CASE REPORT

TYPHOID GLOMERULONEPHRITIS IN A CHILD: A RARE COMPLICATION OF TYPHOID FEVER

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Abstract. We report a child with typhoid glomerulonephritis who presented with fever, gastrointestinal symptoms, edema, hypertension and abnormal urine findings including microscopic hematuria and proteinuria. *Salmonella typhi* resistant to ampicillin and cotrimoxazole was isolated from a blood culture. Renal biopsy was not performed. The child successfully treated with ceftriaxone.

Typhoid fever caused by Salmonella typhi (S. typhi) is still a common public health problem in several developing countries. In Thailand, the number of typhoid patients has been dramatically declined in the past two decades (Division of Epidemiology, 1984-1999). Several uncommon complications of this disease have been reported (Thisyakorn *et al*, 1983; Vejabhuti and Thisyakorn, 1987). However, there is a paucity of published information available on typhoid glomerulonephritis (GN) (Sitprija, 1974; Buka and Coovadia, 1980). We therefore report a case of typhoid GN and review the previously reported English language literatures.

A 14-year-old Thai boy was admitted to Chulalongkorn Hospital on June 1, 2000 because of a 5-day history of fever, vomiting and severe abdominal pain. On examination the patient had a temperature of 40°C, a blood pressure of 130/72 torr and right iliac fossa tenderness with guarding and rebound tenderness. Complete blood count revealed a hematocrit of 41.3 volume%, a white blood cell count of 10,000 cells/mm³ with 86% of neutrophils. Urinalysis showed: protein 2+, 3050 red blood cells(rbc)/high power field (HPF), 2-3 white blood cells (wbc)/HPF, and 1-2 coarse granular casts/HPF. Blood chemistry showed: blood urea nitrogen (BUN) 16 mg/dl, creatinine (Cr) 1.2 mg/dl; Na 128, K 3.8, Cl 95, CO_2 19 mEq/l. On the third day of admission, the patient remained febrile and hypertensive. He developed oliguria, generalized non-pitting edema, and an increased body weight of 3.5 km. Repeated urinalysis showed: protein 4+, 40-50 rbc/HPF, 15-20 wbc/HPF, and 0-3 rbc casts/HPF. Acute pyelonephritis was diagnosed and the patient was treated with parenteral ceftriaxone.

On the fifth day of admission, hemoculture grew Salmonella typhi, which was sensitive to cefotaxime, ceftriaxone and ciprofloxacin, and resistant to ampicillin and cotrimoxazole. The Widal test, Weil-Felix test and leptospirosis titer were negative. Antistreptolysin O titer (ASO), the third component of serum complement (C_3) and anti-DNase B were normal. A clinical diagnosis of typhoid glomerulonephritis (GN) was made. Ceftriaxone was continued for a total of 14 days. The condition of the patient improved after the fifth day of antibiotic therapy and he was discharged on after 16 days.

In the past decades, patients with enteric fever were commonly seen in Thailand. Among 192 children with enteric fever in a previous

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Author, year	Number of cases	Age/ Sex	Clinical of nephritis	Positive blood culture	Urine findings	Positive renal biopsy
Sitprija, 1974	3	16,34,37 yrs M,F,F	No	3/3	Proteinuria (2) Normal (1)	3/3
Buka, 1980	15	2-13 yrs 5M,10F	Yes	9/15	Proteinuria 15/15	2/5
Dhawan, 1992	1	10 yrs/M	Yes	Yes	Proteinuria, red cell casts	ND
Our patient, 200	0 1	14 yrs/M	Yes	Yes	Proteinuria, hematuria	ND

Table 1 Cases of typhoid glomerulonephritis from English language literature review.

Note: yrs = years, M = male, F = female, ND = not done, GN = glomerulonephritis

report, proteinuria with occasional blood cells were seen in the urine of 66% of patients without clinical pictures of GN (Thisyakorn et al. 1987). Even though there is at present a much smaller number of children suffering from typhoid (Wongsawat et al, 2000), this patient presented with typhoid GN. The clinical features of GN, including hypertension, hematuria and proteinuria were seen at the initial presentation on admission, together with edema and oliguria during hospitalization. The co-existing clinical presentation of high fever and prominent gastrointestinal symptoms suggested the probable diagnosis of enteric fever, and this was confirmed by the positive culture of S. typhi.

Poststreptococcal glomerulonephritis (PS GN) is the leading cause of GN in Thailand. Compared with typhoid GN, the course of PSGN is usually shorter and the evidence of group A streptococci (GAS) infection usually disappears by the time of edema (Buka and Coovadia, 1980). This diagnosis is usually confirmed by evidences of GAS infection and immune process including high ASO, anti-Dnase B and/or positive throat swab culture for GAS, and low C_3 . The clinical setting and lack of evidence of GAS infection of our patient helped exclude PSGN from the diagnosis.

Fifteen children aged 2-13 years with typhoid GN were reported (Buka and Coovadia,

1980). Most patients presented with fever, gastrointestinal symptoms, edema, oliguria, high blood pressure and abnormal urine findings. Cultures positive for S. typhi were found in 9 patients. Renal biopsy was performed in two Three patients died. Another repatients. ported case was a 10-year-old Indian boy who presented with fever, acute renal failure, proteinuria and rbc casts in the urine (Dhawan and Marwaha, 1992). Renal biopsy was not performed in this patient. Our patient had clinical presentations and laboratory findings identical to these patients. All reported cases including our patient are summarized in Table 1.

The pathogenesis of typhoid GN is probably caused by direct invasion of *S. typhi* and the process is immune-mediated. This is supported by a previous study on renal biopsies of three typhoid patients who had no clinical manifestations of GN (Sitprija *et al*, 1974). The renal pathology demonstrated immune complex GN, with deposition of immunoglobulins and C_3 , and salmonella Vi antigen in the glomerular capillary wall. Renal biopsy was not performed in our case because this procedure would not have benefited our patient, as commented in a letter to the editor (LoGerfo, 1974).

In summary, clinical GN is a rare complication of typhoid fever whereas subclinical GN may be frequent and overlooked. The patients present with clinical features of typhoid fever together with GN.

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