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Co-Existence of Tuberculosis and Lung Cancer

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Abstract

Background: Tuberculosis (TB) and lung cancer cause significant morbidity and mortality worldwide and pose a global health threat. Each year these two diseases account for more than 1.6 million deaths worldwide. The incidence of both diseases is still high in many developing countries, especially in Asian countries. TB and lung cancer are often confused and misdiagnosed, especially in countries with diagnostic challenges of low TB incidence and risk of missed diagnosis.

Case Presentation: The following is a case report of a 53-year-old male patient diagnosed with pulmonary TB accompanied by right lung cancer, and the same respiratory complaints can be had by lung cancer and TB. However, the presence of facial edema (part of the superior vena cava syndrome) causes clinicians to focus more on lung cancer so that the diagnosis of TB is often overlooked.

Conclusion: Tuberculosis should be a significant concern, especially in patients with malignancies such as lung cancer and located in TB endemic areas. Delay in diagnosis and or miss diagnosis will affect the patient's outcome.

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INTRODUCTION

Tuberculosis (TB) with lung cancer incidence is a significant clinical problem in countries with a high prevalence of TB.[1] Tuberculosis can increase the risk of lung cancer through substantial and prolonged lung inflammation.[1] Epidemiologically, TB is recorded in 2-5% of lung cancer cases, while lung cancer is recorded in 1-2% of TB cases. [2] Shiels' research states that TB will increase the risk of lung cancer in male smokers.[1] Bae's study states that there is a relationship between male sex and the Brikman Index (IB) of heavy smokers with lung cancer incidence in TB history present.[3] The above data suggest that male patients who smoke should be considered not only For lung cancer risk factors but also for the incidence of TB. Another factor that must be considered is that TB is a great imitator, so delays in diagnosis and treatment of TB / lung cancer result in poorer outcomes and lower survival rates.[4] We report a case of TB and lung cancer co-existence to see differences and similarities between TB and lung cancer.

CASE PRESENTATION

A male patient aged 53 y.o came with the chief complaint of shortness of breath since three months ago, not wheezing, increasing with activity, cough previously the patient had been treated for five days at Pariaman Hospital and then referred to Dr. M. Djamil Padang for further management. Cough since three months ago, no phlegm and intermittent. No coughing up blood, no history of coughing up blood. The chest pain has been felt since two months ago, and it is felt in the right chest and does not spread. Night sweats denied. Since one month, there has been a decrease in appetite and a weight loss of ± 5 kg in these two months. Swallowing pain is absent. Hoarse voice has been there for two weeks. The patient's face has been puffy for one month: no nausea, vomiting, urination, and defecation in a normal state. The patient is a cake seller, lives in Pariaman, smokes 20 cigarettes per day for 40 years, and quits these two months. There was no history of hypertension, diabetes mellitus, TB, and no history of malignancy of other organs. There is also no family history of hypertension, diabetes mellitus, TB, or malig-nancy.

Physical examination was moderately ill with a general condition of cooperative composmentis. Blood pressure 130/90, respiration rate 22x/minute, pulse 98x/minute, temperature 36.8^{o} celsius, body weight 60 kg, and height 165 cm. On physical examination, the face looks swollen, with no palpable enlarged lymph nodes. Pulmonary examination by inspection showed venectation on the chest, the right chest looked convex from the left (static) and the right-site chest movement delayed from the left-site (dynamic), palpation revealed weakened right fremitus from the left, dullness in right site percussion, and left site sonor, auscultation on the left bronchovesicular breath sounds no rhonchi no wheezing, and on the right-site breath, sounds weaken until disappear. No clubbing finger was found in the extremities, and no limb edema was found.

Laboratory results obtained: Hemoglobin 13,2g/dl, Leucocyte 10.150x10^3/mm^3, Different counting: 0/0/0/87/7/6, Thrombocytes 338.000 x10^3/mm^3, Hematocrit 42%, SGOT 29µ/L, SGPT 61µ/L, PT 10,6seconds, APTT 21seconds, Ddimer 479µg/mL, Bilirubin Total 0,8mg/dL, Bilirubin Direct 0,4mg/dL, Indirect 0,4mg/dL, Ureum 30mg/dL, Creatinin 0,9 mg/dL, Natrium 138mmol/L, Calium 3,6 mmol/L, Cloride 98 mmol/L.

