for CAPD (intention to treat) was better than in HD patients (79.9 versus 58.3 months, P = 0.03), although CAPD patients are younger (mean age 56.5 versus 60.8 years, P = 0.013), less dependent (mean Carnofska performance score 79.1 versus 70.8, P<0.001) and with less co-morbidity (mean severity 1.46 versus 2.27, P = 0.001). They also started dialysis with higher weekly Kt/Vurea (averaged 1.56 versus 1.38, P<0.001) and at baseline they had slightly higher nPCR (average 0.838 versus 0.798, NS). Results of a one-time Kaplan-Meier survival analysis rangu tests) Vecums <50 50–65 65–75 >(gadi) 75 Nozīmība Vidējā dzīvildze (mēneši) 97,4a 78,5a 44,6 <0.001 Co-morbidity None Moderate Severe Median survival=></0.001 Co-morbidity None Moderate Severe Median> <0.001 KPS group normal= activity need= help dependent median= survival=></0.001 KPS> <0.001 Diabetes Non-diabetic Diabetic Median survival=></0.001 Diabetes Non-diabetic Diabetic Median> <> <0.8 Median survival= (months) 95.5 58.3 p=0.024 Weekly></0.8 Median> <1.4 Median survival= (months) 79.6a 53.8 p=0.005 Serum albumin=></1.4 Median> <35 Median survival= (months) 72.7 51.9 p=0.001 Dialysis mode capd hd median= survival= (months) 79.0 58.3 p=0.03 multivariate= analysis= the= predictors= of= survival= on= the= univariate= analysis= were= used= in= cox= regression= models.= an= npcr= of=></35 Median> <0.8 predicted= adverse= survival= (p=0.015) when= corrected= for= age ,= diabetes= and= other= co-morbidity.= last= pre-dialysis= serum= albumin= was= not= a= significant= factor.= the= addition= of= kt/vurea= to= the= model= abolished= this= effect= and= kt/vurea= became= a= significant= predictor= (p=0.008). age,= diabetes= and= other= co-morbidity= remained= significant= factors=></0.8> <0.001 in= both= models)= (table= 3).= table= 3.results= of= cox= regression= analysis= of= factors= affecting= survival= in= 318= dialysis= patients= . = . b= . se= . wald= . df= . significance= . hazard= ratio= . 95%= ci= . model= 1 step= 1 age 0.043 0.008 27.391 1 0.000 1.044 1.027–1.060 co-morbidity= score 0.316 0.040 63.201 1 0.000 1.371 1.268–1.482 npcr=></0.001> <0.8 0.410 0.168 5.928 1 0.015 1.506 1.083–2.095 Diabetes 0.541 0.172 9.951 1 0.002 1.718 1.227–2.405 Model 2 step= 1 age 0.041 0.008 24.647 1 0.000 1.042 1.025–1.058 co-morbidity= score 0.320 0.041 59.885 1 0.000 1.378 1.270–1.494 diabetes 0.483 0.180 7.153 1 0.007 1.620 1.138–2.308 npcr=></0.8 0.410 0.168 5.928 1 0.015 1.506 1.083–2.095 Diabetes 0.541 0.172 9.951 1 0.002 1.718 1.227–2.405 Model> <0.8 0.217 0.200 1.174 1 0.278 1.242 0.839–1.837 Kt urea −0.353 0.220 2.566 1 0.109 0.703 0.457–1.082 albumin=></0.8 0.217 0.200 1.174 1 0.278 1.242 0.839–1.837 Kt> <35 g 0.129 0.200 0.420 1 0.517 1.138 0.769–1.684 step= 2 age 0.041 0.008 24.958 1 0.000 1.042 1.025–1.059 co-morbidity= score 0.325 0.041 64.156 1 0.000 1.384 1.278–1.499 diabetes 0.514 0.173 8.782 1 0.003 1.672 1.190–2.349 npcr=></35 g> <0.8 0.227 0.198 1.319 1 0.251 1.255 0.852–1.851 Kt/Vurea −0.363 0.220 2.722 1 0.099 0.695 0.451–1.071 Step 3 Age 0.041 0.008 25.563 1 0.000 1.042 1.026–1.059 Co-morbidity score 0.321 0.040 63.603 1 0.000 1.378 1.274–1.491 Diabetes 0.500 0.173 8.356 1 0.004 1.648 1.175–2.313 Kt/Vurea −0.495 0.188 6.956 1 0.008 0.610 0.422–0.881 Predictors of 1- and 2-year survival logistic regression analysis, the factors urea −0.363 0.220 2.722 1 0.099 0.695 0.451–1.071 step= 3 age 0.041 0.008 25.563 1 0.000 1.042 1.026–1.059 co-morbidity= score 0.321 0.040 63.603 1 0.000 1.378 1.274–1.491 diabetes 0.500 0.173 8.356 1 0.004 1.648 1.175–2.313 kt/vurea −0.495 0.188 6.956 1 0.008 0.610 0.422–0.881 = predictors= of= 1-= and= 2-year= survival= using= logistic= regression= analysis,= the= factors=></0.8 0.227 0.198 1.319 1 0.251 1.255 0.852–1.851 Kt/Vurea −0.363 0.220 2.722 1 0.099 0.695 0.451–1.071 Step 3 Age 0.041 0.008 25.563 1 0.000 1.042 1.026–1.059 Co-morbidity score 0.321 0.040 63.603 1 0.000 1.378 1.274–1.491 Diabetes 0.500 0.173 8.356 1 0.004 1.648 1.175–2.313 Kt/Vurea −0.495 0.188 6.956 1 0.008 0.610 0.422–0.881 Predictors of 1- and 2-year survival Using logistic regression analysis , the factors > 24,6 P</50 50–65 65–75 > > survival over 1 year was age (odds ratio 1.02 per year, P = 0.049), co-morbidity (odds ratio 1.53 per point, P<0.001), weekly Kt/Vurea (odds ratio 0.45 per point, P = 0.041) and functional power (odds ratio 0.96 per point per point per Kafsky performance point, P<0.001). 