

A case study of lung cancer patients in term of various risk factors in context of Bangladesh



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DECLARATION

I hereby declare that the thesis is entitled “**A case study of lung cancer patients in term of various risk factors in context of Bangladesh**” is based on my work and it contains no material previously published or written by another person and not accepted for the award of any other degree of a university or other institute of higher education.

This research work was done in the Department of Oncology, Ahsania Mission Cancer Hospital, Mirpur, Dhaka. The work was completed under the supervision of Dr. Mohammad Rafiqul Islam and coordinator Dr. Md Mahboob Hossain, Department of Mathematics and Natural Science, BRAC University and Dr. Khondoker Golam Mostakim, Professor and senior consultant of Oncology at Ahsania Mission Cancer Hospital, Mirpur, Dhaka.

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CERTIFICATE

This is to certify that Musharrat Shabnam has completed the thesis entitled “**A case study of lung cancer patients in term of various risk factors in context of Bangladesh**” as a fulfillment of the requirements for the degree of Bachelors of Science in Microbiology thesis part by the BRAC University Dhaka, Bangladesh. This study has been conducted in the Department of Oncology, Ahsania Mission Cancer Hospital, Mirpur, Dhaka under our joint supervision. Her work is original and the work is up to our full satisfaction.

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LIST OF ABBREVIATIONS

Abbreviations	Elaboration
CT	Computed Tomography
NSCLC	Non-Small Cell Lung Carcinoma
SCLC	Small Cell Lung Carcinoma
SIADH	Syndrome of Inappropriate Antidiuretic Hormone
EGFR	Epidermal Growth Factor Receptor
ALT	Anterior Limited Thoracotomy
AAT	Anterior axillary Thoracotomy
VATS	Video-Assisted Thoracic Surgery
PLT	Posterolateral Thoracotomy
PET	Positron Emission Tomography
PDT	Photodynamic Therapy
VAT	Video-Assisted Thoracoscopy
rhIL-2	Recombinant Human Interleukin-2
IL-2	Interleukin-2
T- lymphocytes	Thymus-Dependent Lymphocytes
CHART	Continuous Hyper fractionated Accelerated Radiotherapy
PCI	Prophylactic Cranial Irradiation
NICRH	National Institute of Cancer Research and Hospital
RSP	Respirable Suspended Particles

ABSTRACT

Lung cancer is the most common cancer among men and women of Bangladesh. People who smoke for more than 10 years, expose to dusty environment, affect by infectious disease like tuberculosis, pneumonia and chronic obstructive pulmonary disease (COPD) are mostly affected by lung cancer. In Bangladesh lung cancer is an important health problem between the age of 50 and 70 with vital signs and symptoms. The present study aim is to provide awareness on the environmental risk factors related to smoking habit which causes lung cancer in Bangladesh. This study was carried out in the Department of Oncology at Ahsania Mission Cancer Hospital, Dhaka. This was a retrospective observational study.

This study was carried out during the period from November 2017 to May 2018 for 7 months of enrollment with a follow up of 3 months up to August 2018 which makes total study period of 10 months. Patients' data were collected from the indoor and outdoor medical records of the AMCH hospital. A representative sample of 300 Bangladeshi cancer Patients aged over 30 was asked about their perception and the level of concern about various environmental lifestyle and genetic risk factor in relation to cancer prevention, as a part of a successful survey.

The patients suffering from adenocarcinoma (42%) is large in number and squamous cell carcinoma (30%) whereas small-cell carcinoma (15%) and carcinoid tumor (3%). Among 28 risk factors, the attributable fraction of lung cancer causing by tobacco smoking (67.3%) and environmental exposure were considered major risk factors. Some infectious diseases like Tuberculosis (39%) and Asthma (18%) were also considered as important risk factors. Patients more than 50 years of age were more likely to have lung cancer. For most risk factors, attributable fraction responses were higher in men (63%) than in women (37%). On the other hand, the attributable fractions of cancer by occupational exposure were considered low compared with other risk factors.

Our result suggests that awareness of the attributable fraction of lung cancer causes in the Bangladeshi cancer patients tends to be dominated by cancer causing infection, men and women hygiene tobacco smoking, chewing tobacco, occupational exposure and life style. In Bangladesh, it appears that most people are aware of the major risk factors of lung cancer. Hence there is need to promote awareness of lung cancer symptoms and develop and evaluate rapid assessment clinics for patients with suspected lung cancers.

INTRODUCTION

1.1. INTRODUCTION

Cancer is the cause of one-quarter of all deaths in developing countries. Lung cancer ranks among the most common and most lethal malignancies worldwide. Lung cancer kills more people than any other cancer. Cancer is a leading cause of cancer death in both men and women which develops over several years and has many causes. Several factors both inside and outside the body contribute to the development of cancer. Cancer is a group of diseases involving abnormal cell growth with the potential to invade or spread to other parts of the body. These contrast with benign tumors, which do not spread to other parts of the body. Possible signs and symptoms include a lump, abnormal bleeding, prolonged cough, unexplained weight loss and a change in bowel movements. While these symptoms may indicate cancer, they may have other causes of indication.

Elements may incorporate investigate to certain medicinal medications, hormones, radiation, infection and natural synthetic concoctions that might be available noticeable all around, water, sustenance and work places. Additionally, weight reduction, physical action are imperative conduct changes to diminish colorectal disease chance, alongside the potential advantage for the decrease of red meat utilization and the expansion in folic corrosive admission. Numerous malignancies can be counteracted by not smoking, keeping up a sound weight, not drinking excessively liquor, eating a lot of vegetables, foods grown from the ground grains, immunization against certain irresistible maladies, not eating excessively prepared and red meat and staying away from an excessive amount of daylight exposure. Early discovery through screening is helpful for cervical and colorectal cancer. The advantages of screening in bosom growth are controversial.

Lung cancer is treated with some blend of radiation treatment, medical procedure, chemotherapy and focused on therapy. Pain and indication administration are a vital piece of care. Palliative care is especially imperative in individuals with cutting edge disease. The shot of survival relies upon the kind of tumor and degree of illness toward the beginning of treatment.

Tobacco use is the reason for around 22% of lung cancer. Another 10% are because of stoutness, terrible eating routine, absence of physical action or unnecessary drinking of liquor. Different variables incorporate certain contaminations, introduction to ionizing radiation and natural pollutants. In the creating scene, 15% of cancers are because of diseases, for example,

Helicobacter pylori, hepatitis B, hepatitis C, human papillomavirus disease, Epstein– Barr infection and human immunodeficiency infection (HIV). These variables demonstration in any event somewhat is by changing the qualities of a cell. Commonly, numerous hereditary changes are required before malignancy creates. Roughly 5-10% of diseases are because of acquired hereditary deformities from a man's folks. Tumor can be identified by specific signs and side effects or screening tests. It is then ordinarily additionally examined by medicinal imaging and affirmed by biopsy.

Smoking cessation is still the most important prevention intervention for reducing lung cancer risk, but recent evidence indicates that increasing physical activity may also be an important prevention intervention for this disease. (World Health Organization, 2006) Most by far 78% of instances of lung growth are because of long haul tobacco smoking. Around 22% of cases happen in individuals who have never smoked. These cases are regularly caused by a blend of hereditary components and presentation to radon gas, asbestos, second-hand smoke, or different types of air contamination. The most common symptoms are shortness of breath, coughing including hemoptysis and weight loss. (Minna, 2004)

Lung growth might be seen on chest radio-graphs and processed tomography filters. The finding is affirmed by biopsy which is normally performed by bronchoscopy or CT-direction. The deferral in finding of lung growth prompts demise, as lung tumor patients encounter considerable postponements from advancement of side effects to first inception of treatment. The quantities of lung growth patients were expanding step by step.

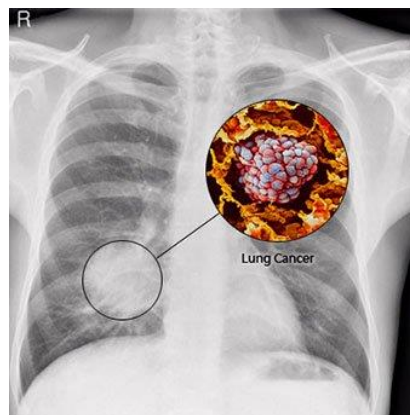


Figure 1: Lung cancer X-ray with callout showing close-up of tumor

1.2.TYPES

Carcinoma is a sort of tumor that creates from epithelial cells. In particular, a carcinoma is a tumor that starts in a tissue that lines the inward or external surfaces of the body, and that emerges from cells beginning in the endodermal, mesodermal or ectodermal germ layer amid developing life beginning. Carcinomas happen when the DNA of a cell is harmed or modified and the cell starts to develop wildly and end up threatening. (Definition of Carcinoma, 2014)

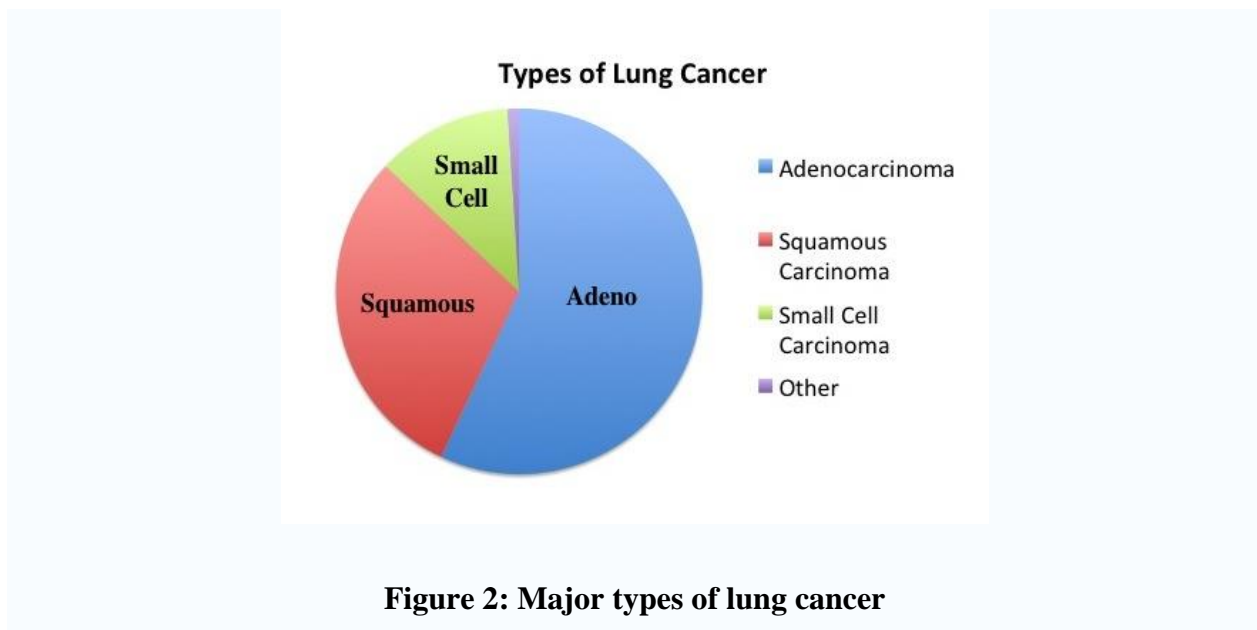
The main types of lung cancer are-

1. Small cell lung carcinoma
2. Non-small cell lung carcinoma

This distinction is important, because the treatment varies; non-small cell lung carcinoma (NSCLC) is sometimes treated with surgery, while small cell lung carcinoma (SCLC) usually responds better to chemotherapy and radiation. The most common cause of lung cancer is long-term exposure to tobacco smoke. (Merck, 2007) The occurrence of lung cancer in nonsmokers is often attributed to a combination of genetic factors. Radon gas, (Catelinois et al., 2006) asbestos, (O'Reilly et al., 2007) and air pollution, (Chiu et al., 2006) including secondhand smoke (Subramanian, 2007).

