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Original Article

Pleural Fluid Interleukin-32 as a Diagnostic Biomarker for Tuberculous Pleural Effusions in Bangladesh: A Cross-Sectional Study

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Abstract:

Background: *Tuberculous pleural effusion (TPE) is one of the most common types of extrapulmonary tuberculosis, and definitive diagnosis based on biomarkers is very challenging. Interleukin 32 (IL-32) is an inflammatory cytokine, crucial for the host immune response to tuberculosis. The purpose of this study is to determine the performance of IL-32 in the diagnosis of TPE.*

Materials and methods: *This cross-sectional observational study was conducted in the Department of Respiratory Medicine, Bangladesh Medical University (BMU), among 50 participants who met the inclusion and exclusion criteria. We assessed pleural fluid for IL-32 and ADA via enzyme-linked immunosorbent assay to determine diagnostic performance and compare the two.*

Results: *A total of 23 TPE and 27 non-TPE were identified by means of pleural biopsy findings, and in both instances, males were predominant. Mean (±SD) age of TPE participants was 34.6 ± 17.3 years, and non-TPE participants were 57.32 ± 11.42 years. The most common symptoms were weight loss, anorexia, and fever in the TPE group. Mean Pleural fluid lymphocyte counts were significantly higher in TPE than in non-TPE (78.4 ± 10.2 vs. 34.8 ± 15.1; P<0.001). Similarly, median (IQR) IL-32 and ADA concentrations were significantly higher in TPE than in non-TPE [321.90 (260.15–490.95)] vs. [113.70 (79.95–152.15)] and [24.11 (10.35–37.76)] vs. [7.22 (4.17–9.28)], respectively; both P<0.001), whereas protein, glucose, and LDH concentrations did not differ significantly between groups. An IL-32 cutoff of >215.9 pg/ml may aid in the diagnosis of TPE with a sensitivity of 95.65, specificity of 92.59, and area under the curve (AUC) of 0.952.*

Conclusion: *IL-32 showed promising diagnostic performance for TPE, with a numerically higher AUC than that of ADA, although the difference was not statistically significant.*

Keywords: *Interleukin 32 (IL-32); Tuberculous Pleural Effusion (TPE), Adenosine Deaminase (ADA).*

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Introduction:

Tuberculosis (TB) is a serious public health hazard, caused by Mycobacterium tuberculosis (MTB), a microorganism that is a leading communicable disease globally and an important cause of morbidity and mortality in the developing world, particularly in Asia and Africa.[1] Although the TB bacilli primarily attack the lungs, they can involve almost all organs of the body. According to the World Health Organization (WHO 2021), there were 10.6 million cases of TB worldwide, with more than two-thirds of cases occurring in eight Asian countries, with Bangladesh being one of the 30 countries with the highest TB burden, contributing an estimated 3.6 percent of all cases.[2] In Bangladesh, about 81% of cases are pulmonary, whereas 19% are extra-pulmonary TB (EPTB), and the sex distribution was 54%, 42%, and 4% for male, female, and children, respectively.[3]

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TPE is the most common type of EPTB and the major cause of pleural effusions in developing countries.[4] Once bacilli involve the pleura, it results in the so-called TPE, defined by an acute-to-chronic buildup of fluid and inflammatory cells within the pleural space.[5] Effusion develops in up to 30 percent of tuberculosis cases, resulting in significant morbidity and presenting with chronic fever, cough, and painful chest, indicating chronic pleural inflammation.[3,5]

Interleukin 32 (IL-32) is a pro-inflammatory cytokine induced in human epithelial cell lines by interferon gamma (IFN-g), IL-1b, and IL-18; in natural killer (NK) cells and CD4+ T lymphocytes by IL-12 and IL-18. When stimulated with CD3 and CD28 monoclonal antibodies, T cells produced IL-32, which was up-regulated in monocyte-derived dendritic cells (DCs) using tumor necrosis factor alpha (TNF-alpha) to mature them6. Granulocyte macrophage colony-stimulating factor (GM-CSF) from eosinophils stimulates IL-32a, IL-32b, IL-32g, and IL-32d in a caspase-1-dependent fashion.[6,7] Mycobacterium tuberculosis (MTB) is a pathogen with which macrophages provide an initial defense. IL-32 influences macrophage responses to MTB by inducing caspase-1-dependent and caspase-1-independent apoptosis (including cathepsin- and caspase-1-mediated pyroptosis), which, unlike cellular necrosis, are key effector mechanisms macrophages use to eliminate intracellular MTB.[8]