0, <0, 8 nPCR was not a 1- or 2-year survival predictor if adjusted for the above factors. nPNA and nPCR As some of our patients had significant proteinuria, we re-analyzed the data using nPNA (calculated with the Randerson equation) [20], which also takes into account protein losses in urine. There was a very high correlation (r = 0.951) between nPNA and nPCR. None of the results, including survival analysis, were significantly altered when nPCR was replaced by nPCR. At the time of the nPNA changes during the pre-dialysis period (figure 3.b 3) were identical to the nPCR (Figure 3a). Dialysis adequacy Most patients were on dialysis. The median total (dialysis + renal) Kt/V was 2.21±0.47 (per week) pd patients; 1.25±0.21 (two pools) three times a week for HD patients; 2.23±0.30 (two pools) twice a week. In the first, second, third, and fourth years of dialysis, differences in dialysis adequacy were examined between the low and high nPCR groups three times a week in HD and PD patients (twice weekly HD patients were small in year 1 and in Year 4 only one patient was twice a week in HD). In the first year of dialysis, except for PD patients with a slightly higher kt/v level in Kt/V (2.38 vs. 2.25; p = 0.018). Discussion We have provided evidence that the sudden decline in nPCR occurs late in the course of the gradual CRF level of creatinine clearance/1.73 m2 <15 ml/min and weekly Kt/Vurea <2.5. We have also confirmed that the expected survival rate is important at the beginning of nPCR dialysis on dialysis, even if it is adjusted according to age, the presence of diabetes and non-renal co-morbidity, although it is not baseline independent of the degree of uema. In addition, we have also shown that patient groups began dialysis with normal NPCR tend to maintain all of the first 3 years of dialysis at least, while groups that initiate low nPCR, although the trend to improve initially, never match their normal NPCR counterparts throughout this period of observation. However, we cannot determine the cause and effect of the relationship between low nPCR and the clinical condition of these patients. We are less confident in what is happening to nPCR earlier in the course of the progressive CRF. When looking at cross-sectional data and showing mean nPCR levels at different creatinine clearance levels according to the creatinine clearance range (Figure 1), it appeared that the decrease in nPCR started at the beginning of the CRF course when creatinine clearance was as much as 50 ml/min. The findings were recovered by Ikizler et al. [7] However, when we looked at what happened to the nPCR longitudinally as a function during the period prior to the start of dialysis (Figure 3), it appeared that the NPCR remained stable until a very late CRF rate, the only significant change that occurred in the 3 months prior to the start of dialysis. Data from the curve fixture provided some support for this last interpretation, best suited to obtaining two-phase exponential expressions, which leads to a very gentle decrease in nPCR during advanced renal failure to a very late CRF at a rapid decline (Figure 2). Cross-sectional studies of the type shown in Figure 1, which show that the mean decrease in NPCR at the start of renal failure, is subject to a number of possible errors. The relationship between nPCR and creatinine clearance is very complex. There is a real physiological relationship to decreased renal function, which reduces protein intake. Artifactal associations may also occur as a result of a mathematical coupling. These can be three broad types [21.22], error coupling, calculation bias and contiguous. We think that the most important of these in this situation is the