Lung malignancy might be seen on chest X-beam and processed tomography (CT check). The finding is affirmed with a biopsy. This is normally performed by means of bronchoscopy or CT-guided biopsy. Treatment and forecast rely on the histological kind of disease, the stage (level of spread), and the patient's execution status. Conceivable medications incorporate medical procedure, chemotherapy, and radiotherapy. With treatment, the five-year survival rate is 14%.

Vast majority of lung cancers are carcinomas malignancies that arise from epithelial cells. There are two main types of lung carcinoma, categorized by the size and appearance of the malignant cells seen by a histopathologist under a microscope: non-small cell (80.4%) and small-cell (16.8%) lung carcinoma. This classification, based on histological criteria, has important implications for clinical management and prognosis of the disease.



1.2.1. Small-cell lung carcinoma (SCLC)

Small-cell carcinoma is a type of highly malignant cancer that most commonly arises within the lung, although it can occasionally arise in other body sites, such as the cervix, prostate, and gastrointestinal tract. Compared to non-small cell carcinoma, small cell carcinoma has a shorter doubling time, higher growth fraction, and earlier development of metastases. It tends to arise in the larger airways like primary and secondary bronchi and grows rapidly, becoming quite large. (Collins et al., 2007) Over 70% of patients with small-cell carcinoma present with metastatic disease; common sites include liver, adrenals, bone, and brain. While initially more sensitive to chemotherapy, it ultimately carries a worse prognosis and is often metastatic at presentation. Small cell lung cancers are divided into limited stage and extensive stage disease. This type of lung cancer is strongly associated with smoking. (Barbone et al., 1997)

1.2.2. Non-small cell lung carcinoma (NSCLC)

Non-small-cell lung carcinoma (NSCLC) is any type of epithelial lung cancer other than small cell lung carcinoma (SCLC). NSCLC accounts for about 85% of all lung cancers. (Riaz et al., 2012) As a class, NSCLCs are moderately obtuse to chemotherapy, contrasted with little cell carcinoma. Whenever possible, they are basically treated by careful resection with corrective

plan, in spite of the fact that chemotherapy is progressively being utilized both pre-operatively (neo-adjuvant chemotherapy) and post-operatively (adjuvant chemotherapy).

The most widely recognized kinds of NSCLC are squamous cell carcinoma, huge cell carcinoma, and adenocarcinoma, yet there are a few different composes that happen less regularly. A couple of the less regular writes are pleomorphic, carcinoid tumor, salivary organ carcinoma, and unclassified carcinoma.

Squamous cell carcinoma

Around 25% to 30% of all lung cancer is squamous cell carcinomas. These growths begin in early forms of squamous cells, which are level cells that line within the aviation routes in the lungs. They are regularly connected to a past filled with smoking and have a tendency to be found in the focal piece of the lungs, close to a principle aviation route (bronchus). Representing 30% of lung growths, (Riaz et al., 2012) squamous cell lung carcinoma ordinarily begins almost a focal bronchus. Cavitation and rot inside the focal point of the growth is a typical finding. All around separated squamous cell lung tumors regularly develop more gradually than other malignancy composes.

Adenocarcinoma

About 40% of lung cancers are adenocarcinomas. It usually originates in peripheral lung tissue. This type of lung cancer occurs mainly in current or former smokers, but it is also the most common type of lung cancer seen in non-smokers. It is more common in women than in men, and it is more likely to occur in younger people than other types of lung cancer. (Barbone, 1997) Adenocarcinoma is normally found in external parts of the lung. In spite of the fact that it has a tendency to develop slower than different kinds of lung malignancy and will probably be found before it has spread, this changes from patient to quiet. Most cases of adenocarcinoma are associated with smoking; however, among people who have never smoked ("never-smokers"), adenocarcinoma is the most common form of lung cancer. (Morandi et al., 2006) A subtype of adenocarcinoma, the bronchioloalveolar carcinoma, is more common in female never-smokers, and may have different responses to treatment. (Subramanian , 2007)

Large cell carcinoma

Around 10% to 15% of lung cancer is large cell lung carcinoma. Large cell lung carcinoma is a heterogeneous gathering of undifferentiated harmful neoplasms starting from changed epithelial cells in the lung. LCLC is separated from small cell lung carcinoma (SCLC) principally by the bigger size of the anaplastic cells, a higher cytoplasmic-to-nuclear size proportion, and an absence of "salt-and-pepper" chromatin. It can show up in any piece of the lung. It has a tendency to develop and spread rapidly, which can make it harder to treat. A subtype of large cell carcinoma, known as extensive cell neuroendocrine carcinoma, is a quickly developing disease that is fundamentally the same as small cell lung tumor.

According to the Nurses' Health Study, the risk of large cell lung carcinoma increases with a previous history of tobacco smoking, with a previous smoking duration of 30 to 40 years giving a relative risk of approximately 2.3 compared to never-smokers, and a duration of more than 40 years giving a relative risk of approximately 3.6. (Kenfield, 2008).

1.2.3. Metastasis

The lungs are a common place for the spread of tumors from other parts of the body. Secondary cancers are classified by the site of origin; for example, breast cancer that has been spread to the lung is called metastatic breast cancer. Metastases often have a characteristic round appearance on chest radiograph. Primary lung cancers also most commonly metastasize to the brain, bones, liver, and adrenal glands. (Kumar 2005)

Immunostaining of a biopsy usually helps determine the original source. The presence of Napsin-A, TTF-1, CK7, and CK20 help confirm the subtype of lung carcinoma. SCLC that originates from neuroendocrine cells may express CD56, neural cell adhesion molecule, synaptophysin, or chromogranin. (Zlotnik, 2011)

1.2.4. Others

Four fundamental histological subtypes are perceived, albeit a few malignancies may contain a blend of various subtypes, for example, adenosquamous carcinoma. Uncommon subtypes incorporate carcinoid tumors, bronchial organ carcinomas, and sarcomatoid carcinomas.

1.3.SIGNS AND SYMPTOMS

Lung growth normally doesn't cause signs and side effects in its most punctual stages. Signs and side effects of lung tumor ordinarily happen just when the sickness is progressed. Signs and side effects of lung disease may include:

- Anorexia (Eating disorder)
- Loss of body weight
- Cachexia (Weakness)
- Coughing
- Hemoptysis (Cough with blood)
- Dyspnea (Shortness of breathing)
- Dysphagia (Difficulty in speech)
- Hoarseness (Voice change)
- Chest Pain
- Lymph Nodes Enlargement
- Liver Enlargement
- Low Hemoglobin
- Anemia
- Bone pain

Individuals with lung disease can encounter shortness of breath if growth develops to obstruct the major aviation routes. Lung malignancy can likewise make liquid amass around the lungs, making it harder for the influenced lung to extend completely when you breathe in. Lung growth can cause seeping in the aviation route, which can make you hack up blood (hemoptysis). Once in a while draining can end up extreme. Medications are accessible to control dying. Propelled

lung disease that spreads to the coating of a lung or to another region of the body, for example, a bone, can cause torment. Lung tumor can make liquid collect in the space that encompasses the influenced lung in the chest pit. Liquid aggregating in the chest can cause shortness of breath. Medications are accessible to empty the liquid out of your chest and lessen the hazard that pleural radiation will happen once more.

Depending on the type of tumor, so-called para neoplastic phenomena may initially attract attention to the disease. (Honnorat, 2007) In lung cancer, these phenomena may include Lambert-Eaton Myasthenic syndrome, hypercalcemia, or syndrome of inappropriate antidiuretic hormone (SIADH). Tumors in the top of the lung, known as Pan coast tumors, (Detterbeck, 2013) may invade the local part of the sympathetic nervous system, leading to changed sweating patterns and eye muscle problems as well as muscle weakness in the hands due to invasion of the brachial plexus.

Lung disease frequently spreads to different parts of the body, for example, the cerebrum and the bones. Growth that spreads can cause torment, sickness, cerebral pains, or different signs and side effects relying upon what organ are influenced. When lung tumor has spread past the lungs, it's for the most part not treatable. Medicines are accessible to diminish signs and side effects and help live more. Around 10% of individuals with lung malignancy don't have side effects at conclusion; these diseases are by chance found on routine chest x-beams.

1.4.CAUSES

The primary driver of lung growth incorporates cancer causing agents, for example, those in tobacco smoke, ionizing radiation, and viral contamination. This introduction makes aggregate changes the DNA in the tissue covering the bronchi of the lungs (the bronchial epithelium). As more tissue ends up harmed, in the long run a malignancy creates. (Vaporciyan et al., 1998)

1.4.1. Smoking

Smoking is a training in which a substance is scorched and the subsequent smoke inhaled is to be tasted and consumed into the circulation system and achieve real tissue. The vaporization permits inward breath and profound infiltration into the lungs where ingestion into the circulatory system

of the dynamic substances happens. Tobacco smoking is the most well-known frame, being rehearsed by more than one billion individuals all around, of whom the lion's share are in the creating nations. Less regular medications for smoking incorporate cannabis, opium and some ordered hard opiates, similar to heroin. Other smoking implements include pipes, cigars, bidis, hookahs, and bongos.

Over the created world, just about 90% of lung cancer is caused by smoking, (Peto et al., 2006). In the United States, smoking is assessed to represent 87% of lung growth cases 90% in men and 85% in ladies. (Samet et al., 1988) Among male smokers, the lifetime danger of creating lung malignancy is 17.2%; among female smokers, the hazard is 11.6%. This hazard is fundamentally lower in nonsmokers: 1.3% in men and 1.4% in ladies (Villeneuve, 1994). Tobacco smoke contains more than 60 known cancer-causing agents, including radioisotopes from the radon rot succession, nitrosamine, and benzopyrene. Also, nicotine seems to discourage the insusceptible reaction to threatening developments in uncovered tissue. (Jemel, 2006)

The time span a man smokes and in addition rate of smoking builds the individual's possibility of creating lung disease. In the event that a man quits smoking, this shot relentlessly diminishes as harm to the lungs is repaired and contaminant particles are bit by bit expelled. Moreover, there is prove that lung disease in never-smokers has a superior anticipation than in smokers, (Thun et al., 2008) and that patients who smoke at the season of conclusion have shorter survival times than the individuals who have stopped (Gorlova et al., 2007).

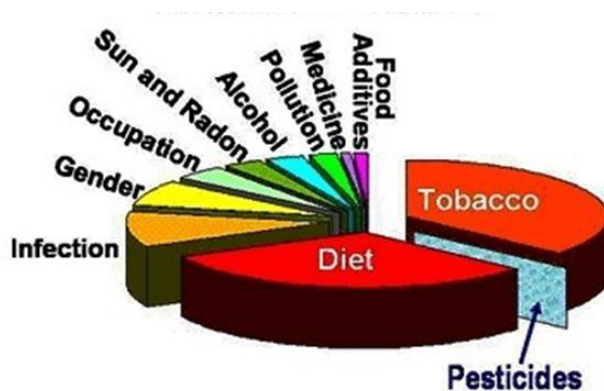


Figure 3: Causes of Lung cancer

1.4.2. Passive smoking

Passive smoking is the inward breath of smoke, called second-hand smoke (SHS), or natural tobacco smoke (ETS), by people other than the expected "dynamic" smoker. (Raz, 2006) It happens when tobacco smoke pervades any condition, causing its inward breath by individuals inside that condition. An aloof smoker can be delegated somebody living or working with a smoker also. Ongoing examination of sidestream smoke proposes that it is more perilous than coordinate smoke inward breath. (Samet, 1988). Presentation to second-hand tobacco smoke causes malady, inability, and demise. Passive smoking increases the risk of asthma and chronic obstructive pulmonary disease (Raz, 2006)

1.4.3. Genetic factor

Similar to many other cancers, lung cancer is initiated by activation of oncogenes or inactivation of tumor suppressor genes. Oncogenes are genes that are believed to make people more susceptible to cancer. Proto-oncogenes are believed to turn into oncogenes when exposed to particular carcinogens. Mutations in the K-ras proto-oncogene are responsible for 10–30% of lung adenocarcinomas. (Brentnall, 2005) The epidermal development factor receptor (EGFR) controls cell multiplication, apoptosis, angiogenesis, and tumor attack. Changes and enhancement of EGFR are normal in non-small cell lung malignancy and give the premise to treatment with EGFR-inhibitors. Her2/neu is influenced less as often as possible.

