PULMONARY HYPERTENSION IN ACTIVE PULMONARY TUBERCULOSIS PATIENTS.

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:***ABSTRACT : Background:*** *Pulmonary tuberculosis is one of the commonest infectious diseases which are encountered in clinical practice, more so in developing countries. Very few studies have been reported regarding the pulmonary hypertension in active pulmonary tuberculosis.*

Aims of study: *To assess the incidence of pulmonary hypertension in active pulmonary tuberculosis patients in the absence of other lung disease or primary cardiac disease.*

Materials and Methods: *During the period from 1st of December 2016 to 30th of May 2017, a cross-sectional study was done on 50 patients with active pulmonary tuberculosis. For all patients, detailed history, careful clinical examination, electrocardiography (ECG), chest x ray (CXR), and routine blood tests were done. All patients evaluated by transthoracic Doppler echocardiography including estimation of pulmonary artery systolic pressure (PASP) and tricuspid annular plane systolic excursion (TAPSE).*

Results: *Among 50 active pulmonary tuberculosis (PTB) patients, 4 had pulmonary artery systolic pressure (PASP) ≥40 mm Hg, 2 of them had right ventricular dilation and TAPSE <16mm. The incidence of pulmonary hypertension (PHT) among active pulmonary tuberculosis (PTB) patients was* ***8%.***

There was a significant association between PTB patients with PHT and higher mean age (p=0.01), higher Modified Medical Research Council dyspnea scale (p<0.0001), chest pain (p=0.01), lower oxygen saturation (SpO2) (p=0.001), sinus tachycardia (p=0.01) and Pericardial effusion (p=0.01).

No significant differences between PTB patients with and without PHT regarding smoking history and CXR finding of pulmonary tuberculosis lesion regarding unilateral and bilateral involvement and cavitary lesion.

Conclusion*: Pulmonary hypertension among active pulmonary tuberculosis patients in the absence of other lung disease or primary cardiac disease is not uncommon in this study*

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INTRODUCTION

Tuberculosis is a common and often deadly infectious disease caused by various strains of mycobacteria, usually *Mycobacterium tuberculosis* in humans (1).

Tuberculosis usually attacks the lungs but can also affect other parts of the body. It is spread through the air when people who have the disease cough, sneeze, or spit (2).

If properly treated, tuberculosis caused by drug-susceptible strains is curable in virtually all cases. If untreated, the disease may be fatal within 5 years in 50–65% of cases (3).

Pulmonary tuberculosis continues to be a major global health problem causing significant morbidity and mortality in spite of modern and effective chemotherapy (4).

TB infection begins when the mycobacteria reach the pulmonary alveoli, where they invade and replicate within the endosomes of alveolar macrophages (1, 9). Tuberculosis is classified as one of the granulomatous inflammatory conditions. Macrophages, T lymphocytes, B lymphocytes and fibroblasts are among the cells that aggregate to form a granuloma, with lymphocytes surrounding the infected macrophages.

Active TB is diagnosed on the basis of a combination of epidemiological (eg, exposure, travel to or residence in a high prevalence area, previous TB), clinical (eg, cough lasting longer than 2-3 weeks,

fever, night sweats, weight loss), radiographic (eg, infiltrates, cavitation), microbiological (eg, positive sputum smear or culture), and histopathologically (eg, caseating granuloma) features. Patients in whom clinical suspicion for TB infections is strong on the basis of clinical criteria should undergo chest radiography. Patients with chest radiographic findings suggestive of pulmonary TB should submit 3 sputum specimens, preferably obtained on different days, for AFB smears and culture8. At least 1 early morning sputum specimen should be submitted.

Radiography demonstrates infiltrates in the apical-posterior segment of the upper lobes, and up to 20% of these infiltrates are associated with cavities (7, 18). Up to 5% of patients with active pulmonary disease may have normal findings on chest radiography (19, 20).