coup for error. This may occur because nPCR and creatinine clearance depend on the overall parameters, the most obvious of which is the volume of urine divided. Accidental errors in urine collection, volume measurement and sampling would affect both parameters in the same direction and to the same extent, thereby leading to a positive correlation of artifacts. Blood creatinine and urea measurements are carried out in the same autoanalyte, which is another possible source of error coupling. Hashing is the introduction of bias with foreign factors associated with each of the parameters of the study. The fact that both nPCR and creatinine clearance return to normal with body size (albeit different) may be a source of error. Calculating biases where random deviations in a study in a case systematically differ from zero in specific situations may have less impact on this situation. When expressing the mean nPCR data longitudinally as a function of time before the start of dialysis, error-tying is effectively excluded and therefore a more reliable indicator of the actual behaviour of the NPCR is likely to be more likely as renal function deteriorates. Worsening of nutritional status has been demonstrated at the beginning of renal failure in studies where nutritional status and renal function were assessed by independent methods and are likely to be more free from the bias of the coupling. In the largest of these [23] in which the initial glomerular filtration rate (GFR) measured with clearance of [121I] iothalamate is corathed with the initial dietary and dietary parameters calculated on the basis of dietary biochemistry measurements and anthropometry, multi-nutritional parameters were found to be multi-nutritional parameters in groups with sequentially lower GLP [>37 (mild), 21-37 (moderate) and <21 ml/min/1.73 m2 (severe)]. However, some patients had evidence of protein and energy malnutrition. The interpretation of this data is complicated by the fact that a significant proportion of patients tried to follow a protein-reduced diet even at baseline and that their proportion was higher in moderate and severe groups. In addition, it was noted that for a number of nutritional parameters the rate of decline was higher than the higher GFR, which corresponds to the data presented here. There are other problems when interpreting NPCR data. The use of NPCR as an indicator of protein intake depends on the steady state when the amount of protein corresponding to the intake of the liver is converted into urea excreted by the kidneys. Thus, it becomes useless as an indicator of protein intake during the disease (catabolic state) and recovery (anabolic state). It depends on normal liver function. Its use before dialysis is assumed to be excreted by the kidneys in patients and none of them is maintained in the body, i.e. the urea is stable in the blood. An increase in urea in the blood causes an underestimate of nPCR. Other mixed agents include the use of diuretics, which may lead to fluid depletion, increased re-absorption of urea in the tubular absorption and increase in blood levels. Despite these rules, nPCR can be a useful tool for assessing the nutritional condition of patients with slowly progressive CRF. Nutrition is an important aspect of preparing patients for dialysis, and the conclusions presented here provide some support for monitoring this parameter in this case, with the aim of starting dialysis before a catastrophic decline in the dietary condition. However, many factors need to be taken into account. Not a panacea. Conflict of interest statement. Not declared. Reference 1Marckmann P. Nutritional status of patients in haemodialysis and peritoneal dialysis. ; : -782Kopt JD. Protein-energy exit pathophysiology in chronic renal failure. ; : –251S3Aparicio M, Cano N, Chauveau P et al. Nutrition status of patients with haemodialysis: French national collaborative study. French study group on dialysis nutrition. ; : –16864Young GA, Kopple JD, Lindholm B et al. 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