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Review Article

Anthracosis, epidemiology, gene and cancer: An updated mini-review

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Abstract

Background: It is believed that anthracosis is one of the most important occupational diseases, mainly seen in the residents of industrial areas and coal mine workers. This bronchial disease is more common in Asia's rural areas, particularly in the Middle East. In this study, we examined the epidemiology and genetic factors affecting this disease and its relationship with different types of cancer.

Methods: In this review article, we searched four databases (Pubmed, the Cochrane Database of Systematic Reviews, Embase and Scopus) up to June 3, 2022, for published articles on anthracosis, epidemiology, gene, and cancer. Non-published studies, studies not published in indexed journals or without peer review, and studies not available in English were all excluded.

Results: The relationship between this disease and tobacco, smoking, air pollution, mycobacterium tuberculosis, gender, and indoor smoke was researched, and its prevalence was cited. Mutations in tumour suppressor genes such as P16 and P53 and expression levels of P16, CDH1, LUNX and RASSF1A genes were researched. Finally, this article discussed the relationship between anthracosis and cancers.

Conclusions: According to the studied literature, antracotic people are more susceptible to pulmonary adenocarcinoma, hepatic nodules, renal cell carcinoma, and esophageal cancer.

Keywords: Anthracofibrosis, Anthracosis, Carcinoma, Environmental factors, Genetic.

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Anthracosis is a kind of pneumoconiosis, mainly a pulmonary disease due to prolonged exposure to carbon (1). This term was first used by coal miners (2, 3). It is an archaic disease discovered in mummies (4). Anthracosis is developed by carbon particle deposits, iron, lead, cadmium, silica, phenol, hydrocarbon complexes, and substances that cause dark pigmentation of the tracheobronchial involving both mucosal and submucosal layers and lung parenchyma, or black pigmentation in macrophages (1, 4). These black pigments are also found as black lesions in the bronchi of coal miners, workers in certain industries, and city dwellers (5). The amount deposited depends on the exposure to soot in the surrounding atmosphere and, theoretically, on the limited capability of the lungs to remove it. Anthracotic pigment in the lung shows the total of environmental exposures. Therefore, age is a determining factor in this illness (6).

The prevalence of anthracosis in Iran and South Korea was reported as 21.8% and 3 %, respectively (7-9). Cough, dyspnea, phlegm, and wheezing can be symptoms (4, 10). The disease, which has symptoms similar to chronic obstructive pulmonary disease, is mostly seen in industrial areas, coal mine workers, and those who work with graphite compounds, carbon black, and electrodes (7). Pulmonary anthracosis has long been associated with urbanization and industrialization. After the developing countries, it was reported in residents of industrialized societies (6). Anthracosis can progress and cause bronchial obstruction in some cases (11). Anthracosis can damage the bronchial mucosa and impair mucociliary clearance, predisposing patients to pulmonary infection (5, 12).

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Some studies suggested that anthracosis is an easy indicator of air pollution or smoking; others believed that exposure to carbon particles in polluted air in metropolitan or direct smoke in workplaces might lead to anthracosis (5). Some studies suggest a strong bond exists between tobacco use and anthracosis (7). Cigarette smoking, urban air pollution, coal mining, occupational exposure to dust and pulmonary tuberculosis, and indoor smoke from oven-based traditional baking are shared among anthracotic cases (13, 14). Anthracosis was also seen in individuals with no occupational exposure or smoking (2, 4). Several studies have found a link between pulmonary tuberculosis and anthracosis. Pulmonary tuberculosis is thought to explain localized anthracosis and anthracofibrosis in patients with no history of smoking or occupational exposure (15).