TB is widespread in Bangladesh, especially in low socio-economic groups due to various reasons, among them malnutrition, overcrowding, and close contact with undetected or untreated smear-positive TB patients are the most common. Also, there is evidence of delay in anti-TB initiation, which increases the risk of poor treatment outcomes, chronic disabilities, and death.[9] Because the clinical manifestations of EPTB are unusual, obtaining tissue samples to confirm the diagnosis can be challenging, and conventional diagnostic methods have poor diagnostic performance; therefore, diagnosis is often delayed. Conventional smear microscopy has low sensitivity (40%), and negative results cannot rule out TB. Mycobacterial culture test reports range from 30% to 80%; however, they can take 2 to 8 weeks, which is too long for treatment.[10] Pleural fluid ADA is a commonly used biochemical test for diagnosing TPE, but it may be elevated in other inflammatory conditions such as parapneumonic effusions, empyema, and lymphoma, creating potential diagnostic uncertainty. IL-32, closely linked to the host immune response to MTB, has also shown promising diagnostic accuracy and could offer greater specificity, but its clinical use and comparative performance with ADA are not fully studied, especially in high-burden areas. To fill this gap, it is necessary to find a fast, simple diagnostic tool that may help in making an early and confident diagnosis, and starting the anti-TB treatment in the shortest possible time. Therefore, this study aims to evaluate the diagnostic accuracy of pleural fluid IL-32 for detecting tuberculous pleural effusions and to compare its performance with the classical TPE marker adenosine deaminase (ADA).

Methodology

This cross-sectional observational study was conducted in the Department of Respiratory Medicine at BMU, Dhaka, Bangladesh, over 1 year after obtaining ethical approval from the BMU Institutional Review Board (IRB). Our sample size was 50. Patients who had previously received anti-TB drugs, antitumor therapy, immunosuppression, recent thoracic surgery or trauma, empyema, and transudative effusion were excluded. After fully explaining the study purpose and procedures, we obtained written informed consent. Fluid aspiration and biopsy of the pleura were done under local anesthesia using an ultrasonography-guided Abram biopsy needle. Saline and formalin were used to prepare tissue for detection of bacilli and histopathological analysis, respectively. Participants were grouped into TPE and non-TPE based on the presence or absence of acid-fast bacilli (AFB) or caseating granuloma in pleural histopathology, respectively. We used a semiautomatic biochemical analyzer with colorimetric enzyme cycling method kits to measure pleural fluid ADA and other biochemical parameters (protein, lymphocytes, glucose, and LDH). We used a sandwich enzyme-linked immunosorbent assay (ELISA) to measure pleural fluid IL-32 concentration. Clinical data were kept confidential from laboratory technicians, and samples were examined only once to prevent degradation. We analyzed data using SPSS version 23.0. We used the Shapiro–Wilk test to assess normality of continuous variables, applied an independent-samples t-test or Mann–Whitney U test to compare groups where appropriate, and used a receiver operating characteristic (ROC) curve to assess diagnostic performance. A p-value of less than 0.05 was taken to be statistically significant.

Results

This study included 50 patients with undiagnosed pleural effusion. Among them, 23 (46%) participants had tuberculous pleural effusion and 27 (54%) had non-tuberculous pleural effusion (Table I).

Regarding age distribution, the mean (± SD) age of TPE participants was 34.6 ± 17.3 years and 57.32 ± 11.42 years in non-TPE participants (p-value <0.001). The sample included 34 male and 16 female participants (Table II).

