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Abstracts

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LEGENDA:	* FIRST EIGHT BEST ABSTRACTS PRESENTED BY YOUNG AUTHORS	■ FIRST EIGHT BEST ABSTRACTS

Abstracts

B: Renal metabolism, hormones, pharmacology

IMPORTANCE OF THE POTENTIAL LOAD OF ALKALI IN THE DIET OF THE RAT FOR ACID-BASE BALANCE

1SH Lin, 1YF Lin, 2SC Dhadli, 2M Gowrishankar, 2ML Halperin
1Renal division, Tri-Service General Hospital, Taipei, Taiwan
2Renal division, St. Michael's Hospital, Toronto, Canada

To gain insights into the nature and physiology of endogenous acid production. Balance studies were performed for 24 hr; urine was collected separately in the day and night portion of the diuranl cycle. A low-electrolyte diet was fed to determine if dietary and urinary organic anions were similar in nature. A titration procedure was developed to gain insights into the pK of urinary unmeasured anions. Despite the fact that normal rats excreted net acid, this excretion was inversely related to the amount of food consumed. The rates of excretion of bicarbonate, citrate, and 'missing' anions were higher while that of net acid was lower in the night portion of diurnal cycle reflecting the net alkali load in the diet. When the dietary alkali removed by consuming a low-electrolyte diet, net acid excretion rose dramatically. Dietary and urinary organic anions were not identical in nature because rats fed the low alkali diet supplemented with potassium solely as its citrate salt excreted < 10% of this alkali load as citrate. When 3000 umol NH4Cl was given intraperitoneally, net acid excretion rose by close to 2000 uEq and the rate of excretion of anions with a pK in the 3-5 pH range fell by close to 1000 uEq; most of these changes occured in the 7 hr after 3000 umol NH4Cl was given. Rat chow provided a net alkali load despite the fact that rats have an appreciable daily excretion of NH4+ and net acid. There are two types of endogenous acid production: the first example is sulphuric acid which requires a rise in net acid excretion for elimination of its proton load. The second example is dietary alkali-driven endogenous acid production which requires renal excretion of a family of organic anions to make these acids end-products of metabolism.

RESULTS OF TREATMENT WITH REAFERON IN PATIENT WITH MESANGIOCAPILLARY GLOMERULONEPHRITIS (MGGN).

A.N. Shishkin, A.V. Sosunov, V.I. Romanova, A.V. Volovnikova.
TherapeuticDepartment,Medical Faculty, State University of St. Petersburg; Dep.of Microbiology, State Academy of Medicine,St.Petersburg; Laboratory of Influencial Medicines, Institute of Influenca, St. Petersburg,Russia

It has been described that recombinant alfa -2 interferone is effective ín treatment of virus associated glomerulonephritis resistant to the traditional therapy. We performed treatment with Reaferon(R) recombinant alfa -2 reaferon in 64 patients with different morphologic forms of glomerulonephritis who were serologically positive to some viruses (HBV, Influenza, adenoidal-pharingeal-conjunctival viruses, arthropod-borne virus and others). In this abstract we present only those results regarding to the treatment of one patient who underwent renal biopsy before and after treatment. A 56-year-old male patient with confirmed on biopsy MCGN manifestated after the acute respiratory disease by hypertension, edema, nephrotic range proteinuria (5 gr/24hrs), hypoalbuminemia (35%), hypercholesterolemia (9,5 mmol/l) and serologically confirmed association with influenza virus A was treated with R.according to the traditional scheme.At the end of treatment decrease of proteinuria to 2 gr/24hrs, normalisation*of* protein levels,release of edema were noted. Within 9 months after treatment respiratory diseases were not noted, proteinuria was 3g/24hrs, glomerular filtration rate was 68 ml/min,, total protein - 49g/l albumins-42%, serum cholsterol- 8,0mmol/l. Investigation of cellular immunity before treatment showed its supression . After the course of R. indications of cellular immuniy were improoved. Repeated renal biopsy showed transformation of lobular form to the simple one, local sclerosis of strome, partial glomerular gialinosis; diffuse segmental MCGN with minimal activity. After the repeated course of R. proteinuria was 1,3g/24hts, glomerular filtration rate- 113ml/min, total protein - 60g/l, albumines - 48%, serum cholesterol 6,4mmolJl, indications of cellular immunity were at norm. In conclusion ,this case is a good confirmation of the efficasy of recombinant alfa-2 interferon in glomerulonephritis associated with virus infection and it can be recommended as an alternative treatment to the traditional one. Also we can suppose the role of viruses in pathogenesis of glomerulonephritis.

