Case Report

Journal of Medical - Clinical Research & Reviews

Typhoid Glomerulonephritis in a Child: A Rare Complication of Typhoid Fever

Mohammed Beshir* and Dulce Maria Garlobo Rosales

Department of Pediatrics, Jimma University, Jimma University Medical Center, Jimma, Ethiopia.

***Correspondence:**
Mohammed Beshir, Department of Pediatrics, Jimma University, Jimma University Medical Center, Jimma, Ethiopia, E-mail: Mohammedbeshir84@yahoo.com.

Received: 17 April 2018; **Accepted**: 09 May 2018

Citation: Mohammed Beshir and Dulce Maria Garlobo Rosales. Typhoid Glomerulonephritis in a Child: A Rare Complication of Typhoid Fever. J Med - Clin Res & Rev. 2018; 2(3): 1-3.

ABSTRACT

Typhoid fever with classical features presents no difficulty in recognition. However, when it presents atypically in the guise of one of its rare complications, diagnosis becomes difficult and appropriate treatment is delayed. Typhoid glomerulonephritis is one such rare complication. we present a 12-year-old boy who presented with high grade fever, abdominal symptoms with signs of glomerulonephritis (edema, oliguria, hypertension and hematuria). Salmonella typhi resistant to chloramphenicol, ampicillin and cotrimoxazole was isolated from a blood culture. He had a complete recovery of his renal abnormality following treatment for his typhoid fever.

Keywords

Typhoid fever, Acute glomerulonephritis, Edema, Hematuria, Oliguria.

Abbreviations

GN: Glomerulonephritis.

Introduction

It is estimated that more than 21.7 million typhoid cases and more than 200,000 deaths reported to occur worldwide annually, it continues to be a major health problem [1]. Amongst various renal complications that it is known to be associated with acute glomerulonephritis (GN) due to typhoid fever in children is rare. It was well documented by Bukaand Coovadia in as early as 1980 but very few cases have been reported thereafter, especially in a resource-limited setting like Ethiopia [2]. I therefore report this case of acute glomerulonephritis associated with typhoid fever.

Case Report

A 12-year old boy presents with a 7 days history of abdominal pain, fever, vomiting, loss of appetite & abdominal distension. He had received chloramphenicol, Amoxicillin & cotrimoxazole during the week prior to hospitalization at private clinics but not improved. There was no history of skin rashes, hemoptysis and recent increase in blood pressure. He denied any history of sore throat in the recent past. His past history and personnel history were unremarkable. On examination the patient was acute sick looking temperature (40°C), tachycardic and tachypneic. His blood pressure was 110/70mmHg (normal for his age) had a normal anthropometric measurement. On abdominal examination he had deep tenderness over RLQ, liver edge is palpable 4 cm below right costal margin & TLS is 8 cm, spleen tip is palpable. On GUS he had CVA tenderness. He had some palmar pallor but no peripheral edema, petechiae, purpura or rash. Examination of other organ system was normal. Initial investigations performed on 7th day of illness had revealed: - CBC-Hb 9.1g/dL; WBC 15,000 cells/mm^3 with 72% neutrophils, Platelets 183,000/cmm. Urinalysis showed: - proteins 2+, 10-16 Pus cells/HPF and no casts. Widal test were positive. Abdominal x ray revealed normal findings. Acute pyelonephritis was diagnosed and the patient was treated with parenteral ampicillin and gentamycin.

On the third day of hospitalization, the patient developed reduced urine output and passage of coke colored urine, BP-130/100mmHg (>95th centile for age) & edema of the face. The patient remained febrile during the three-day hospital stay. There was no clinical evidence of congestive cardiac failure. Rest of the physical examination were normal. Acute renal failure was considered & the patient was revaluated. The investigations performed at the time of hospitalization (i.e. 10th day of illness) demonstrated

CBC -Hb 9.0 g/dl, WBC 22,000 cells/mm^3 with 78% neutrophils, and Platelets 83,000/cmm. On peripheral smear no evidence of microangiopathic hemolytic anemia.

Urinalysis showed: -protein 3+, 40-50 RBCs/HPF, & 5-8 RBCs casts/HPF. Anti-Streptolysin O titer was negative.

Blood chemistry showed: -Cr (1.5 mg/dl), BUN (39 mg/dl), (Na-139, K- 4.1, Cl-97 mEq/L). Serum albumin was 3.2 g/dL. Patient's serum was negative for (HIV), HBV Virus, HCV Virus and for malaria. Patient's blood culture has grown S. typhi which was sensitive to cefotaxime, ceftriaxone and ciprofloxacin, and resistant to chloramphenicol, ampicillin and cotrimoxazole. X-ray of the chest was normal. His echocardiography showed no vegetations. Abdominal ultrasound revealed bilateral enlarged, swollen kidneys with normal echotexture and corticomedullary differentiation. Kidney biopsy, serum c3 level were not done because unavailability of the investigations.

The co-existing clinical presentation of high grade fever and prominent gastrointestinal symptoms with the development GN signs (edema, oliguria, hypertension, hematuria) with the isolation of *S. typhi* from the blood a diagnosis of typhoid fever associated with GN was made. Patient was managed with intravenous frusemide, IV Ceftriaxone, strict input and output of fluid, dietary modification with salt restriction, daily weighing and urinalysis. The clinical condition of the patient the edema, oliguria and blood pressure improved after the 3nd day of IV Lasix. The fever subsided after 5th day of IV ceftriaxone.

