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A STUDY ON CORRELATION OF TRANSFORMING GROWTH FACTOR - β LEVELS WITH PULMONARY FIBROSIS AMONG PULMONARY TB PATIENTS

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ABSTRACT

Background: Pulmonary tuberculosis (PTB) often results in long-term pulmonary sequelae, including pulmonary fibrosis. Transforming Growth Factor-β (TGF-β) is a key pro-fibrotic cytokine implicated in tissue remodeling and fibrosis.

Objectives: To estimate serum TGF-β1 levels among pulmonary TB patients and determine its correlation with the development of pulmonary fibrosis.

Methods: A cross-sectional analytical study was conducted on laboratory-confirmed pulmonary TB patients attending a tertiary care centre in Mysuru. Serum samples were collected, processed, and evaluated for TGF-β1 levels using ELISA. Chest X-rays were assessed for fibrotic changes. Demographic, clinical and laboratory parameters were recorded and correlated.

Results: Among the study participants, a significant proportion demonstrated elevated serum TGF-β1 levels. Patients with radiological evidence of pulmonary fibrosis had markedly higher mean TGF-β1 concentrations compared to those without fibrosis. A positive correlation was observed between TGF-β1 levels and smear grading, chronic disease duration, and radiological fibrosis severity.

Conclusion: Elevated TGF-β1 levels are strongly associated with pulmonary fibrosis in PTB patients. TGF-β1 can serve as a potential biomarker for fibrosis prediction, disease severity assessment, and early clinical intervention.

KEYWORDS: *Mycobacterium tuberculosis*, Pulmonary Fibrosis, TGF-β1.

INTRODUCTION

Tuberculosis (TB) remains a major public health challenge in developing regions, particularly in the Indian subcontinent, where it contributes significantly to morbidity and mortality.[1] As global health threat, TB places a substantial burden on healthcare systems, especially in low- and middle-income countries with limited resources and inadequate healthcare infrastructure. It continues to be among the top ten causes of death worldwide and one of the leading causes of death from infectious diseases.[2]

TB is caused by *Mycobacterium tuberculosis* (MTB), a pathogen transmitted primarily through inhalation of aerosolized droplet nuclei.[3] Although pulmonary tuberculosis (PTB) predominantly affects the lungs, it can also involve extrapulmonary sites such as the kidneys, brain and spine. Common clinical manifestations include chronic cough, hemoptysis, chest pain, fever, weight loss, and night sweats.[3] The infectious nature of TB is notable, as inhalation of as few as 10 bacilli may establish infection, particularly in individuals with weakened immunity. MTB's ability to survive within alveolar macrophages, evade immune responses and persist in a latent state for years contributes to its successful pathogenicity and challenges in treatment.[2] Early phases of anti-TB therapy primarily eliminate rapidly dividing bacilli, whereas the continuation phase is essential for eradicating slow-growing or dormant organisms that may otherwise lead to relapse.[2]

Despite microbiological cures, many recovering TB patients develop long-term pulmonary sequelae. Post-TB lung disease (PTLD) frequently includes structural abnormalities, persistent symptoms and airflow limitation resembling chronic obstructive pulmonary disease (COPD), such as exertional dyspnea.[4] Some patients experience progressive decline in lung function, and permanent anatomical changes—collectively known as TB sequelae—may occur. Pulmonary fibrosis is a major residual lesion formed in the lung parenchyma after TB, contributing significantly to long-term morbidity.[4]

TGF-β1, in particular, has gained prominence due to its central role in fibrosis. It is a multifunctional cytokine involved in regulating cell proliferation, differentiation, and extracellular matrix (ECM) deposition. Recent studies highlight its role in driving lung fibrosis through activation of fibroblasts and differentiation into myofibroblasts, which produce excess ECM proteins.[4] TGF-β1 also enhances α-smooth muscle actin (α-SMA) expression, promotes formation of stress fibers and strengthens adhesion complexes, collectively contributing to progressive scarring and tissue remodeling. Excessive or dysregulated TGF-β1 activity therefore plays a key role in the pathogenesis of TB-associated pulmonary fibrosis.[4]

Given the increasing recognition of post-TB fibrosis and the importance of TGF-β1 in fibrogenesis, understanding its role in PTB patients is clinically relevant. The current study aims to **estimate serum TGF-β1 levels among pulmonary TB patients** and **determine the proportion of individuals who develop pulmonary fibrosis and show elevated TGF-β1 levels** in a tertiary care setting.