Chromosomal harm can prompt loss of heterozygosity. This can cause inactivation of tumor silencer qualities. Harm to chromosomes 3p, 5q, 13q, and 17p are especially regular in small cell lung carcinoma. The p53 tumor silencer quality, situated on chromosome 17p, is influenced in 60-75% of cases. A few hereditary polymorphisms are related with lung growth. These incorporate polymorphisms in qualities coding for interleukin-1, cytochrome P450 and apoptosis promoters, for example, caspase-8 and DNA repair atoms, for example, XRCC1. (Dela, 2015)

1.4.4. Inheritable factors

Most instances of lung growth are not identified with acquired hereditary changes. These malignancies are related with physical transformations that happen just in specific cells in the lung. At the point when lung tumor is identified with acquired hereditary changes, the growth chance takes after an autosomal prevailing example, which implies one duplicate of the modified

quality in every cell is adequate to build a man's possibility of building up the infection. (Dela, 2015) Around 8% of lung disease is caused by acquired variables. In relatives that are determined to have lung tumor, the hazard is multiplied, likely because of a mix of qualities. Not all individuals who acquire transformations in these qualities will create lung disease. (Minna, 2004) Genetic factors are significant contributor, but only a few specific genes and other genetic factors affecting lung cancer have been identified to date. Brothers, sisters and children of those who have had lung cancer may have a slightly higher risk of lung cancer themselves. However, it is difficult to say how much of this excess risk is due to inherited factors and how much is due to environmental tobacco smoke. (Barbone, 1997)

1.4.5. Air pollution

Air pollution from vehicles, industry, and power plants can raise the probability of creating lung tumor in uncovered people. Up to 1-2% of lung cancer spreading are owing to breathing polluted air, and specialists trust that drawn out presentation to very contaminated air can convey a hazard for the advancement of lung growth like that of inactive smoking. (Samet, 1988)

1.4.6. Asbestos

Asbestos fibers are silicate fibers that can continue for a lifetime in lung tissue following presentation to asbestos. Asbestos can cause an assortment of lung cancer, including lung disease. The work environment was a typical wellspring of presentation to asbestos filaments, as asbestos was broadly utilized as a part of the past as both warm and acoustic protection. Both lung disease and mesothelioma are related with introduction to asbestos. There is a synergistic impact between tobacco smoking and asbestos in the development of lung growth. Cigarette smoking radically expands the shot of building up an asbestos-related lung tumor in laborers presented to asbestos; asbestos specialists who don't smoke have a fivefold more serious danger of creating lung disease than nonsmokers, yet asbestos specialists who smoke have a hazard that is fifty-to ninety-overlays more noteworthy than nonsmokers. Today, asbestos utilize is constrained or prohibited in numerous nations, including the U.S. In the UK, asbestos represents 2-3% of male lung disease deaths. (Biesalski, 1998)

1.4.7. Radon gas

Radon gas is a natural radioactive gas that is a natural decay product of uranium that emits a type of ionizing radiation. Radon gas is a known reason for lung cancer, with an expected 12% of lung disease owing to radon gas, or around 21,000 lung-cancer related cases every year in the U.S., making radon the second driving reason for lung growth in the wake of smoking. As with asbestos exposure, concomitant smoking greatly increases the risk of lung cancer with radon exposure. Radon gas can go up through soil and enter homes through holes in the establishment, pipes, channels, or different openings. One out of each 15 homes contains perilous levels of radon gas. Radon gas is imperceptible and scentless; however it can be identified with basic test kits. (Schmid, 2010)

1.4.8. Other factors

Various metals like aluminum, cadmium, chromium, beryllium, iron and steel, nickel, arsenic and inorganic arsenic mixes, and underground hematite mining can likewise be the hazard factors. A few results of ignition like coal, tar, coke, residue, and diesel motor fumes. Some lethal gases methyl ether, sulfur mustard, MOPP and vapor from painting and ionizing radiation, for example, X-radiation, gamma radiation, and plutonium. There is a little increment in the danger of lung disease in individuals influenced by fundamental sclerosis.

1.5.PATHOGENESIS

The pathogenesis of lung disease is started the either the enactment of oncogenes or the inactivation of tumor silencer qualities, which prompts uncontrolled replication and development of the cells in the lungs. There are a few factors that may prompt these hereditary changes: they might be acquired from guardians or gained by presentation to cancer-causing agents. (Tobias, 2010) Mutations in the K-ras proto-oncogene cause roughly 10–30% of lung adenocarcinomas. Nearly 4% of non-small-cell lung carcinomas involve an EML4-ALK tyrosine kinase fusion gene. Epigenetic changes such as alteration of DNA methylation, histone tail modification, or microRNA regulation may result in the inactivation of tumor suppressor genes. (Jakopovic, 2013)

The epidermal growth factor receptor (EGFR) regulates cell proliferation, apoptosis, angiogenesis, and tumor invasion. Mutations and amplification of EGFR are common in non-

small-cell lung carcinoma, and they provide the basis for treatment with EGFR-inhibitors. Her2/neu is affected less frequently. Other genes that are often mutated or amplified include c-MET, NKX2-1, LKB1, PIK3CA, and BRAF. (Herbst, 2008) In the proximal airways, stem cells that express keratin 5 are more likely to be affected, typically leading to squamous-cell lung carcinoma. In the middle airways, implicated stem cells include club cells and neuro-epithelial cells that express club cell secretory protein. Small-cell lung carcinoma may originate from these cell lines or neuroendocrine cells, and it may express CD44. Metastasis of lung cancer requires transition from epithelial to mesenchymal cell type. This may occur through the activation of signaling pathways such as Akt/GSK3Beta, MEK-ERK, Fas, and Par6. (Powell, 2013)

1.6.DIAGNOSIS

Performing a chest x-beam is the initial step if a patient reports side effect that might be suggestive of lung growth. This may uncover a conspicuous mass, enlarging of the mediastinum (suggestive of spread to lymph hubs there), atelectasis (crumple), union (pneumonia), or pleural emanation. In the event that there are no x-beam discoveries yet the doubt is high, (for example, a substantial smoker with blood-recolored sputum), bronchoscopy and additionally a CT output may give the vital data. Bronchoscopy or CT-guided biopsy is frequently used to recognize the tumor type.

The differential conclusion for patients who give variations from the norm on chest x-beam incorporates lung tumor and in addition nonmalignant ailments. These incorporate irresistible causes, for example, tuberculosis or pneumonia, or incendiary conditions, for example, sarcoidosis. These infections can bring about mediastinal lymphadenopathy or lung knobs, and some of the time copy lung cancers. (Minna, 2004) Lung cancer can also be an incidental finding: a solitary pulmonary nodule (also called a coin lesion) on a chest x-ray or CT scan taken for an unrelated reason.

Staging

Lung cancer staging is the assessment of the extent to which a lung cancer has spread from its original source. Likewise with most tumors, organizing is a critical determinant of treatment and visualization. By and large, further developed phases of disease are less amiable to treatment and

have a more terrible prognosis. The underlying assessment of non-small cell lung disease arranging utilizes the TNM grouping. This is based on the size of the primary tumor, lymph node involvement, and distant metastasis. After this, using the TNM descriptors, a group is assigned, ranging from occult cancer, through stage 0, IA, IB, IIA, IIB, IIIA and IIIB to IV. This stage group assists with the choice of treatment and estimate of prognosis. (Rami, 2009)

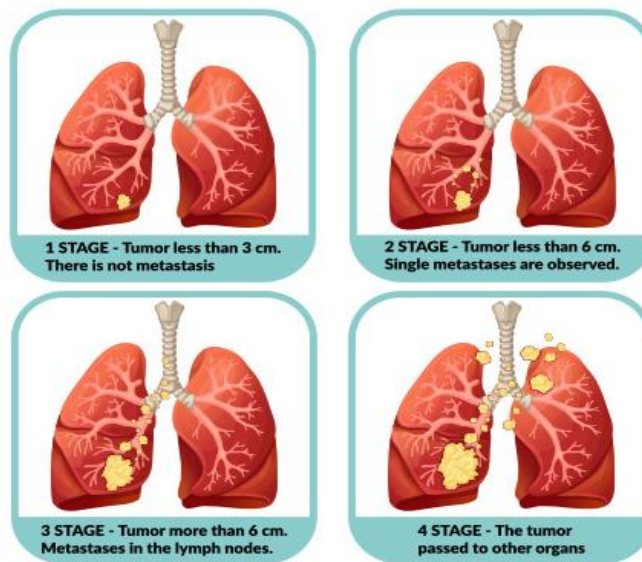


Figure 4: Stages of Lung cancer

The TNM staging system of lung cancer is:

T Tumor

T1a: Primary tumor is ≤ 2 cm in greatest dimension.

T1b: Primary tumor is >2 but ≤ 3 cm in greatest dimension.

T2a: Primary tumor is >3 and ≤ 5 cm in greatest dimension.

T2b: Primary tumor is >5 and ≤ 7 cm in greatest dimension.

T3 size: Primary tumor is >7 cm in greatest dimension;

T3 inv: Primary tumor invades the chest wall, diaphragm, phrenic nerve or pericardium;

T3: Primary tumor is <2 cm to the carina or there is atelectasis of the entire lung;

T3: Primary tumor is associated with separate tumor nodule(s) in same pulmonary lobe;

T4 inv: Tumor invading the heart, great vessels, trachea, recurrent laryngeal nerve, esophagus, or spine;

T4: Tumor of any size with additional tumor nodule(s) in a different ipsilateral lobe

N Lymph node

N1: Nodal metastasis in ipsilateral pulmonary or hilar lymph nodes;

N2: Nodal metastasis in ipsilateralmediastinal/subcarinal lymph nodes.

1.7.PREVENTION

The most ideal approach to prevent lung cancer is not to smoke. People should not start smoking, and those who already smoke should quit. Everyone should avoid breathing in another people's smoke. People should find out about cancer-causing chemicals they may be exposed to at work and take appropriate protective measures. (Dela, 2011)

Over the previous century, lung growth has gone from a dark illness to the main source of tumor demise around the world. At first a scourge sickness among men in industrialized countries, lung tumor currently has turned into the main disease executioner in both genders. Lung tumor rate to a great extent reflects smoking pervasiveness, with an inertness time of a very long while.

Other critical hazard factors for the advancement of lung cancer include ecological presentation to tobacco smoke, radon, word related cancer-causing agents, and prior nonmalignant lung infection. Concentrates in sub-atomic science have illustrated the part that hereditary elements play in altering a person's hazard for lung cancer.