Active tuberculosis22 means a disease that is caused by Mycobacterium tuberculosis in any part of the body and that is in an active state as determined by either:

1) A smear or culture taken from any source in the person's body tests positive for tuberculosis and the person has not completed the appropriate prescribed course of medication for active tuberculosis disease.

2) Radiographic, current clinical, or laboratory evidence is sufficient to support a medical diagnosis of tuberculosis for which treatment is indicated.

Pulmonary hypertension has been defined as an increase in mean pulmonary arterial pressure (mPAP) ≥ 25 mmHg at rest as assessed by right heart catheterization (RHC) (15). Regardless of the initial genetic or pathogenic trigger, the increased pulmonary vascular resistance seen in pulmonary hypertension can be attributed to the collective effects of sustained vasoconstriction, vascular remodeling, in situ thrombosis, and increased arterial wall stiffness (28).

Clinical Presentation

The **symptoms** of PH are non-specific and mainly related to progressive right ventricular (RV) dysfunction. Initial symptoms are typically induced by exertion. They include shortness of breath, fatigue, weakness, chest pain and syncope. Less commonly patients may also describe dry cough and exercise-induced nausea and vomiting. Symptoms at rest occur only in advanced cases. Abdominal distension and ankle edema will develop with progressing

RV failure. The presentation of PH may be modified by diseases cause or are associated with PH as well as other concurrent diseases15.

The **physical signs** of PH include left parasternal lift, an accentuated pulmonary component of the second heart sound, a pansystolic murmur of tricuspid regurgitation and a diastolic murmur of pulmonary regurgitation. Elevated jugular venous pressure, hepatomegaly, ascites, peripheral edema and cool extremities characterize patients with advanced disease. Wheeze and crackles are usually absent15. **Chapter One Introduction**

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Electrocardiogram

The ECG may be normal and may provide suggestive or supportive evidence of PH by abnormalities range from sinus tachycardia through to overt right ventricular hypertrophy and strain. **RV strain** [[ST depression / T wave inversion in the right precordial (V1-4) and inferior (II, III, aVF) leads]]. **Right bundle branch block** [[Wide QRS >0.12 sec, slurred S wave in lead I and V6, RSR'-pattern in V1 where R' > R]]. **Right axis deviation** [[QRS is positive (dominant R wave) in Lead II, Lead III and aVF QRS is negative (dominant S wave) in Lead I]]. Right atrial enlargement produces a peaked P wave (**P pulmonale)** >2.5 mm in the inferior leads (II, III and AVF) and > 1.5 mm in V1 and V2). The absence of these findings does not exclude the presence of PH nor does it exclude severe haemodynamic abnormalities15.

Transthoracic Doppler Echocardiography

Transthoracic Doppler echocardiography **(DE**) is recommended as the initial noninvasive modality in the screening and evaluation of PH (54).

DE estimates pulmonary artery systolic pressure (PASP) and can provide additional information about the cause and consequences of PH (54). Echocardiography can be used to evaluate right-sided chamber size and function and the presence of pericardial effusion, which is known to impact survival (55-57).

Table 1: Sociodemographic characteristics of PTB patients.

Variable	No.	%
Age mean±SD (40.4±19 years)		
15-19 years	**4**	**8.0**
20-29 years	**15**	**30.0**
30-39 years	**8**	**16.0**
40-49 years	**4**	**8.0**
≥50 years	**19**	**38.0**
Total	**50**	**100.0**
Gender		
Male	**26**	**52.0**
Female	**24**	**48.0**
Total	**50**	**100.0**
Occupation		
Unemployed	**14**	**28.0**
Retired	**5**	**10.0**
Public servant	**7**	**14.0**
Self employed	**14**	**28.0**
Student	**10**	**20.0**
Total	**50**	**100.0**

Table 2: ECG findings of PTB patients.

