Case Reports

Mesangial Proliferative Glomerulonephritis and Nephrotic Syndrome with Hepatitis A Virus Infection

R.C. Mathur
N.C. Mathur

The association of Hepatitis A, B and C viruses with renal dysfunction has been reported in literature. Renal failure has also been documented with Hepatitis A virus infection(l-4). In this case report we are describing a 7 year old boy with Hepatitis A virus infection who developed immune complex mesangial proliferative glomerulonephiritis, nephrotic syndrome and acute renal failure.

Case Report

A 7-year-old boy was admitted with seven day history of fever, anorexia, abdominal discomfort and vomiting. On examination, the child was febrile (10FF) with a respiratory rate of 34/min and a normal blood pressure. Icterus was present. He had epigastric and right upper quadrant tenderness. There was no hepatomegaly. Serum bilirubin was 2.3 mg/dl with a direct fraction of 0.4 mg/dl. Urine for bile pigments, salts and urobilinogen was positive. Further serial laboratory data is shown in *Table I.* Viral serology demonstrated the presence of antiHAV IgM antibodies. Other viral serological markers were absent. Ultrasound of the abodomen was normal.

On the 3rd day after admission he developed oliguria which was corrected by intravenous saline infusion. The 24 hours urine Na^+ done during this period was 230 mmol/1. The serum creatinine was 1.8 mg/dl and BUN 12 mg/dl. By day 6, he developed pedal edema, bilateral pleural effusion and hypertension of 140/100 mmHg. Urine output was normal. Serum total proteins were 2.4 g/dl, serum albumin 1.2 g/ dl, serum globulin 1.2 g/dl, serum creatinine 2.7 mg/dl and 24 hours urine protein 3.2 g/24 hours. Hemogram showed a hemoglobin of 12 g/dl, total count of 12,,800 cells/cu mm (neutrophilis 71%, lymphocytes 25% and eosinophils 4%). Blood culture was negative. C3 and C4 were decreased to 73 mg/dl and 20 mg/dl, respectively. Urine analysis showed albuminuria with granular casts. Serum cholestrol was raised. Renal ultrasound was normal. Renal scintigraphy with radio tracer Tc 99 DTPA showed normal vascular and excretory phases. Uptake phase showed moderate decrease in concentration bilaterally, with normal shape and sized kidneys. Relative function was 36% in the right kidney and 34% in the left kidney. This suggested a bilaterally symmetrical reduction in renal function.

The child was given albumin, nifedipine and blood pressure control was satisfactory. There was diuresis and clinical improvement. By day 9, the child's BUN rose to 40 mg/dl and serum creatinine to 3.5 mg/dl. On day 10, a kidney biopsy was done. Light microscopy showed glomeruli with prominent mesangial matrix with hypercellularity and focal prominence of epithelial cells. Capillary membranes were normal. Tubules revealed protein casts. Immunofluroscent study showed intense

From the Department of Pediatrics, Aditya Hospital, Hyderabad.

Reprint requests: Dr. Rajiv Chandra Mathur, 5, Subhodaya Apartments, Boggulkunta, Hyderabad 500 001.

Received for publication: January 9,1996; Accepted: August 14,1996

TABLE I–*Pertinent Laboratory Data*

Laboratory data	Day 1 (Admission)	Day 3	Day 6	Day 9	Day 14	Day 19 (Discharge)	Day 36
Serum bilirubin (mg/dl)	2.3		2.2	2.0	1.2		1.1
SGPT (IU/L)	603		432	310	52		32
SGOT (IU/L)	240		132	104	30		28
PT (sec)	12.2			11.1			
Serum albumin (g/dl)			1.2			2.0	3.2
Serum creatinine (mg/dl)		1.8	2.7	3.5		2.1	1.2
BUN (mg/dl)		12	35	40		24	16
24 h Urine protein (g/24h)				3.2			56 mg
Urine analysis							
Albumin			4+	4+		1+	Nil
Microscopy			Granular casts	Granular casts		Nil	Nil
Serum cholestrol (mg/dl)			300				150
Viral markers							
Anti HAV IgM	+ve						–ve
Anti HAV IgG	-ve						+ve

* Other viral hepatitis markers were negative.

focal granular deposits of IgM and C_r These findings suggested immune complex measangial proliferative glomerulo-nephritis. ANA was negative. Liver function tests were normalized by day 14. With the improving renal function by day 19 the child was discharged and reviewed weekly. Two weeks later, viral hepatitis serology showed presence of anti HAV IgG and absence of IgM antibody. The liver and renal function tests were normal. He was followed up bi-monthly for 6 months.

Discussion

There have been reports of association of hepatitis B with various forms of glomerulonephropathies in children(5). HAV glomerulonephropathy is uncommon. Association has occasionally been documented with mild proteinuria, hematuria and renal failure (1-4). One report demonstrated association with nephrotic syndrome and renal failure(6) and 2 reports documented IgM glomerulo-nephritis with hepatitis A infection(7,8).

The support for the association of HAV and immune complex glomerulo-nephritis in this child are: *(i)* its development after HAV infection, *(ii)* no prior renal disease, *(iii)* no evidence of systemic disease, *(iv)* remission of nephrotic syndrome in the convalescent stage of hepatitis A infection, and *(v)* biopsy consistent with immune complex glomerulonephritis. Electron microscopy was not done in this case.

Acute renal failure is an infrequent complication of HAV infection. It may be due to transient hypotension and decreased plasma volume, which leads to decreased renal perfusion. Improvement in renal function in the recovery stages of HAV infection may be due to the removal of the

viral antigen. The prognosis of these cases is unknown.

In conclusion, this case suggests that rarely mesangio proliferative glomerulonephritis, nephrotic syndrome and acute renal failure may occur as a complication of hepatitis A virus infection.

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