

Renal profile among Sudanese Patients with Tuberculosis in Kassala State, Eastern Sudan: Cross-sectional study

¹⁻ Abbas E. Nogd-allh¹
²⁻ Ahmed T. Mohammed²
³⁻ Morad M. Ahmed³
⁴⁻ Taha Y. Fadul⁴

1,2,3, and4: Sudan international university department of clinical chemistry, Khartoum, Sudan

5,6- Husham O. Elzein -ORCID: 0000-0001-8517-0348.

5-Department of Medical Laboratory, Faculty of Applied Medical Sciences, Northern Border University, Arar, Saudi Arabia.

6-Faculty of medical laboratory science, University of Science and Technology, Khartoum, Sudan.

7-Amna.O.M. Elzein⁵ Corresponding Author- PhD

AlzaemAlazhari University- faculty of medical laboratory sciences, department of clinical chemistry, Khartoum, Sudan.

Abstract:

Introduction

Tuberculosis (TB) known as one of the most common health problems worldwide. *Mycobacterium tuberculosis* considered the main cause of TB that mainly affects the lungs and leading to pulmonary tuberculosis which associated with increased morbidity and mortality cases. The aim of this study is to assess the renal profile among tuberculosis patients in Kassala state, Eastern of Sudan.

Materials andMethods

This is a cross-section study enrolled 200 individuals, aged 15 to 60 years (100 as Tuberculosis and 100 as a control group). the study conducted at the Out-Patient clinic in Kassala Teaching Hospital during the period of April-July 2017. Serum Urea and creatinine measured by the spectrophotometric method while sodium (Na⁺) and potassium (K⁺) levels measured by using EasyLyte automated Analyzer (Medica Corporation, 5 Oak Park Drive Bedford, MA 01730, USA). Also, the study calculated the GFR by applying the formula of Cockcroft - Gault formula (1973).

Results

The main findings of this study were a significant increase in the mean levels of Urea (69.33 ± 26.44), Creatinine ($1.52 (\pm 0.68)$), and GFR (47.03 ± 17.64) when compared to control group (20.04 ± 4.11), 0.61 ± 0.24 , and 87.38 ± 5.44 , respectively, $P.V < 0.05$). The study also revealed that the mean values of patient's Na⁺ (131.64 ± 4.59) and K⁺ (3.00 ± 0.51) were significantly decreased when compared to control group ($140.77 (\pm 2.58)$ and $4.42 (\pm 0.23)$, respectively, $P.V = 0.00$).

Conclusions

This study confirmed that patients with TB are revealed abnormal renal profile results. These results may highlight clearly why patients with renal disease are more likely to present with active TB infection.

Keywords: Creatinine; Electrolytes; Sudan; Tuberculosis; Urea.

Date of Submission: 14-08-2022

Date of Acceptance: 27-08-2022

I. Introduction

Tuberculosis (TB) is one of the most public infectious health problems worldwide [1,2]. *Mycobacterium tuberculosis* considered the principal cause of TB that predominantly affects the lungs and leading to pulmonary tuberculosis [3]. TB is well-known as one of the main causes of the high numbers of morbidity and mortality cases, especially within low-income regions. In 2014 around 1.5 million TB deaths occurred, nine of ten of the death's cases take place in Sub Saharan Africa, and Southeast Asia [4]. It has been found that the rate of TB in Africa was reached 263–341/100,000 population [5]. WHO stated that in Sudan exactly in the year 2013 TB was estimated 108 cases per 100,000 population [6]. Renal disease, in particular, chronic kidney disease has supposed to be a typical relevant factor for TB progress; due to immunodeficiency, and malnutrition status of the patients [7-11]. Previous studies reported that the incidence of TB in renal patients

is high [12]. The mechanisms by which TB causes renal failure have occurred in some circumstances such as; during the disease progression, wide areas of papillary necrosis can cause cavities formation that can destroy the renal parenchyma, which may lead to endarteritis with dystrophic calcification and may cause secondary renal amyloidosis, and both of these outcomes can cause renal defect [13]. The infection can cause some early changes including vascular insufficiency in renal papillae, which leading to papillary necrosis [14,15]. The bacilli organisms can also migrate to the cortico-medullary junction and build cortical granulomas of ≤ 3 mm in size. These granulomas remain stable for long time, and during reactivation, the organisms invade the renal medulla and cause papillitis [16,17]. The dissemination of infection to the renal pelvis can cause tuberculous pyelonephritis, which can progress to pyonephrosis. The infection generally dispenses from the ureter to the bladder, producing lesions associated with fibrosis [18]. The current study aimed to assess the changes in renal profile among Sudanese patients with tuberculosis in Kassala state, Eastern of Sudan.