Variable	No.	%
Heart rate mean±SD (89.4±19.6 b/m)		
Normal (60-100 b/m)	**30**	**60.0**
Sinus tachycardia>100b/m	**20**	**40.0**
Total	**50**	**100.0**
P. pulmonale		
No	**50**	**100.0**
Total	**50**	**100.0**
RAD		
Yes	**1**	**2.0**
No	**49**	**98.0**

Total	**50**	**100.0**
RV-strain		
No	**50**	**100.0**
Total	**50**	**100.0**
RBBB		
Yes	**1**	**2.0**
No	**49**	**98.0**
Total	**50**	**100.0**

RAD: Right Axis Deviation, RBBB: Right Bundle Branch Block, RV: Right Ventricle.

Table 3: Echocardiography finding of PTB patients.

Variable	No.	%
Pulmonary Artery Systolic Pressure (PASP)		
Normal (<40mmHg)	**46**	**92.0**
Mild (40-50mmHg)	**2**	**4.0**
Moderate (51-70mmHg)	**2**	**4.0**
Severe(>70mHg)	**0**	**-**
Total	**50**	**100.0**
Tricuspid Annular Plane Systolic Excursion (TAPSE)		
Normal	**48**	**96.0**
Abnormal (<16mm)	**2**	**4.0**
Total	**50**	**100.0**
Evidence of right atrium dilation		
No	**50**	**100.0**
Total	**50**	**100.0**
Evidence of right ventricle dilation		
Yes	**2**	**4.0**
No	**48**	**96.0**
Total	**50**	**100.0**
Pericardial effusion		
Yes	**6**	**12.0**
No	**44**	**88.0**
Total	**50**	**100.0**
PHT		
Yes	**4**	**8.0**
No	**46**	**92.0**
Total	**50**	**100.0**

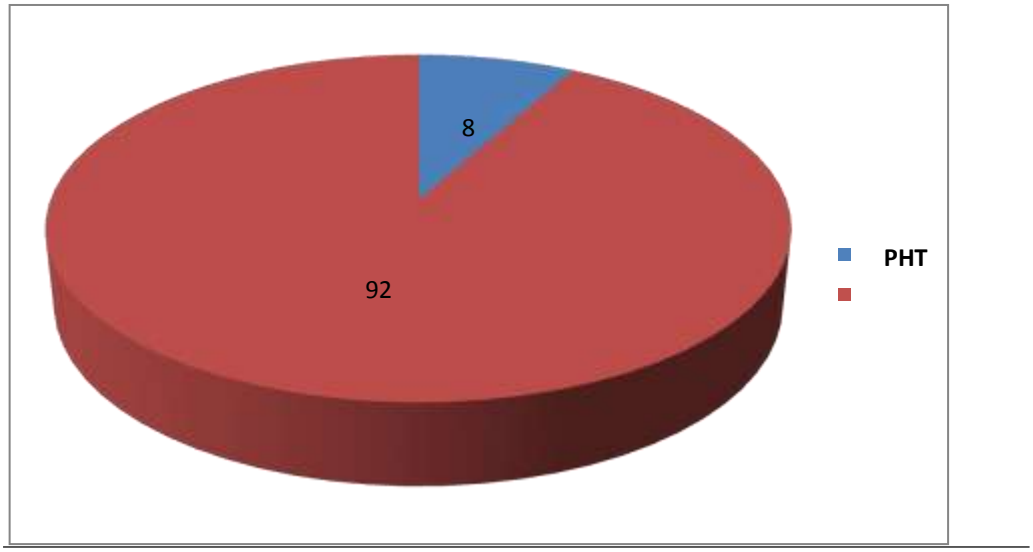

Figure 1: Distribution of PHT among PTB patients.

Table 4: Distribution of Sociodemographic characteristics of PTB patients according to PHT.

Variable	PHT		No PHT		P value
	No.	%	No.	%	
Age					
Mean±SD	63.5±6.9		38.4±18.3		**0.01****
Gender					0.9*
Male	2	50.0	24	52.2	
Female	2	50.0	22	47.8	
Occupation					0.4*
Unemployed	2	50.0	12	26.1	
Retired	0	-	5	10.9	
Public servant	0	-	7	15.2	
Self-employed	2	50.0	12	26.1	
Student	0	.0	10	21.7	

** Fisher's exact test, **t-test.*

Table 5: Distribution of mMRC dyspnea scale and SpO2 of PTB patients according to PHT.