Despite the fact that chemo preventive agents might be created to anticipate lung cancer, counteractive action of smoking commencement and advancement of smoking suspension are presently the best weapons to fight lung disease. In view of present and anticipated smoking

examples, it is foreseen that lung cancer will remain the main source of disease demise on the planet for a considerable length of time to come. (Manser, 2013)

1.7.1. Smoking ban

While in many nations mechanical and local cancer causing agents have been distinguished and prohibited, tobacco smoking is as yet across the board. Dispensing with tobacco smoking is an essential objective in the anticipation of lung disease, and smoking end is a critical preventive apparatus in this procedure. (Goodman, 1990) Limitations after smoking in bars and restaurants can considerably enhance the air quality in such foundations. Approach mediations to diminish detached smoking in broad daylight regions, for example, eateries and work environments have turned out to be more typical in numerous Western nations. Bhutan has had a total smoking boycott since 2005 while India presented a restriction on smoking in broad daylight in October 2008. Research has created confirm that second-hand smoke causes an indistinguishable issues from coordinate smoking, including lung tumor, cardiovascular malady, and lung diseases, for example, emphysema, bronchitis, and asthma. (UN Health, 2008)



Figure 5: Quit Smoking

1.7.2. Screening

Screening recognizes early lung cancer before they cause indications, at a point where they will probably be reparable. Screening studies for lung cancer have only been done in high risk populations, such as smokers and workers with occupational exposure to certain substances. CT screening has been related with a high rate of dishonestly positive tests which may bring about unneeded treatment. (Aberle, 2013) For each evident positive sweep there have been upwards of 19 erroneously positives filters. When screening is done with regards to a procedure of indicative

tests, false positives have been lessened to roughly 12%. Different concerns incorporate radiation introduction and the cost of testing alongside the follow up of tests. Research has not discovered two other clinically accessible tests-sputum cytology or chest radiograph (CXR) screening tests to decrease the general number of individuals who kick the bucket from lung malignancy. (Manser, 2013)

1.7.3. Supplement

The long- term utilization of supplemental vitamin A, vitamin C, vitamin D or vitamin E does not lessen the danger of lung disease. A few examinations propose that individuals who eat diets with a higher extent of vegetables and natural product have a tendency to have a lower hazard, yet this might be because of perplexing with the lower chance in reality because of the relationship of a high foods grown from the ground eat less carbs with less smoking. (Sun, 2016)

1.8.TREATMENT

Once a lung cancer has been arranged, the doctor and patient can examine treatment choices. An individual at that point has a superior thought of the estimation of various types of treatment. Different variables that are considered incorporate the individual's general wellbeing, restorative issues that may influence treatment, for example, chemotherapy, and tumor attributes. The qualities of the lung tumor help to isolate people into two groups:

- those who are at low risk of cancer recurrence
- those who are at high risk of cancer recurrence

In particular, the histopathologic groupings of small cell lung carcinoma (SCLC) versus non-small cell lung carcinoma (NSCLC) may be used to better predict a patient's prognosis and response to therapy.

1.8.1. Surgery

Not all lung cancers are suitable for surgery. The stage, location and cell type are important limiting factors. In addition, people who are very ill with a poor performance status or who have inadequate pulmonary reserve would be unlikely to survive. Even with careful selection, the overall operative death rate is about 4.4%. In non-small cell lung cancer staging, stages IA, IB,

IIA, and IIB are suitable for surgical resection. Horn, 2015) Pulmonary reserve is measured by spirometry. If there is no evidence of undue shortness of breath or diffuse parenchymal lung disease, and the FEV1 exceeds 2 liters or 80% of predicted, the person is fit for pneumonectomy. If the FEV1 exceeds 1.5 litres, the patient is fit for lobectomy. Types of surgery given below:

- Lobectomy (removal of a lobe of the lung)
- Sublobar resection (removal of part of lobe of the lung)
- Segmentectomy (removal of an anatomic division of a particular lobe of the lung)
- Pneumonectomy (removal of an entire lung)
- Wedge resection (remove a localized portion of diseased lung)
- Bronchoplastic resection (removal of tubular section of the main bronchial passage)
- VATS lobectomy (minimally invasive approach to lobectomy).

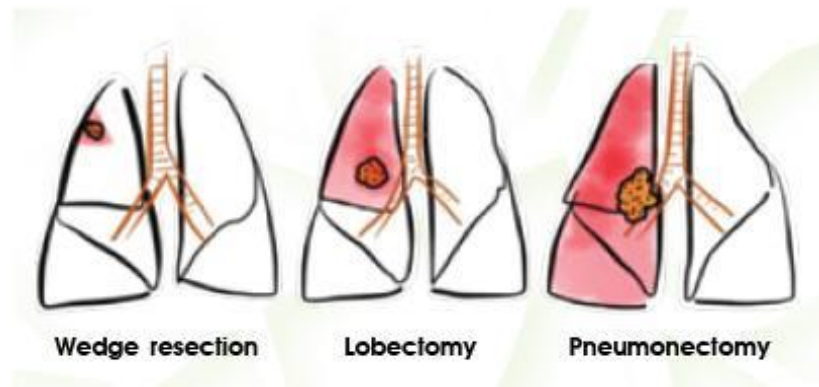


Figure 6: Types of Lung cancer surgery

In most cases of early-stage NSCLC, removal of a lobe of lung (lobectomy) is the surgical treatment of choice. In people who are unfit for a full lobectomy, a smaller sub-lobar excision (wedge resection) may be performed. However, wedge resection has a higher risk of recurrence than lobectomy. Radioactive iodine brachytherapy at the margins of wedge excision may reduce the risk of recurrence. Rarely, removal of a whole lung (pneumonectomy) is performed. Video-assisted thoracoscopic surgery (VATS) and VATS lobectomy use a minimally invasive approach to lung cancer surgery. VATS lobectomy is equally effective compared to conventional open lobectomy, with less postoperative illness. (Rueth, 2010)

In SCLC, chemotherapy and/or radiotherapy are typically used. However the role of surgery in SCLC is being reconsidered. Surgery might improve outcomes when added to chemotherapy and radiation in early stage SCLC. (Goldstein, 2011)

1.8.2. Electrosurgery

Electrosurgery is medical procedure performed utilizing a needle, knob, or circle anode, Nd-YAG laser treatment, cryotherapy, and brachytherapy are extra tumor debulking, or measure diminishing, methods that might be performed amid bronchoscopy. Such techniques are particularly helpful for obstructive, inward depression (intraluminal) lung tumors.

1.8.3. Radiosurgery

Radiosurgery likewise called stereotactic radiosurgery or radiation medical procedure is a kind of outer radiation treatment that might be utilized to treat inoperable lung tumor. In this treatment, a solitary vast dosage of radiation is directed accurately to the tumor, making little harm to sound tissue. Despite its name, stereotactic radiosurgery is anything but a surgery.

1.8.4. Chemotherapy

Small cell lung carcinoma is dealt fundamentally with chemotherapy and radiation, as medical procedure has no self-evident impact on survival. Essential chemotherapy is likewise given in metastatic non-small cell lung carcinoma.

The blend regimen relies upon the tumor compose. Non-small cell lung carcinoma is regularly treated with cisplatin or carboplatin, in mix with gemcitabine, paclitaxel, docetaxel, etoposide, or vinorelbine. In little cell lung carcinoma, cisplatin and etoposide are most regularly utilized. Blends with carboplatin, gemcitabine, paclitaxel, vinorelbine, topotecan, and irinotecan are likewise utilized.

Adjuvant chemotherapy alludes to the utilization of chemotherapy after medical procedure to enhance the result. Amid medical procedure, tests are taken from the lymph hubs. On the off chance that these examples contain cancer, the patient has arranged II or III ailment. In this circumstance, adjuvant chemotherapy may enhance survival by up to 15%. Standard practice is to offer platinum-based chemotherapy. Adjuvant chemotherapy for patients with arranges IB tumor is dubious, as clinical preliminaries have not plainly exhibited a survival advantage.

1.8.5. Radiotherapy

Radiotherapy is frequently given together with chemotherapy and might be utilized with remedial goal in individuals with NSCLC who are not qualified for medical procedure. This type of high-force radiotherapy is called radical radiotherapy. (Arriagada, 2002) A refinement of this procedure is Consistent Hyper-Fractionated Quickened Radiotherapy, in which a high measurement of radiotherapy is given in a brief span period. Postoperative thoracic radiotherapy for the most part ought not to be utilized after therapeutic goal medical procedure for NSCLC. (Fairchild, 2008).

In the event that malignancy development hinders a short area of bronchus, brachytherapy might be given specifically inside the aviation route to open the entry. Contrasted with outside shaft radiotherapy, brachytherapy permits a diminishment in treatment time. Confirmation for brachytherapy, in any case, is not as much as that for outer pillar radiotherapy. (Reveiz, 2012)

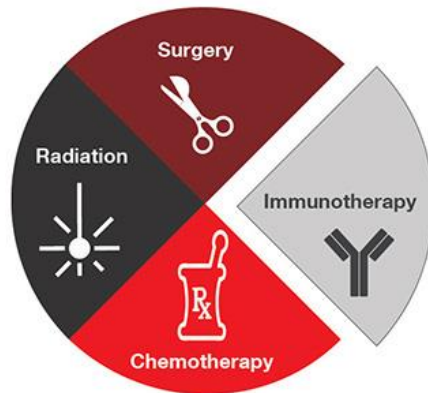


Figure 7: Treatments of Lung cancer

1.8.6. Targeted therapy

A few medications those done for sub-atomic pathways in lung cancer are accessible, particularly for the treatment of cutting edge sickness. Erlotinib, gefitinib and afatinib hinder tyrosine kinase at the epidermal development factor receptor. Denosumab is a monoclonal

counter acting agent coordinated against receptor activator of atomic factor kappa-B ligand. It might be helpful in the treatment of bone metastases. (D'Antonio, 2014)

1.8.7. Photodynamic therapy

Photodynamic therapy might be particularly valuable for the care of people with inoperable lung tumor. Photodynamic treatment starts with the infusion of a light-actuated medication e.g., photofrin/polyhaematoporphyrin, lumin. At that point, amid bronchoscopy, the lung tumor is enlightened by a laser fiber that transmits light of a particular wavelength.

1.9.PROGNOSIS

Overall, fewer than 10% of people with primary lung cancer survived 5 years after diagnosis. But 5-year survival rates may be as high as 35 to 40% among patients who undergo surgical resection for cancer that has not spread beyond the lung. General estimates of stage-specific median survival times the points at which 50% of patients are still living are as follows:

- Stage 1a - more than 60 months (> 5 years)
- Stage 1b - about 36 months (3 years)
- Stage 2a - about 24 months (2 years)
- Stage 2b - about 20 months (< 2 years)
- Stage 3a - about 15 months (< 1.5 years)
- Stage 3b - about 12 months (1 year)
- Stage 4 - about 8 months (< 1 year)

Recent findings indicate that 5-year survival rates approach 85% among patients who are younger than 30 years of age and have surgically removable lung cancers. Researchers believe that genetic mutations may be responsible for differences in survival rates between older and younger people with lung cancer.

Nearly half of limited-stage SCLC patients who have been treated aggressively are alive after 2 to 3 years. By contrast, untreated patients with localized disease show median survivals of approximately 3 to 4 months. Extensive-stage SCLC patients who undergo comfort care have expected median survivals of 4 to 6 weeks. (Merck, 2007)

1.10. ARSENIC AND LUNG CANCER IN BANGLADESH

Arsenic is a known carcinogen but the risk of lung cancer from the widespread contamination of drinking water in rural Bangladesh has not been estimated. To determine whether estimated exposure of villagers in Bangladesh to arsenic in drinking water differed between those with lung cancer and those with non-malignant lesions.

Information was gotten from 7286 subjects who experienced lung biopsy in 2003-2006 at an indicative focus taking referrals from all through Bangladesh. Examination was constrained to 5372 individuals living in towns throughout the previous 10 years who detailed utilizing tube well water. Of these, 3223 with an essential lung tumor were selected as cases and 1588 with non-threatening sores as referents in an unmatched investigation. Arsenic presentation was assessed by normal focuses for every one of 64 areas. Strategic relapse was utilized to test the impacts of age; arsenic and smoking on hazard and to explore relationship to cell compose.