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The patient was diagnosed with suspect right lung cancer unknown cell type T4NxM1a (pleural effusion) stage IV PS ECOG 1 with SVCS. The patient was treated with IVFD NaCl 0.9%/12 hours, furosemide injection 1x1amp, dexamethasone injection 3x1amp, n.acetylcysteine 200mg 2x1 (p.o). Patients were planned for sputum cytology, thoracentesis, chest ultrasound, bronchoscopy with preparation, chest CT scan with contrast.

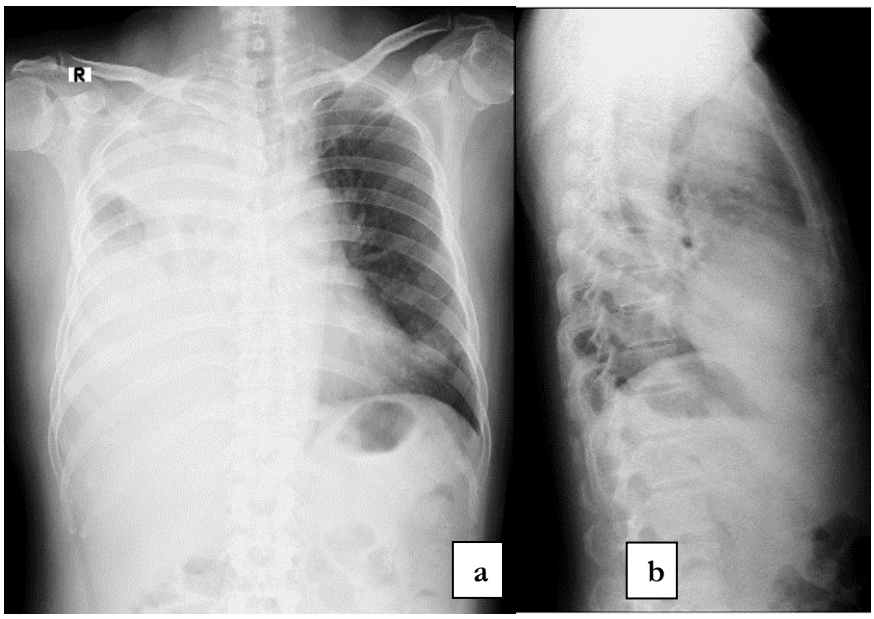

Figure 1. Chest X-ray: Right pleural effusion with atelectasis component a. Posterior Anterior position, b. Lateral position

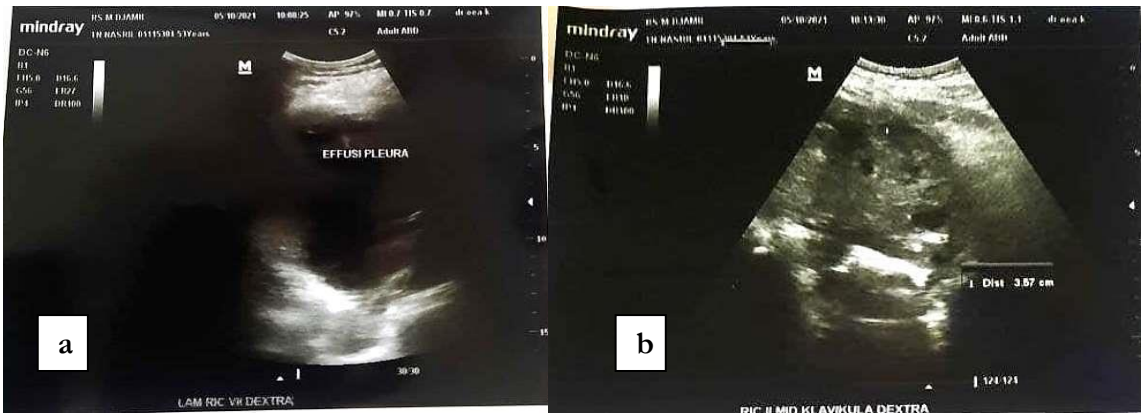

Figure 2. Thoracic ultrasound (a) Right pleural effusion, and (b) right lung mass

On the second day of hospitalization, a thoracic ultrasound was performed; right pleural effusion and right lung mass. Then the patient was done thoracocentesis of 100cc hemorrhagic obtained analysis of pleural fluid exudate chronic process as listed at table 1. Then a cytologic examination of the pleural fluid was performed. The patient was planned for bronchoscopy under general anesthesia pro bronchoscopy and cryobiopsy.