Antibiotics were continued for the total duration of 14 days. He was discharged home for follow up in the clinic after 17 days of hospitalization. Antihypertensive treatment was tapered and discontinued after 4 weeks. Follow up at 8 weeks demonstrated normal blood pressure, normal urinalysis & normal renal function.

Discussion

Reports dating back more than three decades have placed overall incidence of renal involvement in typhoid fever at 2-3%. However, in most cases mild proteinuria is the only manifestation [3]. Various other forms of renal involvement include hemolytic-uremic syndrome, pyelonephritis, nephrotic syndrome, cystitis, interstitial nephritis and acute renal failure. However, acute GN during typhoid fever is uncommon [4,5]. After the series of 15 children of typhoid GN reported by Buka and Coovadia in 1980 very few cases have been reported in the literature later on particularly in resource limited settings [2].

In the present case the co-existing clinical presentation of high grade fever (>10 days) and prominent gastrointestinal symptoms with the development of GN signs (edema, oliguria, hypertension, hematuria) and isolation of *S. typhi* from the blood a diagnosis of typhoid fever associated with GN was made [1]. Poststreptococcal glomerulonephritis (PS GN) is the leading cause of GN in Ethiopia. The clinical setting and lack of evidence of GAS infection of our patient helped to exclude PSGN from the diagnosis though serum c3 level and renal biopsy were not done.

Acute GN due to/associated with typhoid fever develops during the course of infection as early as the first week. In the present case evidence of GN was apparent in the second week of illness. There is no latent period between the infection and the manifestations of GN caused by/associated with typhoid fever and thus typhoid GN is not a post-infectious phenomenon. Almost all the cases of typhoid GN reported so far were actually during an active stage of the disease where Salmonella organisms were either isolated or patients had a positive serology and a continuous febrile state while signs of GN were noted [2,8]. In contrast, in acute post-streptococcal GN, there is a latent period between the streptococcal infection and signs and symptoms of GN. Of the 15 cases reported by Buka and Coovadia serum C3 levels were reported to be low in 13 and normal in 2 cases [2].

The mechanism of glomerular injury in typhoid GN is possibly immune complex mediated; but deposition of Vi antigen has been documented in only few cases. Mesangial deposition of IgA, IgG and C3 is common [5]. Histological examination of renal biopsy may be considered necessary for confirmation of GN and to rule out other conditions closely resembling GN especially like IgA nephropathy. However, in the present case the patient showed rapid resolution of edema, hematuria and oliguria. His hypertension also resolved completely and there was no persistent microscopic hematuria at 8 weeks.

Prognosis is generally good in acute GN due to/associated with typhoid fever. In majority of cases it resolves completely and does not lead to any long-term sequele [2]. The index patient had a complete recovery of his renal abnormality following treatment for his typhoid fever.

Conclusion

Typhoid fever with classical features presents no difficulty in recognition. However, when it presents atypically in the guise of one of its rare complications, diagnosis becomes difficult and appropriate treatment is delayed. In order to reduce the morbidity and mortality from the disease, a meticulous approach is necessary especially in a resource-limited setting where delays in obtaining antibiotic cultures has led to a treat-first-diagnose-later approach.

Author Contribution

Mohammed Beshir: management and writing of the manuscript.
Dulce María Garlobo Rosales: Manuscript editing.
Both authors admit and managed the patient, both authors read and approved the final manuscript.

Acknowledgment

Thanks for the patient's mother & father for giving this permission to publish the case report.

References

1. Background document The Diagnosis, Treatment and

Prevention of Typhoid Fever. The Department of Vaccines and Biologicals; 2003.
2. Buka I, Coovadia HM. Typhoid glomerulonephritis. Arch Dis Child. 1980; 55: 305e307.
3. Khosla SN. Changing patterns of typhoid: a reappraisal. Asian Med J. 1982; 25: 185e198.
4. Bhutta ZA. Enteric fever (typhoid fever). In: Kleigman, Stanton, St. Geme, Schor, Behrman, eds. Nelson Textbook of Pediatrics. 19th edn. Elsevier; 2011: 954e958.
5. Lesavre Philippe, Davison Alex M. Infection related glomerulonephritis. In: Davison Alex M, Stewart Cameron J, Ritz E, et al., eds. Oxford Textbook of Clinical Nephrology. 3rd edn. Oxford University Press; 2005: 601e624.
6. Khajehdehi P, Tastegar A, Kharazmi A. Immunological and clinical aspects of kidney disease in endemic typhoid fever in Iran. Q J Med 1984; 53:101 7.
7. Yashodhara BM, Yousuf UA. A case report of typhoid fever masquerading as acute glomerulonephritis. Int J Case Rep Images. 2011; 2:1 4.
8. Pancharoen C, Wongsawat J, Phancharoen S, et al. Typhoid glomerulonephritis in a child: a rare complication of typhoid fever. Southeast Asian J Trop Med Public Health. 2001; 32: 869-871.