Male cases were more established than referents and more inclined to smoke, to smoke >20 units/day and to smoke biri (little, hand-moved cigarettes). Chances proportions for lung cancer expanded relentlessly with mean arsenic focus, yet the certainty interim avoided 1.0 just at fixations >100 $\mu\text{g/l}$ (OR 1.45, 95% CI 1.16 to 1.80). This pattern was seen just in smokers where the expanded hazard at >100 $\mu\text{g/l}$ was 1.65 (95% CI 1.25 to 2.18). A comparable pattern was found in ladies smokers. Squamous cell lung cancer was more successive in smokers and, having balanced for smoking, in locale with arsenic focuses >100 $\mu\text{g/l}$.

Among Bangladeshis who smoke, those whose drinking water is contaminated with arsenic at concentrations >100 $\mu\text{g/l}$ are at increased risk of lung cancer. With high levels of exposure misclassification and short latency of exposure, the study cannot estimate or exclude the likely long-term risk in non-smokers and at lower arsenic concentrations. (Health Research Ethics Board of the University of Alberta, 2008)

The Global Organization for Exploration on Malignancy finished up in 2004 that arsenic in drinking water was presumably causally identified with lung growth in man. Be that as it may, the investigations referred to were constrained both as respect geological zone (Taiwan, Chile) and in data on potential confounder. The biggest populaces presented to high centralizations of arsenic through drinking water live in Bangladesh and West Bengal (India), yet the danger of

lung growth in these territories has not yet been evaluated and may vary due to other water borne contaminants, smoking propensities and perhaps hereditary variables. Affirmation of a causal association with ingested arsenic is critical deductively; in that exclusive breathed in cancer-causing agents have already been identified with essential lung growth. It is likewise a noteworthy general medical problem in a district with extremely constrained social insurance limit.

The collaboration amongst arsenic and tobacco utilize is questionable: past investigations of natural (ingested) and word related (breathed in) arsenic recommend a synergistic impact, with tobacco and arsenic having a hazard that is more noteworthy than added substance. The present examination tends to these inquiries encourage in patients with lung opacities explored in a solitary symptomatic focus in Dhaka, Bangladesh. Our goal is to decide if subjects whose biopsy showed an essential lung disease will probably have lived in a town with high arsenic defilement than those whose biopsy result was considerate, and to evaluate the part of tobacco use in this relationship.

- Arsenic sullying in drinking water from tube wells in country Bangladesh is related with lung growth in guys.
- Having balanced for smoking, squamous cell lung diseases are more probable in zones with arsenic fixations over 100 $\mu\text{g/l}$
- In the two people, the impact of arsenic in drinking water on lung cancer is most noteworthy in the individuals who smoke.

Among men living in towns and drinking tube well water, just 13.8% were in regions in which the assessed presentation met the WHO rule of 10 $\mu\text{g/l}$, however a further 47.8% met the Bangladesh rule of 50 $\mu\text{g/l}$; the staying 38.4% of male patients lived in regions with fixations over that level, the most noteworthy being 366 $\mu\text{g/l}$. By and large, for men, the extent of cases expanded with mean arsenic focus in savoring water their locale, however the related 95% CI barred 1.0 just at $>100 \mu\text{g/l}$ (OR 1.45, 95% CI 1.16 to 1.80) contrasted and 0– 10 $\mu\text{g/l}$. In an investigation stratified by smoking propensity and balanced for age and tobacco item and amount, the expansion in chances proportions with arsenic focus was seen just in men who smoked.

1.11. EPIDEMIOLOGY

Lung cancer has been the most common cancer in the world for several decades. There are estimated to be 1.8 million new cases in 2012 (12.9% of the total), 58% of which occurred in the less developed regions. The disease remains as the most common cancer in men worldwide (1.2 million, 16.7% of the total) with the highest estimated age-standardized incidence rates in Central and Eastern Europe (53.5 per 100,000) and Eastern Asia (50.4 per 100,000). Notably low incidence rates are observed in Middle and Western Africa (2.0 and 1.7 per 100,000 respectively). In women, the incidence rates are generally lower and the geographical pattern is a little different, mainly reflecting different historical exposure to tobacco smoking. Thus, the highest estimated rates are in Northern America (33.8) and Northern Europe (23.7) with a relatively high rate in Eastern Asia (19.2) and the lowest rates again in Western and Middle Africa (1.1 and 0.8 respectively). (IARC, 2015)

Lung cancer is the most common cause of death from cancer worldwide, estimated to be responsible for nearly one in five (1.59 million deaths, 19.4% of the total). Because of its high fatality (the overall ratio of mortality to incidence is 0.87) and the relative lack of variability in survival in different world regions, the geographical patterns in mortality closely follow those in incidence.

According to the latest WHO data published in 2017 Lung Cancers Deaths in Bangladesh reached 12,075 or 1.53% of total deaths. The age adjusted Death Rate is 11.33 per 100,000 of population ranks Bangladesh is 99 in the world. (World Health Organization, 2017)

LITERATURE REVIEW

2. LITERATURE REVIEW

Women were more likely to be never-smokers than men, particularly those with the squamous type cancer (8.3% for women versus 2.9% for men 55 years old or older). Men started smoking earlier, reported inhaling more deeply, and smoked more cigarettes per day than women. In contrast, dose response ORs over cumulative exposure to cigarette smoking were 1.2-fold to 1.7-fold higher in women than in men for the three major histologic types; these differences were more pronounced for small cell carcinomas and adenocarcinomas than for squamous carcinomas. Adjustments for weight, height, or body mass index did not alter the ORs. The ORs for major lung cancer types are consistently higher for women than for men at every level of exposure to cigarette smoke. Furthermore, this gender difference cannot be explained by differences in baseline exposure, smoking history, or body size, but it is likely due to the higher susceptibility to tobacco carcinogens in women.

Depression was self-rated in 322 patients (33%) before treatment and persisted in more than 50% of patients. 25 % SCLC patients had a three-fold greater prevalence of case depression than those with 9% NSCLC. An increased rate for women was found for good performance status patients (PS of 0 or 1) but the sex difference reduced for poor PS patients (PS of 3 or 4) because of increased depression rates for men (χ^2 for trend, $P < .0001$). Multivariate analysis showed that depression increased by 41% for each increment on the impairment scale. Pretreatment physical symptom burden, fatigue, and clinician-rated PS were also independent predictors, but cell type was not. Depression is common and persistent in lung cancer patients, especially those with more severe symptoms (Journal of Clinical Oncology, February 2000).

Fine particulate and sulfur oxide related pollution were associated with all cause, lung cancer, and cardiopulmonary mortality. Each 10- $\mu\text{g}/\text{m}^3$ elevation in fine particulate air pollution was associated with approximately 4%, 6%, and 8% increased risk of all cause, cardiopulmonary, and lung cancer mortality, respectively. Measures of coarse particle fraction and total suspended particles were not consistently associated with mortality. Long-term exposure to combustion related fine particulate air pollution is an important environmental risk factor for cardiopulmonary and lung cancer mortality.

A total of 1867 patients underwent randomization; 36.5 percent had pathological stage I disease, 24.2 percent stage II, and 39.3 percent stage III. Of the 932 patients assigned to chemotherapy,

73.8 percent received at least 240 mg of cisplatin per square meter of body-surface area. The median duration of follow-up was 56 months. Patients assigned to chemotherapy had a significantly higher survival rate than those assigned to observation (44.5 percent vs. 40.4 percent at five years). There were no significant interactions with prespecified factors. Seven patients (0.8 percent) died of chemotherapy-induced toxic effects. Cisplatin-based adjuvant chemotherapy improves survival among patients with completely resected non–small-cell lung cancer.

Patients with inoperable non–small cell lung cancer who received stereotactic body radiation therapy had a survival rate of 55.8% at 3 years, high rates of local tumor control and moderate treatment-related morbidity. While anatomical resection is the standard treatment for early stage lung cancer, some patients cannot tolerate surgery due to comorbidities such as emphysema and heart disease. These patients are deemed medically inoperable and are generally offered conventional radiotherapy, most commonly given during 20-30 outpatient treatments or observed without specific cancer therapy. Outcomes are not ideal with either approach. Conventional radiotherapy fails to durably control the primary lung tumor in 60% to 70% of patients. More than half of patients ultimately die specifically from progressive lung cancer with observation and 2-year survival is less than 40% with either approach. (International Adjuvant Lung Cancer Trial, 2004)

METHODS
&
MATERIALS

3. METHODS & MATERIALS

3.1.SUBJECTS AND PLACE OF STUDY

A hospital-based case study of **“A case study of lung cancer patients in term of various risk factors in context of Bangladesh”** was reviewed and approved by Ahsania Mission Cancer Hospital was conducted.

Briefly, all newly diagnosed cases of cancer patients aged 30-100 from November 2017 to May 2018 were identified by collaborating physicians in Ahsania Mission Cancer Hospital, to ensure completeness of case ascertainment, records of the cancer registry were also checked regularly.

For each case, patients’ files obtained and reviewed. After an initial visit to obtain oral informed consent and schedule a visit for the interview, the survey was conducted by face to face interview.

The methodology followed in preparing the study consisted of the following:

- Critical scrutiny of auxiliary sources including books, article, reports, unpublished postulation, figures, official records, sites and so forth.
- Primary information gathering through field interviews, contextual analyses and organization of surveys among chosen respondents in basic regions that need considerable information.
- A point by point survey of the primary draft of the report through counsel with the chief.
- The point by point surveys were produced based on the current writing, sentiment and proposal got from the manager and seniors.
- The respondents were asked to reply by the poll after appropriately disclosing to them the goals of the examination and the substance of the survey.

3.2.INTERVIEWS

Face to face, structure interviews were directed were control cases and the closest relative of expired cases. An aggregate of 300 growths tolerant took part in the investigation. Data was gotten about statistic qualities, occupation and living arrangement narratives, youth living condition, history of chose therapeutic condition and family history of malignancy, tobacco and liquor utilization. An ever smoker was characterized as a smoker of no less than one cigarette for every day for a half year or more. At first therapeutic history of patient was asked as the statistic profile may once in a while lead the patient to a humiliating circumstance and they may decline to give the appropriate response. At that point they were gotten some information about their nourishment propensity lastly their statistic profile was accumulated from the enrollment type of doctor's facility records. In each inquiry there were some choices to help replying.

3.3.STATISTICAL ANALYSIS

Our sampling procedure is non-probabilistic and convenience sampling. Percentage and frequency of the attributable fractions were calculated for each risk factor of cancer. For analyses, the percentage and frequency of each risk factor were presented by pie diagram, bar diagram tables and interpreted.

RESULTS

&

DISCUSSION

4. RESULTS AND DISCUSSION

A total of 300 subjects responded to the survey, with a higher response rate in men (89%) than in women (11%). Response rate was lower in the 30s strata than in the other age groups, but no trend to an increase in response rate with increasing age was observed overall no significance differences in area age distribution was between the sample population and survey respondents.

Risk factors associated with lung cancer among the Bangladeshi patients

Age, Gender, Weight, Height, Blood pressure, Pulse Rate, Respiratory rate, Temperature, Pneumonia, Tuberculosis, Bronchitis, Asthma, Heart disease, Diabetes, Hypertension, Cachexia, Anorexia, Loss of body weight, Coughing, Hemoptysis, Dyspnea Hoarseness, Dysphagia, Chest Pain, Anemia, Hemoglobin, Liver Enlargement, Lymph Nodes Enlargement Bone pain, Chest X-ray, Computed Therapy, Scan of chest, FNAC, Bronchoscopy, Stage group, Surgery, Radiation therapy, Chemotherapy, Combined therapy, Histological Type, Smoking, Passive smoking, Co-existing lung disease, Genetic factors, Environmental factors.

