

1630 UNEXPECTED TUBULOINTERSTITIAL INVOLVEMENT IN GLOMERULAR DISEASE - DIAGNOSIS USING B2 MICROGLOBULIN. Ronald J. Portman, Anne Richardson, and Alan M. Robson, Washington University School of Medicine, St. Louis Children's Hospital, Department of Pediatrics, St. Louis, MO

Fractional excretion (FE) of B2 Microglobulin (B2M) was studied in children with glomerular (n=112) or tubular (n=77) diseases. FE-B2M (nl<0.4%) was significantly lower in glomerular diseases than in tubular lesions (0.17±0.53% vs 16.7±23.5%; p<.001). Unexpectedly, several patients with glomerular diseases were found to have increased values for FE-B2M. To determine whether this was due to a tubular component in a primary glomerular disease process, FE-B2M was measured in 30 children, with various glomerulonephritides who underwent renal biopsy. The biopsy was reviewed, without knowledge of the FE-B2M results, for evidence of tubulointerstitial disease (TID). No patients were receiving drugs, or had other diseases, known to modify B2M excretion. FE-B2M in patients without biopsy evidence of TID was 0.10±0.02% (n=17); all values were in the normal range. FE-B2M in patients with biopsy evidence of TID averaged 16.3±29.3% (n=13); only 2 results were normal. There were patients with the same glomerular diseases in both groups. FE-B2M was used to follow successfully the course and treatment of a patient with membranous glomerulonephritis and an immunologically mediated Fanconi's Syndrome. These data demonstrate that TID occurs more often in apparently pure glomerular disease than has been recognized and that FE-B2M represents a reliable noninvasive method to diagnose such involvement. Preliminary data demonstrate that FE-B2M is of value in monitoring the progress and response to therapy in TID.

1631 VALUE OF MATERNAL SONOGRAPHY IN THE DETECTION OF CONGENITAL ABNORMALITIES OF THE URINARY TRACT. F Ramirez, R Kessler, A Cepero. Miami Children's Hospital, Divisions of Nephrology and Radiology, Miami, Florida.

During the 1 year period from 1982-1983, 9 cases of congenital urinary malformation referred to our hospital had been detected by maternal ultrasonography. All sonograms were done in the last trimester of pregnancy to evaluate fetal maturity and without suspicion of a possible malformation. All babies had further radiological investigations in the immediate postnatal period including renal sonogram, VCUg and radionuclide renal scan, which correlated well with the prenatal sonogram findings.

All 9 patients had unilateral urinary malformations including 2 multicystic kidneys, 1 dysplastic kidney, 3 UPJ obstructions, 2 V.U. Reflux and 1 mesoblastic nephroma.

Elective surgical procedures were performed in all cases. These patients accounted for 36% of the congenital urinary malformation diagnosed in the newborn period in our institution during this period of time.

The maternal sonogram therefore, represents an important diagnostic tool in the detection of congenital urinary anomalies and may significantly affect the peri-natal management and thus minimize renal damage.

†1632 PULSE METHYLPREDNISOLONE (MP) THERAPY OF NEPHROTIC SYNDROME WITH FOCAL GLOMERULOSCLEROSIS (FGS) Vivian M. Reznik, Bruce M. Tune, William R. Griswold, Martha Vasquez, and Stanley A. Mendoza. U.C.S.D. School of Medicine and Stanford University School of Medicine, Depts. of Pediatrics, La Jolla and Stanford, Ca. and Dept. of Pathology, VA Hospital, La Jolla, Ca.

Seven children with nephrotic syndrome resistant to 8 weeks of oral prednisone at a doses of 2 mg/kg/day and biopsy-proven FGS were treated with intravenous MP (30 mg/kg/dose) and alternate day prednisone. MP was given three times a week for two weeks and then at increasing intervals. All seven patients had a marked decrease in edema and urinary protein excretion and an increase in serum albumin concentration. Three patients were treated with an alkylating agent in addition to the high dose MP to produce a sustained remission. The clinical remissions lasted from one to twenty months. Six out of seven patients are in remission and edema free after a mean of 10.5 months on therapy. One patient developed a cataract shortly after being started on MP and immediately after a prolonged course of high-dose daily prednisone. Another patient developed hypertension early in the course of MP therapy which resolved as the interval between MP infusions was increased. No other adverse reactions were observed. It would appear that intravenous MP in high doses causes a significant improvement in the short-term morbidity of FGS. The long-term effects of this treatment remain to be determined.

†1633 ALDOSTERONE--A POTENT STIMULATOR OF THE RENAL KALLIKREIN-KININ SYSTEM DURING FETAL LIFE. Jean E. Robillard, Kenneth T. Nakamura, Oliva McWeeny, Sindy Wear and William Lawton, University of Iowa, Departments of Pediatrics and Medicine, Iowa City, IA.