Additional hypotheses attempt to explain the prevalence of pulmonary tuberculosis in patients with anthracosis as a result of silica particle-induced macrophage dysfunction (2). It is not yet agreed upon that mycobacterium tuberculosis (TB) is a predisposing factor for anthracosis or vice versa. However, it is reported that 27%-60% of anthracotic patients have TB (4). In previous published studies in Iran, the prevalence of pulmonary tuberculosis in anthracosis patients ranged from 6.9 percent to 57.8 percent (2, 16, 17). In Iran, no tuberculosis report in anthracotic patients was found in Tabriz and Sanandaj. On the other hand, it was 44% in Zahedan, 25%-30% in Mashhad, and 6.9% in Kerman (4, 18). Besides, the disharmonious prevalence of TB and anthracosis in Iran causes an immature correlation between these two, resulting in the fact that there is more need for sufficient evidence to establish a relation between them (4). The comorbidity rate of TB and anthracosis was 37 % (7, 19). Anthracotic people are 2.6 more likely to get TB than healthy ones (16).

The silicosis theory explains the high prevalence of pulmonary tuberculosis in patients with anthracosis by causing impaired alveolar macrophage function and a decrease in the body's tolerance to mycobacterium tuberculosis (20). In other words, anthracosis is a risk factor for pulmonary tuberculosis (2). Anthracofibrosis is characterized by the deposition of carbon particles as well as fibrosis, adhesion, narrowing, and collapse (7). Anthracofibrosis, a chronic tracheobronchial tree and lung disease, had its first reports in 2008 in Europe and in 2014 in Spain (21-23). This disease is more common in Asia's rural areas, particularly in the Middle East. The majority of cases have been reported in Iran and Korea (4). In Iran, it is reported that it is more prevalent in women than in men and men, in which farmers and manual workers comprise the majority of the male gender (4). Anthracofibrosis was predominantly seen in older women without exposure to coal dust or tobacco smoke and was associated with active TB, confirmed on bacteriological or histopathological examination in more than 60% of patients (21). Women were reported to be more likely to get anthracosis (17), and noted that the female sex is an influential factor in anthracosis (2). This disease is common in developing countries but uncommon in developed countries. The majority of those affected by this disease are immigrants from third-world countries (24).

According to developing-country epidemiology, exposure to wood smoke can play an etiologic role in a wide range of adult medical diseases (7, 24-27). According to Amoli's case study, exposure to indoor smoke from traditional baking in ground ovens was widely reported in the history of anthracotic patients. Women in this category were more likely to contract anthracosis as a result of baking (3, 13). As a result, having good ventilation in the kitchen, a place for cooking and heating with biomass fuels, is critical in avoiding this disease, particularly in rural homes in Iran and Korea (4, 28). A summary of the aforementioned context is shown in figure 1. Due to the high prevalence of anthracosis in Asia and its importance, and the fact that not many researches have been done regarding its genetics and related cancers, in this article, we investigated the genetics of anthracosis and its connection to different cancers.

Methods

In this review, we searched all published articles in four databases (Pubmed, the Cochrane Database of Systematic Reviews, Embase and Scopus) up to June 3, 2022, in which anthracosis, epidemiology, gene and cancer have been studied. The study excluded non-published studies, studies that were not published in indexed journals or published without peer review, and studies that were not available in English. The authors gathered the findings of studies that met the inclusion and exclusion criteria (51 articles). The study was approved by the Hormozgan University of Medical Sciences Ethics Committee under the ethical code IR.HUMS.REC.1402.309.

Genetics of anthracosis: Observations suggest that a history of exposure to chemical carcinogens is significantly associated with the degree of anthracosis (29), the production of reactive oxygen species, and oxidative stress damage at the cell membrane level as a central stimulus mechanism in inducing cellular responses (30). With these interpretations, two possibilities can be raised: one is that

adenocarcinoma grows in a highly anthracotic lung, that is, cancer grows faster in a damaged lung, and the second is that tumours are more prone to develop in severe anthracotic lungs (29). The most common type of lung cancer is lung adenocarcinoma. Many genetic changes have been identified that are effective in this cancer. Cases such as dominant oncogene point mutation, retinoblastoma, and mutations in tumour suppressor genes such as P16 and P53 and expression levels of P16, CDH1, LUNX (lung-specific X protein) and RASSF1A gene are now evaluated (31).

