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## CASE REPORT

# Hepato-Renal Syndrome

*by*

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**Abstract**

*Herein we report a case of hepato-renal syndrome, the diagnosis established was based on chronic hepatic disease accompanied by impairment of renal function and histopathologic changes.*

*Clinical pictures and histopathologic examination of the liver showed that the hepatic disease was an obstructive jaundice due to congenital malformation. On the other hand, clinical pictures, laboratory examination, renogram, light microscopy and electron microscopy of the kidney showed that there were severe impairment of renal function and tubular changes together with focal interstitial fibrosis.*

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## Introduction

During the past three decades, many investigators had described characteristic renal glomerular alterations in a variety of acute and chronic liver disease. Anuria and uremia had been observed in four cases after cholecystectomy, diffuse cortical necrosis of both kidneys with severe oliguria following a crushing injury of the liver, were reported by Bell (1950), and this report was confirmed by other authors (Heptinstall, 1966, Kaplan and Drumond, 1975).

In viral hepatitis, by an immunologic mechanism the renal alterations were oliguria, anuria, uremia, proteinuria, hematuria and may be renal failure could occur. Anatomical alterations were tubular defect, membrano-proliferative glomerulonephritis, focal glomerulonephritis, epimembranous glomerulonephritis (Bridi et al., 1972; Myers et al., 1973; Knieser et al., 1974).

These renal disease were also observed in some kind of congenital hepatic disease such as polycystic of the liver, small liver, chronic bile duct obstruction and congenital hepatic fibrosis, (Anand et al, 1975; Dobrin et al., 1977; Better et al., 1972).

Callard et al. (1975), detected the glomerular lessions in 9 of 10 patients with liver cirrhosis.

The pathogenesis of renal alteration in hepatic disease such as chronic active hepatitis or others remains unclear up to now. Knieser et al., (1974), note that the renal alterations in hepatic disease were due to hyperimmunoglobulinemia. Kaplan and Drumond (1975), suggested that the renal alterations may be due to the toxic substances normally
the liver.

## Case report

An Indonesian female, 3½ years old, with a body weight of 11kg, was hospitalized for the second time. The first hospitalization was in 1974 because of congenital bile duct obstruction and her parents refused surgical intervention (1½ months old). On this second admission, deep unconsciousness (coma) was observed, with marked jaundice, convulsions occurred one hour before admission. On physical examination ascites and hepatosplenomegaly were observed, the cerebro spinal fluid was normal.

The conventional treatment was instituted with glucose 10%, tutofusin intra venously, antibiotic and corticosteroid were given immediately. On the fourth day of hospitalization she was alert but still icterus and oliguria were detected.

On further investigation severe impairment of the liver function was observed (with direct bilirubin of more than 6 mg% and indirect bilirubin of more than 4 mg%), the liver enzymes increased and the histopathological examination showed cellular infiltration and severe obstruction of the biliary system.

In the first three weeks of hospitalization there were oliguria and high blood urea and creatinin content, low clearence of ureum and creatinin. The renogram revealed severe impairment of the left renal function and slow excretion of the right kidney. Pathological examina-

tion of the renal specimen (obtained by needle biopsy), showed evidence of slight glomerular dysfunction together with tubular changes (examined by light microscopy and electron microscopy).

HBAg examination was negative and immunological examination revealed elevation of IgM (700 mg%) and IgA (154 mg%) with normal values of IgG (1570 mg%) and BIC globulin (150 mg%).

## Discussion

The term hepato-renal syndrome was known as any kind of liver disease such as congenital or acquired, acute or chronic, infection or traumatic due to accident or surgical intervention, which was followed by the alteration of renal function with or without the presence of histopathologic changes of the renal tissue.

Our patient presented with features of characteristic congenital chronic bile duct obstruction in association with alteration of the renal function. There were elevation of blood urea and creatinin with low clearance, and renogram examination showed severe impairment of the renal function, in which slight glomerular dysfunction and tubular changes were noticed on light and electron microscopic examination.

