Original Article

Evaluate The Effect Of Tuberculosis And Pneumonia On Kidney

Rimsha Naveed[1] Usama Attique[2] Dr. Abid Ali[3]

[1] Doctor of Medical Laboratory Science, University of Lahore Chenab Campus, Gujarat.
[2] Doctor of Medical Laboratory Science, University of Lahore Chenab Campus, Gujarat.
[3] Head of Department of Doctor of Medical Laboratory Sciences, Assistant professor in Allied Health Sciences, University of Chenab, Gujarat.
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Abstract

BACKGROUND: Tuberculosis (TB) caused by the bacterial pathogen Mycobacterium tuberculosis, remains one of the deadliest infectious diseases with over a billion deaths over the past 200 years. M. tuberculosis is an intracellular pathogen transmitted by inhalation of aerosolized droplets containing bacteria. Pneumonia is a common acute respiratory infection that affects the alveoli and distal bronchial tree of the lungs. Lower respiratory tract infection was the second most common cause of death. The most common symptoms of pneumonia are cough, shortness of breath, chest pain, expectoration, and fatigue.

Methodology: A cross sectional study design was used. For this study a total 200 patients were selected and drawn their sample by using sterile condition. Out of 200 samples 100 was pneumonia patient samples and 100 was Tuberculosis (TB) patients. Renal Profile test Urea and Creatinine performed to evaluate the current condition of kidneys. Test was performed on state-of-the-art instrument after confirmation of Quality Control (QC) status of the instrument

Results: Data analysis shows that a minimum range of urea in pneumonia patients which was 10 mg/dl and maximum was 326mg/dl with mean of 68.18 ± 52.59 STD. and creatinine was minimum 0.32mg/dl and Maximum was 7.9mg/dl with 2.1907 ± 9.14 STD. In Tuberculosis (TB) patient's data shows that Urea minimum was 13.0mg/dl and maximum was 310.0mg/dl with mean value of 55.58 ± 52.26 and creatinine minimum values was 0.40mg/dl and maximum value was 7.9mg/dl with mean 1.43± 1.40 STD. According to this analysis there was a significant difference from normal values, mean values of urea a creatinine in both pneumonia and TB patients indicates that both diseases affect the renal conditions and can lead to severe state.

Conclusion(s): The risk of kidney disorder is positivity correlated with the patients who were diagnosed with TB and Pneumonia disorders. Renal function determined by performing urea and creatinine tests. Results of both parameters in TB and Pneumonia disease were clearly defined that these diseases can lead to renal end stage disorder.

Keywords: Tuberculosis, Chronic Kidney Disease, Estimated Glomerular Filtration Rate, Creatinine.

INTRODUCTION

Tuberculosis (TB), caused by the bacterial pathogen Mycobacterium tuberculosis, is one of the deadliest infections, killing more than 1 billion people over the past 200 years [1]. There were an estimated 12 million (11-13 million) tuberculosis cases in 2012, equivalent to about 169 per 100,000 populations. 1 million tuberculosis cases were reported by the National Tuberculosis Program (NTP). The ratio of reported cases to the estimated number of new and recurrent tuberculosis cases that are thought to have occurred in a given year is a problematic indicator in tuberculosis epidemiology, but very useful regarding the `diagnostic capacity' with which tuberculosis can provide control programs[2, 3]. The most common tuberculosis pathogen, Mycobacterium tuberculosis, is an immobile, aerobic rod-shaped bacterium[4]. The mycobacterial cell envelope consists of four main layers. (i) the plasma membrane or inner membrane, (ii) the peptidoglycan-arabinogalactan complex, (iii) the asymmetric outer membrane or fascia covalently linked to AGP via mycolic acids, and (iv) the outermost shell[5].Mycobacterium tuberculosis is spread almost exclusively from person to person by aerosolized particles. Infectious droplet sizes from Mtb-infected patients range from 0.65 (small) to 7.0 μm[6]. Mycobacterium tuberculosis is an intracellular pathogen that is transmitted