II. Materials and Methods

Ethical issue: This study was approved by the research ethics board at the faculty of medical laboratory science, Alzaiem Alazhari University, Khartoum, Sudan. All the participants signed the informed consent; data were collected on their clinical history, socio-demographic by using a structured questionnaire.

Design and subjects:

This is a cross-sectional analytical study which is conducted at the Out-Patient clinic in Kassala Teaching Hospital during the period of April-July 2017. Kassala hospital is a tertiary hospital concerned with the referred patients. The study groups included 200 participants (100 patients with TB and 100 as a healthy control group). The participants age ranges from 15 to 60 years. Subjects with a history of hypertension, diabetes mellitus, and HIV are excluded from the study. To get a proper serum sample, 5 ml of venous blood were obtained under all septic conditions from all the participants in plain blood containers, and the sample was allowed to clot, then centrifuged at (3000 rpm) for 5 minutes. Serum was extracted and stored at (-20 °C) until the assay time of urea, creatinine by spectrophotometer and sodium, potassium by using EasyLyte automated Analyzer (Medica Corporation, 5 Oak Park Drive Bedford, MA 01730, USA). The Glomerular Filtration Rate (**GFR**): was calculate by using Cockcroft _ Gault formula (1973):

$$GFR = \frac{(140 - \text{Age}) \times \text{weight (Kg)}}{\text{Serum Creatinine mmol/L}}$$

Statistical analysis:

Data were entered and analyzed by a programmed computer system using a statistical package for social science (SPSS, version 11.5 for Windows). Data were expressed as mean \pm S.D, and the results were shown in tables and figures. The *t*-test was used to compare the two groups and the ANOVA test was used to compare the different variables. A P value of <0.05 was considered significant.

III. Results

This study includes 200 participants, 100 as tuberculosis patients, and 100 as a healthy control group. The study showed a significant finding between case and control groups, regarding age, and gender while there was a significant result concerning the residence as shown in **Table 1**. The description statuses of the TB patients are illustrated in **Fig.1**. The mean and standard deviation for blood Urea and Creatinine for the patient's group were statically significant ($P.V= 0.00$) when compared with the control group as given in **Table 2**. In electrolyte tests, the mean values of serum sodium (Na⁺) and serum potassium (K⁺) were statically significant ($P.V= 0.00$) **Table 2**. Also, in GFR the mean value for the patient group was statically significant ($P.V = 0.00$) as shown in **Table 2**. The study also determined the correlation between age and renal profile in TB group, and revealed that there were significant, positive correlations between age with urea ($r= 0.456$, $P.V= 0.00$), Creatinine ($r= 0.526$, $P.V= 0.00$) and GFR ($r = -0.160$, $P.V= 0.023$). While, there was insignificant, negative correlation of age with Na⁺ ($r = -0.139$, $P.V =0.169$), and K⁺($r = -0.014$, $P.V= 0.893$), respectively. According to the gender and residence, there were insignificant differences in urea, creatinine, Na⁺, K⁺, and GFR levels among the males, females, urban, and rural in the patient's group ($P.V> 0.05$) **Table 3**. In a comparison of the renal profile according to the tuberculosis patient's status, there were significant differences in urea and creatinine levels $P.V = 0.00$ as given in **Table 4**. On the other hand, the levels of Na⁺, K⁺, and GFR were not significantly affected ($P.V> 0.05$) **Table 4**.

Table 1: The demographic information of the study group (n= 200).