Variable	PHT		No PHT		P value
	No.	%	No.	%	
mMRC dyspnea scale					**<0.0001***
<2	0	-	42	91.3	
≥2	4	100.0	4	8.7	
Mean±SD	2.3±0.5		0.9±0.6		**<0.0001****
SpO2					**0.001***
≥95%	1	25.0	41	89.1	
<95%	3	75.0	5	10.9	
Mean±SD	91.8±4.2		96.5±2.5		**0.001****

**Fishers exact test, **t-test. mMRC: Modified Medical Research Council, SpO2: Oxygen saturation*

Table 6: Distribution of clinical features of PTB patients according to PHT.

Variable	PHT		No PHT		P value
	No.	%	No.	%	
Fever					0.5*
Yes	4	100.0	42	91.3	
No	0	-	4	8.7	
Night Sweats					0.8*
Yes	3	75.0	32	69.6	
No	1	25.0	14	30.4	
Weight loss					0.2*
Yes	4	100.0	35	76.1	
No	0	-	11	23.9	
Anorexia					0.08*
Yes	4	100.0	26	56.5	
No	0	-	20	43.5	
Generalized weakness					0.7*
Yes	3	75.0	37	80.4	
No	1	25.0	9	19.6	
Cough					0.1*
Yes	4	100.0	32	69.6	
No	0	-	14	30.4	
Hemoptysis					0.1*
Yes	0	-	14	30.4	
No	4	100.0	32	69.6	
Chest pain					**0.01***
Yes	4	100.0	16	34.8	
No	0	-	30	65.2	

** Fisher's exact test.*

Table 7: Distribution of smoking status and CXR findings of PTB patients according to PHT.

Variable	PHT		No PHT		P value
	No.	%	No.	%	
Smoking					**0.8***
Never	**3**	**75.0**	**33**	**71.7**	
Current	**1**	**25.0**	**9**	**19.6**	
Former	**0**	**-**	**4**	**8.7**	
Pack year					**0.2***
≤10 pack year	**0**	**-**	**8**	**61.5**	
>10 pack year	**1**	**100.0**	**5**	**38.5**	
Mean±SD	**30±00**		**13.6±8.4**		**0.08****
Unilateral involvement					**0.2***
No	**2**	**50.0**	**10**	**21.7**	
Right	**0**	**-**	**20**	**43.5**	
Left	**2**	**50.0**	**16**	**34.8**	
Bilateral involvement					**0.2***
Yes	**2**	**50.0**	**10**	**21.7**	
No	**2**	**50.0**	**36**	**78.3**	
Cavitary lesion					**0.5***
Yes	**1**	**25.0**	**19**	**41.3**	
No	**3**	**75.0**	**27**	**58.7**	

**Fishers exact test, **t-test.*

Table 8: Distribution of ECG characteristics and echocardiography finding of Pericardial effusion of PTB patients according to PHT.

Variable	PHT		No PHT		P value
	No.	%	No.	%	
Heart rate					**0.01***
Normal	0	-	30	65.2	
Sinus tachycardia	4	100.0	16	34.8	
Mean±SD	108.7±2.5		87.7±19.5		**0.03****
RAD					**0.001***
Yes	1	25.0	0	-	
No	3	75.0	46	100.0	
RBBB					**0.001***
Yes	1	25.0	0	-	
No	3	75.0	46	100.0	
Pericardial effusion					**0.01***
Yes	2	50.0	4	8.7	
No	2	50.0	42	91.3	

**Fishers exact test, **t-test. RAD:Right Axis Deviation, RBBB: Right Bundle Branch Block*

Tricuspid annular plane systolic excursion (TAPSE) is a validated parameter of global right ventricular (RV) function (11). Echocardiographic assessment of (RV) function remains difficult because of its complex geometry. Standard echocardiographic parameters of RV function, such as ejection fraction (RVEF), are excellent theoretical entities but due to suboptimal RV endocardial definition may have limited value in current clinical practice (13).

