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Reply

We thank Dr. Srivastava for his interest in the case report of acute glomerulonephritis in typhoid fever(1). Reasons beyond our control precluded a renal biopsy. The absence of confirmatory histological findings naturally generates controversy regarding the renal pathology. We would like to reiterate that the presence of red cell casts in the urine and the low serum C3 give credence to a glomerular lesion. Sitprija *et al.*(2) were able to demonstrate immune complex glomerulitis in three unselected cases of typhoid fever with no clinical or laboratory evidence of renal dysfunction and postulated a direct role of *S. typhi* in the pathogenesis of the glomerular lesion. If this is true, then recovery in renal function could parallel the amelioration of other symptoms and signs as was observed in the case reported. We accept criticism of the obvious omission in our case but it may prompt a systematic evaluation of the pathogenetic mechanism(s) of renal dysfunction in typhoid fever and of the course and pattern of recovery in cases with renal failure.

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Trichuris Dysentery

Infection with whipworm, *Trichuris trichiura* is one of the commonest helminthiases of childhood. In tropical countries where other diseases obscure the source of symptoms, this parasite is usually more or less ignored, with the assumption that it is non-pathogenic(1). That such is not always true is being demonstrated by the following case report.

A five-year-old male child was admitted with history of prolonged diarrhea with blood streaked stool, abdominal pain, tenesmus for the last 3 years. The child had suffered from generalized swelling of the body two years back for which he had received three bottles of blood transfusion. He was also suffering from rectal prolapse. Apart from this, the child was treated outside also with metronidazole, cotrimoxazole, furazolidine, nalidixic acid and Inj. emetine without any appreciable result.

Physical examination revealed a moderately anemic child with weight of 14 kg, height 100 cm and mid-arm circumference 13 cm. No edema or clubbing was observed. Abdominal examination showed

that liver was enlarged 3 cm below the right costal margin, firm and nontender. Spleen was not palpable. Neither any abdominal tenderness nor any thickened colon was detected. The boy had exhibited prolapse rectum.

Investigations showed a hemoglobin of 7.8 g/dl, total leucocyte count 7700/mm^3 with 68% neutrophils, 19% lymphocytes, 1% monocyte and 12% eosinophils and adequate platelets. Peripheral picture showed some degree of anisopoikilocytosis and hypochromia of red cells. Repeat examination of stools revealed ova of *Trichuris trichiura.* Ova count per g of stool was more than 25000. Ova of *A. lumbricoides* was detected on one occasion. Barium meal examination of stomach and duodenum and follow through showed no abnormality. Barium enema *X*-ray was also normal. Sigmoidoscopic examination (upto 20 cm from anus) showed hyperemic, edematous and friable bleeding mucosa. Colonic biopsy showed fragments of colonic mucosa with nonspecific inflammatory infiltration and many intestinal nematodes probably *T. trichiura.*

This patient was treated with two courses of mebendazole 100 mg twice daily for 3 days at 7 days interval. He showed great symptomatic improvement and was discharged in good condition. Though complete eradication of infection is extremely difficult but mebendazole is considered to be the drug of choice for this infestation as it is active against both larval and adult stages of the worms. It is also ovicidal for *Ascaris* and *Trichuris*(2). The adult worm consumes about 0.005 ml of blood from the host each day and may precipitate an iron deficiency anemia in childrden already undernourished(3). In a tropical country like ours *Trichuris trichiura* infestation should, therefore, be borne in mind in dealing with cases of blood streaked diarrhea in children.

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