MATERIALS AND METHOD

Study Population

This study was analytical, cross-sectional study conducted in the Department of Microbiology, JSS Medical College & Hospital, Mysuru, Karnataka. A total of 80 patients diagnosed with pulmonary tuberculosis were recruited to evaluate the correlation between the serum Transforming Growth Factor-β(TGF-β)levels and pulmonary fibrosis.

Inclusion Criteria

Participants were included based on the following criteria:

1. Patients diagnosed with pulmonary tuberculosis confirmed by sputum smear microscopy, CBNAAT or culture (MGIT/LJ), consistent with national TB diagnostic guidelines.[5]
2. Patients in the pretreatment or early intensive phase of anti-tuberculosis therapy.[6]
3. Willingness to participate after receiving information about the study (written informed consent obtained).

Exclusion Criteria

The following individuals were excluded:

1. HIV-positive individuals due to immune dysregulation influencing cytokine levels.[7]
2. Patients who were diagnosed with (Extra-pulmonary tuberculosis) EPTB.

Procedure

All eligible participants were contacted and briefed about the study. Written informed consent was obtained prior to enrollment. Detailed demographic information and clinical historyincluding symptom duration, prior TB history and comorbidities, were recorded, as recommended in previous TB clinical studies.

A 3–5 mL venous blood sample was collected aseptically. After clot formation, samples were centrifuged, and the serum was aliquoted into sterile cryovials and stored at –20°C, following standardcytokine stabilityprotocols.[10] Serum TGF-β1 levels were quantified using a Sandwich ELISA, performed as per the manufacturer's instructions. Optical density values were plotted against a standard curve to determine TGF-β concentration, in accordance with established cytokine detection methods.[10]

Pulmonary fibrosis was assessed via Chest X-ray (PA view). Radiological evaluation considered fibrotic bands, parenchymal scarring and volume loss, similar to previously published PTB fibrosis classification systems.

Statistical Analysis

Data were compiled in Microsoft Excel and processed using SPSS version 23. The Kolmogorov–Smirnov test was applied to assess the normality of data distribution. Statistical significance was set at $p < 0.05$, consistent with standard biomedical research methodology.[11]

RESULTS

A total of 80 pulmonary tuberculosis (PTB) patients who met the inclusion criteria were included in the study. None of the subjects fulfilled the exclusion criteria. The study population was assessed for demographic characteristics, smoking history, educational status, occupation, and radiological findings. The distribution of these characteristics is shown in Table 1.

Table 1: Characteristics of Study Participants (N = 80).

Variable	N (%)
Gender	
Male	64 (80)
Female	16 (20)
Age Group (Years)	
11-20	3 (3.75)

21–30	6 (7.5)
31-40	16 (20)
41-50	17 (21.25)
51-60	17 (21.25)
61-70	13 (16.25)
71-80	4 (5)
81-90	4 (5)
Total	**80 (100)**
Personal Habits	
Smoking	17 (21.25)
Alcoholic	8 (10)
Both Smoking and Alcoholic	33 (41.25)
None	22 (27.5)
Education Level	
Uneducated	33(41.25)
< Senior High School	32(40)
Senior High School	8 (10)
Bachelor	7 (8.75)
Occupation	
Indoor	15 (18.75)
Outdoor	65 (81.25)

Table 1 shows the demographic details of the study which includes a total of 80 pulmonary tuberculosis (PTB) patients, among whom males predominated with 64 (80%) patients compared to 16 (20%) females. The majority of patients belonged to the middle-aged and older adult groups, with the highest proportions observed in the 41–50 years and 51–60 years categories, each contributing 17 patients (21.25%), followed by the 31–40 years age group with 16 patients (20%). Regarding personal habits, 33 patients (41.25%) reported both smoking and alcohol consumption, while 17 (21.25%) were smokers, 8 (10%) consumed alcohol alone, and only 22 (27.5%) had no such habits. Educational status assessment revealed that 33 patients (41.25%) were uneducated, 32 (40%) had education below senior high school level, 8 (10%) had completed senior high school, and 7 (8.75%) held a bachelor's degree. Occupational analysis showed that outdoor workers constituted the majority with 65 patients (81.25%), whereas indoor workers accounted for only 15 patients (18.75%). Overall, the study population primarily consisted of adult males engaged in outdoor occupations, many of whom had lower education levels and high-risk personal habits, collectively highlighting a group with increased vulnerability to tuberculosis.

