

# A case report of typhoid fever masquerading as acute typhoid glomerulonephritis

B M Yashodhara, Uduman Ali Mohamed Yousuf

## ABSTRACT

**Introduction:** Typhoid fever is a common infectious disease worldwide, especially in developing countries. Typhoid fever presenting as nephritis is rare. The common renal complications of typhoid fever are pyelonephritis and cystitis. Patients with typhoid glomerulonephritis may present with acute renal failure, hypertensive encephalopathy or nephritic syndrome. Presence of typhoid nephritis increases mortality to 20-30% in untreated typhoid fever. **Case report:** We report a case of a 29-year-old patient in this case report who presented with fever, hypertension, pedal oedema, and hematuria. Widal was positive in high titres and blood culture grew *Salmonella typhi*. His fever, hypertension and oedema resolved on treatment for the infection. **Conclusion:** Typhoid fever presenting as typhoid glomerulonephritis is a rare occurrence and has to be considered in patients in endemic areas, presenting with nephritis and concomitant fever. High index of suspicion, prompt early diagnosis is beneficial, in preventing morbidity and mortality related to with *Salmonella typhi* infection.

**Keywords:** Typhoid glomerulonephritis, Typhoid fever, Nephritis, Acute renal failure

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## INTRODUCTION

Typhoid fever is a very common infective disease in Asian and underdeveloped countries [1, 2]. Typhoid fever, in the first week of illness, manifests with fever, headache, abdominal pain, gastrointestinal symptoms like anorexia, nausea, vomiting, constipation with or without any specific signs. The common signs like relative bradycardia, hepatomegaly, splenomegaly, abdominal tenderness may develop in second week of illness. Complications like acute abdomen, intestinal perforation, pneumonia, psychosis, ataxia, altered sensorium and nephritis are likely to develop in third to fourth week of illness [3-5]. Initial presentation as acute glomerulonephritis is a very rare manifestation of typhoid fever [6-8].

## CASE REPORT

A 29-year-old male presented with fever of eight days, periorbital puffiness, gross hematuria and oliguria of four days. There was no past history of leg swelling, oliguria, hematuria, loin pain, facial puffiness, skin or joint complaints, hypertension,

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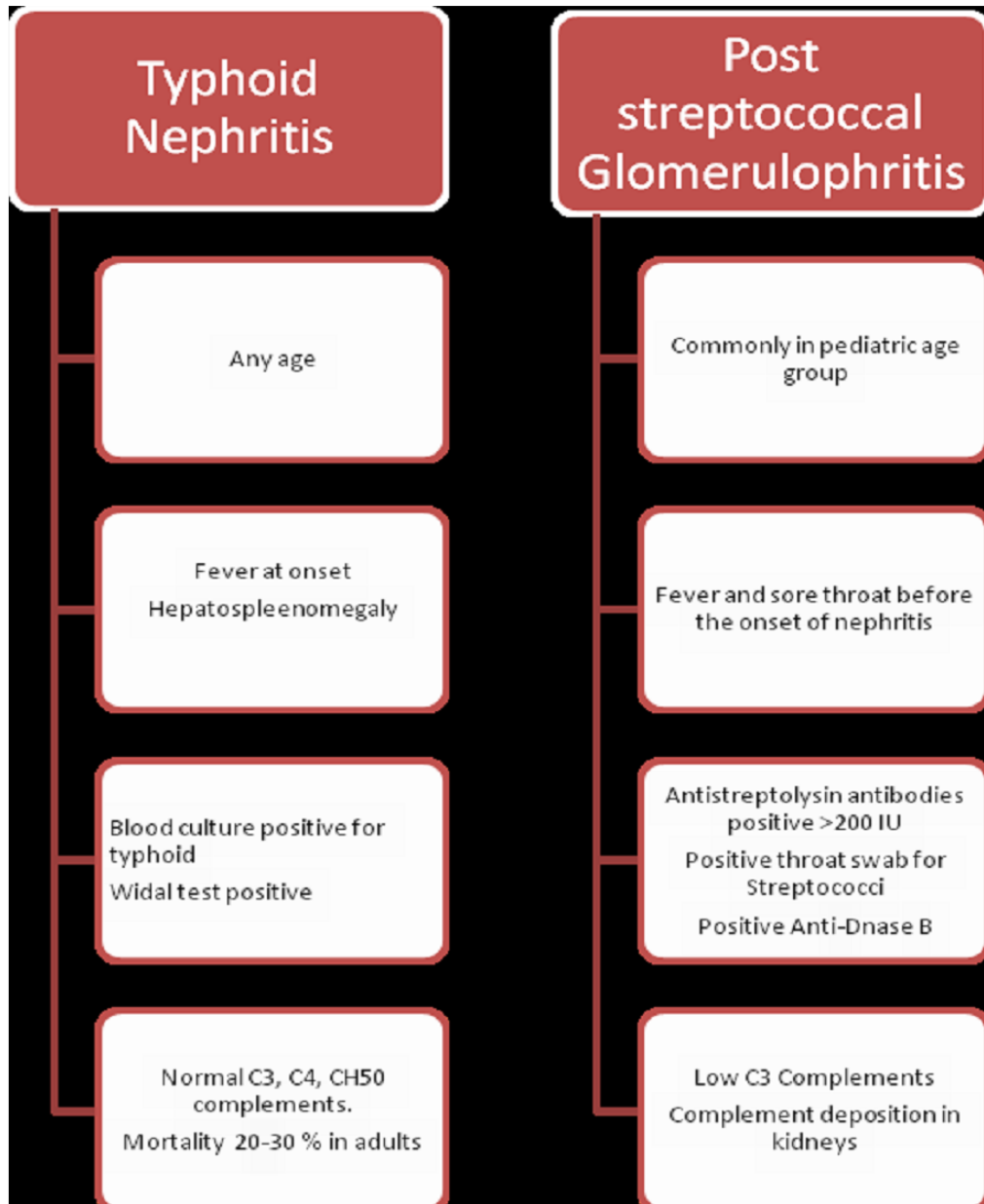