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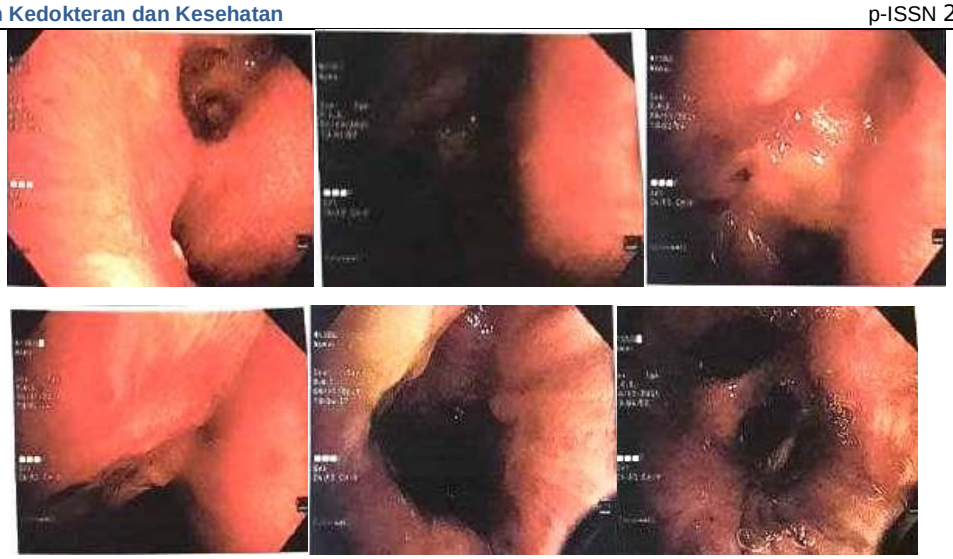

Figure 3. Macroscopic overview of bronchoscopy

Table 1. Pleural Fluid Analysis

	Pleural Fluid	**Serum**
Glukosa	142.5	167
Protein	2.9	4.2
Albumin	1.6	3.4
LDH	1292	408
PMN/MN	15/85	
Rivalta	(+)	
Expertise	**Exudate Chronic**	

The fifth day of treatment. Pleural fluid cytology results: no malignant cells were found. Figure 3 showed bronchoscopy, in trachea: open, smooth mucosa and not hyperemic. Carina: Symmetrical taper. Second Carina : open, smooth mucosa, hyperemia, intraluminal mass is seen less than 2cm from the carina, bleeds easily, rinse and cryobiopsied. Truncus intermedius: open smooth mucosa not hyperemic. Lower Medius Dextra: open, smooth mucosa and not hyperemic. Lower Bronchus Dextra: open, smooth, and not hyperemic mucosa. Main Bronchus Sinistra: open, smooth mucosa and not hyperemic. Lower Bronchus Sinistra : open, smooth, and not hyperemic mucosa. Conclusion: Mass in the right main bronchus (T4), then a post bronchoscopy brushing and cryobiopsy were performed.

On the sixth day of treatment, the gene Xpert sputum TB results: Mtb detected medium and rifampin resistance not detected. The patient was then diagnosed with right lung cancer with unknown cell type T4NxM1a (pleural effusion + contralateral nodule) stage IV PS ECOG 1 with SVCS + new case lung TB bacteriolo-gically confirmed. Patients are planned to treat with anti TB drugs sensitive starting tomorrow morning.

On the seventh and eighth day of treatment, Clinically, there is nausea, no vomiting, shortness of breath has reduced, the patient can lie

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down supine. A bedsite ultrasound was returned to view the pleural fluid, and the impression was minimal, and a sample for ADA could not be obtained. The patient was then discharged and planned to evaluate OAT treatment and wait for the results of cytology and histopathology. The patient was planning for a chest CT scan with contrast. The control patient was brought to the polyclinic with the results of histopathological bronchoscopy. Cytological results of bronchial washings showed no malignant tumors were found, but the results of cryobiopsy showed a microscopic picture showing a combination of small cell carcinoma and large cell neuroendocrine carcinoma.