Age/Gender/Residence	Tuberculosis (mean ± SD)	Control (mean ± SD)	P. value
• Age (Years)	49.2 (±10.13)	47.9 (±11.38)	0.608
• Gender (%)			0.336
Male	46 (46%)	50 (50%)	
Female	54 (54%)	50 (50%)	
Total	100 (100%)	100 (100%)	
• Residence (%)			0.001
Urban	67 (67%)	57 (57%)	
Rural	33 (33%)	43 (43%)	
Total	100 (100%)	100 (100%)	

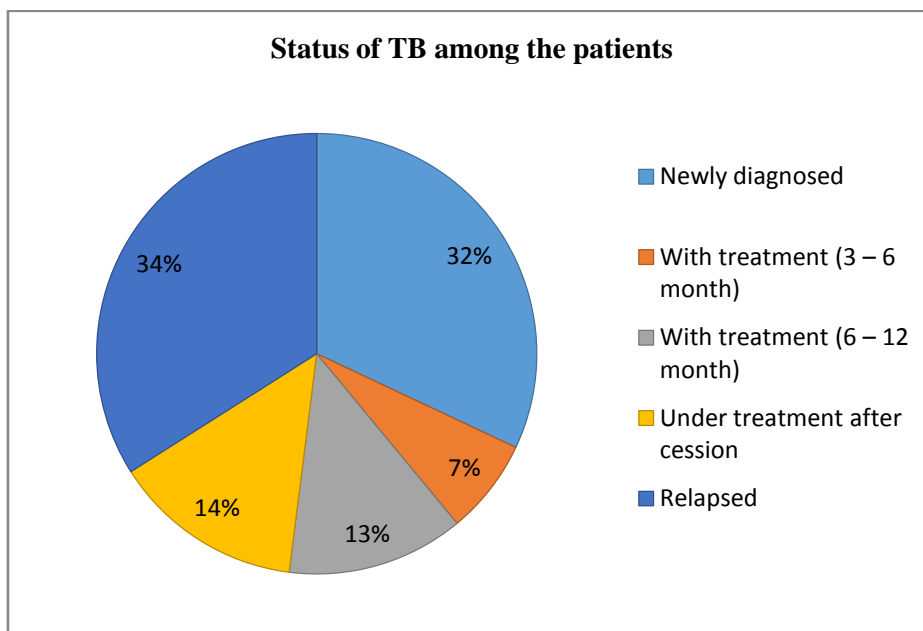


Fig1: TB status among the patients (n=100)

Table 2: Shows the Mean ± SD of Urea, Creatinine, Sodium, Potassium and GFR among tuberculosis patients and healthy control groups (n= 200)

Renal profile	Tuberculosis Pts	Control	P. value
Urea (mg/dl)	69.33 (± 26.44)	20.04 (± 4.11)	0.000
Creatinine (mg/dl)	1.52 (± 0.68)	0.61 (± 0.24)	0.000
Sodium (Na ⁺) (mmol/L)	131.64 (± 4.59)	140.77 (± 2.58)	0.000
Potassium (K ⁺) (mmol/L)	3.00 (± 0.51)	4.42 (±0.23)	0.000
GFR (ml/min)	47.03 (± 17.64)	87.38 (± 5.44)	0.000

Table 3: Shows the comparison of renal profile according to the gender and residence in TB group (n= 100)

	Urea (mg/dl)	Creatinine (mg/dl)	Sodium (Na ⁺) (mmol/L)	Potassium (K ⁺) (mmol/L)	GFR (ml/min)
• Male	71.19 ± (24.55)	1.64 ± (0.58)	131.32 ± (4.68)	3.06 ± (0.49)	67.36 (± 24.75)
• Female	67.74 ± (28.08)	1.14 ± (0.75)	131.90 ± (4.54)	2.95 ± (0.51)	67.06 (±023.50)
P. value	0.518	0.101	0.532	0.253	0.929
• Urban	65.74 ± (26.51)	1.44 ± (0.66)	131.09 ± (4.67)	2.98 ± (0.51)	63.84 (± 23.51)
• Rural	76.60 (± 25.13)	1.66 ± (0.71)	131.90 ± (4.44)	3.05 ± (0.53)	62.32 (±24.19)
P. value	0.053	0.138	0.404	0.508	0.744

Table 4: The comparison of renal profile according to the TB status in TB group (n= 100)