P16 is a protein that effectively controls the cell cycle in phase G by inhibiting CDK4 and CDK6 due to cancer progression (31). In other words, this protein acts as a tumour suppressor by inhibiting tumour growth. Inactivating P16 by eliminating point mutation or methylation of its promoter leads to tumour progression (5). The findings show it decreased P16 in cancerous tissues compared to nearby healthy tissues. We discovered a high frequency of methylation of the P16 gene in tumours in patients with severe anthracnose. In addition, cases with abnormal gene expression have a significantly higher level of background anthracnose. However, methylation is observed in the early stages of pulmonary adenocarcinogenesis because there was no significant relationship between P16 methylation and pathological stage. In pulmonary adenocarcinogenesis, the level of background anthracosis is thus closely associated with DNA methylation of the p16 promoter region and inactivation of p16 expression (5, 31).

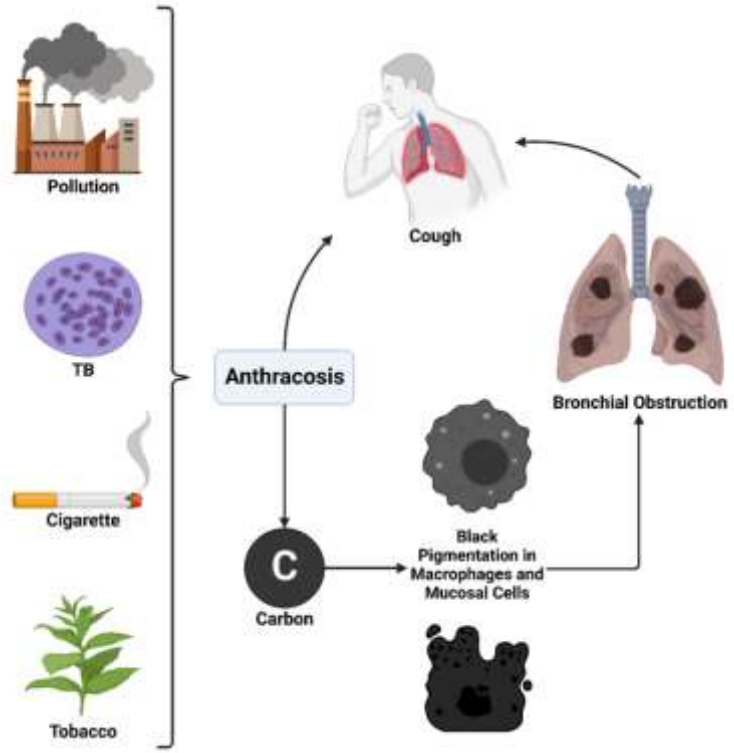

Figure 1. **Anthracosis predisposing factors and pathogenesis. Created with BioRender.com.**

The findings show it decreased P16 in cancerous tissues compared to nearby healthy tissues. We discovered a high frequency of methylation of the P16 gene in tumours in patients with severe anthracnose. In addition, cases with abnormal gene expression have a significantly higher level of background anthracnose. However, methylation is observed in the early stages of pulmonary adenocarcinogenesis because there was no significant relationship between P16 methylation and pathological stage. In pulmonary adenocarcinogenesis, the level of background anthracosis is thus closely associated with DNA methylation of the p16 promoter region and inactivation of p16 expression (5, 31).

CDH1 is one of the E-cadherin family of adhesive molecules. Because it enables cell binding and is effective in the cancer process by regulating the cell cycle and interaction with other cells. It can also inhibit metastasis and invasion of cancer cells, which naturally inhibit metastasis (5). The following gene is the EGFR (epidermal growth factor receptor). Its mutation is primarily influenced by smoking. Research has shown that in cases with an anthracnose background, most tumours were EGFR +, and

the amount of anthracnose was positively correlated with the percentage of EGFR-positive tumour cells (30).