TAPSE correlates closely with the RVEF52 and has been found to be both highly specific and easy to measure (53).

Estimated PASP derived from tricuspid regurgitant jet velocity is not prognostic. The tricuspid annular plane systolic excursion (TAPSE) and pericardial effusion have been reported to be of prognostic value (15).

The pathogenesis of PHT in active pulmonary tuberculosis is not clear. Active tuberculosis by unknown mechanisms may be directly responsible for rising pulmonary arterial pressure. PH may be related to parenchymal abnormalities, inflammation and hypoxia in the course of the disease especially in case of delayed diagnosis and initiation of anti-TB treatment (29).

Many authors stated that the possible causes for the development of PH in these patients are the destruction of vascular bed due to parenchymal abnormalities, inflammation, vasculitis, and endarteritis, leading to the reduced cross-sectional area of the pulmonary vasculature (27).

Aims of Study

1. To assess the incidence of pulmonary hypertension in active pulmonary tuberculosis patients in the absence of other lung disease or primary cardiac disease.
2. To compare different variables (as age, gender, dyspnea scale and oxygen saturation) among active pulmonary tuberculosis patients with and without pulmonary hypertension.

Patients & Methods

Study design & settings

A cross sectional study carried out in the Chest and Respiratory Disease National Specialized Center in Baghdad for a period from 1st of December 2016 to 30th of May 2017.

Inclusion Criteria

Patients with active pulmonary tuberculosis. Tuberculosis was considered active when a patient with the symptoms, signs and radiologic lesions of pulmonary tuberculosis, with positive Ziehl Neelsen staining of sputum showing acid-fast bacilli (AFB).

Exclusion Criteria

1. Patients with a history of previous treatment for tuberculosis were not studied in order to exclude those with previous lung fibrosis.
2. Primary lung diseases other than pulmonary tuberculosis
3. All primary cardiac diseases
4. Hypertension.
5. Other causes of pulmonary hypertension were considered in the exclusion criteria

Sampling

A convenient sample of fifty patients with the symptoms, sign and radiological lesions of pulmonary tuberculosis with positive Ziehl Neelsen staining of sputum showing acid-fast bacilli presented to the Chest and Respiratory Disease National Specialized Center in Baghdad was the sample of the study.

Data collection

The data were collected in the specially designed questionnaire filled by the researcher. Diagnosis of active pulmonary tuberculosis was made on symptoms, sign and radiological lesions of pulmonary tuberculosis with positive Ziehl Neelsen staining of sputum showing acid-fast bacilli.

An oral permission from the individuals who participate in this study was taking and all of them were wearing the mask .

Early morning sputum was collected in a sterile container on three consecutive days. The sputum smears were prepared and stained by the Ziehl-Neelsen method and acid-fast bacilli were identified.

A detailed history was taken and a detailed clinical examination was conducted.

The main data and parameters included in our study are the followings:

1. Sociodemographic characteristics: Age, gender and occupation.

2. **Smoking status**: current, never and former smoker12.

- **Current smoker:** An adult who has smoked 100 cigarettes in his or her lifetime and who currently smokes cigarettes at least monthly.
- **Never smoker:** An adult who has never smoked, or who have smoked less than 100 cigarettes in his or her lifetime and does not currently smoke.
- **Former smoker:** An adult who has smoked at least 100 cigarettes in his or her lifetime but who had quit smoking at the time of interview.

3. Pack-years: A way to measure the amount a person has smoked over a long period of time. It is calculated by multiplying the number of packs of cigarettes smoked per day by the number of years the person has smoked16.

4. Clinical presentation.

5. Modified Medical Research Council dyspnea scale (mMRC) dyspnea scale.

RESULTS

A total of 50 active pulmonary tuberculosis (PTB) patients were included in this study with mean age of 40.4±19 years; Males were more than females with the male to female ratio as 1.08:1. The occupation of PTB patients was distributed as followings; unemployed (28%), self-employed (28%), students (20%), public servants (14%) and retired (10%). All these findings were shown in table 1.

