115 GLOMERULAR LESIONS IN ASYMPTOMATIC CHILDREN WITH RENAL DISEASE DISCOVERED ON ROUTINE URINARY SCREENING.

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The aim of this study is to investigate histopathologically the incipient lesions of the insidious glomerular diseases. Under the care of our division of pediatric nephrology, there are about 300 children with urinary abnormalities pointed out initially by the routine urinary screening. Renal biopsied from 87 patients among them were studied with light and electron microscopy and immunofluorescent techniques. Their histological classification is shown in the table. It is noteworthy that many patients with potentially progressive renal disease such as crescentic GN, MPGN, MN, FGS, IgA Nephropathy, Alport's synd. and so on were found in asymptomatic children. Since 1973, the annual urinary screening at school has been carried out under government throughout Japan. Although it is difficult to decide whether medical management is required for asymptomatic children or not, the histological findings may make it possible to give pertinent guidance for planning appropriate therapeutic program in early stages of various types of glomerular diseases respectively. In addition, early detection of so-called incurable renal disease in children may offer better understanding of its histopathological change.

Minimal lesions	15
Focal GN	2
Focal glomeruloscrelosis	5
Mesangial GN	28
Endocapillary GN	8
Membranoproliferative GN	13
Membranous GN	1
Crescentic GN	2
miscellaneous lesions	13
TOTAL	87

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Fifteen patients with 5-23 years duration of IgA glomerulonephritis were studied. Four children were serially investigated from the onset. Clearance of inulin and PAH, and urinary excretion of sodium were studied during hydropenia and isotonic saline volume expansion.

GFR was depressed at the onset of the disease but normalized during the following months. Sodium excretion was low still after six months but then became normal. At the investigation at one and two years renal function was normal but all patients had signs of active disease and residual biopsy changes indicating an adaptation. At 5 years GFR in all four children had increased to high normal and sodium excretion during volume expansion was somewhat decreased.

Of the nine patients investigated after 9-23 years two had decreased GFR, 15 and 29 ml/min/1.73 m^2 10 years from onset. Both had hypertension. The others had normal or high GFR and in three fractional sodium excretion was increased. One of the latter had manifest and one had inconstant hypertension. Except more pronounced proteinuria the clinical course of the patients with decreased GFR did not differ from the others.

IgA glomerulonephritis thus could be a serious disease with risk of development of hypertension as well as renal insufficiency. The course of IgA glomerulonephritis can not be predicted within 5 years from onset.

117 PROLIFERATIVE GLOMERULONEPHRITIS ASSOCIATED WITH OSTEOGENIC SARCOMA.

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Tumor-associated glomerulonephritis, mediated by immune complex deposition occurs in carcinoma and hematopoeitic neoplasms but is not described in sarcoma. A 12 year old black girl with chest pain, fever and dyspnea was found to have massive cardiomegaly without murmur or pericardial rub. She also had proteinuria. Pericardiocentesis did not clarify the cardiac lesion. Two months later, cardiac symptoms recurred with pericardial effusion and left ankle swelling. X-ray showed an osteolytic lesion in the distal left tibial metaphysis, later diagnosed as poorly differentiated sarcoma by bone biopsy. Proteinuria was 530 mg/24hr. Renal biopsy revealed proliferative glomerulonephritis granular deposits of IgG and C-3 in glomerular capillary walls and mesangium, and glomerular osmiophilic deposits in subepithelial intramembranous and mesangial zones. Later she developed multiple nodular densities in the lungs and a right sides hemorrhagic pleural effusion. Samples of epicardial tumor implants obtained at thoracotomy revealed telangiectatic osteosarcoma. This pediatric case represents osteosarcoma associated with immune complex proliferative glomerulonephritis.

118 The Treatment of Glomerular Disorders

The treatment of glomerulonephritis is one of the most important therapeutic questions that nephrologi -st are facing today. In all countries glomeruloneph -ritis usually results in renal failure requiring dia -lysis or transplantation at the end stage. In recent years the anticoagulant treatment are widespread use on trials. Among 14 cases of glomerular disorders wi -th persistent glomerulonephritis, asymptomatic prot -einuria, and chronic glomerulonephritis, 7 cases sh -owed a reduction in proteinuria as the result of the treatment with dypiridamole. Histologically, they con -sist 4 cases of proliferative glomerulonephritis, 2 of membranoprolifetative glomerulonephritis, 1 of IgA nephropathy. 49 glomerular diseases were treated with carbocromen, among them 15 cases showed 25% or more reduction in proteinuria, but its effective ratio was relatively lower than in dypiridamole treatment under those quantities of trials. Histologically, they con -sisted 7 cases of proliferative glomerulonephritis, 2 of membranoproliferative glomerulonephritis, each 1 of IgA nephropathy and minimal change. A trial of kanpo treatment for glomerular disorder was also stu -died.

119 GLOMERULAR INJURY ASSOCIATED WITH MYCOPLASMA PNEUMONIAE INFECTION.

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We have studied 2 patients in whom glomerular injury was associated with M. pneumoniae infection. Case 1 (Nephron 21:284, 1978), an 11 y. girl, had pneumonia, pleural effusion and hematuria. Case 2, a 13 y. boy, had gross hematuria and diffuse pneumonitis. BUN and serum creatinine concentrations were normal in Case 1 and slightly elevated in Case 2 (30 and 1.1 mg/dl). ASOT, throat culture and C3 were normal or negative in both. Evidence for M. pneumoniae infection was:

		CASE 1	CASE 2
Cold agglutinin titer	initial	1:64	1:4
	subsequent	1:256	1:256
Mycoplasma CFT	initial	0	0
	subsequent	1:128	1:2048

In Case 1 the renal biopsy revealed an acute glomerulonephritis, IgG, C3 and mycoplasma antigen along glomerular capillary loops and in the mesangium.

In Case 2 the biopsy revealed a focal glomerulonephritis. IgA, C3 and weak fluorescence for mycoplasma antigen were seen in the mesangium.

We conclude that M. pneumoniae may be associated with an immune complex glomerulonephritis as well as an IgA nephropathy (Berger's disease).

120 SERIAL PATHOLOGICAL CHANGES OF MPGN

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Seventeen patients of MPGN have been followed for 1.5-7.5 years. The age at onset or discovery ranges from 3 to 25 years of age. Initially 9 had edema, 2 had anemia and hypertention and 15 had hypocomplementemia. Five had previous diagnosis of AGN, and 6 were nephrotic. In 9 patients abnormalities were found by routine annual urinalysis at school. Ten patients were treated with multiple small doses of steroid and immunosuppressants. None of these patients had so-called dense deposit disease. A 29 y/o nephrotic girl had intramembranous and subendothelial deposits which were characterized as segmental pattern. A 9 y/o boy had persistent hypocomplementemia although his initial hematuria and proteinuria have disappeared for 2 years. His renal biopsies showed subepithelial and mesangial deposits without any evidence for lupus nephritis serologically. Another 13 y/o girl had focal segmental GBM changes on initial specimen, which progressed to diffuse and global changes after 2 years. After a mean of 3.5 years follow-up one is in complete remission and one in CRF. Because of prevailing routine urinalysis at school, it becomes possible to detect and treat in its earlier stage of MPGN and this may give a clue to investigate pathogenesis of this disease.