DISCUSSION

Tuberculosis is an infectious disease caused by Mycobacterium Tuberculosis infection as described at figure 4.[5]

More than one-third of the world's population is infected with TB germs. In 2018, global TB Reports reported 1.3 million deaths in HIV-negative TB patients and 374,000 deaths in HIV-positive TB patients worldwide.[7] Lung cancer is the second leading cause of malignancy globally after breast cancer. In 2020 according to a report from Globocan, there were 19.3 million new cases, of which 11.4% were cases of lung cancer and accounted for the highest number of all cancers at 18%. The prevalence of lung cancer is relatively high in developing countries, which is as much as 58%.[9] Incidence of lung cancer in Asia reported as many as 1.03 million cases with 926,000 deaths in 2012. The most burden of lung cancer in Asia is in countries in Southeast Asia.[8]

The prevalence co-existence of lung cancer with TB is not yet fully known. A report from the National Cancer Institute shows a 2% prevalence of TB and lung cancer co-existence and is most commonly found in the superior lobes of the lung. Saulius et al. reported TB co-existence in 2.1% of patients diagnosed with lung cancer in Lithuania during 1990-2015.[10] Beyhan et al. reported a higher prevalence of 4% of lung cancer and TB co-existence cases in 374 patients from 2009 to 2014 in Turkey. Although the two rarely occur together, a relationship has been established between them. A study conducted by the National Cancer Institute found that patients with TB had an increased risk of lung cancer, and others estimated a two-times increase in the risk of lung cancer in men with TB.[11] It spreads through the air when people who have an active MTB infection cough, sneeze or transmit their saliva through the air. Lung cancer is a complex etiological disease in which several genes are involved in pathogenesis through different pathways. When these genes interact with environmental factors, individuals can develop lung cancer.[4] There are several common risk factors such as smoking for TB and lung cancer. Smoking can facilitate the manifestation of adverse effects of TB through a variety of mechanisms. First, smokers tend to experience chronic cough, a typical symptom of TB. A later diagnosis of tuberculosis can be delayed leading to a worse prognosis and a higher chance of relapse. Second, smoking is a cause of comorbidities, such as chronic bronchitis, chronic airway obstruction, pulmo-nary emphysema, and coronary heart disease, which can facilitate the development of TB infection into disease, but also interfere with lung function in the absence of TB disease alone, leading to a worse prognosis.

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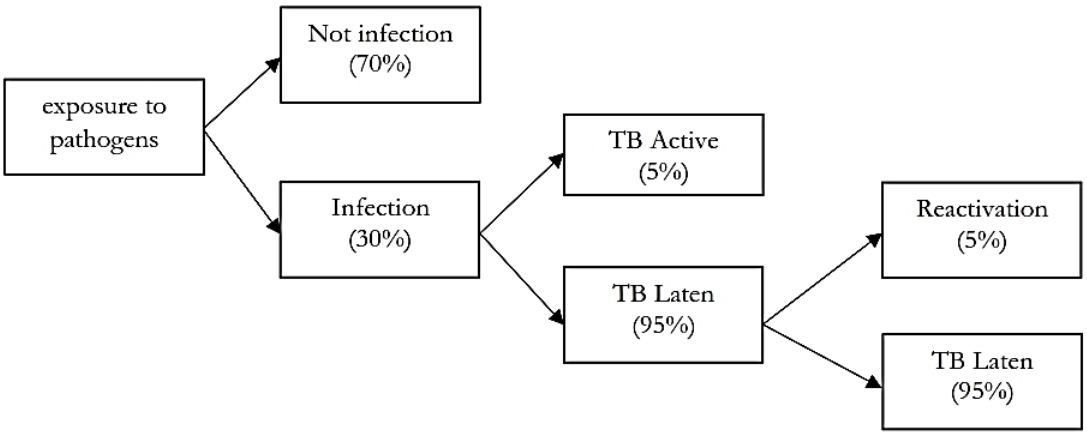

Figure 4. The percentage of people exposed to TB germs that develop into TB disease. [5]

Third, excess iron in macrophages in lung tissue is discussed as a direct effect impairs cellular response to microorganisms. And finally, one could speculate that smokers had poor therapy adherence to therapy, at least in certain places or in certain areas, although this may not be a problem in areas that use directly observed therapy for most or all patients.[4]

Smoking is the most critical risk factor for lung cancer. In patients with lung cancer, a history of active smoking was found in 87% of men and 85% of women. There are ten times increased risk of lung cancer in smokers and 20 times more risk in heavy smokers (>20 sticks/day). The relative risk of developing lung cancer was 2.64 for kretek smokers and 2.23 for cigarette smokers, with 2.45 as overall relative risk.[4] In these patients include smokers with a heavy brikman index. The all risk factors for TB and lung cancer can be seen ata table 2.

