PROGNOSTIC FACTORS IN SEGMENTAL GLOMERULOSCLEROSIS.
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Thirty-four children with segmental glomerulosclerosis (SGS) were studied retrospectively to establish prognostic factors. At latest follow-up they were divided into 3 groups: 15 children had absent or minimal proteinuria and normal blood pressure and renal function ("recovered"); 9 had heavy proteinuria and/or hypertension ("active disease"); and 10 children had either died or showed chronic renal failure (CRF).

In 21 patients biopsy was performed within 6 months of onset. In 16 of these, less than 20% of glomeruli showed segmental or global sclerosis; 12 had recovered, 3 had active disease and one showed CRF. In contrast, 4 of 5 children with > 20% sclerotic glomeruli showed CRF and the other one active disease (p <0.01).

There was no difference among the 3 groups of patients in regard to the clinical findings at onset, or mesangial cellularity. Since later biopsies may show > 20% sclerosis even with clinical recovery, early renal biopsy has greater prognostic value when SGS is suspected.

 $122~{\rm MEMBRANOUS}$ GLOMERULONEPHRITIS AND HB-e ANTIGEN

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The study was done in 6 patients with glomerulopathy accompanying with positive HBsAg, Core-Ab, eAg in serum, in whom 4 were found from 70,000 school children by urine-massscreening and 2 were younger than school age and found in clinic. 4 of 6 were with membranous glomerulonephritis (MG) and 2 were with minimal change (MC). By immunofluorescence (1F), eAg was stained in glomerular deposit together with IgG was stained in glomerular deposit together with IgG and C3 in all 4 patients with MG, whereas HBs-AB was not detected. Circulating immune complex (CIC) was detected in patients in active stage of MG, but not in recovery stage after steroid treatment. In 2 patients with MC, all IF staining and CIC were negative.

THREE DIFFERENT CLINICAL PRESENTATIONS FOR FOCAL GLOMERULOSCLEROSIS (FGS). Bacheyie, G.S., Baumal, R., Arbus, G.S. The Hospital for Sick Children, University of Toronto, Toronto, Ontario, Canada. Twenty-seven children in whom idiopathic FGS was diagnosed on the basis of at least one kidney biopsy were studied retrospectively. Patients were grouped by response to therapy. Group 1

Twenty-seven children in whom idiopathic FGS was diagnosed or the basis of at least one kidney biopsy were studied retrospectively. Patients were grouped by response to therapy. Group l (Gpl) children consistently responded to prednisone or cyclophosphamide; Gp2 were intermittently clear of proteinuria (P) from 0.75-18 months (mean 7.7) before showing persistent P; Gp3 manifested persistent P from the outset,

	GPI	Gp2	<u> </u>
No. of Patients	8	5	14
Male/female	6/2	4/1	8/6
Hematuria, † BP or azotemia	2	4	4
Terminal renal failure	0	4	8
Initial biopsy - minimal lesion	1	1	3
- FGS (segmental)	4	1	10
- FGS (global)	3	3	1
Mean age (years) at outset	3.3	2.9	6.8

Except for a slightly higher incidence of hypertension, initial presenting symptoms in Gp2 resembled those of Gp1 or minimal lesion nephrosis. Yet, terminal renal failure developed in 4/5 of Gp2 patients. Caution is needed in making a prognosis, since persistent P and eventual renal failure may develop in nephrosis patients who have periods free of P early in their disease.

CLINICOPATHOLOGICAL OBSERVATION OF PURPURIC NEPHRITIS
- REVIEW OF 41 CASES -

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PATIENTS AND METHODS

133 patients with anaphylactoid purpura between Jan. 1960 and Dec. 1979 were retrospectively reviewed in regard to renal involvement. Renal involvement were 41 cases (30.8%). Their clinico-patho-immunological correlation were analized, as follows.

1) Clinical, pathological classification and their prognosis.

- ?) Correlation with streptococcal infection, serum IgA. and
- 3) Presentation of 2 patients with CRF.