Renal profile	Newly diagnose	Under treatment			Relapsed	P. value
		3 - 6 months	6 - 12 months	After cession		
Urea (mg/dl)	44.75 (±17.17)	59.57 (± 15.96)	69.15 (±14.33)	87.92 (±9.04)	86.88 (±24.44)	0.00
Creatinine (mg/dl)	1.01 (±0.42)	0.95 (±0.25)	1.62 (±0.76)	1.97 (±0.50)	1.89 (±0.68)	0.00
Sodium (Na ⁺) (mmol/L)	132.12 (±3.50)	132.85 (±5.27)	131.23 (±5.52)	133.00 (±5.67)	130.52 (±4.49)	0.389
Potassium (K ⁺) (mmol/L)	3.01 (±0.48)	2.80 (±0.57)	2.73 (±0.51)	3.05 (±0.54)	3.12 (±0.49)	0.139
GFR (ml/min)	51.45 (± 15.58)	52.28 (± 16.64)	44.58 (± 22.87)	44.33 (± 17.65)	43.83 (±17.36)	0.368

IV. Discussion

To our knowledge, this is the first study carried out in Kassala, Eastern Sudan, which examines the renal profile in patients with TB. The main findings of the current study were, the patient's serum Urea was 69.33 ± 26.44 , and Creatinine was 1.52 ± 0.68 which are significantly increased when compared to control group (20.04 ± 4.11 and 0.61 ± 0.24), respectively, $P.V < 0.05$. The explanation this elevation may be due to patients treatment with Rifampicin drugs, as per Chogtu B et al study^[19], which revealed that rifampicin induced renal toxicity and induced acute renal injury and then cause of these elevations in a majority of cases besides that the regular exposure to rifampicin may stimulate the immune system to forms drug antibody complexes which lead to cell damage and the immune complexes can cause glomerular endotheliosis that may lead to tubular injury and a decrease in renal function as reported by Cheesbrough M et al study^[20]. The current study revealed that the mean values of patient's serum Na⁺ (131.64 ± 4.59) and K⁺ (3.00 ± 0.51) were significantly decreased when compared to control group (140.77 ± 2.58) and 4.42 ± 0.23), respectively, $P.V = 0.00$). This observed decrease in Na⁺ and K⁺ levels in the patients might be attributed to dehydration due to muscle wasting and fever. These findings are in agreement with Figueiredo AA, et al study^[21]. The current study showed that there were significant correlations of age with urea ($r = 0.456$, $P.V = 0.00$), creatinine ($r = 0.526$, $P.V = 0.00$) and GFR ($r = -0.160$, $P.V = 0.023$). Also, there were insignificant correlations of age with Na⁺ and K⁺, $P.V > 0.05$). This study showed that patients under treatment; after cession and relapsed have high urea and creatinine levels, while the levels of Na⁺ and K⁺ are not significantly affected ($P.V > 0.05$). The present study has some limitations that should be considered. First, this study only described a few demographic variables and might not represent all populations, so we need to cover further variables and increases the sample size to obtain more recommended results.

V. Conclusion:

We found steady evidence demonstrating that patients with TB exhibited abnormal renal profile results. These results may explain clearly why patients with renal disease are more likely to present with active TB infection.

Acknowledgment: The authors would like to thank all the patients who were included in this study, and the medical staff of Kassala Teaching Hospital, Kassala State, Sudan, for their collaboration.

Funding: None of the authors received fund.

Conflicts of Interest: The authors declare that they have no conflict of interest.