Diagnosis of TB and lung cancer is classified by time as follows:[12] 1. Simultaneous (simultaneous) – when the diagnosis of tuberculosis and lung cancer coincides or when the time between the two diagnoses < 2 months; 2. Sequential (sequential): a. First lung cancer – when tuberculosis is diagnosed after lung cancer diagnosis and within 12 months after completion of lung cancer treatment; b. First tuberculosis – when lung cancer is diagnosed two months after the diagnosis of tuberculosis, indefinitely, due to the possibility of cancer scars and chronic inflammation. The incidence of TB and lung cancer coexisting is called co-existence. However, the co-existence of TB and lung cancer remains controversial. Chronic inflammation due to TB is thought to be responsible for the occurrence of cancer. Cancer and TB co-existence can cause delays in diagnosis. Patients with cancer are susceptible to developing TB due to immuno-suppression due to the use of intensive treatment modalities, such as aggressive chemotherapy, radiotherapy, or malnutrition.[13]

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Table 2. Risk factors for TB and lung cancer:[4]

Tuberculosis	Lung cancer
1. An known history of close contact with someone suffere from TB. Breathing air from an infected person is proportional to the amount of time spent in the same air circulation, the proximity of the person, and the level of ventilation. 2. Immunocompromised status (e.g., those infected with HIV, cancer, organ trans-plants, and prolonged high doses of corti-costeroid therapy) 3. Substance abuse (injectionecting or IV drug users and alcoholics). 4. Everyone without adequate health care (homelessness, especially children under the age of 15 and young adults between the ages of 15 and 44) 5. Preexisting medical conditions or specialized treatments (e.g., diabetes mellitus, chronic kidney failure, malnutrition, hemodialysis, or organ transplantation) 6. Institutionalization (e.g., long-term care facilities, psychiatric institutions, prisons) 7. Living in overcrowded and substandard housing 8. Become a health worker who performs high-risk activities: Administration of pentamidine aerosols and other drugs, sputum induction procedures, bronchoscopy, suction, cough procedures, treating patients with immunosuppression, and administration of anesthesia and related procedures (e.g., intubation, suction)	1. Patients aged >40 years with a history of smoking ≥30 years and stop smoking within 15 years before the examination or patients ≥50 years with a history of smoking ≥20 years and the presence of at least one other risk factor. 2. Exposure to radiation/atmospheric agents and jobs have known carcino-gens such as Radon (an established lung carcinogen), asbestos, arsenic, bischlo-rometil ether, chromium, nickel, polycyclic aromatic compounds 3. Occupational exposure to carcinogenic chemicals 4. History of cancer in patients or families 5. An environment of Tobacco Smoker (ETS) 6. Exposure to certain metals (chromium, cadmium, arsenic), some organic chemi-cals, radiation, air pollution 7. Medical history of tuberculosis 8. Cytogenetic studies have identified many chromosomal changes in lung cancer with numerical abnormalities and structural deviations, including deletion and translocation. Small cell lung can-cers are associated with oncogenes, such as c-myc, L-myc, N-myc, c-raf, and tumor suppressor genes, such as p53 and Rb. Non-small cell lung cancers are associated with the K-ras, N-ras, H-ras, c-myc, c-raf, and suppressor tumors such as the p16 and rb genes.

Lung cancer is associated with inflammatory diseases such as pneumonia and tuberculosis. The mechanism of the relationship between the two remains unclear, but the inflammatory process and involvement of macrophages are considered important in disease development. Tuberculosis can increase the risk of lung cancer through substantial and prolonged lung inflammation, leading to host tissue damage, fibrosis, scar formation, and genetic changes. The incidence of lung cancer accompanied by TB has been clearly described in several pieces of literature, ranging from autopsy reports to epidemiological data.[9] Some studies show that TB is associated with an increased risk of lung cancer, especially adenocarcinoma, but a study from Vesna et al. suggests adenocarcinoma ranks third after planocarcinoma and squamous cell carcinoma as described at figure 5. In these cases, small cell carcinoma became the four highest cases in the incidence of TB. The biological link between TB and lung cancer broadly focuses on the role of chronic inflammation and fibrosis in lung carcinogenesis, as in figure 6.