In some cases, it has been hypothesized that age, obesity, and anthracnose as surrogates for smoking can predict RASSF1A promoter methylation in normal kidney tissue (32). RASSF1 is a tumour suppressor gene, and RASSF1A is one of its eight isoforms (33). RASSF1A is involved in cell cycle control in normal cells, and depletion promotes several cellular changes that increase cancer risk (32). Epigenetic inactivation of RASSF1A has been identified in various types of cancers. RASSF1A is usually inactivated by promoter hypermethylation (33). It is said to be used with another gene as a valuable biomarker pair in lung cancer screening (34). The human PLUNC (palate, lung and nasal epithelium clone) is also known as LUNX. PLUNC is a gene family, and its gene product SPLUNC1 is a secretory protein that is only expressed in the human lungs. It can be used to diagnose Non-Small Cell Lung Cancer (NSCLC) patients as an effective and unique marker. Of course, its mechanism is not yet fully understood, but it may be inherent in immunity (5, 35, 36). A summary of the aforementioned context is shown in figure 2.

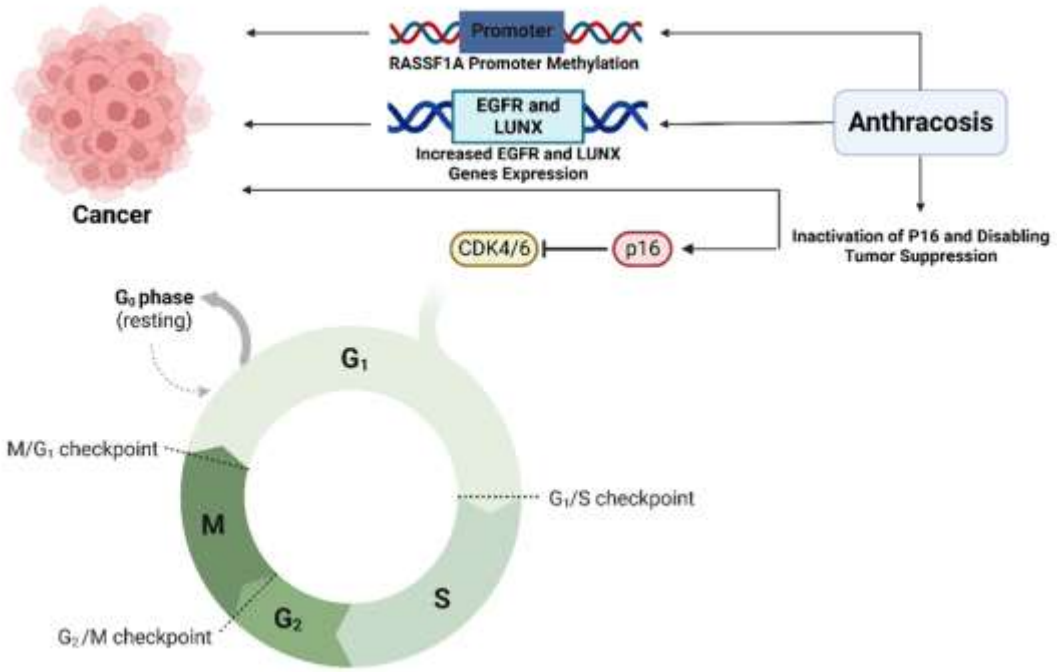

Figure 2. Anthracosis' genetical background. Created with BioRender.com

Anthracosis-related cancers

Lung cancer: Lung cancer is the leading cause of cancer-related death. Cigarette smoking and other tobacco products, secondhand tobacco smoke exposure, occupational lung carcinogens, radiation, and indoor and outdoor air pollution are all risk factors for lung cancer (37, 38). Intrapulmonary particulate pollutants (IPP) from autopsy cases and patients lobotomized due to lung cancer must be separated by alkali digestion of the lung tissue and analyzed using a wavelength-dispersive x-ray fluorescence spectrometer to investigate anthracosis of the human lung, particularly its causal relationship with atmospheric pollution and the occurrence of lung cancer. Silicon is the most abundant mineral component in PPI's non-carbon part, followed by calcium, magnesium, iron, aluminium, and other trace elements. Iron, calcium, copper, lead, chromium, and nickel levels in PPIs are higher in lung cancer patients than in non-lung cancer patients. As a result, lung anthracnose is thought to be linked to the aetiology of lung cancer. Anthracosis patients were also found to have consolidated right upper lobes (RUL) and nonspecific mediastinal lymphadenopathy.