References

- [1]. Fader T, Parks J, Khan NU, Manning R, Stokes S. Extrapulmonary tuberculosis in Kabul, Afghanistan: a hospital-based retrospective review. *Int J Infect Dis.* 2010;14:e102–10. [PubMed] [Google Scholar]
- [2]. Tiemersma EW, van der Werf MJ, Borgdorff MW. Natural history of tuberculosis: duration and fatality of untreated pulmonary tuberculosis in HIV negative patients: a systematic review. *PLoSOne.* 2011;6:e17601. [PMC free article] [PubMed] [Google Scholar]
- [3]. T, Minias A, Van Ingen J, Rastogi N, Brzostek A, Żaczek A, Dziadek J. Methodological and clinical aspects of the molecular epidemiology of Mycobacterium tuberculosis and other mycobacteria. *Clin Microbiol Rev.* 2016 Apr 1;29(2):239-90.
- [4]. World-Health-Organisation Global tuberculosis report 2015 (20th ed.), WHO press, Geneva (2015), pp. 5-86 Google Scholar
- [5]. WHO. Global Tuberculosis Report 2015. Geneva: World Health Organization; 2015
- [6]. Sudan WH. Country statistics summary. WHO Regional Office for the Eastern Mediterranean. Cairo, Egypt: World Health Organization. 2013.
- [7]. Romanowski K, Clark EG, Levin A, Cook VJ, Johnston JC. Tuberculosis and chronic kidney disease: an emerging global syndemic. *Kidney Int.* 2016;90:34–40.
- [8]. Al-Efraij K, Mota L, Lunny C, Schachter M, Cook V, Johnston J. Risk of active tuberculosis in chronic kidney disease: a systematic review and metaanalysis. *Int J Tuberc Lung Dis.* 2015;19:1493–9.
- [9]. Kumari P, Meena LS. Factors affecting susceptibility to Mycobacterium tuberculosis: a close view of immunological defence mechanism. *Appl Biochem Biotechnol.* 2014;174:2663–73.

- [10]. Hussein MM, Mooij JM, Roujouleh H. Tuberculosis and chronic renal disease. *Semin Dial.* 2003;16:38–44.
- [11]. Milburn H, Ashman N, Davies P. Guidelines for the prevention and management of Mycobacterium tuberculosis infection and disease in adult patients with chronic kidney disease. *Thorax* 2010;65:559–70. [PubMed] [Google Scholar]
- [12]. Ostermann M, Palchaudhuri P, Riding A, Begum P, Milburn HJ. Incidence of tuberculosis is high in chronic kidney disease patients in South East England and drug resistance common. *Ren Fail.* 2016 Feb 7;38(2):256-61.
- [13]. Rose BD. Renal disease in tuberculosis, 2007. Available from: www.uptodate.com .
- [14]. Chun HM, Hale B. Renal tuberculosis: three cases and a review of the literature. *Infect Dis Clin Pract.* 2004 Mar 1;12(2):117-22.
- [15]. Daher Ede F, Silva Júnior GB, Damasceno RT, Santos GM, Corsino GA, Silva SL, et al. End-stage renal disease due to delayed diagnosis of renal tuberculosis: A fatal case report. *Braz J Infect Dis.* 2007;11:169–71. [PubMed] [Google Scholar]
- [16]. Jung YY, Kim JK, Cho KS. Genitourinary tuberculosis: Comprehensive cross-sectional imaging. *AJR Am J Roentgenol.* 2005;184:143–50. [PubMed] [Google Scholar]
- [17]. Merchant S, Bharati A, Merchant N. Tuberculosis of the genitourinary system-Urinary tract tuberculosis: Renal tuberculosis-Part I. *Indian J Radiol Imaging.* 2013 Jan;23(1):46.
- [18]. Figueiredo AA, Lucon AM, Júnior RF, Ikejiri DS, Nahas WC, Srougi M. Urogenital tuberculosis in immunocompromised patients. *Int Urol Nephrol.* 2009 Jun 1;41(2):327.
- [19]. Chogtu B, Surendra VU, Magazine R, Acharya PR, Yerrapragada DB. Rifampicin-induced concomitant renal injury and hepatitis. *J Clin Diagn Res.* 2016 Sep;10(9):OD18.
- [20]. Cheesbrough M. District laboratory practice in tropical countries, part 2. Cambridge university press; 2006 Mar 2.
- [21]. Figueiredo AA, Lucon AM, Arvellos AN, Ramos CO, Toledo AC, Falci Jr R, et al. A better understanding of urogenital tuberculosis pathophysiology based on radiological findings. *Eur J Radiol.* 2010 Nov 1;76(2):246-57.

Dr: Amna.O.M. Elzein, et.al. "Renal profile among Sudanese Patients with Tuberculosis in Kassala State, Eastern Sudan: Cross-sectional study." *IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)*, 21(08), 2022, pp. 06-10