and leucopenia absent. In endemic areas a high index of suspicion and appropriate investigations are required to establish the diagnosis.

REFERENCES

1. Maegraith B. Leishmaniasis. *In:* Clinical Tropical Diseases. 8th edn. Great Britain, ELBS, 1984, pp 189-310.
2. Bahr M. Leishmaniasis. *In:* Tropical diseases, 18th edn. London, ELBS and Bailliere Tindall, 1982, pp 93-115.
3. Mishra M, Choudhary RR. Kala azar with peripheral eosinophilia. J Assoc Phys India 1987, 35: 319-320.
4. Talukdar B, Kanwar A, Saini L, Singh T, Chauhan S. Lymphadenopathy in Indian kala azar. Indian Pediatr 1987, 24: 1043-1045.
5. Mishra V, Singh AN. Jaundice as presenting feature of Indian kala azar. J Assoc Phys India 1985, 33: 22.
6. Prasad LSN. Kala azar in Bihar. Indian J Pediatr 1987, 54: 103-112.
7. Barat D, Bhattacharya PK. Clinical profile of kala azar in North Bihar. J Indian Med Ass 1986, 84: 367-369.
8. Thakur CP. Epidemiological, clinical and therapeutic features of Bihar kala azar. Trans Royal Soc Trop Med Hyg 1984, 78: 391-398.

Acute Glomerulonephritis in Multi-Drug Resistant *Salmonella Typhi* Infection

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Typhoid fever still remains a perplexing public health problem in the developing world. In 25-30% of cases, the illness is complicated by involvement of one or more major organs of the body(1,2). The emergence, in recent months of a multi-drug resistant (MDR) strain of *Salmonella typhi*(3,4) is likely to increase the frequency of encounters with atypical manifestations of this disease. This report of a child with acute renal failure in association with MDR *S. typhi* infection aims to focus attention on renal involvement in enteric fever.

Case Report

A 10-year-old school-going boy, presented to the Pediatric services of Nehru Hospital, P.G.I.M.E.R, Chandigarh with an acute illness, characterized by remittent fever, of a fortnight's duration. A few days after the onset of the fever, he had intermittent, ill-localised pain in the abdomen and occasional vomitings. The appearance of hematuria and oliguria, 5 days prior to hospitalization, necessitated a referral from the local physician, who, however, failed to provide specific treatment details.

Examination revealed a febrile, ill-looking child with periorbital puffiness of both eyes, edema feet and deep acidotic breathing. He was normotensive (90/60 mm of Hg in the supine position) and had a hepatosplenomegaly of 3 cm and 1 cm below the costal margin, respectively.

The hemoglobin was 6.9 g/dl, white blood cell count 8.0 $\times$ 10^9/L with 70% neutrophils and the platelet count was nor-

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mal. The coagulation screen did not reveal any abnormality. He had normal serum electrolytes, a markedly elevated blood urea nitrogen (BUN) of 260 mg/dl and a serum creatinine of 4.0 mg/dl. The urinary sediment showed plenty of RBCs/HPF with RBC casts and an occasional granular cast. The 24-hour urinary protein was 200 mg. MDR *S. typhi* (*i.e.*, resistant to chloramphenicol, cotrimoxazole and amoxycillin) was isolated on hemoculture whilst the urine culture was sterile. The titres of TH and TO antigens were 1 : 1280 and 1 : 320, respectively. The serum complement (C3) level was 85 mg/dl against a control of 100 mg/dl.

Treatment for typhoid fever was begun with injectable ciprofloxacilin in a dose of 10 mg/kg every 12 hours. Fluids were initially restricted to 400 ml/m^2 and peritoneal dialysis was instituted on 3 occasions before he entered a diuretic phase and his BUN and serum creatinine showed a progressive decline to normal levels. The course of events during his hospital stay and the biochemical parameters are illustrated in the *Fig.* Improvement in parameters of renal function and an increase in urinary output coincided with defervescence. Therapy with ciprofloxacilin was continued for a total of 14 days. At discharge, the child had recovered completely. Permission for a renal biopsy was refused and we did not force the issue once the biochemical and the urinary parameters were entirely normal. The child has been followed up for over 6 months now and remains entirely well clinically and biochemically. The repeat C3 level, estimated after an interval of 6 weeks was 110 mg/dl.

Discussion

The clinical manifestation and laboratory findings in our patient were characteristic of an acute glomerulonephritis leading to acute renal failure in association with a MDR *S. typhi* infection. Although conclusive evidence based on renal histological findings has not been obtained, it will be logical to presume a cause and effect relationship between *S. typhi* and nephritis. Specific antimicrobial therapy and repeated peritoneal dialysis culminated in recovery from typhoid fever and the simultaneous return of completely normal renal function.