by inhalation of aerosol droplets containing the bacteria. Small Mtb aerosol particles are expected to pass through the nasopharynx or tracheobronchial region and deposit in the distal airways, while larger particles may become trapped in the upper respiratory tract or oropharynx, leading to tuberculosis (TB) [7] . Transmission occurs almost exclusively by droplet transmission. Whether an infection is present or not basically depends on frequency of contact with people with infectious pulmonary tuberculosis [8]. Patients with active pulmonary or laryngeal tuberculosis produce droplet nuclei containing Mycobacterium tuberculosis through coughing, singing, screaming, sneezing, or other respiratory movements that shear airway secretions from the airways. Coughing is the most efficient way to generate infectious aerosols [9]. After inhalation, extracellular and intracellular bacterial growth occurs preferentially in the upper layers of the well-ventilated lung, especially alveolar macrophages. Three to four weeks after infection, an intracellular proliferation of the bacterium. However, the risk of reactivation is also increased by other immunosuppressive conditions, Diabetes mellitus, end-stage renal disease [10]. The most common diagnostic test is a microscopic scan for acid-fast bacteria on sputum swabs [11]. The prevalence of tuberculosis in dialysis patients is closely related to and many times higher than that in the general population. The main symptoms of tuberculosis, anorexia, weight loss, low-grade fever, and general weakness, are also common in patients with chronic renal failure. It has been reported that extra pulmonary tuberculosis occurs more frequently in dialysis patients than pulmonary tuberculosis and may play an important role in delaying the diagnosis of tuberculosis in dialysis patients [12].

Host resistance to infection is primarily mediated by cell-mediated immunity, which is deficient in CKD patients. Therefore, such patients have a higher incidence of infections, including tuberculosis. Impaired cell-mediated immunity suppresses lymphocyte mutagenic responses. Protein malnutrition, zinc and pyridoxine deficiencies, and defects in leukocyte function after contact with dialysis membranes increase the susceptibility to tuberculosis in dialysis patients[13]. The linkage between CKD and active TB by investigating a large-scale predialysis CKD cohort including people with CKD confirmed with sequential laboratory examinations and comparing their TB risks [14]. It is well established that dialysis-dependent patients or kidney transplant recipients have an elevated risk for TB [15] . . It was suggested that TB risk may also be higher in those people with mild to moderate CKD. However, large-scale evidence regarding TB risk in people with predialysis CKD has rarely been reported [16]. The presence of predialysis CKD was determined when evidence of CKD (i.e., the presence of dipstick albuminuria or eGFR<60 ml/min per 1.73 m2 calculated. To stratify the predialysis CKD stages, we defined four subgroups with the eGFR and albuminuria values from the first included health examination. CKD stage 1 (eGFR≥90 ml/min per 1.73 m2) and stage 2 (eGFR≥60 and <90 ml/min per 1.73 m2) included individuals who had successive albuminuria but had no reduced eGFR at baseline. Health examinees with CKD stage 3 (eGFR≥30 and <60 ml/min per 1.73 m2) and stage 4 or 5 without dialysis (eGFR<30 ml/min per 1.73 m2) were also identified[17] .

The word pneumonia comes from the ancient Greek word pneumon, which means lung, hence the term pneumonia. Medically, it is inflammation of one or both lung parenchyma, more commonly, but not always, caused by infection. Many causes of pneumonia include bacteria, viruses, and fungi [18]. The epidemiology of pneumonia is constantly changing with molecular diagnostic tests, development of antimicrobial therapies, and implementation of preventive measures [19]. In 2013, the Global Burden of Disease Study based on data from 188 countries around the world, reported that lower respiratory tract infection was the second most common cause of death[20].