Pathological examination of the RUL lesion obtained from a transbronchial biopsy may reveal invasive adenocarcinoma. A cytology of the tracheal duct lymph nodes may reveal macrophages filled with patient pigment on a background of lymphocytes, but no evidence of malignancy. One of the demonstrations could be an unintentional discovery of a dark pigment lining the bronchial mucosa. On a lymphoid background,

macrophages' black pigmentation can also be seen. Bronchoscopy of these patients reveals a dark brown pigment lining the endobronchial mucosa, primarily at the right middle lobe and right upper lobe bronchus levels (39). Transesophageal endoscopy (EUS) guided by fine needle aspiration (FNA; EUS-FNA) is a valuable procedure for diagnosing suspected lung cancer, malignancy (adenocarcinoma, small cell, squamous cell, and lymphoma), and benign disease (inflammation, sarcoidosis, and anthrax).

Pleural anthracosis has been linked to pathological changes in the pleural lymphatics as well as lung cancer. EUS is a practical, safe, minimally invasive procedure that does not require general anaesthesia or a hospital stay and has a high sensitivity in detecting lesions as small as 1 cm. Complication rates are low (0.5 to 2.3 percent), and no complications have been reported in several studies. Because of its inherent ability to image the entire posterior portion of the mediastinum, including the subclavian and inferior mediastinal lymph node stations and the arterial window, EUS is essential in assessing the mediastinum. Nevertheless, due to the presence of air, it has limited utility in the pretracheal region and a portion of the trachea (5, 40, 41).

Pulmonary adenocarcinoma: Adenocarcinoma that develops in severe coal lungs progresses quickly to an advanced stage. Adenocarcinoma with a poor prognosis is more common in severe coal lungs. Specimens should be fixed in 10% formalin and embedded in paraffin for a more accurate diagnosis. Hematoxylin, eosin, and van Gieson should be used to stain thinly sliced samples. Finally, they can be classified histologically based on the subtype of small lung adenocarcinoma. The GS-700 image densitometer can measure spot density (29).

Hepatic nodules: Hepatocellular carcinoma can also be caused by anthracosis. According to reports, patients who have had lobectomy with pericardial resection due to localized invasion and mediastinal lymphadenectomy can develop contralateral pulmonary lesions and a synchronous nodule in the liver. The primary working diagnosis can be tumour recurrence. A lung biopsy in these patients reveals a chronic inflammatory response with anthracosis and dystrophic calcification foci. A new pulmonary lesion discovered in a patient with a history of bronchial cancer raises the possibility of another primary tumour or tumour recurrence. When this specific population was studied, it was discovered that 80 percent of the patients had another primary tumour and 2 percent had tumour recurrence. This indicates a high likelihood of cancer, which warrants further investigation (42, 43).

Spindle cell pseudotumors: Mycobacterial spindle cell pseudotumor is an immunocompromised patient-related reactive lesion. It can happen anywhere in the body. It is a rare disorder distinguished by the proliferation of spindle-shaped histiocytes containing intracytoplasmic acid-fast bacilli (44, 45). One type can be anthrax-associated mediastinal spindle cell proliferation. This complication shows different clinical features in different patients. Clinically, the lesions form masses that are visible on radiographs. In some cases, a striking pattern of interlocking rhabdoid cells can be found microscopically. This proliferation can be extended beyond the ganglion capsule and surrounds nerves in some patients. The diagnosis would be malignancy in some conditions. The cases may have nodular hyaline scars and contain silica-like polar material. Lesions may be enlarged or not during the 6 to 48-month follow-up period in these patients. The surgeon may comment on the magnitude of anthracoid pigment in the thoracic cavity in anthracosis-related cases. Also, in anthrax-associated types, spindle cells become immunologically active for histiocytic markers and contain mainly fine anthrax pigments. A variety of rhabdoid tumors, including malignant fibrous histiocytoma, follicular dendritic cell tumor, rhabdoid melanoma, and Kaposi's sarcoma, are on the differential diagnosis (46).