Her primary liver disease manifested as presistent jaundice, elevation of serum liver enzymes, severe impairment of the liver function, and histopathological examination revealed marked cellular infiltration and obstruction of the bile duct system.

The pathogenesis of hepato-renal syndrome is up to now uncertain. Better and Messry (1972), stated that the effect of chronic bile duct obstruction was the alteration on renal handling of salt and water, manifested as low sodium excretion, low water excretion, oliguria, hypotonic urine, increase of urine osmolality and decrease of the concentration ability of the kidney. Kaplan and Drumond (1975) stated that the possibility of renal function disorder was due to renal vascular vasoconstriction caused by a vasoactive compound normally detoxified by the liver. In cases of chronic active viral hepatitis, Myers et al. (1973) ,Knieser et al. (1974), Andre and Andre (1976), Lassen and Thomson (1958), stated that in such cases the evidence of hyper-immunoglobulinemia was considered as the cause of the alteration of the kidney. In this case the hyper- bilirubinemia may have toxic effect and produce cholemic nephrosis as stated before by Lassen and Thomson (1958) and Kaplan and Drumond (1975).

On the other hand the hyper-immunoglobulinemia in this case was also concerned as the cause of the entity as stated by some authors, Callard et al. (1975) and Dubrin et al. (1977). Unfortunately we were unable to demonstrate the immunoglobulin deposit in the renal tissue with immunofluorescence technique.

## Conclusion

This patient presented with congenital chronic bile duct obstruction, accompanied by alteration of the renal function

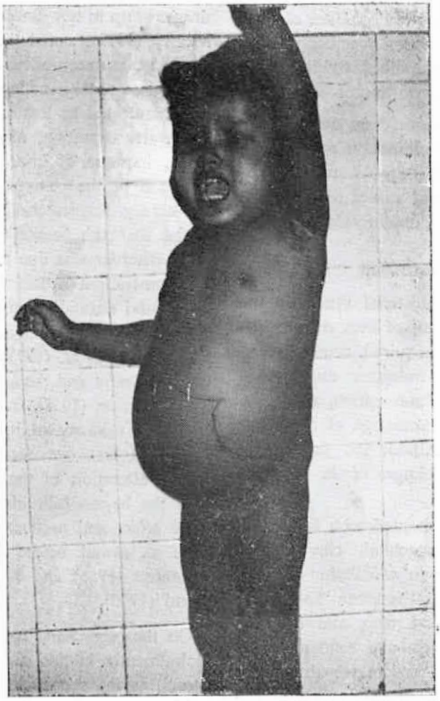

FIG. 1 : *The patient, a little girl of 3½ years old.*

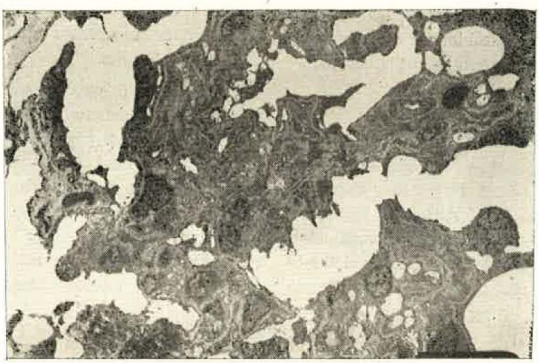

FIG. 2 and 3 : *The electron microscopic examination, with magnification 3,500 x and 16,800 x.*

with renal tissue changes. We are of the opinion that the renal disorder was caused by hyperbilirubinemia and hyperimmunoglobulinemia due to the chronic liver disease.

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Two glomeruli were examined. The mesangium is unaltered but some capillary loops are widely patent while others are collapsed. Some podocytic processes are fused. The peritubular capillaries are dilated while the tubular cells posses prominent lysosomes. There is evidence of slight glomerular dysfunction and together with the tubular changes.

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