Pneumonia is a common acute respiratory infection that affects the alveoli and distal bronchial tree of the lungs. It is broadly divided into hospital-acquired pneumonia, which includes community-acquired pneumonia and ventilator-associated pneumonia. Aspiration pneumonia accounts for 5-15% of all cases of CAP. However, its prevalence in HAP patients is unknown[21]. Community-acquired pneumonia can be caused by an extensive list of pathogens, including bacteria, viruses, fungi and parasites. Bacteria have classically been divided into two categories based on etiology: 'typical' and 'atypical' organisms. Typical organisms can be cultured on standard media and seen on Gram stains, but 'atypical' organisms do not have such properties [22]. The most common symptoms of pneumonia are cough, shortness of breath, chest pain, expectoration, and fatigue . Most cases of bacterial pneumonia are caused by microorganisms that migrate from the nasopharynx to the lower respiratory tract [23]. The lower respiratory tract is not sterile and is constantly exposed to environmental pathogens. Bacterial pneumonia is caused by the entry and spread of the above bacteria into the lung parenchyma at the alveolar level. On the other hand, the body's inflammatory response causes the clinical syndrome of pneumonia. Several host defense mechanisms work in concert in the lung to prevent this microbial growth. For example, mechanical defense mechanisms (such as nostril hairs, nasopharyngeal and oropharyngeal mucus) and chemical defense mechanisms (such as surfactants - proteins produced by alveolar epithelial cells such as protein A, D has the unique property of opsonizing bacteria). Another component of the lung defense system consists of immune cells such as alveolar macrophages. These cells engulf and kill proliferating bacteria, but when the bacteria overcome the capacity of host defenses, they begin

to multiply. This inflammatory response is the major cause of the clinical manifestations of bacterial pneumonia. Cytokines are released in response to inflammatory responses and cause systemic symptoms. For example, IL-1 (interleukin-1) and TNF (tumor necrosis factor) cause fever. Colony-stimulating factors such as chemokine-like IL-8 (interleukin-8) and G-CSF (granulocyte colony-stimulating factor) promote neutrophil chemo taxis and maturation, leading to leukocytosis in the serum laboratory and purulent discharge. These cytokines are responsible for leakage of alveolar-capillary membranes at sites of inflammation, leading to poor compliance and shortness of breath [2023/6/10].Acute kidney injury (AKI) is a common problem in critically ill patients. AKI occurs in up to 70% of critically ill patients and has a mortality rate in this group more than twice that of similar patients without AKI[25]. A short episode of AKI may predispose the patient to permanent kidney damage and observed an association between AKI and chronic kidney disease[26].AKI in our study adhered to the Kidney Disease, which defined AKI as an increase in Serum Creatinine levels by ≥ 1.5-fold from baseline within 7 days of illness onset or an increase in Serum Creatinine levels by ≥ 0.3 mg/dL (26.4 μmol/L) within 48 h of illness onset. According to Kidney Disease criteria, AKI was classified into three stages: stage 1 (defined by an increase in Serum Creatinine by at least 0.3 mg/ dL or a 1.5 to 1.9 fold increase from baseline); stage 2 (an increase in Serum Creatinie by 2.0–2.9 fold from baseline); and stage 3 (an increase in Serum Creatinine by ≥ 3.0 fold from baseline, by ≥ 4.0 mg/dL or by initiation of dialysis)[27]

Material and methods:

Study design, duration and setting

Retrospective cross-sectional study was conducted. A sample of 200 people confirmed with tuberculosis and pneumonia infection were included in my research.

Inclusion:

- Recently diagnosed Tuberculosis patients and Pneumonia patients.
- Age: All age groups will be included.
- Gender: Both male and female.

Exclusion:

- Exclusion Criteria include those patients who have any kind of other infection.
- Patients having incomplete data are excluded from the study.

Ethical approval and Consent

Ethical approval was taken from Institutional Review Board (IRB) of University of Lahore, Punjab, Pakistan. An informed was taken from the participants before collecting data. Ensured that data would be used for only research purpose. The research project was approved by research and ethics committee of University of Lahore

Data collection procedure and tools

The request form was design as a data collection tool in order to collect the information from the Laboratory. The form will be comprised of data of chemistry test form the lab.

Samples collection and processing:

Procedure for sample collection

Sample collection procedure is as follow

1. Demographic data of patients was noted

2. Clinicians prepared the patient for sampling and made him/her comfortable.

4. Disinfected arm before insertion of needle

5. Collected the sample via venipuncture

6. Draw required amount of blood and collected them in red top vial

7. After that, samples were centrifuge to separate serum from whole blood and run on chemistry analyzer and recorded the results. Creatinine normal result is 0.7 to 1.3 mg/dL for men and 0.6 to 1.1 mg/dL for women. Urea normal results in adult men: 8 to 24 mg/dL and adult women: 6 to 21 mg/dL and children 1 to 17 years old: 7 to 20 mg/dL.