Figure 1 showed that altogether 44 (88%) patients reported complaints about their weight loss, 43 (86%) about their cough, and 42 (84%) about their anorexia. Others included fever in 56%, chest pain in 52%, breathlessness in 32%, and hemoptysis in 26%.

When pleural fluid was analyzed, mean (± SD) pleural concentration of lymphocytes was significantly higher in patients with TPE (78.4 ± 10.2) than in patients with non-TPE (34.8 ± 15.1) (p-value <0.001), and the difference in mean pleural concentration of protein and glucose and median LDH concentration between patients with TPE and non-TPE were non-significant (p-value>0.05). Additionally, median (IQR) pleural concentrations of IL-32 were

significantly higher in patients with TPE [321.90 (260.15–490.95)] than in patients with non-TPE [113.70 (79.95–152.15); P<0.001]. Similarly, median (IQR) levels of ADA were significantly higher in patients with TPE than in patients with non-TPE [24.11 (10.35–37.76) vs. 7.22 (4.17–9.28); p-value <0.001] (Table III).

Table IV shows the diagnostic performance of IL-32 and ADA in the study. At a cutoff value of >215.9 pg/ml, IL-32 diagnosed TPE with a sensitivity of 95.65 %, specificity of 92.59%, and area under the curve (AUC) of 0.952. The corresponding values for ADA at a cut-off of >23.1 U/L were 91.3%, 74.1%, and 0.873, but they were not statistically significant.

ROC curve analysis showing better diagnostic performance of IL32 with an AUC of 0.952, though statistically not significant (p-value - 0.235) (Figure 2)

Table V showed that the percentage of lymphocytes in the PF remained an independent predictor of TBE in multivariable analysis (adjusted OR 1.127, 95% CI 1.047–1.214; p=0.001). A borderline inverse association was observed between age and TBE (adjusted OR 0.921, 95% CI 0.845–1.003; p=0.058), while sex, ADA and IL-32 were not independently associated with TBE after adjustment.

Table 1: Etiological frequency of the studied participants (N=50)

Variables	Etiology	Number (n=50)	Percentage (%)
Tuberculous pleural effusion (TPE)	Tuberculosis	23	46
Non tuberculous pleural effusion (non-TPE)	Malignant	18	36
	Parapneumonic	6	12
	Others	3	6

Table 2: Age and gender distribution of the studied participants (N=50)

Variables		TPE*	Non-TPE**	p-value[s]
Number of patients (n) with percentage (%)		23 (46%)	27 (54%)	
Age in year (Mean ±SD)		34.6+17.3	57.32+11.42	**<0.001**
Gender	Male	16 (69.57%)	18 (66.6%)	
	Female	7 (30.43%)	9 (33.3%)	

*TPE: Tuberculous pleural effusion **Non-TPE: Non tuberculous pleural effusion
[s]p-value less than 0.05 is statistically significant

Table 3: Comparison of pleural fluid analysis in between tuberculous and non-tuberculous pleural effusion in the studied participants (N=50)

Variables	TPE (n=23)	Non-TPE (n=27)	p-value[s]
Total protein (g/l) (Mean ± SD)	43.6 +9.9	40.8 +11.2	0.353
Lymphocyte differential count (Mean ± SD)	78.4 ± 10.2	34.8 ± 15.1	0.001
Glucose (mmol/l) (Median-IQR)	4.82 (4.11–6.72)	4.76 (2.98–8.25)	0.930
LDH* (U/l) (Median-IQR)	241.5 (223.3–308.3)	255.1 (235.0–344.6)	0.284
ADA** (U/l) (Median-IQR)	24.11 (10.35–37.76)	7.22 (4.17–9.28)	0.001
IL-32* (Median-IQR)	321.90 (260.15–490.95)	113.70 (79.95–152.15)	0.001

*LDH-Lactate dehydrogenase **ADA- Adenosine deaminase
[s]p-value was calculated using independent-samples t-test and Mann–Whitney U test where appropriate a value < 0.05 was considered significant.