Levels of TGF β1 estimated using ELISA method distributed age wise

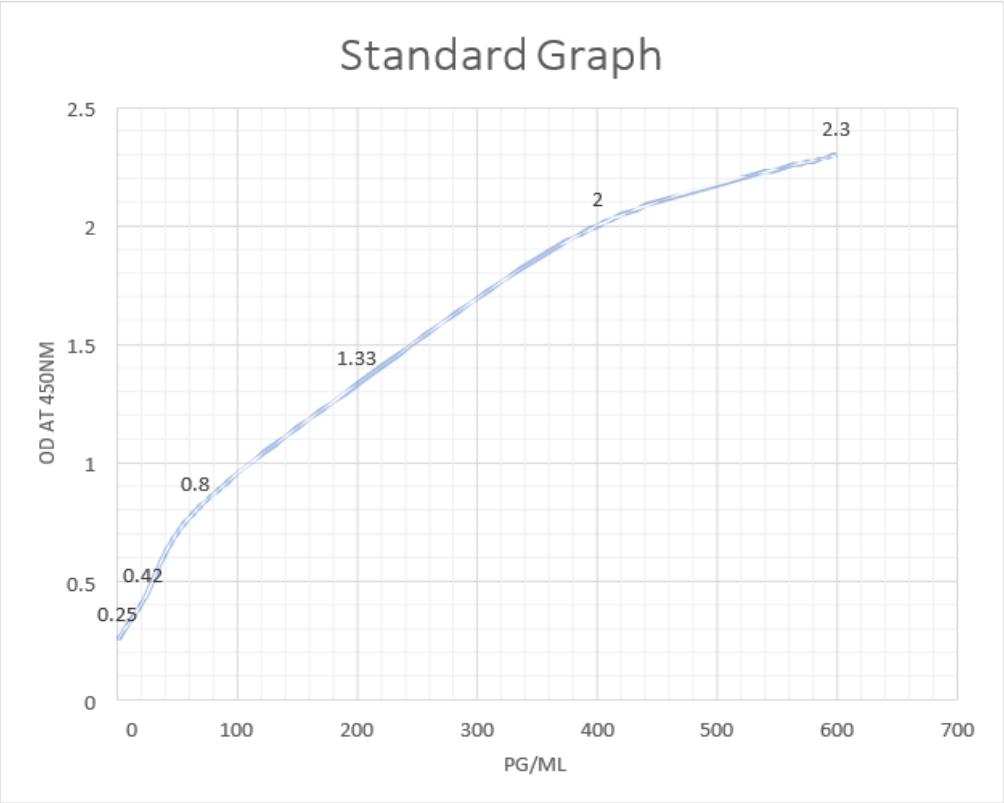
The ELISA findings are depicted in Table 2 and Graph

Table 2: TGF-β1levelsin serum of TB patients.

SL. No	AGE	ODAT 450nm	pg/ml	*Dilution factor (300)	ng/ml
1	55	0.9692	105	31500	**31.5**
2	33	1.6446	295	88500	**88.5**
3	58	0.8471	70	21000	**21**
4	58	1.4238	230	69000	**69**
5	80	0.658	45	13500	**13.5**
6	45	0.8016	60	18000	**18**
7	67	0.9419	100	30000	**30**
8	22	0.893	85	25500	**25.5**
9	65	1.1016	140	42000	**42**
10	53	1.2164	170	51000	**51**