After collecting the samples and required data I performed the data analysis.

Chemistry analyzer

The analyzer measures the signal produced by each channel and compares it with a calibration curve to determine the concentration of the target analyze in the sample. The calibration curve is generated by analyzing known concentrations of analyze under similar conditions.

Centrifuge machine

It is used to separate red blood cells and other blood components from whole blood. A centrifugation machine spins at 3000rpm for 2 minutes.

Statistical analysis:

The Statistical Package for Social Sciences (SPSS) 20.0 was used to examine the data (SPSSA Inc. Chicago, USA). The categorical value was expressed in the form of frequency and percentages. The data was shown using pie charts and bar charts. To examine the data, appropriate statistical methods were used.

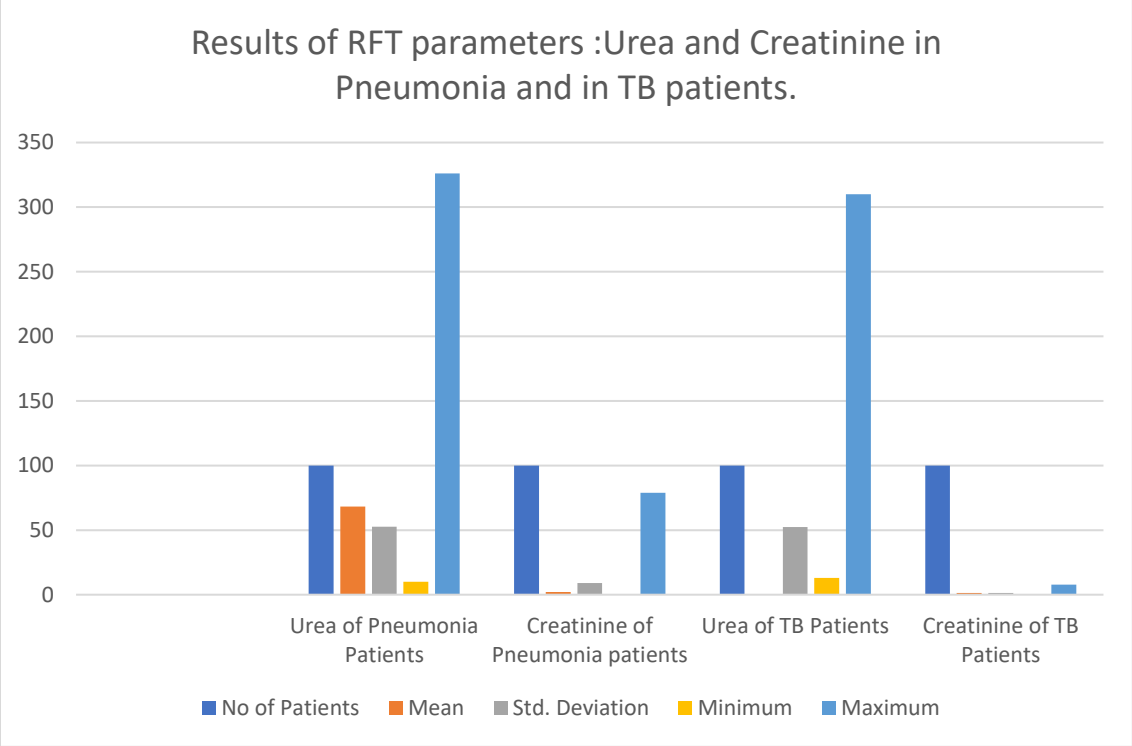
Results:

The purpose of this study was to find out the effects of pneumonia and Tuberculosis (TB) on renal. For this study a total 200 patients were selected and drawn their sample by using sterile condition. Out of 200 samples 100 was pneumonia patient samples and 100 was Tuberculosis (TB) patients. Renal Profile test Urea and Creatinine performed to evaluate the current condition of kidneys. Test was performed on state-of-the-art instrument after confirmation of Quality Control (QC) status of the instrument. Data analysis shows that a minimum range of urea in pneumonia patients which was 10 mg/dl and maximum was 326mg/dl with mean of 68.18 ± 52.59 STD. and creatinine was minimum 0.32mg/dl and Maximum was 7.9mg/dl with 2.1907 ± 9.14 STD. In Tuberculosis (TB) patient's data shows that Urea minimum was 13.0mg/dl and maximum was 310.0mg/dl with mean value of 55.58 ± 52.26 and creatinine minimum values was 0.40mg/dl and maximum value was 7.9mg/dl with mean 1.43± 1.40 STD. According to this analysis there was a significant difference from normal values, mean values of urea a creatinine in both pneumonia and TB patients indicates that both diseases effect the renal conditions and can lead to severe state.

TABLE 1

Variables	Total No of Samples	Minimum	Maximum	Mean	Std. Deviation
Urea of Pneumonia Patients	100	10.00	326.00	68.1800	52.59803
Creatinine of Pneumonia	100	.32	7.90	2.1907	9.14258
Urea of TB Patients	100	13.00	310.00	55.5800	52.26277
Creatinine of TB Patients	100	.40	7.90	1.4350	1.40945

Table No 4.1: Results of RFT parameters: Urea and Creatinine in Pneumonia and in TB patients.



Graph No 4.1: Results of RFT parameters: Urea and Creatinine in Pneumonia and in TB patients.

Table No 4.2: Urea Results in Pneumonia Patients

Variables	Total No Samples	Minimum	Maximum	Mean	Std. Deviation
Urea of Pneumonia Patients	100	10.00	326.00	68.1800	52.59803

Table 3

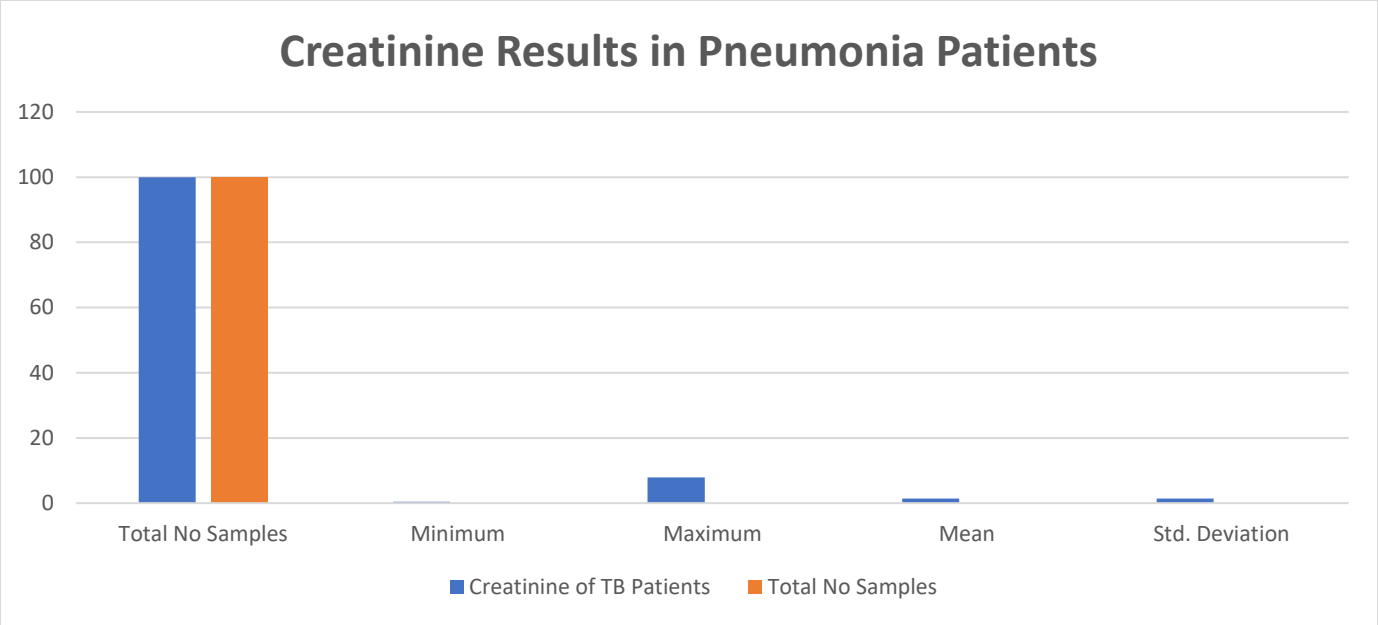
Table No 4.3: Creatinine Results in Pneumonia Patients