Table 4: Comparison of the diagnostic performances of IL-32 and ADA in the studied participants (N = 50)

Variables	Cutoff value	AUC	Sensitivity (%)	Specificity (%)	PLR	NLR	PPV	NPV	p -value*
IL-32 (pg/ml)	≥215.9	0.95	95.65	92.59	12.91	0.04	91.67	96.15	0.2
ADA (U/l)	>23.1	0.87	91.30	74.10	3.52	0.12	74.1	91.3	

AUC: Area under the curve; PLR: Positive likelihood ratio;
NLR: Negative likelihood ratio; PPV: Positive predictive value;
NPV: Negative predictive value
*p-value was calculated using DeLong test and a value < 0.05 was considered significant.

Table 5: Multivariate logistic regression analysis

Variable	OR (95% CI) **	P value	Adjusted OR (95% CI)	P value*
Age, years	0.905 (0.855–0.957)	<0.001	0.921 (0.845–1.003)	0.058
Male sex	1.130 (0.342–3.731)	0.841	0.387 (0.026–5.678)	0.488
Lymphocyte percentage	1.180 (1.071–1.300)	<0.001	1.127 (1.047–1.214)	0.001
ADA (U/L)	1.167 (1.058–1.288)	0.002	0.993 (0.909–1.085)	0.879
IL-32 (pg/ml)	1.020 (1.009–1.031)	<0.001	0.998 (0.993–1.003)	0.428

We used Firth-penalized logistic regression because the conventional logistic model showed separation.
*A p-value <0.05 is taken as statistically significant. **OR: Odd ratio

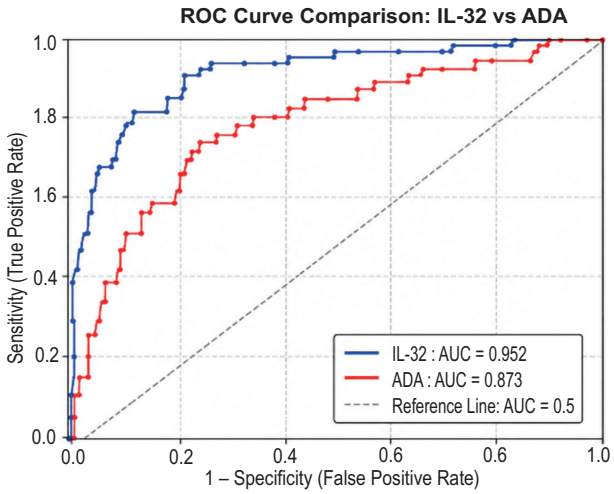

Figure 2: *ROC curve showing comparison of diagnostic efficacy of IL-32 and ADA.*

In pleural fluid, IL-32 showed an AUC of 0.952 with 95.65% sensitivity and 92.59% specificity, while adenosine deaminase showed an AUC of 0.873 with 91.3% sensitivity and 74.1% specificity. AUC difference (IL-32 – ADA): 0.0789; Standard error (SE): 0.0665; 95% CI of difference: -0.0515 to o.2093; z-statistics: 1.186; p-value: 0.235.

Discussion:

Tuberculous pleural effusion (TPE) remains difficult to diagnose using conventional clinical and biochemical criteria, particularly in high-burden regions such as Bangladesh where invasive microbiological confirmation is often unavailable. In this study, pleural fluid interleukin-32 (IL-32) demonstrated excellent diagnostic accuracy for TPE (AUC 0.952, sensitivity 95.65%, specificity 92.59% at a cutoff of >215.9 pg/mL), performing at least as well as, and numerically better than, ADA (AUC 0.873). These findings are discussed below in relation to the small but growing body of literature specifically evaluating IL-32 and ADA as pleural fluid biomarkers of TPE.