The present study focused at the Bangladeshi cancer patients, demonstrated that the inferable division of disease among the cancer patients had a tendency to be higher for tobacco smoking, biting, cleanliness, malignancy causing bacterial and viral contamination, age air contamination, water containing arsenic than real way of life factors, for example, dietary elements.

In numerous underdeveloped nations cancer frequency seems much lower, in all likelihood in view of the higher passing rates because of irresistible damage. With the expanded control over tuberculosis in some underdeveloped nations, occurrence of tumor is relied upon to rise; this is termed the epidemiology transition in epidemiological terminology. Cancer epidemiology closely mirrors risk factor spread in various countries. Tobacco smoking becomes more common in various Third world countries like Bangladesh lung cancer incidence has increased in a parallel portion.

In the present study the attributable fraction was considered highest for tobacco smoking which has been correspondents to the findings of other reports from other parts of the worlds. When a cigarette burns it releases thousands of different chemicals, many of them are harmful to health. The three main components of cigarette smoke are nicotine a fast acting drug that makes smokers crave cigarettes.

- **Carbon monoxide** -A poisonous gas that reduces oxygen in the blood stream causing breathing problems
- **Tar**- A sticky black residue made up of thousands of chemicals that stays in the smoker's lung and causes cancer
- **Nicotine** -Very addictive one constitutes approximately 0.6–3.0% of the dry weight of tobacco
- **Acetone** - The colorless, volatile, flammable organic liquid compound. It is used for cleaning purposes such as nail polish remover
- **Ammonia**– A colorless gas with a characteristic pungent smell which is served as food, fertilizers and for cleaning purposes
- **Arsenic** -Used in pest control and insecticides however the use of arsenic is declining.
- **Cadmium** -used in batteries
- **Formaldehyde**- A naturally occurring organic compound. It is used in the production of industrial resins, e.g., coatings and to preserve dead bodies from decaying.

Pathological changes of lung cancer in miners of Yunnan Tin Mine were studied, and additionally, mineral dusts in the miners' lung were also investigated by using scanning electronic microscope, energy disperse X-ray spectrometer and electronic probe. The results showed:

- Mineral dust caused active hyperplasia, atypical hyperplasia, metaplasia and atypical metaplasia of the epithelial of alveoli and bronchi, which was able to induce cancer.
- Pneumoconiosis-like changes in the miners' lung are correlated with the high incidence of lung cancer.
- Correlated also with copper, lead, zinc and iron may be the pathological changes of lung cancer in miners of Yunnan Tin Mine were studied, and additionally, mineral dust in the miners' lung were also investigated by using scanning electronic microscope, energy high incidence of lung cancer.
- Transition form from hyperplasia and atypical hyperplasia of alveolar epithelia to malignancy was observed.
- Lung squamous cell carcinoma probably originates from the alveolar epithelia of the lung.

GRAPHS RELATED TO LUNG CANCER RISK FACTORS

Table 1: Frequency distribution of lung cancer patients based on age

Age	Frequency	Percentage
30-44	26	8.7%
45-54	43	14.3%
55-64	72	24%
64-96	159	53%
Total	300	100%

With ages the risk of lung cancer increases in time. From our present survey it is observed that the higher number of patients was aged more than 64 years old, which was 53%. About 8.7% patients were aged less than 45, 14.3% between 45 to 54 years and 24% between 55 to 64 years.

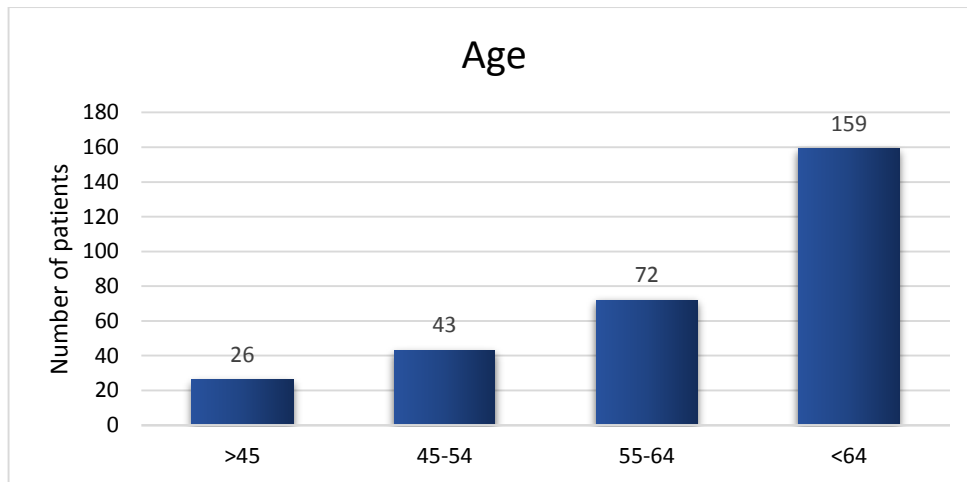


Figure 8: Lung cancer related to age (years)

Table 2:Frequency distribution of lung cancer patients based on gender

Gender	Frequency	Percentage
Male	189	63%
Female	111	37%
Total	300	100%

The majority of advanced lung cancer cases were found in men (63%) over age 55. Men were approximately 1.7 times more likely to get lung cancer than women (37%).

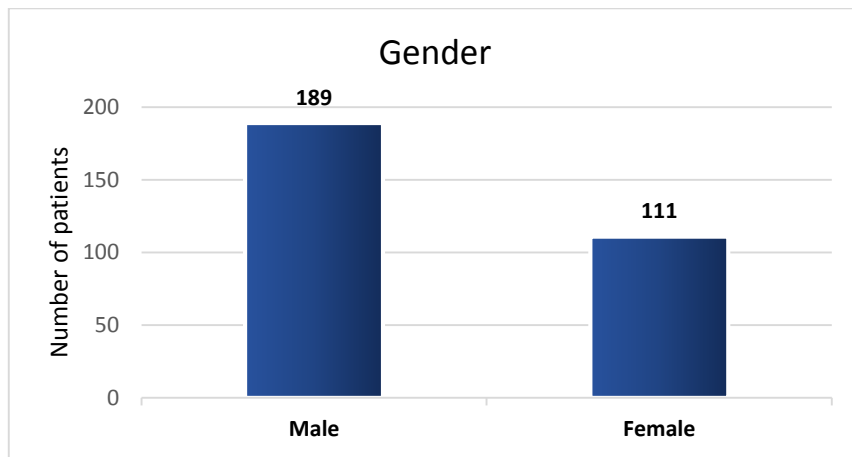


Figure 9: Lung cancer related to gender

Table 3:Frequency distribution of lung cancer patients based on weight

Weight (kg)	Frequency	Percentage
30-45	40	13.3%
46-54	150	50%
55-64	74	24.7%
65-75	36	12%
Total	300	100%

Due to loss of appetite and physical condition, the weight began to decrease. In most cases, highest number that is 50% of people fall between 46 to 54 kg, 13.3% fall below 46 kg, 24.7% between 55 to 64 kg and 12% above 64 kg.

Table 4: Frequency distribution of lung cancer patients based on height

Height (cm)	Frequency	Percentage
156	66	22%
157-170	159	53%
171-185	75	25%
Total	300	100%

In most cases, highest number that is 22% of people fall less than 157 cm, 53% between 157 to 170 cm, 25% above 170 cm.

Table 5: Frequency distribution of lung cancer patients based on BMI

BMI	Frequency	Percentage
Underweight (Below-18.4)	15	5%
Normal (18.5-24.9)	144	48%
Overweight (25-29.9)	108	36%
Obese (30- Above)	33	11%
Total	300	100%

Among 300 patients, 5% were underweight, 48% were normal, 36% were overweight and 11% were obese according to the BMI format with the help of weight and height of the lung cancer patients.

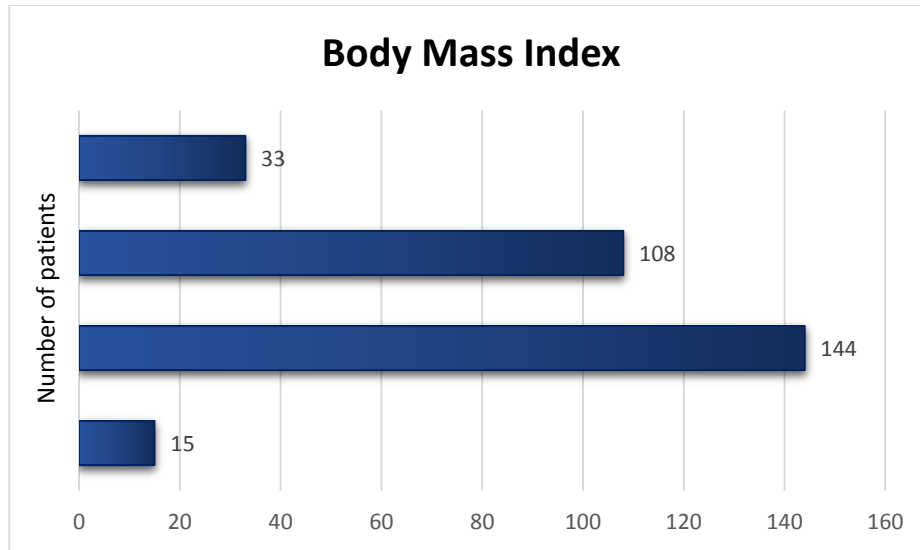


Figure 10: Lung cancer related to BMI

Table 6: Frequency distribution of lung cancer patients based on blood pressure

Blood Pressure	Frequency	Percentage
Low	176	58.7%
Normal	64	21.3%
High	60	20%
Total	300	100%

Blood pressure is vital for health. Maximum number (58.7%) of people had low blood pressure which is below 60/90 mmHg, 21.3% had normal blood pressure of 80/120 mmHg and 20% had high blood pressure of above 90/140 mmHg.

Table 7: Frequency distribution of lung cancer patients based on pulse rate

Pulse Rate (beat per minutes)	Frequency	Percentage
60-84	30	10%
85-99	75	25%
100-130	195	65%
Total	300	100%

Among 300, 10% had pulse rate slower than 85 bpm, 25% had between 85 to 99 bpm and 65% had pulse rate faster than 99 bpm.

Table 8: Frequency distribution of lung cancer patients based on respiratory rate

Respiratory Rate (breaths per minutes)	Frequency	Percentage
10-14	30	10%
15-24	153	51%
24-30	117	39%
Total	300	100%

About 10% cases had low R/R below 14 bpm, 51% had normal rate between 15 to 24 bpm and 39% high rate above 24 bpm.

Table 9: Frequency distribution of lung cancer patients based on temperature

Temperature (°F)	Frequency	Percentage
85-96	30	10%
97-99	150	50%
100-106	120	40%
Total	300	100%

According to survey, approximately 10% patients had temperature below 97°F, 50% had between 97 to 99°F and 40% had temperature above 99°F.

Table 10: Frequency distribution of lung cancer patients based on respiratory disease

Respiratory disease	Frequency	Percentage
Pneumonia	81	27%
Tuberculosis	13	39%
Bronchitis	48	16%
Asthma	54	18%
Total	300	100%

According to the survey of lung cancer patients, the percentages of each respiratory disease were different. Among 300 patients, 27% had pneumonia, 39% had tuberculosis, 16% had bronchitis and 18% had asthma.