11	44	0.889	80	24000	**24**
12	38	1.0124	115	34500	**34.5**
13	78	0.6011	40	12000	**12**
14	42	1.2026	170	51000	**51**
15	48	1.3157	200	60000	**60**
16	33	0.9929	115	34500	**34.5**
17	34	0.7481	55	16500	**16.5**
18	50	1.4248	230	69000	**69**
19	19	0.9214	90	27000	**27**
20	63	1.2294	175	52500	**52.5**
21	50	1.5276	260	78000	**78**
22	39	0.9731	110	33000	**33**
23	54	1.442	240	72000	**72**
24	40	1.3697	215	64500	**64.5**
25	63	1.2871	190	57000	**57**
26	45	0.7835	60	18000	**18**
27	48	1.1157	140	42000	**42**
28	58	1.6654	300	90000	**90**
29	36	0.9246	90	27000	**27**
30	40	1.4862	250	75000	**75**
31	52	0.7276	50	15000	**15**
32	75	0.9013	85	25500	**25.5**
33	35	0.8214	65	19500	**19.5**
34	36	1.1217	150	45000	**45**
35	59	1.0134	115	34500	**34.5**
36	37	0.9025	85	25500	**25.5**
37	32	1.5287	260	78000	**78**
38	60	1.7219	320	96000	**96**
39	70	0.9213	90	27000	**27**
40	50	0.9246	90	27000	**27**
41	30	0.7832	60	18000	**18**
42	63	1.1247	150	45000	**45**
43	81	0.7514	55	16500	**16.5**
44	38	0.944	100	30000	**30**
45	19	1.3851	220	66000	**66**
46	45	1.4547	245	73500	**73.5**
47	40	1.0169	115	34500	**34.5**
48	30	0.8239	65	19500	**19.5**
49	29	0.7749	55	16500	**16.5**
50	49	0.8863	80	24000	**24**
51	35	1.6224	290	87000	**87**
52	34	0.8414	70	21000	**21**
53	37	0.9016	85	25500	**25.5**
54	60	0.7827	60	18000	**18**
55	55	1.2843	195	58500	**58.5**
56	59	1.5216	260	78000	**78**
57	25	1.6019	285	85500	**85.5**
58	70	1.2024	170	51000	**51**
59	82	0.8534	70	21000	**21**
60	50	1.247	180	54000	**54**
61	50	1.1967	140	42000	**42**
62	46	1.3952	225	67500	**67.5**
63	57	0.9421	100	30000	**30**
64	48	0.8653	75	22500	**22.5**
65	80	1.0216	115	34500	**34.5**
66	70	0.8892	80	24000	**24**
67	23	1.4278	225	67500	**67.5**
68	53	1.5519	270	81000	**81**

69	59	1.1358	140	42000	**42**
70	67	1.2197	170	51000	**51**
71	55	0.7652	55	16500	**16.5**
72	53	1.7294	320	96000	**96**
73	67	0.9275	90	27000	**27**
74	62	0.8897	80	24000	**24**
75	70	0.6124	40	12000	**12**
76	42	1.483	250	75000	**75**
77	65	1.0463	125	37500	**37.5**
78	41	0.7856	60	18000	**18**
79	73	0.9501	100	30000	**30**
80	**17**	**1.5925**	**255**	**76500**	**76.5**



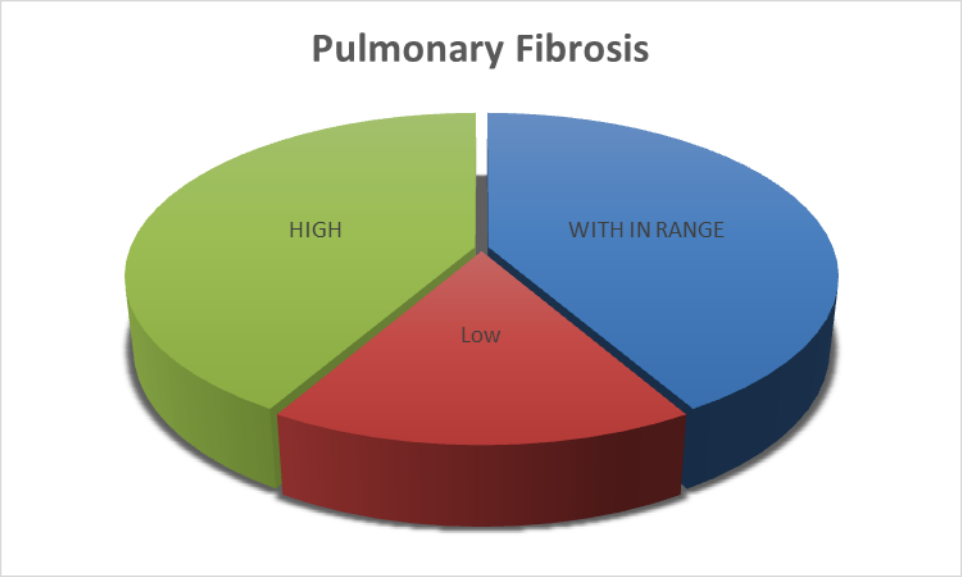
Graph 1: Standard Graph of ELISA.

X-ray findings correlation with TGF - β1level

The MIC DistributionofTGF-β1 levels incorrespondencetotheirX-rayfindings are depicted in Table 3 and graph 2 highlighting the statistics of pulmonary fibrosis.

Table 3: TGF-β1levels in correspondence to X-ray findings.