The ECG findings of PTB patients included the mean heart rate of 89.4±19.6 b/m, 40% of PTB patients had sinus tachycardia. No PTB patient had P. pulmonale and RV-strain. Only one PTB patient had RAD and one PTB patient had RBBB. All these findings were shown in table 2.

The echocardiography findings of PTB patients included the followings; four PTB patients had raised PASP, so Pulmonary hypertension (PHT) was found among 8% of PTB patients. Two PTB patients had mild PHT, two patients had moderate PHT and no patient had severe PHT. Two of PTB patients with raised PASP had abnormal TAPSE (<16mm) and RV dilation. No PTB patient had RA dilation. Pericardial effusion was detected among six PTB patients. All these findings were shown in table 3-3 and figure 1.

Mean age of PTB patients with PHT was significantly higher than PTB patients with no PHT (p=0.01). No significant differences between PTB patients with and without PHT regarding their gender and occupation. All these findings were shown in table 4.

There was a highly significant association between higher mMRC dyspnea scale of PTB patients and PHT (p<0.0001). A significant association was observed between lower SpO2 of PTB patients and PHT (p=0.001). All these findings were shown in table 5.

As shown in table 6, no significant differences between PTB patients with and without PHT regarding fever, night sweats, weight loss, anorexia, generalized weakness, cough and hemoptysis. A significant association was observed between PTB patients with PHT and chest pain (p=0.01).

As shown in table 7, no significant differences between TB patients with and without PHT regarding smoking status & pack-year and CXR finding of PTB lesion regarding unilateral and bilateral involvement and cavitary lesion.

As shown in table 8, there was a significant association between PTB patients with PHT each of sinus tachycardia and Pericardial effusion (p=0.01). There was a significant association between TB patients with PHT and each of RAD and RBBB (p=0.001).

Discussion

The present study in 50 active pulmonary tuberculosis patients, four patients had pulmonary artery systolic pressure ≥40 mm Hg, two of them had the right ventricular dilation and TAPSE <16mm.

The present study showed that pulmonary hypertension (PHT) incidence among patients with active pulmonary tuberculosis (PTB) was 8%. This incidence is close to the result reported by Marjani et al 29 study in Iran of 9.5%.

In a study with 52 new cases of pulmonary tuberculosis 44.2% had systolic pulmonary artery pressure above 25 mmHg by echocardiography. All the patients were young and the majority of them had disseminated disease or involvement of intra-thoracic lymphatics46.

In Patel et al.44 study have described PH in six out of 50 (12%) cases of PH to develop from pulmonary tuberculosis (active/healed).

The pathogenesis of PHT in active pulmonary tuberculosis remains poorly understood. Active tuberculosis by unknown mechanisms may be directly responsible for rising pulmonary arterial pressure. PHT may be related to parenchymal abnormalities, inflammation and hypoxia in the course of the disease especially in case of delayed diagnosis and initiation of anti-TB treatment (29).

Many authors stated that the possible causes for the development of PH in these patients are the destruction of vascular bed due to parenchymal abnormalities, inflammation, vasculitis, and endarteritis, leading to a reduced cross-sectional area of the pulmonary vasculature (27).

In our study, two of active PTB patients (4%) had RV dilation and TAPSE <16mm.

A similar incidence of RV dilation was found in Rajesh et al 51 study 50 cases of sputum-positive pulmonary tuberculosis 2 had RV dilation.

In Dasti et al 21 study 3 out of 50 cases of sputum-positive pulmonary tuberculosis had RV dilation.

In Kotresh et al 50study in India, 10 out of 100 patients (10%) had TAPSE < 16mm, this higher result probably due to that all cases of pulmonary tuberculosis, irrespective of duration and type of treatment received included in the study

The four PTB patients with PHT in our study were distributed according to the PASP level as followings; two patients with mild PHT (40-50mmHg) and two patients with moderate PHT (51-70mmHg) . No PTB patient had severe PHT (>70mmHg) .