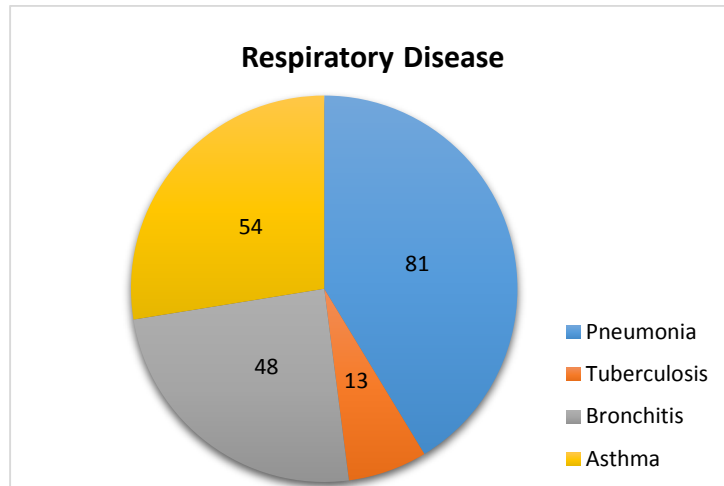


Figure11: Lung cancer related to respiratory disease

Table 11: Frequency distribution of lung cancer patients based on history of diseases

History of diseases	Frequency	Percentage
Heart disease	115	38.3%
Diabetes	100	33.3%
Hypertension	85	28.4%
Total	300	100%

Out of 300, about 38.3% patients were found diseased regarding heart problem, 33.3% had diabetes and 28.4% had hypertension

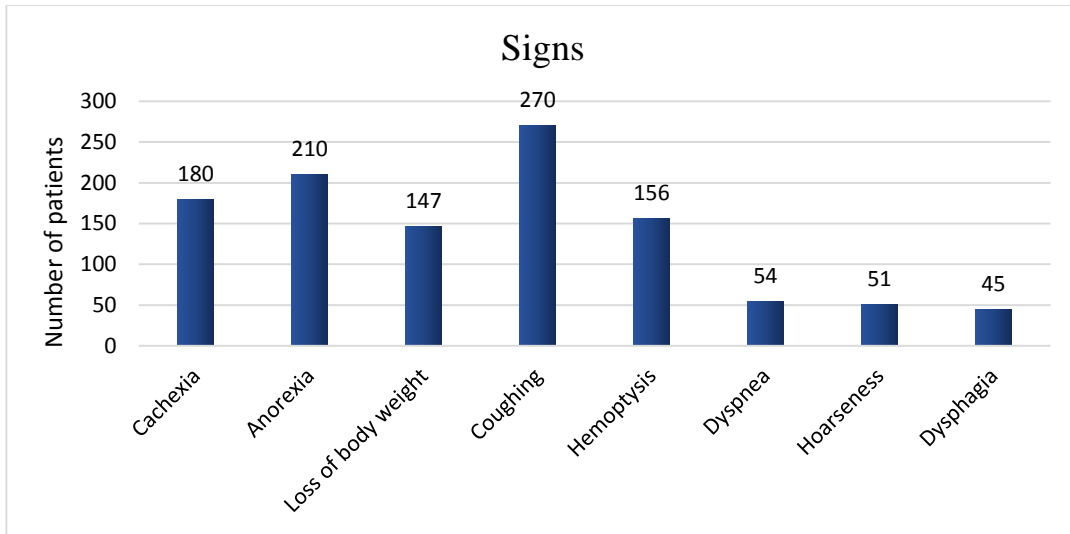


Figure 12: Lung cancer's signs

About 60% lung cancer patients had cachexia, 70% anorexia, 49% loss of body weight, 90% coughing, 52% hemoptysis, 18% dyspnea, 17% hoarseness and 15% dysphagia. More or less every patient shows more than three signs.

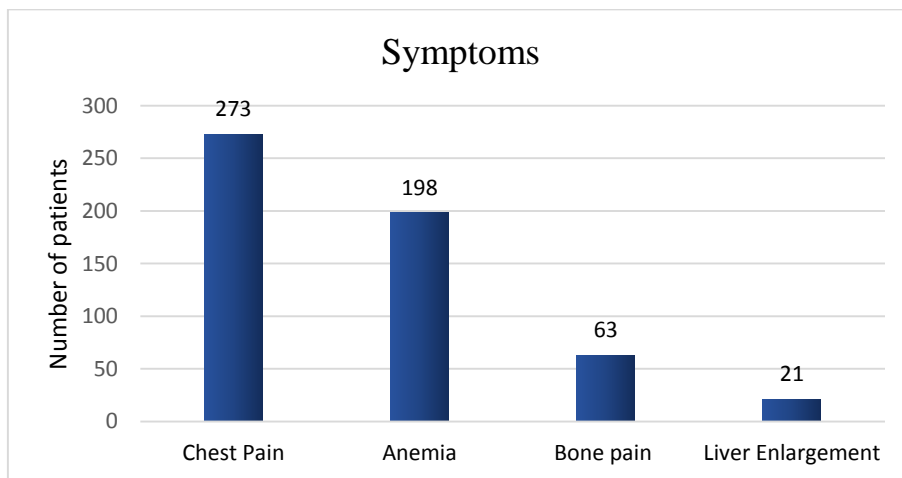


Figure 13: Lung cancer's symptoms

Most of the cases, more than one symptom were seen, among them about 91% had chest pain, 66% anemia, 21% bone pain and 7% liver enlargement.

Table 12: Frequency distribution of lung cancer patients based on hemoglobin

Hemoglobin (g/dL)	Frequency	Percentage
6-9	148	49.3%
10-14	98	32.7%
15-19	54	18%
Total	300	100%

Out of 300 cases, 49.3% had hemoglobin less than 10 g/dL, 32.7% had normal hemoglobin between 10 to 14 g/dL and 18% had hemoglobin higher than 14 g/dL.

Table 13: Frequency distribution of lung cancer patients based on lymph nodes

Lymph nodes enlargement	Frequency	Percentage
Cervical	225	75%
Supraclavicular	27	9%
Axillary	30	10%
Other	18	6%
Total	300	100%

About 75% had cervical lymph nodes, 9% had supraclavicular, 10% had axillary and 6% had other types of lymph nodes enlargement.

Table 14: Frequency distribution of lung cancer patients based on diagnosis

Diagnosis	Frequency	Percentage
Chest X-Ray	24	8%
Computed Therapy	75	25%
FNAC	124	41.3%
Bronchoscopy	77	25.7%
Total	300	100%

Out of 300 cases, 8% had chest X-ray, 25% had computed therapy, 41.3% had FNAC and 25.7% had bronchoscopy.

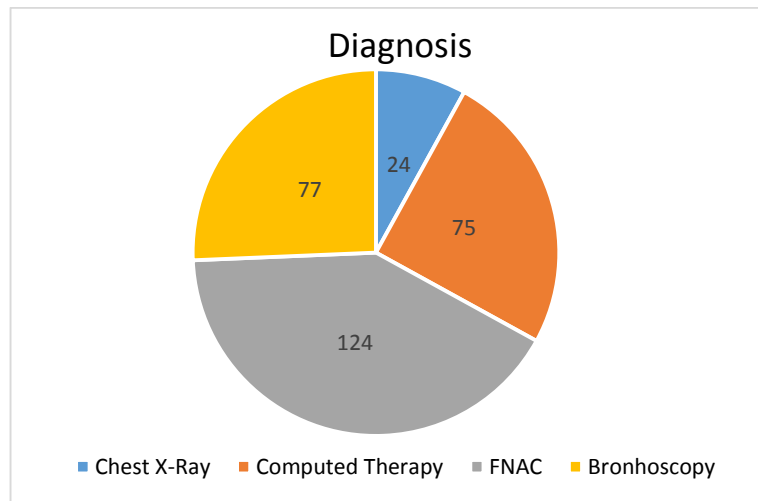


Figure 14: Diagnostic procedure in lung cancer

Table 15: Frequency distribution of lung cancer patients based on treatment

Treatment	Frequency	Percentage
Surgery	107	35.7
Radiation Therapy	64	21.3
Chemotherapy	53	17.7
Combined Therapy	76	25.3
Total	300	100%

Out of 300 cases, 35.7% had surgery, 21.3% had radiation therapy, 17.7% had chemotherapy and 25.3% had combined therapy.

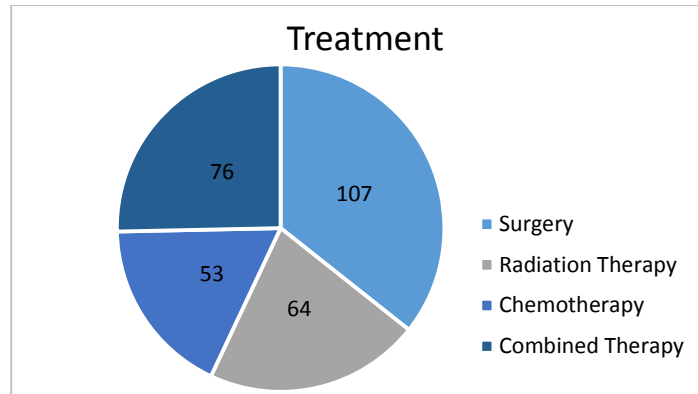


Figure 15: Treatment of lung cancer

Table 16: Frequency distribution of lung cancer patients based on histological type

Histological Type	Frequency	Percentage
Small-Cell Lung Carcinoma	45	15%
Squamous-Cell Carcinoma	90	30%
Adenocarcinoma	126	42%
Large-Cell Carcinoma	30	10%
Carcinoid Tumor	9	3%
Total	300	100%

About 15% had small-cell lung carcinoma (SCLC), in non-small-cell lung carcinoma (NSCLC) 30% had squamous-cell carcinoma, 42% had adenocarcinoma, 10% had large-cell carcinoma and 3% had carcinoid tumor.

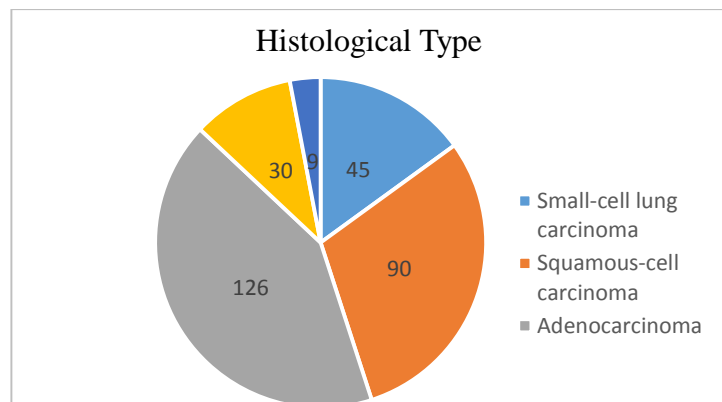


Figure 16: Histological type of lung cancer

Table 17: Frequency distribution of lung cancer patients based on smoking habit

Smoking habit	Frequency	Percentage
Smoker	181	60.3%
Ex-smoker	21	7%
Non-smoker	39	13%
Passive Smoker	59	19.7%
Total	300	100%

Lung cancer can happen to anyone but among them smokers are greatly affected. About 60.3% were smokers, 7% were ex-smokers, 13% were non-smokers and 19.7% were passive smokers.

Table 18: Frequency distribution of lung cancer patients based on smoking types

Smoking	Frequency	Percentage
Tobacco	86	28.7%
Cigarette	152	50.7%
Cannabis	49	16.3%
Other	13	4.3%
Total	300	100%

Maximum men are addicted to smoking and in or country mostly women are interested in chewing tobacco. Out of 300 cases in survey, 44% were used to have tobacco, 78% used to take cigarette, 25% had cannabis and 7% take other drugs.

Table 19: Frequency distribution of lung cancer patients based on smoking duration

Smoking Duration	Frequency	Percentage
7 years and less	77	25.7%
8-18 years	121	40.3%
18 years and above	102	34%
Total	300	100%

Tendency to have lung cancer also depend on the duration of smoking. About 25.7% were smoking for 7 years or less, 40.3% for 8 to 18 years and 34% for more than 18 years.