The markedly elevated pleural IL-32 concentrations observed in TPE participants compared with non-TPE participants in this study are consistent with the underlying immunobiology of IL-32 in tuberculous infection. IL-32 is produced predominantly by monocytes and macrophages within the pleural fluid mononuclear cell compartment, where its production is positively correlated with other Th1-associated mediators such as interferon-gamma (IFN-ã) and is itself inducible by IFN-ã.[13] This places IL-32 mechanistically downstream of the same delayed-type hypersensitivity response to Mycobacterium tuberculosis antigens that drives lymphocytic pleural exudation, providing biological plausibility for its diagnostic elevation in TPE. Notably, experimental work has also shown that IL-32 regulates ADA expression in macrophages, with IL-32 knockdown reducing, and IL-32ã stimulation inducing, ADA production.[11] This functional link offers a legible explanation for why IL-32 and ADA concentrations tracked together in the present cohort, and suggests the two markers reflect overlapping rather than entirely independent components of the same macrophage-driven inflammatory pathway.

The diagnostic performance of IL-32 reported here closely parallels the largest previous evaluation of this biomarker. In a two-center Chinese cohort of 131 patients with undetermined pleural effusion, Du and colleagues[11] reported an IL-32 AUC of 0.933 for TPE, with 88.4% sensitivity and 93.4% specificity at a cutoff of 247.9 ng/mL, and further showed that a multivariate model combining age, ADA, lactate dehydrogenase and IL-32 reliably distinguished TPE across a range of disease prevalence. The specificity obtained in the present study (92.59%) is almost identical to that reported by Du et al.[11], and the somewhat higher sensitivity observed here may reflect differences in cohort composition and sample size between the two studies.

A more recent, larger evaluation by Wang and colleagues[14] specifically examined how age modifies the diagnostic accuracy of IL-32ã, ADA and IFN-ã in 188 patients with pleural effusion. They found that IL-32ã performed only modestly overall (AUC 0.812) and was inferior to both ADA (AUC 0.991) and IFN-ã (AUC 0.992) in patients aged 65 years or younger, but became clearly superior to both markers in patients older than 65 years (IL-32ã AUC 0.984 versus ADA 0.817 and IFN-ã 0.578), a pattern attributed to an age-related decline in ADA and IFN-ã accuracy that IL-32ã did not share[14]. This contrasts with the present findings: the cohort reported here was considerably younger (mean age 34.6 years in TPE participants), yet IL-32 still outperformed ADA in both sensitivity and specificity. This disparity may

reflect differences in the composition of the non-TPE comparator group, assay methodology, and the inherent limitations of a much smaller sample, and it highlights that the relative performance of IL-32 versus ADA cannot yet be assumed to generalize consistently across age groups or populations without further comparative data.

For ADA alone, the sensitivity recorded in this study (91.3%) is consistent with pooled estimates from systematic reviews and meta-analyses of ADA for TPE, which have generally reported sensitivities in the region of 88-93% across a range of diagnostic thresholds4. The specificity observed here (74.1%), however, was considerably lower than the pooled specificity of around 90-91% reported in these meta-analyses[15], and lower than the 91% pooled ADA specificity reported in a comparative meta-analysis of ADA against IFN-ã, which found IFN-ã to be the more accurate of the two established markers overall (relative diagnostic odds ratio 2.22 in favor of IFN-ã).[16] The comparatively low ADA specificity in the present cohort is most plausibly explained by the size and composition of the non-TPE comparator group (n=27), since even a small number of false-positive ADA elevations, for example from parapneumonic or other infected effusions, can markedly shift specificity estimates in a small sample. Taken together with meta-analytic evidence that IFN-ã outperforms ADA, and that IL-32 is mechanistically and functionally linked to the same Th1 pathway[15,16], the present results broadly support the hypothesis that IL-32 may offer a diagnostic advantage over ADA in some settings, though this requires confirmation in adequately powered, directly comparative studies.

