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Acute Kidney Injury in Patients Receiving Anti-Tuberculosis Treatment

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Abstract

Objectives: Although life-saving, anti-tuberculosis therapy (ATT) carries the risk of acute kidney injury (AKI), potentially complicating its management. This study highlights the frequency of AKI in patients taking ATT.
Methods: 102 subjects starting on ATT were followed for 2 months. Presence of AKI was documented with monitoring of kidney functions.
Results: AKI was observed among 14.7% patients, predominantly affecting middle-aged males.
Conclusion: Early recognition of AKI by monitoring for kidney function is crucial for reducing the risk of long-term sequelae of kidney failure among patients taking ATT.

Keywords: AKI, tuberculosis, nephrotoxicity, rifampicin, anti-Tuberculous.

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Introduction

Tuberculosis (TB) remains a major global health issue, with millions affected annually.[1-3] Standard Anti-tuberculosis therapy (ATT) includes isoniazid, rifampicin, pyrazinamide, and ethambutol. Among these, rifampicin has been implicated as a leading cause of drug-induced nephrotoxicity, specifically acute kidney injury (AKI).[4-6] Although infrequent, AKI associated with ATT can range from mild impairment to severe renal failure requiring dialysis.

Drug-induced AKI typically manifests as acute tubular necrosis or acute interstitial nephritis.[7] Studies from Asia and Europe have shown variable frequencies of AKI during ATT, but local data in our population remain scarce. This study aimed to document the frequency of AKI in patients who initiated ATT.

Methods

This descriptive case series was conducted at the Nephrology Department in collaboration with the Pulmonology Department, Mayo Hospital, Lahore, Pakistan over a period of 6 months. Following the approval of the research synopsis by the research review committee and approval of the synopsis. The primary objective of this study was to determine the frequency of AKI in patients initiating ATT. We

hypothesized that the frequency of AKI would be significantly higher than expected in the study population.

A sample size of 102 patients was calculated using a 95% confidence level, 5% margin of error, and an expected AKI frequency of 7.1%. Non-probability consecutive sampling was used for patient enrolment.

Inclusion Criteria

- Patients aged 14 – 65 years with a confirmed diagnosis of pulmonary tuberculosis, which was defined as Clinical history of cough, Chest X-ray showing consolidation, Sputum was positive for Acid-Fast Bacilli (AFB).
- Patients initiating ATT.
- Patients with normal baseline renal function tests (RFTs).

Exclusion Criteria

- Patients with abnormal kidney function prior to initiating ATT.
- Patients concurrently taking nephrotoxic drugs (e.g., NSAIDs).
- Patients with infections known to cause renal injury.
- Patients presenting with hypotension.
- Patients with obstructive uropathy (e.g., urolithiasis or prostatic enlargement).
- Non-compliant patients regarding treatment and follow-up.

AKI was defined by KDIGO criteria as any one of the following during he first 2 months of ATT:
1. Increase in serum creatinine levels by ≥0.3 mg/dl within 48 h.
2. Increase in serum creatinine to ≥1.5 times baseline, presumed to have occurred within the previous 7 days.
3. The reduction in urine output was 0.5 ml/kg/h for 6 consecutive hours.

Ethical review certificate (issued 03/06/2021) was obtained before patient recruitment. Thus, a total of 102 adult patients diagnosed with pulmonary TB and normal baseline kidney function were enrolled., Participants underwent regular monitoring (serum creatinine and electrolytes) at baseline and fortnightly for two months.

Data were analysed using SPSS 22 (IBM Corp., Armond, New York, USA). Age, sex, and frequency of AKI were described, and chi-square test was used to assess associations (significance at $p \leq 0.05$).

Results

The mean age was 40.77 ± 12.45 years, with males comprising n=69 (67.6%) of the cohort. The occurrence of AKI and its relationship with age and sex are shown in Table 1. Middle-aged men appeared to be more susceptible to AKI.

AKI developed in 14.7% of patients, with the majority (12.7%) presenting on the 45th day of treatment (Figure 1). Hypernatremia was observed in 9.8% of patients, clustering around the 45th day, and

hypokalemia was significantly more prevalent in patients with AKI, although the total number was too small.

Table 1: Data stratification by age and gender among 102 patients taking anti tuberculous treatment and presence or absence of acute kidney injury

Variable	AKI Absent (n)	AKI Present (n)	Total (n)	Chi-Square	P-value
Gender					
Male	57	12	69	**1.226**	**0.268**
Female	30	3	33		
Age Groups					
16 – 30 yrs	22	4	26		
31 – 45 yrs	33	5	38	**0.1177**	**0.95**
46 – 65 yrs	32	6	38		
Total (All)	**87**	**15**	**102**		

Table 2: Frequency distribution of Electrolytes among 102 patients taking anti tuberculous treatment and presence or absence of acute kidney injury.