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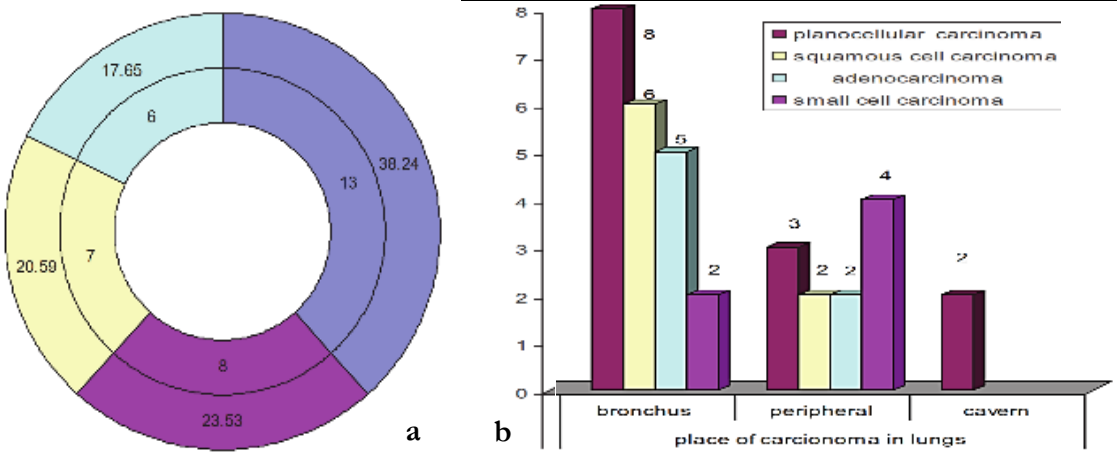

Figure 5. The ratio between histology and the location of lung cancer in TB patients.[13] a) histologic rasio of lung cancer, and b) location of lung cancer

Cytokines, in particular Tumor Necrosis Factor (TNF) and Interleukin-1 (IL-1) mediated by infiltration of lymphocytes and macrophages, will lead to the proliferation of pulmonary epithelial cells. Tb infection will occur in the process of DNA damage due to the role of Nitrite Oxide Synthase (NOS) synthesized by infected macrophages.[15] Reactive Oxygen Spe-cies (ROS) that appear in inflammatory cells will cause rupture of chromosome chains and accumulated changes in deoxyribose nucleic acid (DNA) mutations. Oxidative stress increases the activity of the E2 factor resulting in increased cell proliferation. Recent research reported that Mtb germs can synthesize B-cell lymphoma 2 (BCL-2), antiapoptosis, thus preventing the death of infected cells. Mtb germs can also increase the activity of vascular endothelial growth factor (VEGF), angiogenic that plays a role in the neovascularization of tumor cells.[16] Increase in cell proliferation also occurs due to Mtb germ resistance in the synthesis of the enzyme p21.[17]

Activation of Nuclear Factor-kappa B (NF-$\varkappa$B) in macrophages infected with TB germs will block the activity of the pro-apoptosis enzyme p53. Resistance to the activity of the p53 enzyme will cause damaged DNA cells that are usually destroyed through apoptosis G2 checkpoint/M cells will still proliferate and cause the onset of cancer cells.[17] TB is associated with EGFR mutations primarily in 19th exon deletion in adenocarcinoma patients. Trials of chronic TB infection in mice may induce squamous cell aggregation in the lungs with the potential for malignancy mediated by DNA damage and epiregulin production. Epiregulin is a peptide hormone bonded to EGFR and Human Epidermal Growth Factor Receptor (HER4) that serves as a signal regulator of proliferation, migration, differentiation, cyto-kine secretion, and innate immunity. Epiregulin is also produced by macrophages infected with TB germs to modulate Toll-like receptor (TLR). The high rate of epiregulin is associated with proliferation, invasion, metastasis, angiogenesis, and apoptosis resistance of cancer cells.[18]