The difference in AUCs between IL-32 and ADA in this study did not reach statistical significance (p=0.235), and given that even the much larger studies by Du et al.[11] and Wang et al.[13] reported meaningfully different absolute AUCs for the same biomarker, the point estimates from a 50-patient cohort should be regarded as imprecise. The age-dependence of IL-32 accuracy demonstrated by Wang et al.[13] was not examined here, and given that the diagnostic advantage of IL-32 over ADA in that study was concentrated in older patients, while an advantage was observed here in a considerably younger cohort, the mechanism underlying IL-32's diagnostic performance across different age structures remains incompletely understood. IL-32 regulates ADA expression (1) and shares upstream regulatory cytokines with ADA-associated pathways (3); our multivariable finding that neither marker remained an independent predictor of TPE once lymphocyte percentage was accounted for argues against interpreting IL-32 as a wholly distinct diagnostic axis from ADA.

This study has some limitations. First, it enrolled a very small sample size and relied on clinical and biochemical criteria rather than universal microbiological confirmation to diagnose TB in pleural fluid.

Conclusion:

Based on this study, IL-32 demonstrated good diagnostic value for discriminating tuberculous pleural effusion (TPE) from non-TPE, with a numerically higher AUC than ADA. However, the difference in diagnostic performance between IL-32 and ADA was not statistically significant. Multivariable analysis showed that pleural fluid lymphocyte percentage was independently associated with TPE, whereas ADA and IL-32 did not remain statistically significant after adjustment. These findings suggest that IL-32 may serve as a promising adjunct biomarker for diagnosing TPE rather than a superior replacement for ADA. When interpreting diagnostic estimates, one has to remember the very small sample size. Further large-scale, prospective, multicenter studies are warranted to validate the incremental diagnostic value of IL-32 and determine its potential role alongside existing biomarkers before routine clinical implementation.

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Conflict of interest:

None declared

Data availability statement:

The data that support the findings of this study are available on request from the corresponding author. The data are not publicly available due to privacy or ethical restrictions.

Ethics statement:

This study was conducted in accordance with the Declaration of Helsinki. The Institutional Review Board (IRB), Bangladesh Medical University, Dhaka, Bangladesh, approved this human study (approval ID: 948). All adult

participants provided written informed consent to participate in this study.

Authors contribution:

MAR was responsible for the study conception and overall design, methodology development, data collection, validation, and formal statistical analysis. MAR also led the investigation and prepared the initial manuscript draft, followed by critical revision and editing. SM contributed to research resources, investigation, oversight, methodology development, data collection, validation, and formal statistical analysis, and participated in critical revision and editing. JC contributed to methodology development, statistical analysis, and manuscript revision and editing. SR contributed to providing research resources and investigation and participated in critical manuscript revision and editing. MMR contributed to providing research resources and investigation and participated in critical manuscript revision and editing. SI contributed to providing research resources and investigation and participated in critical manuscript revision and editing. KRF contributed to providing research resources, investigation, and validation and participated in critical manuscript revision and editing. SS contributed to providing research resources, investigation, validation, and participated in critical manuscript revision and editing. SHC contributed to providing research resources, investigation, validation, and participated in critical manuscript revision and editing. AN contributed to research resources, investigation, validation, and participated in critical manuscript revision and editing. MRA, MKI participated in critical manuscript revision and editing. SA developed visual representations of the data, contributed to study supervision and validation, and participated in manuscript revision and editing. RC coordinated project activities, supervised the research process, provided research resources, oversight, and validation of the findings, and participated in critical manuscript revision and editing. All authors reviewed the final manuscript and approved it for publication.