The effects of acute (A) (0.25 ug/min/kg body wt. over 2 hrs) and chronic (C) (0.50 ug/kg over 4 days) aldosterone infusion (Aldo-I) on active urinary kallikrein (Uka1) and Na⁺ excretion (UNa⁺V) were studied in young (95-115 days, n=6) and old (125 days, n=8) fetal lambs (term 145 days) chronically instrumented. A-Aldo-I was not associated with changes in plasma electrolytes concentration but produced a significant decrease in UNa⁺V in both groups of fetuses and a rise in Uka1 in young fetuses. C-Aldo-I produced a significant (p<0.05) rise in plasma Na⁺ (142±1 to 147±1 meq/l meq/l) and decline in plasma K⁺ (4.7±0.3 to 4.0±0.2 meq/l) in old fetuses and was associated with decreases in UNa⁺V and increases in Uka1 in both groups of fetuses.

	<115 days			>125 days		
	Baseline	A	C	Baseline	A	C
Plasma aldosterone ng/100 ml	4.3±1	87±16*	83±26*	5.7±1	136±25*	129±30*
GFR ml/min	2.1±0.3	2.4±0.4	2.3±0.2	2.3±0.4	2.1±0.5	3.4±0.6
UNa ⁺ V ueq.min ⁻¹ .ml GFR ⁻¹	8.8±2.3	4.2±1.2*	2.6±1.1*	14.3±2.6	6.6±1.9*	1.9±1.7*
Active Uka1 mEU.min ⁻¹ .gkw ⁻¹	4.5±1.0	8.1±1.0*	26.4±7.3*	6.7±2.6	7.9±4.3	23.9±5.1*

* different from baseline at p<0.05. All values were back to baseline levels 3 days after stopping C-Aldo-I. The present results demonstrate that Aldo is a potent modulator of Na⁺ reabsorption and Uka1 production very early during gestation and suggests a link between the kallikrein-kinin and renin-angiotensin-aldosterone system during fetal life.

1634 ALUMINUM-RELATED BONE DISEASE AND SERUM ALUMINUM IN CHILDREN UNDERGOING CAPD. I.B. Salusky, J.W. Coburn, L. Paunier, D.J. Sherrard, & R.N. Fine.

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Al-related osteomalacia has been reported in young uremic children not yet on dialysis; however, the effect of CAPD on Al metabolism is uncertain. Therefore, we studied 16 children with a mean age of 8.9±1.0 (SE) years and body weight, 24±2.4 kg. Serum (S) Al levels, measured after 8 and 18 mos of CAPD, were 55±11 and 60±10 ug/l, respectively (normal (N) children, 8.2±1.1 ug/l, p<0.001). The estimated oral Al intakes from Al hydroxide gels were 98±20 and 104±32 mg/kg/day at the same times; CAPD fluid Al was <6 ug/l. S-Al correlated with oral Al intake (r=0.86, p<.001) and indirectly with body weight (r=-0.68, p<.01) and age (r=-0.54 p<.01). The youngest patient (2.5 years) had the highest S-Al (174-204 ug/l) and largest Al intake (192-310 mg/kg/day); he failed to respond to calcitriol despite S-Ca of 11.2-13.2 mg/dl. A bone biopsy was characteristic of Al-related bone disease, with marked Al staining, greatly increased osteoid surface, and undetectable bone formation as determined from double tetracycline labelling; findings of 2° hyperparathyroidism were absent. Thus, Al accumulation occurs in young CAPD patients receiving large doses of Al gels despite the removal of significant but small quantities of Al in CAPD fluid. These observations indicate that Al-containing phosphate binders should be used cautiously in young children treated with CAPD.

1635 EFFECTS OF VARYING PROTEIN INTAKE ON RENAL FUNCTION IN PREMATURE INFANTS. Uana Sanocka, Jose Straus, Salha Daniel, Karl Schulze, Sudha Kashyap, Stanley James.

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Ingestion of an acute protein load by a normal adult has been shown to increase Glomerular Filtration Rate (GFR) by 20-30%, while in the patient with diabetic nephropathy GFR decreases with protein loading. It has been hypothesized that the kidneys of a newborn behave in a similar fashion to those of diabetic patients.

The effects of different protein loads on the renal function of the premature infant were studied in six babies (PA 33-38 wks) on 8 occasions. Group I infants received 3.8 gm/kg/day of protein. Group II infants received 2.25 gm/kg/day of protein (whey/casein ratio 60/40). All infants were normally growing prematures taking 180 cc/kg/day by mouth or 120 cal/kg/day; protein constituted 12.7% of total caloric intake in Group I and 7.5% in Group II. Each study consisted of a six hour urine collection started just prior to feeding. All urine voided spontaneously during the study period was analyzed. The following results were obtained:

	Pr.Intake mg/kg/dy	C _{Cr} ml/min/kg	P _{Cr} mg%	UV _{UN} ug/min/kg	P _{UN} mg%
GRP. I(n=5)	3.8	1.38±.12	0.56±.07	30.9±8.82	4±1.12
GRP.II(n=3)	2.25	1.15	0.53	15.7	2.1

No significant difference was observed in GFR between Groups I and II while total nitrogen excretion was doubled in Group I. These preliminary observations indicate that GFR is not modified by the two protein loads and is therefore different from both the normal adult and those with diabetic nephropathy.