GROWTH IN SMALL CHILDREN RECEIVING CONTINUOUS AMBULATORY PERITONEAL DIALYSIS (CAPD)
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Growth failure is common in end-stage renal disease (ESRD). CAPD is a treatment of choice in small patients awaiting transplantation. CAPD improves some of the metabolic and nutritional problems associated with growth failure in ESRD.

The aim of the study was to evaluate growth in 20 patients (13 males and 7 females), aged 14.8 months ($r = 4 - 24$ months). All had been treated on CAPD over ($X + SD$) 23.2 ± 2.9 months ($r = 6 - 62$ months) and early enteral tube feeding.

Weight and height were assessed at the beginning and every 3-6 months during follow-up. The measures were converted in the Z scores for age and sex. Growth velocity was determined by the changes in the Z score for height.

<table>
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<th>Initial</th>
<th>Final</th>
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<tr>
<td>$X ± SD$</td>
<td>$X ± SD$</td>
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<tr>
<td>$Z$ weight/age</td>
<td>$-2.23 ± 0.24$</td>
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<tr>
<td>$Z$ height/age</td>
<td>$-1.84 ± 0.4$</td>
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These results show that patients on CAPD under 2 years of age ameliorated their nutritional state but have severe impairment of growth. New therapeutic strategies or supportive measures must be developed in order to improve their chances of growth.

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CLASSICAL HAEMOLYTIC UREMIC SYNDROME (HUS) IN ARGENTINEAN CHILDREN: RESULTS AFTER 10 YEARS OF FOLLOW-UP AND PROGNOSTIC FEATURES
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HUS is a main cause of acute renal failure and the third cause of end-stage renal failure (ESRF) in children, in Argentina. Among 300 cases of HUS seen since 1986 we conducted a retrospective study of 118 patients (1968-1984), to evaluate their evolution after at least 10 years. All the patients, 63 boys and 55 girls were under regular medical review. Mean follow-up was 156.1 months (13 years). Four evolution patterns at the end of the follow-up were found:

1. Complete recovery (creatinine clearance (CCl) > 80 ml/min 1.73m2, normal blood pressure (BP), no proteinuria (PR), 75 patients (63.8%) - 2) Significant PR, normal CCl, with/without high BP: 18 patients (15%, 2%) - 3) Chronic renal failure (CRF) (CCl > 80 ml/min 1.73m2 and > 5 ml/min 1.73m2): 21 patients (17.8%) - 4) ESRF (CCl < 5 ml/min 1.73m2) 4 patients (3.4%).

We investigated the association between several variables of the acute stage and the long-term evolution. From those which did not required peritoneal dialysis (PD) ($n = 48$), 91.7% recovered completely, while from 42 children who were under PD for 1-10 days and 26 dialyzed for more than 10 days, complete recovery was achieved in 54.7% and 32.1% respectively (Chi Square for lineal trend $p<0.0001$). Only 2 patients who did not required PD developed CRF, and one of them was transplanted. From the cases with significant PR at 12 months control, 86.4% had either CCl < 80 ml/min 1.73m2, and/or significant PR and/or hypertension in the last control while only 25% of those without PR at 12 months showed those findings (Chi square $p<0.0001$). We did not find association between low CCl at 1 year and functional renal sequelae at the end of follow-up ($p=0.1633$).

CONCLUSION: We determined that after more than 10 years at follow-up at 116 patients with classical HUS, 43 (36.4%) developed permanent functional sequelae, 21 of them CRF and 4 ESRF. A close association was found between the percentage of patients with permanent functional renal sequelae at the end of follow-up, the days that PD was required and the presence of PR at 12 months control and the acute episode. These appear to be prognostic parameters.

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ETHIOLOGY, MANIFESTATIONS AND MOLECULAR PATHOLOGY OF CHRONIC ALLOGRAFT REJECTION
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Among the risk factors predisposing the recipient for chronic rejection are histoincompatibility, duration of periparative period, donor age, frequency and intensity of acute rejection episodes, infections (particularly CMV), and possibly lipid abnormalities. Additional parameters, not studied so far, may include hypertension and diabetes.

Considering the two most important manifestations in chronic rejection histology, inflammation and arteriosclerosis, the following working hypothesis arises: immune response characterized by perivascular inflammation induces persistent low-grade damage to vessel endothelium, which in turn begins to secrete growth factors to repair the damage. This results in smooth muscle cell replication in the vessel wall and influx of monocytes from the media into the intima and generation of an arteriosclerotic lesion. All intramural arteries of a transplant are affected during allograft arteriosclerosis which is concentric and generalized in contrast to the usually focal and eccentric classical atherosclerosis. Acute inflammation (rejection) is a prerequisite for chronic changes. There are several molecular cascades participating in the generation of chronic rejection. The final effector molecules are most likely growth factors synthesized as a response of injury by the parenchymal and endothelial cells of the transplant. There will be no single therapy for the prophylaxis or treatment of chronic rejection, and several parameters must be overcome to counteract these alterations. However, if the current 7-8-year hait-tite of renal transplants could be doubled, chronic rejection would factually be overcome.