	Total No Samples	Minimum	Maximum	Mean	Std. Deviation
Creatinine of Pneumonia	100	.32	79.00	2.1907	9.14258
Total No Samples	100				
Total No Samples	100				

Data analysis shows that a minimum range of urea in pneumonia patients was 10 mg/dl and maximum was 326mg/dl with mean of 68.18 ± 52.59 STD.

Graph No 4.2: Urea Results in Pneumonia Patients

Creatinine analysis shows that a minimum result was 0.32mg/dl and Maximum was 7.9mg/dl with 2.1907 ± 9.14 STD.



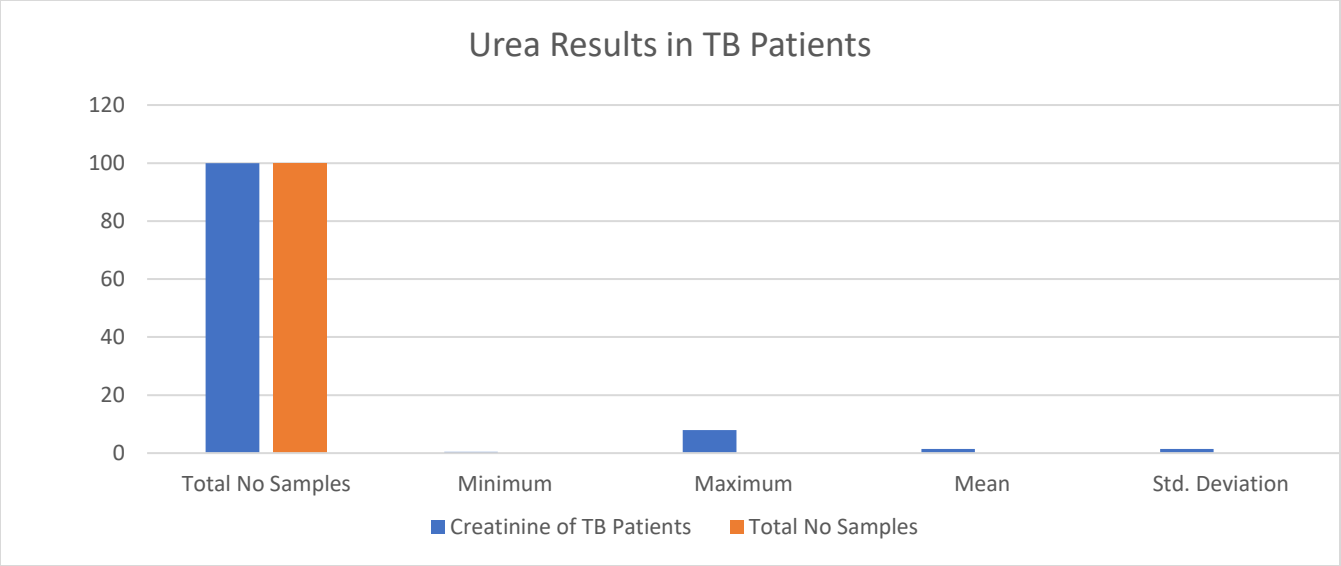
Graph No 4.3: Creatinine Results in Pneumonia Patients

Table 4

Table No 4.4: Urea Results in TB Patients

Variables	Total No Samples	Minimum	Maximum	Mean	Std. Deviation
Urea of TB Patients	100	13.00	310.00	55.5800	52.26277
Total no of Samples	100				

In Tuberculosis (TB) patient's data shows that Urea minimum was 13.0mg/dl and maximum was 310.0mg/dl with mean value of 55.58 ± 52.26