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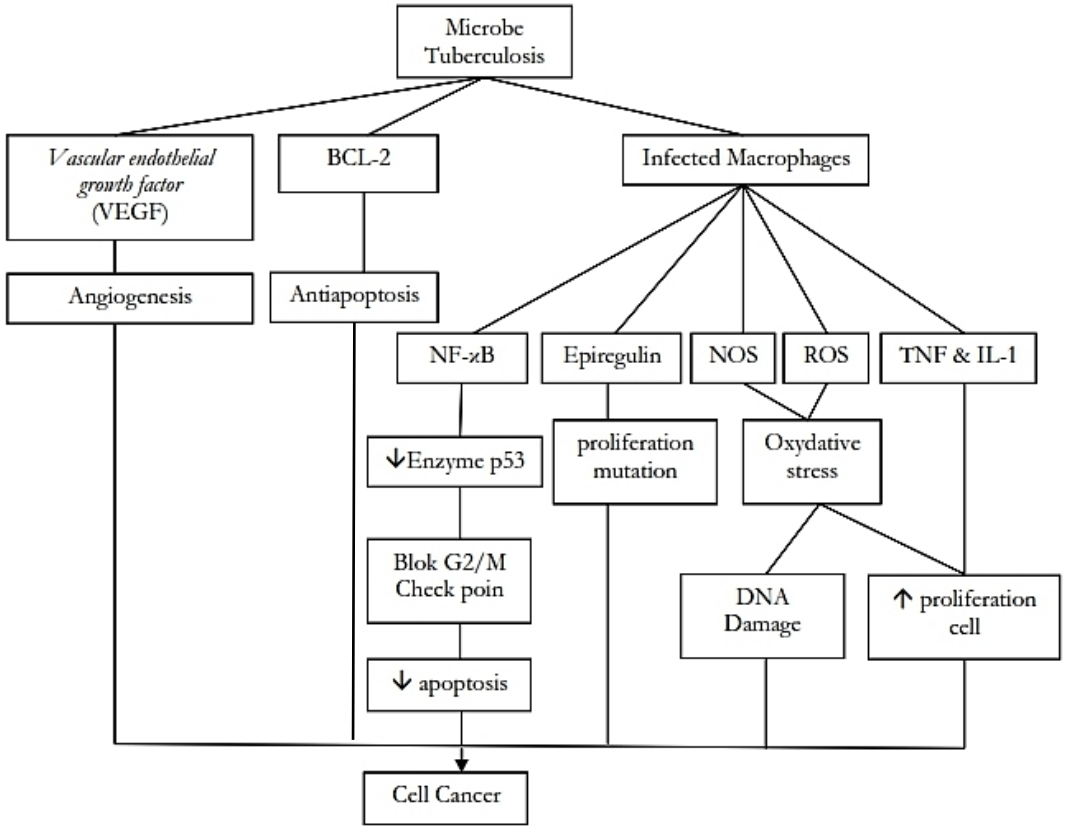

Figure 6. Biological relationship of TB and lung cancer.[14]

Lung cancer, like other malignancies, makes sufferers experience immunocompromised status that can lead to the reactivation of TB germs. Lung cancer risk factors such as smoking can also increase the risk of TB infection. The duration and dose of a cigarette can cause mucous clearance disorders and decrease the ability of macrophages to phagocytes that can cause Mtb germs to escape the pulmonary defense system.[16] Invasion into the focus of old TB lesions by lung cancer cells can also result in TB reactivation. In areas where the prevalence of TB and lung cancer are the same, lung cancer and TB infection can coincide. Radiotherapy in patients with long-standing TB lesions should be done with caution because it can cause granuloma degranulation so that TB germs will proliferate. Radiation damages the DNA of cancer cells and the surrounding healthy cells. The effects of radiation on the immune system can appear on several factors: damage to healthy cells in the radiation area that triggers the body's immune reaction, depletion of peripheral lymphocytes, and changes in the balance of cellular immunity (B cells, T cells, and natural killers).[19] Lung cancer invasion of old TB lesions and systemic treatment in lung cancer is associated with an increased risk of infection or reactivation from TB.[9,27]

CONCLUSION

Tuberculosis should be a significant concern, especially in patients with malignancies such as lung cancer and located in TB endemic areas. Clinical symptoms, radiological appearances that are almost similar require doctors to more intensively and comprehensively think about tuberculosis in malignancy patients, especially epidemiologically in the case of male smokers.

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Delay in diagnosis and or miss diagnosis will affect the patient's outcome. TB screening prior to bronchoscopy should be a mandatory requirement to prevent aerosol transmission during the procedure.

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