Figure 1: Compares acute typhoid nephritis with acute post-streptococcal glomerulonephritis.

generalised weakness and other renal related problems. There was also no history of any drug intake, sore throat or skin infections in the recent past. On examination oral temperature was 101 degree F, pulse rate was 90 beats/min, and blood pressure was 170/100 mm of Hg. He had periorbital and facial puffiness, bilateral pedal oedema, elevated jugular venous pulse of 6 cm from sterna angle and 4 cm non-tender hepatomegaly from right costal margin, 3 cm spleenomegaly from the left costal margin. There was no free fluid in the abdomen and no tenderness anywhere in the abdomen including renal angle tenderness. Other systemic examination for cardiovascular, respiratory, central nervous and

musculoskeletal system were essentially normal. His urine output from 1<sup>st</sup> to 5<sup>th</sup> day in the hospital was 300 to 400 ml/day with gross hematuria.

**Laboratory investigations:** Haemoglobin was 11 gm/dl, total leukocyte count was 4300 cells/cumm with 40% neutrophils, 58% lymphocytes and 2% basophils and ESR was 40 mm/1<sup>st</sup> hr. Urine examination showed hematuria on gross examination, confirmed by microscopic examination, with crenated red blood cells (RBCs) and RBC casts. There was urine proteinuria of 860 mg/24 hours of urine collection. His serum creatinine was 3.5 mg/dl, and blood urea was 45 mg/dl. His liver function tests, ECG, Chest X-ray were within normal limits. His first Widal test

yielded antibody titre for O antigen of 1:160, H antigen 1:160 which was done on the first day of admission and rose to a titre of 1:640 on the 8<sup>th</sup> of admission. Blood culture was positive for *Salmonella typhi*; sensitive to ceftriaxone, cefotaxime, and quinolones but resistant to amoxicillin and ampicillin. At all times, titres for AH and BH (Paratyphoid A and B) were negative. Urine culture for urinary tract infection did not yield any growth. Serology for HIV, HBsAg, and hepatitis C were negative. Antinuclear antibody, malaria parasite antigen and peripheral smear for malarial parasite were all negative. C-reactive protein was 6 mg/dl and Anti-streptolysin antibody was negative. Ultrasonography showed mild hepatosplenomegaly and normal sized kidneys.

He was treated with intravenous (IV) ceftriaxone 2 gm/day for 10 days, salt and fluid restriction, oral atenolol 50 mg/day once a day and frusemide 40 mg/day and bed rest. He became afebrile on 4<sup>th</sup> day of admission. He started passing good volume of urine from day five of admission with gradual disappearance of hematuria. Blood pressure was well controlled to 130/80 mm of Hg from day two of admission. Blood culture done on 6<sup>th</sup> day of illness was sterile. Microscopic hematuria was improving although persisted upto 10 days of admission and then disappeared during outpatient follow up. His blood urea and serum creatinine started to normalise and were completely normal on discharge. However, mild proteinuria persisted at 120 mg/day during outpatient follow up, but it was much lower as compared to time of presentation. Kidney biopsy was not done in view of rapid recovery.