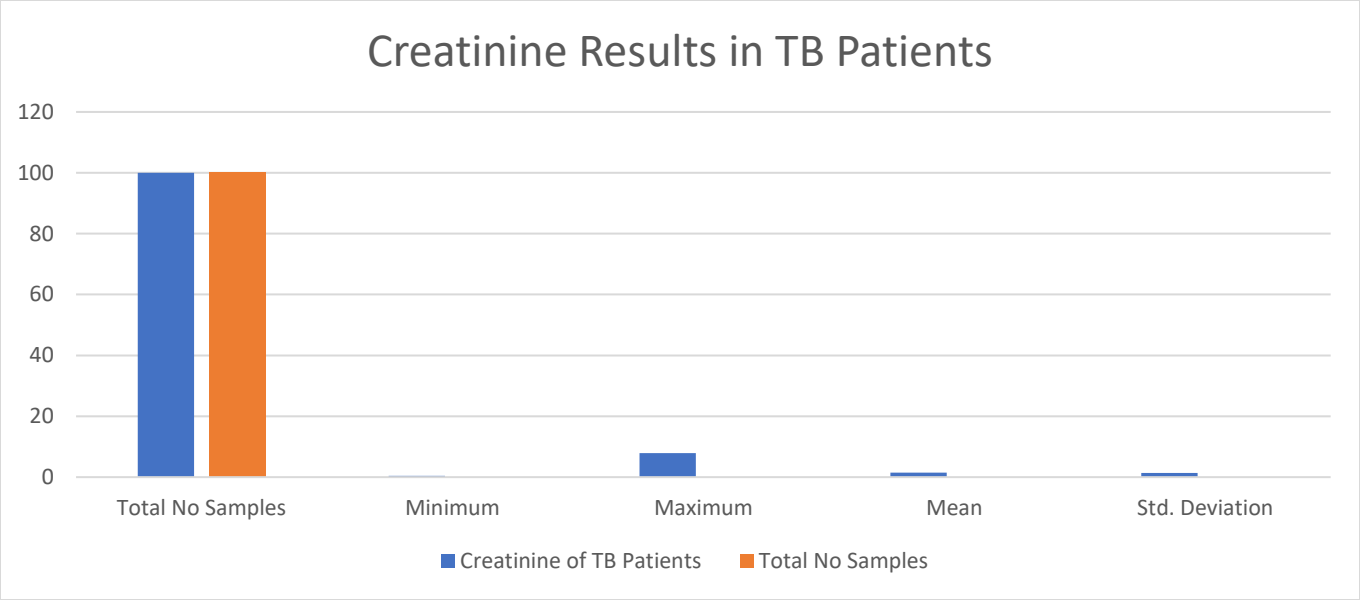
Graph No 4.4: Urea Results in TB Patients

Table 5

Table No: 4.5 Creatinine Results in TB Patients

Variables	Total No Samples	Minimum	Maximum	Mean	Std. Deviation
Creatinine of TB Patients	100	.40	7.90	1.4350	1.40945
Total No Samples	100				

In TB patients' creatinine minimum values was 0.40mg/dl and maximum value was 7.9mg/dl with mean 1.43± 1.40 STD this difference is significant.



Graph No: 4.5 Creatinine Results in TB Patients

Discussion:

Tuberculosis (TB), caused by the bacterial pathogen Mycobacterium tuberculosis (Mtb), remains one of the deadliest infectious diseases with over a billion deaths over the past 200 years. M. tuberculosis is an intracellular pathogen transmitted by inhalation of aerosolized droplets containing bacteria. Small Mtb aerosol particles are expected to pass through the nasopharyngeal or tracheobronchial region to be deposited in the distal airways, larger particles may become trapped in the upper airway or oropharynx and potentially lead to Tuberculosis (TB). Transmission occurs almost exclusively through droplet infection. The vulnerability of the exposed person this series propose a simple cascade for transmission of tuberculosis in which initial case of tuberculosis (1) produces infectious particles (2) which survive in the air and (3) are inhaled by one become a vulnerable person (5) who can become infected and (6) who then has the potential to develop tuberculosis. Innate immune cells in the lungs, mainly macrophages, dendritic cells, monocytes, and neutrophils, phagocytes readily in M. tuberculosis and are the earliest defenders against the pathogen. Mycobacterium Tuberculosis can also transfer during dialysis in renal failure patients. The prevalence of TB in dialysis patients is closely related to the prevalence in the general population and is several times higher. Anorexia, weight loss, low-grade fever, and general weakness, which are the main symptoms of TB, are also commonly seen in patients with chronic kidney failure. It is reported that extra pulmonary TB occurs more frequently than pulmonary TB in dialysis patients and may play a significant role in delaying the diagnosis of TB in dialysis patients. Pneumonia is a common acute respiratory infection affecting the alveoli and distal bronchial tree of the lungs. The disease is roughly divided into community-acquired pneumonia (CAP) or hospital-acquired pneumonia (HAP), which includes ventilator-associated pneumonia (VAP). Aspiration pneumonia accounts for 5–15% of all cases of CAP; but its prevalence in patients with HAP is unknown. The most common symptoms of pneumonia are cough, shortness of breath, chest pain, sputum production and fatigue. CAP is usually clinically suspected when there are acute (7-days) symptoms of LRTI, such as cough, sputum production, fever and dyspnea, and new infiltrates on chest radiographs (CXRs). In elderly patients, symptoms are usually less pronounced and fever may be absent in up to 30% of patients. Symptoms may also be less pronounced in patients treated with steroids, NSAIDs and antibiotics. In 2016 Lin, T.-Y., et al they stated that Pneumococcal disease leads to renal complications ranging from persistent proteinuria to end-stage renal disease. Studies on the association between pneumococcal pneumonia (PP) and acute kidney injury (AKI) are scant. This study assessed the relationship between PP and risk of AKI. This nationwide population-based cohort study examined data from the Taiwan National Health Insurance Research Database for the period 2000–2011. We identified in patients with newly diagnosed PP according to the International Classification of Diseases, Ninth Revision, Clinical Modification (ICD-9-CM) codes. In addition, we selected a comparison cohort from inpatient claims without the diagnosis of PP that was randomly frequency-matched with the PP cohort according to age, sex, index year and comorbidities. We analyzed the risks of AKI by using Cox proportional hazards regression models, adjusted for sex, age, and comorbidities. A total of 10,069 patients with PP and 10,069 controls were enrolled in this

study. After adjustments for age, sex, and comorbidities, patients with PP had a 1.11-fold risk of developing AKI compared with the comparison cohort. This study indicates that AKI risks are higher in patients with PP compared with the comparison cohort. Careful follow-up observation and aggressive treatment are necessary for patients with PP to reduce the risk of AKI. Main purpose of this study was to find out whether TB and pneumonia affects the renal health. For this study a total 200 patients were selected and drawn their sample by using sterile condition. Out of 200 samples 100 was pneumonia patient samples and 100 was Tuberculosis (TB) patients. Renal Profile test Urea and Creatinine performed to evaluate the current condition of kidneys. Test was performed on state of the art instrument after confirmation of Quality Control (QC) status of the instrument. Data analysis shows that a minimum range of urea in pneumonia patients which was 10 mg/dl and maximum was 326mg/dl with mean of 68.18 ± 52.59 STD. and creatinine was minimum 0.32mg/dl and Maximum was 7.9mg/dl with 2.1907 ± 9.14 STD. In Tuberculosis (TB) patient's data shows that Urea minimum was 13.0mg/dl and maximum was 310.0mg/dl with mean value of 55.58 ± 52.26 and creatinine minimum values was 0.40mg/dl and maximum value was 7.9mg/dl with mean 1.43± 1.40 STD.

CONCLUSIONS

Kidney disorder itself a big disorder, if it develops due to cause of other disease this can lead to very serious complication. In this study we have talked about Tuberculosis and pneumonia and effects of these diseases on patient kidneys. On the behalf of above-mentioned s results, we can say that, the risk of kidney disorder is positivity correlated with the patients who were diagnosed with TB and Pneumonia disorders. Renal function determined by performing urea and creatinine tests. Results of both parameters in TB and Pneumonia disease were clearly defined that this disease can lead to renal end stage disorder.

Conflict of Interest: The authors declare that there is no conflict of interest regarding this study.

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Data availability statement: Supported data will be provided on request from the corresponding author.

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