X-Ray Findings	Frequency	TGF		β 1 Levels
		High	Low	Within Range
Pulmonary fibrosis	24	10	4	10
Cavitary lesion	14	4	2	8
Cavitary consolidation	1	1	-	-
Pleuralempyema	19	5	4	10
Calcifiednodule consistent with calcified granuloma	2	-	-	2
Cannotestablish contact / unwilling to answer	12	1	5	6
Normal	8	1	3	4
Total	80	22	18	40



Graph 2: TGF-β1levels in correspondence to Pulmonary fibrosis.

DISCUSSION

This study analyzed the serum levels of TGF-β1 in 80 active pulmonary tuberculosis (PTB) patients to understand its role in inflammation, disease progression, and the development of pulmonary fibrosis. A significant gender difference was observed, with males comprising 80% of cases, a trend consistent with Putoo A.N. et al.[2], who reported a higher prevalence of TB among males (54.8%). This disparity may be attributed to increased exposure, higher rates of smoking and alcohol consumption, and occupational risk among men. Age also played a critical role in disease distribution. The majority of patients belonged to the 41–50 and 51–60 age groups (21.25% each), followed by 31–40 years (20%), highlighting the susceptibility of middle-aged individuals. Similar conclusions were drawn by An et al.[12], who emphasized the vulnerability of individuals above 50 years.

Occupational data revealed that a considerable proportion of PTB cases occurred among individuals working outdoors, particularly farmers (28.75%) and public servants (16.25%). This finding aligns with Amin et al.,[13] who reported a strong association between PTB and farming due to environmental exposure, poor ventilation, and limited access to healthcare. Personal habits significantly contributed to TB risk: 41.25% of patients were both smokers and alcohol consumers, followed by smokers (21.25%) and alcohol users (10%). While a study from Ethiopia suggested that smoking was not a major risk factor, our findings support the evidence from J. Bras Pneumol et al.,[14] which indicates higher TB mortality among smokers.

Comorbidities such as diabetes mellitus (17.5%), COPD (11.25%), asthma and lower respiratory tract infections (8.75%), and hypertension (7.5%) significantly influenced TB progression. ME´DA et al.[15] also highlighted the impact of comorbidities in worsening TB outcomes. Education status further revealed an important risk determinant. A large proportion of the patients (41.25%) were uneducated, with only 18.75% having studied beyond primary school. Limited education is often associated with poor health-seeking behavior, inadequate awareness of TB transmission, and poor treatment compliance, as supported by Malays J et al.[16]

Measurement of serum TGF-β1 using ELISA demonstrated that its levels were elevated across all age groups, with higher concentrations in the 31–40 and 51–60-year cohorts. Among the 80 patients, 22 had high TGF-β1 levels, 18 had

low levels, and 40 fell within the normal range. Studies by Teklu et al.,[17] Pavan Kumar et al.,[18] and Olobo et al.[19] similarly reported elevated TGF-β1 in active TB, contributing to immunosuppression and tissue remodeling.

Importantly, 27.25% (22/80) of patients developed pulmonary fibrosis. Increased TGF-β1 is known to downregulate cell-mediated immunity while enhancing fibroblast proliferation, extracellular matrix deposition, and tissue scarring. This mechanism aligns with findings from Sasse et al.,[15] who demonstrated TGF-β1's role in promoting fibrosis and pleural space remodeling. In the present study, chest X-ray evaluation showed pleural empyema in 19 patients and cavitary lesions in 14 patients, both of which are complications associated with prolonged inflammation and TGF-β1-mediated tissue damage. Among 24 patients with fibrosis, TGF-β1 levels were high in 10 and low in 4, indicating a clear trend toward elevated cytokine levels being associated with fibrotic changes consistent with the findings of Teixeira et al.[20] in Germany.

Despite treatment availability, only 62.5% of patients achieved cure, with 27.5% still undergoing treatment and a mortality rate of 10%. Poor treatment adherence, persistent personal habits, comorbidities, and delayed diagnosis may contribute to adverse outcomes. Given the established association between elevated TGF-β1 and fibrosis, monitoring this cytokine may serve as a valuable prognostic marker in PTB patients.

CONCLUSION

The expression of TGF-β1 levels was found to be high in 22 patients (27.5%), low in 18 patients (22.5%) and within range in 40 patients (50%). In conclusion, there is a correlation between TGF-β1 levels and the occurrence of pulmonary fibrosis. Out of 22 patients having high levels TGF-β1, 10 of them progressed to secondary complication i.e, pulmonary fibrosis. Therefore, TGF-β1 could be introduced as a suitable biomarker in tracking the progression rate of TB patients.

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