Electrolyte Disturbance	Total (n=78)	AKI Present (n=11)	AKI Absent (n=67)	P-Value
Hypernatremia	5 (6.4%)	2 (18.2%)	3 (4.5%)	0.143
Hyponatremia	9 (11.5%)	1 (9.1%)	8 (11.9%)	1.000
Hyperkalemia	3 (3.8%)	1 (9.1%)	2 (3.0%)	0.387
Hypokalemia	8 (10.3%)	4 (36.4%)	4 (6.0%)	**0.007**

Discussion

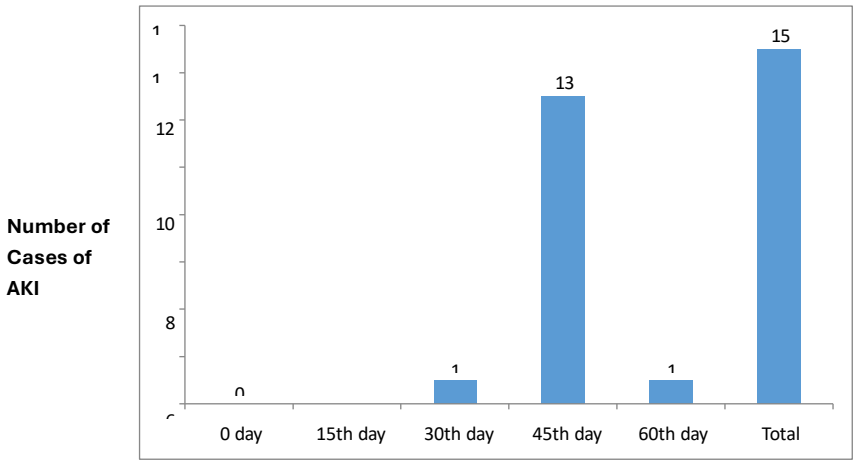
This study investigated the occurrence of AKI in patients undergoing ATT. Using the KDIGO criteria, this study found a 14.7% incidence of AKI, which was higher than expected. Consistent with earlier findings, AKI was more frequent in males (12 out of 15 cases) and predominantly developed around the 45th day of treatment, suggesting a possible acute phase reaction.[6]

Histological data from other studies have implicated acute interstitial nephritis and acute tubular necrosis as ≥common pathological findings in rifampicin-induced AKI.[4,5,9,10] However, kidney biopsies were not performed in our study.

Most prior research indicates that over 80% of patients recover from AKI within 120 days, although our study lacked follow-up data to assess recovery rates.[9] Similarly, prognostic factors such as anuria duration or leukocytosis were not analyzed here. A key limitation of this study was its small sample size (102 patients), single-center design, and short follow-up period, which limited the generalizability of the results. Additionally, the descriptive case series design lacked a control group and outcome follow-up period. Nevertheless, these findings underscore the importance of close monitoring during

the first two months of ATT and emphasize the need for proper regimen adjustment to prevent renal injury in high-risk patients.

Figure 1: Number of cases of acute kidney injury as they were reported on days during the follow up of 102 patients on anti-tuberculous treatment, maximum observed at 45th day.



Conclusion

AKI in patients receiving ATT is not uncommon and requires vigilant renal function monitoring, particularly in the sixth week of therapy. Awareness of this complication enables timely intervention, potentially preventing permanent renal damage

References

1. World Health Organization. Global Tuberculosis Report 2024. https://www.who.int/publications/m/item/top-findings-and-messages-in-the-2024-report?utm_source=chatgpt.com
2. Schwalb A, Samad Z, Yaqoob A, Fatima R, Houben RMGJ. Subnational tuberculosis burden estimation for Pakistan. PLOS Glob Public Health. 2024 Sep 23;4(9):e0003653.
3. WHO. *Treatment of Tuberculosis: Guidelines 4th Edition. https://iris.who.int/handle/ 10665/44165
4. 4.Covic A, Goldsmith DJ, Segall L. Rifampicin-induced acute renal failure: a series of 60 patients. Nephrol Dial Transpl. 1998;14(4):924 – 9.
5. Beebe A, Seaworth B, Patil N. Rifampicin-induced nephrotoxicity in a tuberculosis patient. J Clin Tuberc Other Mycobact Dis. 2015 Oct 24;1:13-15.
6. Chang, CH., Chen, YF., Wu, VC.*et al.*Acute kidney injury due to anti-tuberculosis drugs: a five-year experience in an aging population.*BMC Infect Dis*14, 23 (2014).
7. Perazella MA, Rosner MH. Drug-Induced Acute Kidney Injury. Clin J Am Soc Nephrol. 2022 Aug;17(8):1220-1233.
8. Kidney Disease: Improving Global Outcomes (KDIGO). *Clinical Practice Guideline for Acute Kidney Injury*. 2012. https://kdigo.org/guidelines/acute-kidney-injury/](https://kdigo.org/guidelines/acute-kidney-injury/)
9. De Vriese AS, Robbrecht DL, Vanholder RC, Vogelaers DP, Lameire NH. Rifampicin-associated acute renal failure: pathophysiologic, immunologic, and clinical features. Am J Kidney Dis. 1998 Jan;31(1):108-15.
10. Sakashita K, Murata K, Takahashi Y, Yamamoto M, Oohashi K, Sato Y, Kitazono M, Wada A, Takamori M. A Case Series of Acute Kidney Injury During Anti-tuberculosis Treatment. Intern Med. 2019 Feb 15;58(4):521-527.