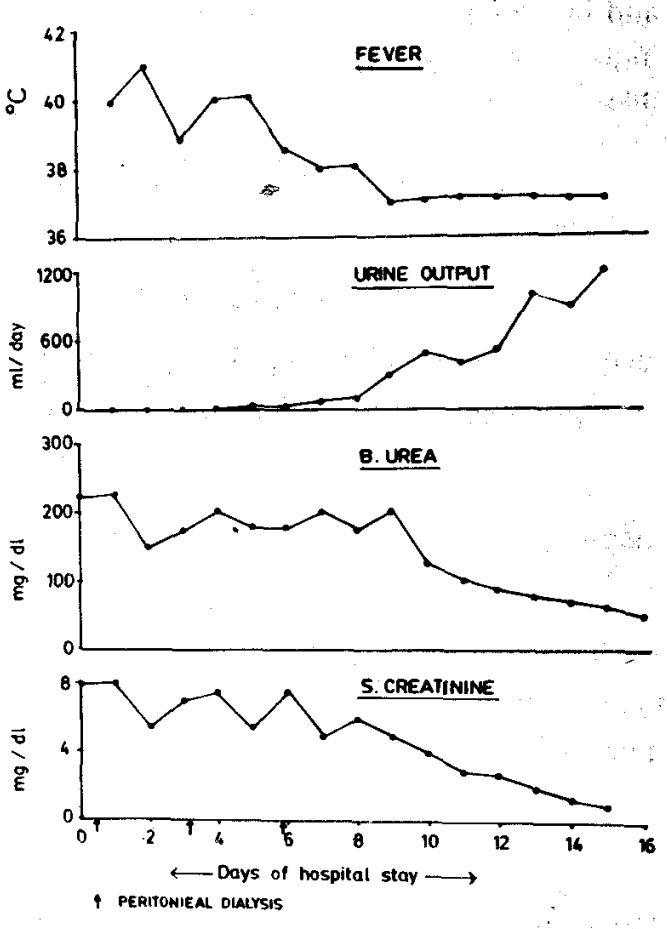

Fig. Course of events during hospital stay

Renal involvement in typhoid fever is an uncommon event with an overall incidence of 2-3% having been reported(1,2). In most cases, it manifests as mild proteinuria. Glomerulonephritis has been rarely reported. Gulati *et al.* recorded two cases of nephritis in a series of 98 patients. Glomerulonephritis with overt clinical re-

nal manifestations in patients with typhoid fever is the subject of case reports(5-7). However, Sitprija *et al.*(8) have postulated that glomerulitis with typhoid fever may be frequent but is overlooked because of lack of clinical manifestations. They carried out renal biopsies in 3 unselected cases of typhoid fever with no clinical or laboratory evidence of renal dysfunction and were able to demonstrate immune complex glomerulitis in all.

The main histologic findings in typhoid nephritis are a diffuse tubular damage, localized small cell infiltration of the interstitium with relative sparing of the glomeruli(5,9). Bacilli are generally not demonstrated in the kidneys and a toxin-induced nephritis has been postulated as the pathogenetic mechanism. More recently, however, Sitprija *et al.*(8) have suggested a direct role of *S. typhi* in the pathogenesis of the glomerular lesion. They observed deposition of immunoglobin and complement (C3) on the glomerular basement membrane besides detecting *Salmonella* VI antigen in the glomerular capillary wall. Amerio and associates(6) demonstrated similar deposition of circulating immune complexes in the glomerular lesion but failed to show the typhoid antigen. In a recent study of renal functions in typhoid fever, Khosla and Lochan(10) correlated the disappearance of circulating immune complexes with recovery in renal functions. We were unable to perform a renal biopsy but speculate that the low C3 during the acute illness suggests a similar pathogenetic basis for the kidney involvement in our case.

The kidneys may be much more frequently involved in typhoid fever than is commonly believed. In most cases, the dysfunction is restricted to a non-specific mild proteinuria. Our case serves to highlight the relatively rare complication of severe glomerulonephritis with acute renal failure. Prompt recognition and repeated peritoneal dialysis with specific antimicrobial therapy resulted in complete recovery.

REFERENCES

1. Gulati PD, Saxena SN, Gupta PS, Chuttani HK. Changing patterns of typhoid fever. Am J Med 1968, 45: 544-548.
2. Khosla SN. Changing patterns of typhoid: A reappraisal. Asian Med J 1982, 25: 185-198.
3. Koul PB, Murali MV, Sharma PP, Ghai OP, Ramachandran VG, Talwar V. Multidrug resistance *Salmonella typhi* infection: Clinical profile and therapy. Indian Pediatr 1991, 28: 357-361.
4. Sen S, Goyal RS, Dev R. Ciprofloxacilin in the management of multiple drug resistant typhoid fever. Indian Pediatr 1991, 28: 417-419.
5. Faierman D, Ross FA, Seckler SG. Typhoid fever complicated by hepatitis, nephritis and thrombocytopenia. JAMA 1972, 221: 60-61.
6. Amerio A, Campese VM, Coratelli P. Glomerulonephritis in typhoid fever (Abstract). Fifth International Congress of Nephrology, Sao Paulo, 1972, p 316.
7. Choudhry VP, Singh BM, Sinclair S. Salmonella nephritis. Indian Pediatr 1977, 14: 857-859.
8. Sitprija V, Pipatanagul V, Boonpucknavig V, Boonpucknavig S. Glomerulitis in typhoid fever. Ann Int Med 1974, 81: 210-13.
9. Henke F, Lubarsh O. Handbuch der speziellen pathologischen Anatomie and Histologie. Berlin, Verlag Yon Julicis Springer, 1968, pp 570-573.
10. Khosla SN, Lochan R. Renal dysfunction in enteric fever. J Assoc Phys India 1991, 39: 382-384.