THE INFLUENCE OF rHuEPO THERAPY ON THYROID FUNCTION IN CHILDREN WITH END-STAGE RENAL FAILURE

M. Roszkowska-Blaim, M. Sieniawska
Dep. of Pediatrics & Nephrology, University Children's Hospital, Warsaw, Poland

The aim of the study was to assess the influence of rHuEPO therapy on thyroid function in dialysed children.
The study group comprised 19 children (11 on HD, 8 on CAPD), aged 4-17.5 years, mean 11.5±3.4. The period of renal replacement therapy was in HD group 1.5±0.75 years, in CAPD 2.7±1.4 years. All children had received sc rHuEPO in individually adjusted effective doses. When Hb was above 10g% (period I), rHuEPO was withdrawn, Hb was monitored every week and the therapy of rHuEPO was restarted when Hb decreased below 8 g% (period II). The interval between I and II periods was 5 to 14 weeks, mean 9.0±2.5. In all children, during the period I and II, the following parameters were estimated: thyrotropin (TSH), triiodothyronine (T_3), thyroxine (T_4).
Results: In most investigated children the values of T_3, T_4 and TSH were normal in both periods.

Periods	T_3 [ng/ml]	T_4 [ng/ml]	TSH [µU/ml]
(Hb >10g%)	0.95±0.29 *	82.90±21.71 **	1.62±1.10
(Hb <8 g%)	1.19±0.31	59.21±19.41	1.71±1.54

* T_3 (I) vs T_3 (II) p <0.01, ** T_4 (I) vs T_4 (II) p <0.001
Normal values: T_3 - 0.55 ÷ 1.7 ng/ml, T_4 - 42 ¸ 130 ng/ml, TSH - 0.5 ¸ 4.8 µU/ml
Conclusions: The mean values of T_3, T_4 serum levels decreased significantly during the break of rHuEPO therapy, but were still in normal range. No changes in serum TSH levels were observed.

FENOFIBRATE INCREASES CREATININEMIA BUT DOES NOT ALTER GLOMERULAR FILTRATION RATE IN PATIENTS WITH MILD RENAL INSUFFICIENCY.

Hottelart C, El Esper N, Achard JM, Pruna A, Fournier A.
Service de Néphrologie, CHU Amiens, France.

Fenofibrate is a potent hypolipemic agent, widely used in patients with mild to severe renal failure in whom hyperlipoproteinemia is frequent. A moderate reversible increase in creatinine plasma levels has been reported with fenofibrate therapy; however, it is not known whether this increased creatininemia reflects a fenofibrate induced alteration of renal function or if fenofibrate interfere with creatinine tubular handling. We prospectively examined the effect of 2 weeks fenofibrate treatment (200 mg daily) on renal function in 13 hyperlipidemic patients with normal renal function or mild to moderate renal failure (Creat C1: 110 to 30 mVmin).
Results:

	control	fenofibrate	p (paired t-test)
plasma cholesterol	6.43±0.34	5.31±0.2	0.0004
plasma triglycerides	1.92±0.12	1.43±0.13	0.009
plasma creatinine	147±12 mmol/l	170±15 mmol/l	0.014
PAH clearance	304±56 ml/min	311±49 ml/min	NS
inulin clearance	51.7±6 ml/min	52.3±7 ml/min	NS
creatinine clearance	69±8 ml/min	68±8 ml/min	NS
24 h creatininuria	13.7±5 mmol	15.4±4 mmol	0.03

This study confirms that fenofibrate therapy significantly increases creatininemia in patients with mild to moderate renal failure, but does not alter renal hemodynamic nor glomerular filtration rate as assessed by the stability of PAH and inulin clearances. The increase in creatininemia is neither due to an inhibition of creatinine tubular excretion, since no change in creatinine clearance was observed, but appears to be associated to a parallel increase in creatinine daily urinary excretion.
Conclusions: fenofibrate therapy in renal patients does not worsen renal function, nor diminish the reliability of creatinine clearance for its follow-up in spite of a significant rise in creatininemia. The mechanism of the fenofibrate-induced increase in urinary creatinine excretion (increased metabolic production or decreased extra-renal elimination) remains to be determined.