Renal cell carcinoma: The most common histologic subtype of sporadic kidney tumors, renal cell carcinoma, is associated with age, obesity, and smoking (47, 48). RASSF1A hypermethylation is one of the most common epigenetic changes found in human cancers, including renal cell carcinoma. RASSF1A regulates cell cycle in normal cells, and its deficiency promotes several cellular changes that increase cancer risk. Age, adiposity, and anthracnose, as alternatives to smoking, are thought to be predictors of RASSF1A promoter methylation in normal renal tissue (32).

Esophageal cancer: Esophageal cancer is the sixth leading cause of cancer-related deaths worldwide. It is the eighth most common cancer in the world (49, 50). It is one of the deadliest cancers in the world and is characterized by differences in prevalence between different populations. Anthracosis can be one of the causes of this cancer. These patients should be treated with esophagectomy. During the diagnosis, resected specimens and harvested lymph nodes should be formalin-fixed and paraffin-embedded, and esophageal lymph nodes may be examined histologically for the presence of anthrax pigment (51). A summary of the aforementioned cancers and their relation with anthracosis is shown in figure 3. The role of genes in determining the course and severity of disease is well-known. The condition

can be exacerbated by mutations in the genes P16, EGFR, CDH1, and LUNX. Thus, in the center of anthracotic lesions, there is a decrease in the expression of P16 and CDH1 and an increase in the expression of LUNX tumor genes. In cases with anthracosis, the proportion of EGFR-positive tumours was significantly higher. The percentage of EGFR-positive tumor cells was positively correlated with the amount of anthracosis in anthracotic lungs. Furthermore, some studies suggest that EGFR signalling pathways are involved in air pollution-related carcinogenesis. It has been demonstrated in the studies of the relationship between anthracotic lungs and pulmonary adenocarcinoma that the presence of anthracosis causes the progression of adenocarcinoma. It is also hypothesized that age, adiposity, and anthracosis, as a smoking surrogate, can be predictors of RASSF1A promoter methylation in normal kidney tissue. Furthermore, anthracotic pigments have been found in the lymph nodes of esophageal cancer patients.

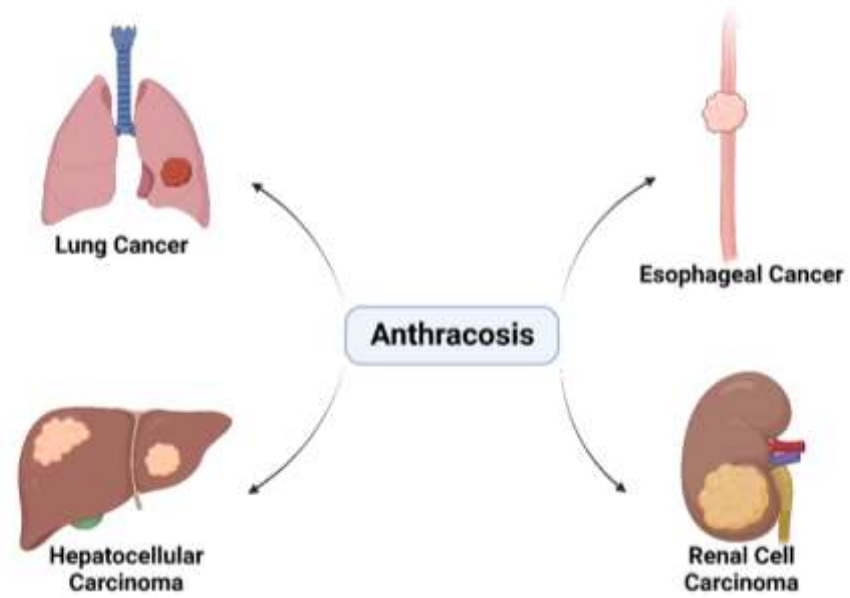

Figure 3. Anthracosis-related cancers. Created with BioRender.com.

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