RESULTS

The group; AGN 46%, CGN 34%, NS 17%, CRF 2.4%, RPGN 0% The clinical manifestation and the pathological diagnosis were compaired. The patients of 52.1% had positive ASLO titer. Positive throat swab culture; 23%. 80% had high or significant increase in the value of Ig A and C3, but there was no significant difference between every groups. In the series two patient with NS went on to develop chronic renal failure.

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HBS ANTIGENEMIA ASSOCIATED NEPHROPATHY IN CHILDREN Yamashita, F. Matsuo, H., Yoshimoto, M., Shindo, S., Araki, H., Fujimoto, T., Nakano, M., Fujisawa, N., Nagayama, K., and Ito, Y. Kurume Univ. Sch. of Med., Dep. Ped., Kurume, Japan To know the role of HBs antigenemia in renal diseases in child-

To know the role of HBs antigenemia in renal diseases in children, the incidences of positive HBs antigenemia by histology were compared, and clinico-immuno-pathological study was performed in 9 cases of positive antigenemia with nephrotic s.or abnormal urine.

HISTOLOGY :	MC	PGN	FGS	MPGN	MN	CS* TOTAL#	-
HBs Ag(+)/total N	: 2/42	1/45	0/2	3/8	3/5	0/6 9/108	
2(+)/total_N.:	4,8	2.2	0	37.5**	60**	08.3	
persistent HBsAg		1/1		3/3	3/3	7/9	
persistent e Ag	0/2	1/1		2/3	3/3	6/9	

*(CS:chronic sclerosing N;**p=0.005 btw.MPGN &/or MN vs. others)

HBs antigenemia was highly positive in MPGN and/or MN groups, although no significant difference between 2 entities. A case with acute HB hepatitis c hematuria with transient HBs for 6 ms after hepatitis and a case with nephrotic S. and elevated serum IgE with transient HBs for 1 month both showed MC, wheras the other 7 cases with persistent HBs antigenemia and high incidence of persistent e antigenemia(6/7) revealed marked renal pathology:MPGN(3 cases), MN(3 cases) and PGN(1 case). HBs antigen was positive by IF only in a case with acute hepatitis and negative in 2 cases examined. Immune complex was positive in 3 cases in 3 examined by Rajii cell method and C1q solid phase EIA.Low C3,4,C1q were confirmed in 7/9. The data suggests pathognomonic role of HBs antigenemia in MPGN

COAGULATION

as well as MN in chidren.

HFMOLYTIC-UREMIC SYNDROME -A Report of an Outbreak-

Seven cases of hemolytic-uremic syndrome occurred in a small area in Japan within a short period in summer, 1978. The main clinical and laboratory findings are summarized in the Table. Bloody diarrhea, hemolytic anemia, proteinuria and hematuria were observed in all 7 cases, thrombocytopenia in 6 cases and uremia in 5 cases. Based on the grade of severity, 3 cases were considered as severely affected, one mildly affected and three subclinical or very mild. No common causative agents were detected. Histopathological finding was confirmed in severe case, revealing fibrosis of 20 % glomeruli and deposition of C3 and fibrinogen/fibrin along GBM. Electron microscopic examination was also performed. Packed red blood cells were transfused in 6 cases. Peritoneal dialysis and/or heparin were used in 3 severely affected cases. All cases recovered within 2 weeks to 3 months and were well in February 1980.

Table Clinical and Laboratory Findings in 7 Cases

Case No.	Age	Date of Admission in 1978	Hb g/d1	Platelet per c.mm	BUN mg/d1	Heparin Therapy	Peritoneal Dialysis
1	7y.	June 28	8.2	24x10 ⁴	32	-	-
2	11m.	July 1	6.2	7x10 ⁴	69.1	-	i -
3	4y. 2m.	July 3	3.1	7x10 ⁴	126	+	-
4	2y. 3m.	July 10	6.2	3.1x10 ⁴	22.2	-	-
5	2y. 7m.	July 10	6.6	4x10 ⁴	24	-	_
6	1y.5m.	July 21	3.6	5.6x10 ⁴	72	+	+
7	2y.	July 31	5.8	3x10 ⁴	108	+	+