In Ahmed et al (34) study in Sudan 14 patients who were treated for PTB and were found to have PHT, were distributed as followings; 4 patients with mild, 9 patients with moderate and 1 patient with severe PHT. Their different from ours as none of their cases had active PTB and the mean interval since PTB diagnosis was 9.4 years.

The pericardial effusion was in 12% of PTB patients in this study. This is similar to results of Casas et al 35 study in Spain which found that 14.1% of patients with tuberculosis would have pericardial effusion. Previous South Korean study 36 documented that pulmonary tuberculosis represented one of the common etiologies of pericardial effusion. The anatomical relationship between the pulmonary system and the pericardium may determine the existence of pericardial effusion as this occurs most

commonly following contiguous areas of adjacent lymph nodes and less often due to haematogenous spread (59).

In our study, the pericardial effusion was significantly higher among those PTB patients with PHT (p=0.01).

Current study reported a significant association between elderly age PTB and development of PHT (p=0.01). Akgün et al 37study in USA revealed that pulmonary diseases were more prevalent and more severe in symptoms and outcomes among elderly patients. Despite equal age distribution of respiratory diseases on population, the prevalence of lung diseases is increased with the increase of population age and it is till now underestimated in older persons 38. Atypical clinical manifestations of tuberculosis in older persons can result in the delay in diagnosis and initiation of treatment; thus, unfortunately, higher rates of morbidity and mortality from this treatable infection can occur (47).

The mMRC dyspnea scale of PTB patients in our study was significantly higher among those PTB patients with PHT (p<0.0001).

This is similar to results of Soliman et al 45 study which documented that dyspnea scale is significantly correlated positively with pulmonary hypertension.

In Patel *et al*.44 study dyspnea on exertion was the commonest symptom in pulmonary hypertension patients, dyspnea on exertion may be due to underlying pulmonary hypertension and associated respiratory diseases.

Oxygen saturation (Spo2) at rest of PTB patients with PHT in this study was significantly lower than PTB patients without PH (p=0.001).

Some authors suggested an important role of hypoxia. Hypoxia, of all pathogenic factors, represents the most serious limitation of pulmonary diffusion, though other agents may not be primarily important for the onset and development of pulmonary hypertension23.

This finding is in agreement with results of Raina et al (43) study in India which found that oxygen saturation percentage is commonly lower among PTB patients with deteriorated status, respiratory distress and pulmonary hypertension.

Chest pain was a significant symptom of PTB patients with PHT in our study (p=0.01). This is consistent with results of Marjni et al 29 study in Iran.

The electrocardiography was done in all 50 PTB patients in our study, sinus tachycardia was seen in 20 patients (40%). In Dasti et al 21 study the electrocardiography showed sinus tachycardia in 22 out of 72 patients (30.55%). Tachycardia probably was due to fever, toxaemia and anaemia.

Current study reported a significant association between sinus tachycardia and development of PHT (p=0.01).

In our study, only one PTB patient had right axis deviation and one PTB patient had right bundle branch block, both patients had PHT.

Conclusions

- Pulmonary hypertension among active pulmonary tuberculosis patients in the absence of other lung disease or primary cardiac disease is not uncommon.
- There was a significant association between pulmonary hypertension and elderly age, high dyspnea scale and low oxygen saturation.
- There was a significant association between pulmonary hypertension and chest pain.
- There was a significant association between pulmonary hypertension and sinus tachycardia, pericardial effusion, right axis deviation and right bundle branch block.

RECOMMENDATIONS

- Public education on risks of tuberculosis and its sequale with preventive measures is mandatory for vulnerable population groups (e.g. prisoners).
- Encouraging peoples infected with tuberculosis on adhesion to treatment protocols and echocardiography monitoring especially those peoples at risk of pulmonary hypertension (e.g. elderly).
- Further national wide sized studies on the prevalence of pulmonary hypertension among active pulmonary tuberculosis must be supported

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