References

1. Centers for Disease Control and Prevention (CDC). *Tuberculosis (TB): basic TB facts* [Internet]. Atlanta (GA): CDC; 2019 Jun 19 [cited 2023 Apr 14]. Available from: https://www.cdc.gov/tb/topic/basics/default.htm
2. *World Health Organization. Global tuberculosis report 2022 [Internet]. Geneva: World Health Organization; 2022 [cited 2023 Apr 14]. Available from: https://www.who.int/teams/global-tuberculosis-programme/tb-reports/global-tuberculosis-report-2022*
3. World Health Organization. *Bangladesh TB profile 2021* [Internet]. Geneva: World Health Organization; 2021 [cited 2023 Apr 15]. https://www.aidsdatahub.org/sites/default/files/resource/bangladesh-tb-profile-2021.pdf
4. Porcel JM. Tuberculous pleural effusion. Lung. 2009 Oct;187(5):263-70. https://doi.org/10.1007/s00408-009-9165-3
5. Hasaneen NA, Zaki ME, Shalaby HM, El-Morsi AS. Polymerase chain reaction of pleural biopsy is a rapid and sensitive method for the diagnosis of tuberculous pleural effusion. Chest. 2003 Dec 1;124(6):2105-11. https://doi.org/10.1378/chest.124.6.2105
6. Shoda H, Fujio K, Yamaguchi Y, Okamoto A, Sawada T, Kochi Y, et al. Interactions between IL-32 and tumor necrosis factor alpha contribute to the exacerbation of immune-inflammatory diseases. Arthritis research & therapy. 2006 Nov 1;8(6):R166. https://doi.org/10.1186/ar2074
7. Jeong HJ, Shin SY, Oh HA, Kim MH, Cho JS, Kim HM. IL 32 up regulation is associated with inflammatory cytokine production in allergic rhinitis. The Journal of pathology. 2011 Aug;224(4):553-63. https://doi.org/10.1002/path.2899
8. Bai X, Kim SH, Azam T, McGibney MT, Huang H, Dinarello CA, et al. IL-32 is a host protective cytokine against Mycobacterium tuberculosis in differentiated THP-1 human macrophages. The Journal of Immunology. 2010 Apr;184(7):3830-40. https://doi.org/10.4049/jimmunol.0901913
9. National Tuberculosis Control Programme (NTP), Directorate General of Health Services, Government of the People's Republic of Bangladesh. '*National Guideline and Operational Manual for Tuberculosis*'. 6th ed. Dhaka, Bangladesh: Ministry of Health and Family Welfare; October 2021. https://www.ntp.gov.bd/guidelines/.
10. Lee JY. Diagnosis and treatment of extrapulmonary tuberculosis. Tuberculosis and respiratory diseases. 2015 Apr 2;78(2):47. http://dx.doi.org/10.4046/trd.2015.78.2.47.
11. Du J, Shao MM, Yi FS, Huang ZY, Qiao X, Chen QY et al. Interleukin 32 as a Potential Marker for Diagnosis of Tuberculous Pleural Effusion. Microbiology Spectrum. 2022 Aug 31;10(4):e0255321. doi: 10.1128/spectrum.02553-21.
12. Haque AF, Kaes MI, Islam AA, Mohammad N. Clinicopathological Study of Pleural Effusion in A Tertiary Care Hospital. Khwaja Yunus Ali Medical College Journal [Internet]. 2022 Sep. 5;13(2):98-101. Available from: https://banglajol.info/index.php/KYAMCJ/article/view/61339
13. Wang X, Yang C, Quan C, Li J, Hu Y, Liu P, Guan L, Li L. The regulation and potential role of interleukin-32 in tuberculous pleural effusion. Frontiers in Immunology. 2024 May 13;15:1342641. doi: 10.3389/fimmu.2024.1342641
14. Wang Q, Ma L, Zhai K, Yi FS, Shi HZ, Shao MM. Diagnostic accuracy of interleukin 32ã for tuberculous pleural effusion. Journal of Thoracic Disease. 2025 Apr 30;17(4):2339-49. DOI: 10.21037/jtd-24-1676.
15. Aggarwal AN, Agarwal R, Sehgal IS, Dhooria S. Adenosine deaminase for diagnosis of tuberculous pleural effusion: A systematic review and meta-analysis. PloS one. 2019 Mar 26;14(3):e0213728. DOI: 10.1371/journal.pone.0213728
16. Aggarwal AN, Agarwal R, Dhooria S, Prasad KT, Sehgal IS, Muthu V. Comparative accuracy of pleural fluid unstimulated interferon-gamma and adenosine deaminase for diagnosing pleural tuberculosis: a systematic review and meta-analysis. PLoS One. 2021 Jun 24;16(6):e0253525. https://doi.org/10.1371/journal.pone.0253525