## DISCUSSION

Our patient was a 29-year-old adult. He presented with fever, oliguria, oedema, proteinuria, hypertension and azotemia. Urine microscopy showed crenated red cells. All these features suggested glomerulonephritis with acute renal failure, which promptly recovered with treatment of typhoid. Typhoid fever is a very common infective disease. Common early presentation of typhoid fever is insidious onset of continuous fever, with nonspecific malaise, myalgia, arthralgia, headache, nausea, anorexia, constipation. In first week of illness signs are usually not present [2-5]. Hepatosplenomegaly, relative bradycardia and tenderness in right iliac fossa tend to develop in second week of illness. Uncommon manifestations like altered sensorium, prolonged pyrexia, pneumonia, intestinal perforation, intestinal hemorrhage, myocarditis, septic shock, meningitis, parotitis, osteomyelitis, orchitis, nephritis may present by third to fourth week as complications of untreated or multi drug resistant cases [2-4, 9]. They are very rare nowadays owing to availability of early diagnosis and treatment [4, 10]. Typhoid fever masquerading as acute glomerulonephritis is a very rare entity and occurs in

Table 1: Renal Manifestations of Typhoid fever

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Bacilluria
Proteinuria
Hematuria following typhoid vaccination
Acute tubular necrosis with acute renal failure
Cystitis
Pyelonephritis
Acute interstitial nephritis
Acute glomerulonephritis
Ig A nephropathy
Postvaccinal nephropathy

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2-3% of all cases [7]. Renal involvement in typhoid is called 'nephrotyphoid' [4, 10]. Renal manifestations of typhoid fever are shown in the table 1.

The usual renal manifestations in typhoid are presence of red blood and white blood cells in urine, albuminuria, azotemia, nephritis, renal failure and pyelonephritis [6-8, 10, 11]. Glomerulonephritis and acute interstitial nephritis due to typhoid are very uncommon [11-13].

Glomerulonephritis in typhoid is due to immune complex mediated glomerular damage and it can cause hematuria and/or proteinuria. Biopsy shows diffuse proliferative glomerulonephritis with deposits of IgM, complement C3 and Vi antigen in glomerular capillary wall [2, 4, 6, 10]. Renal failure in typhoid may be a part of septicemia or shock. Symptomless bacilluria may occur in 20-25% of typhoid fever. This is not a complication as this bacilluria is temporary and kidneys purely act as organs of excretion, without itself being the seat of typhoid lesions [4]. Typhoid nephritis in adults, as a complication of untreated typhoid carries a very high mortality of 20-30% [6, 7]. Hypocomplementemia is uncommon in typhoid nephritis in comparison to acute streptococcal glomerulonephritis (Figure 1). This could be due to host factors, although HLA haploid-type link has not been proven, for normal complement levels in typhoid glomerulonephritis [14]. In certain geographic locations typhoid nephritis needs to be considered as a possibility in patients with nephritis and concomitant fever [7, 14]. Other types of renal involvement in typhoid fever are: Ig A nephropathy [15] and post-vaccination nephropathy [16] following typhoid vaccination. Most patients with enteric glomerulonephritis present with acute renal failure, hypertensive encephalopathy or nephritic syndrome and prompt recovery on treating typhoid fever with IV ceftriaxone or other antibiotics [8]. Interestingly complications of typhoid fever like hepatitis and nephritis were more common in females in one particular study, implicating host's immune response to acute typhoid infection [17]. Jaundice due to typhoid hepatitis, which may be seen in upto 50% patients with typhoid [18], may precipitate the occurrence of typhoid glomerulonephritis [19]. As such, the typhoid hepatitis and typhoid nephritis occurring simultaneously in a

patient with typhoid is very rare [20], but may be more common in HIV positive patients with typhoid fever [21]. Very rarely acute renal failure in typhoid fever may occur due to intravascular hemolysis caused by typhoid fever [18]. Awareness of rare manifestations of this common infectious illness, early recognition and prompt treatment is instrumental in reducing morbidity and mortality.

## CONCLUSION

Typhoid nephritis is a rare complication of typhoid fever with significant morbidity and mortality. In endemic areas for typhoid fever, typhoid nephritis should be suspected in patients with nephritis and concomitant fever. In patients presenting with fever, diarrhea, hepatosplenomegaly, positive blood culture for typhoid fever and positive Widal test, typhoid nephritis and renal failure could be easily detected by urine examination and renal function tests. Early treatment of typhoid fever with intravenous antibiotics and fluid management prevents further progression of typhoid nephritis and impending dialysis.

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## Author Contributions

Yashodhara BM – Substantial contributions to conception and design, Acquisition of data, Drafting the article, Revising it critically for important intellectual content, Final approval of the version to be published

Uduman Ali Mohamed Yousuf – Substantial contributions to conception and design, Analysis and interpretation of data, Drafting the article, Final approval of the version to be published

## Guarantor

The corresponding author is the guarantor of submission.

## Conflict of Interest

Authors declare no conflict of interest.

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