Table 20: Frequency distribution of lung cancer patients based on smoking sticks per day

Sticks Per Day	Frequency	Percentage
1-10	110	36.7%
11-20	121	40.3%
Above 20	75	25%
Total	300	100%

Lung cancer stages depend on the amount of smoking cigarette per day, which can be regular or occasionally. Based on the survey around 36.7% smoked 1 to 10 sticks per day, 40.3% smoked 11 to 20 sticks and 25% more than 20 sticks per day.

Table 21: Frequency distribution of lung cancer patients based on quit smoking

Quit Smoking	Frequency	Percentage
Less than 3 years	48	16%
3-6 years	152	50.7%
Above 6 years	55	18.3%
Total	300	100%

Out of 300, many of them quit smoking for years, among the quitters 16% quit for less than 3 years, 50.7% for 3 to 6 years and 18.3% for more than 6 years.

Table 22: Frequency distribution of lung cancer patients based on co-existing lung disease

Co-Existing Lung Disease	Frequency	Percentage
Diffuse Pulmonary Fibrosis	21	7%
Chronic Infection	180	60%
Chronic Obstructive Pulmonary Disease	99	33%
Total	300	100%

There are some diseases which increase the risk of having lung cancer, among them 7% were affected by diffuse pulmonary fibrosis, 60% by chronic infection and 33% by chronic obstructive pulmonary disease.

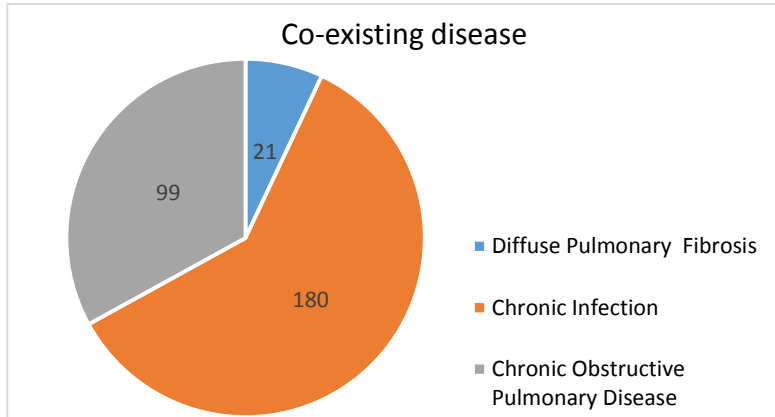


Figure 17: Co-existing lung disease related to lung cancer

Table 23: Frequency distribution of lung cancer patients based on genetic factor

Genetic factor	Frequency	Percentage
Grandfather	24	8%
Grandmother	20	6.7%
Father	33	11%
Mother	30	10%
Brother	15	5%
Sister	13	4.3%
Total	300	100%

Out of 300, 135 had a background history of family cancer which lead to their lung cancer, among them 8% were from grandfather, 6.7% grandmother, 11% father, 10% mother, 5% brother and 4.3% sister.

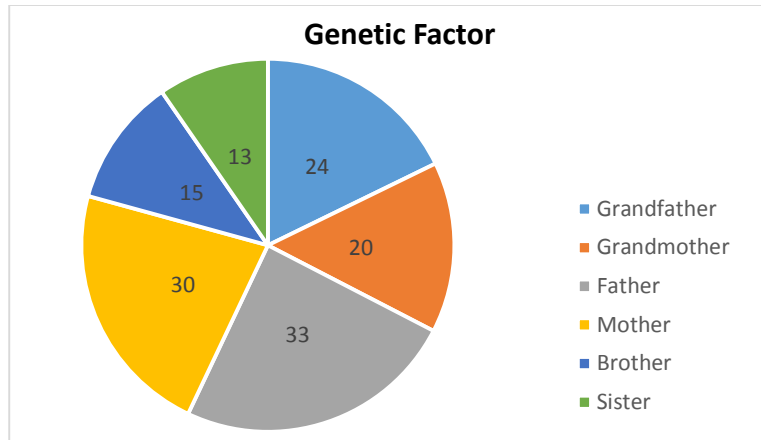


Figure 18: Genetic factor related to lung cancer

Table 24: Frequency distribution of lung cancer patients based on environmental factors

Environmental factors	Frequency	Percentage
Asbestos	69	23%
Radon	35	11.7%
Chromium	25	8.3%
Arsenic	48	16%
Others	15	5%
Total	300	100%

Environmental factors can also be the reason behind lung cancer. About 23% people were affected by asbestos exposure, 11.7% by radon gas, 8.3% by chromium and 16% by arsenic present in nature. Other 5% was the reason for cancer like pesticides, harmful chemical and polluted air.

Table 25: Frequency distribution of lung cancer patients based on stages

Stages	Frequency	Percentage
IA	81	27%
IB	63	21%
IIA	68	22.7%
IIB	40	13.3%

IIIA	30	10%
IIIB	15	5%
IV	3	1%
Total	300	100%

People of different stages got admitted in the hospital, most got cured in first and second stages but very less amount of people survived at the very last stages. These type of people got maximum 5 years in life. About 27% had IA, 21% had IB, 22.7% had IIA, 13.3% had IB, 10% had IIIA, 5% had IIIB and 1% had IV stage.

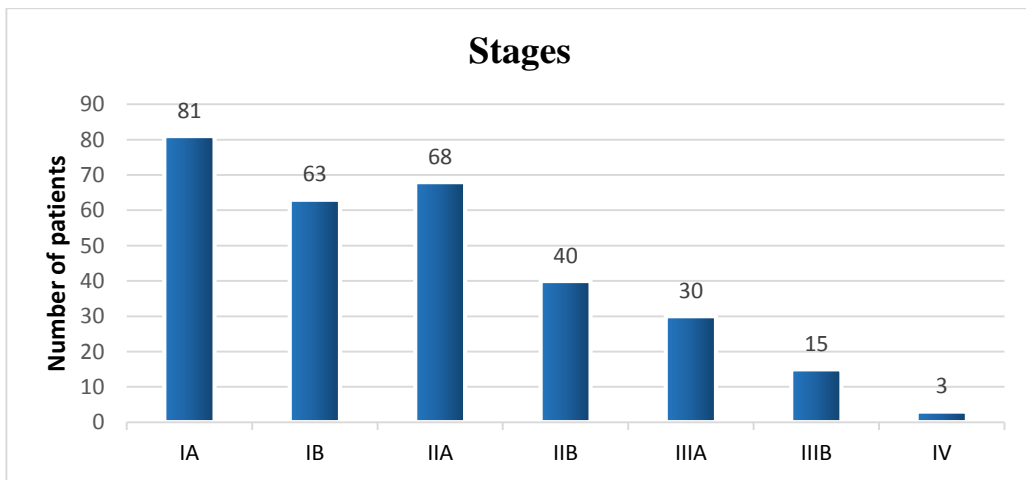


Figure 19: Stages of lung cancer

CROSS-CHECK WITH THE GENDER OF LUNG CANCER PATIENTS

Table 26: Cross table analysis between gender and lung cancer related to other diseases

Gender	Male	Female	Total
Heart Disease	50 (45.5%)	60 (54.5%)	110
Diabetes	57 (60%)	38 (40%)	95
Hypertension	54 (69.2%)	24 (30.8%)	78

Among 300 patients, 110 had heart disease from which 50 were male and 60 were female. Out of 95 patients, 57 male and 38 female had diabetes. Out of 78, 54 male and 24 female had hypertension.

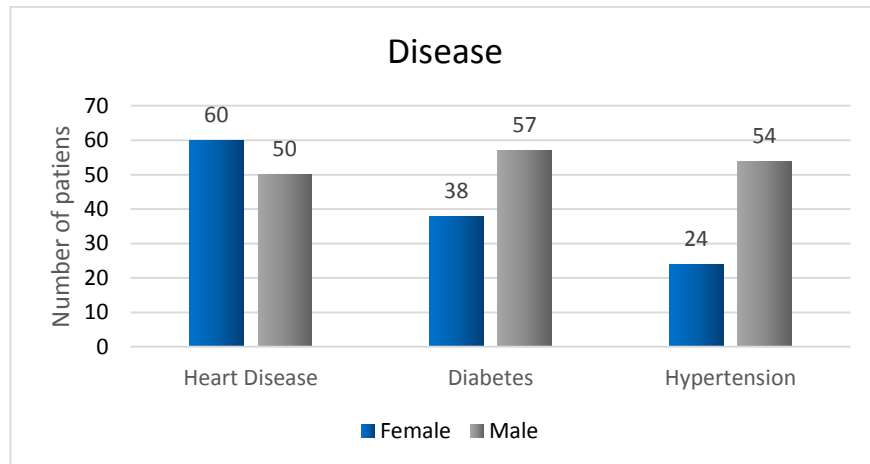


Figure 20: Gender ratio related diseases to lung cancer

Table 27: Cross table analysis between gender and lung cancer related respiratory diseases

Gender	Male	Female	Total
Pneumonia	49 (60.4%)	32 (39.6%)	81
Tuberculosis	9 (69.2%)	4 (30.8%)	13
Bronchitis	30 (62.5%)	18 (37.5%)	48
Asthma	35 (64.81%)	19 (35.19%)	54

Among 300 patients, 81 had pneumonia from which 49 were male and 32 were female. Out of 13 patients, 9 male and 4 female had tuberculosis. Out of 48, 30 male and 18 female had bronchitis. And out of 54, 35 male and 19 female had asthma.

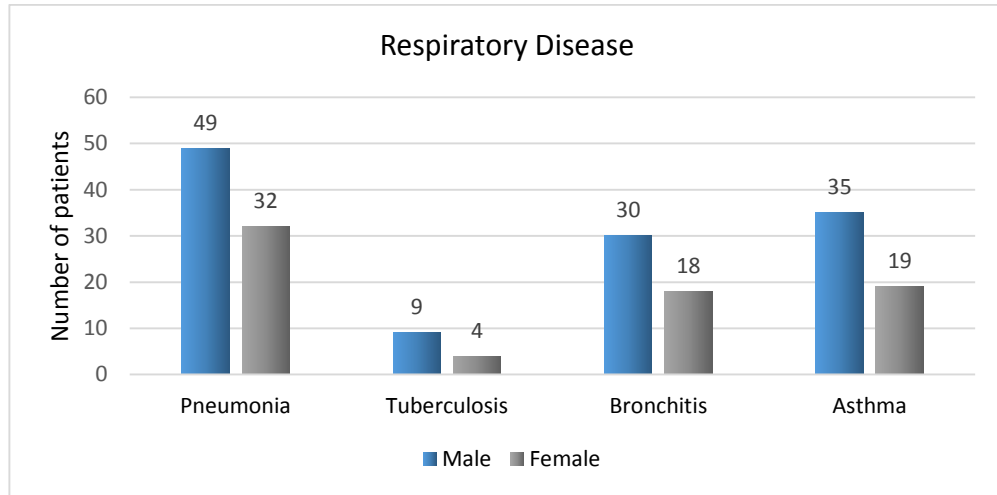


Figure 21: Gender ratio related to respiratory diseases

Table 28: Cross table analysis between gender and lung cancer related to diagnosis

Diagnosis	Male	Female	Total
Chest X-Ray	16 (66.7%)	8 (33.3%)	24
Computed Therapy	49 (65.3%)	26 (34.7%)	75
FNAC	80 (64.5%)	44 (35.5%)	124
Bronchoscopy	45(58.4%)	32 (41.6%)	77

Among 300 patients, 24 had chest x-ray from which 16 were male and 8 were female. Out of 75 patients, 49 male and 26 female had computed therapy. Out of 124, 80 male and 44 female had FNAC. And out of 77, 45 male and 32 female had bronchoscopy.

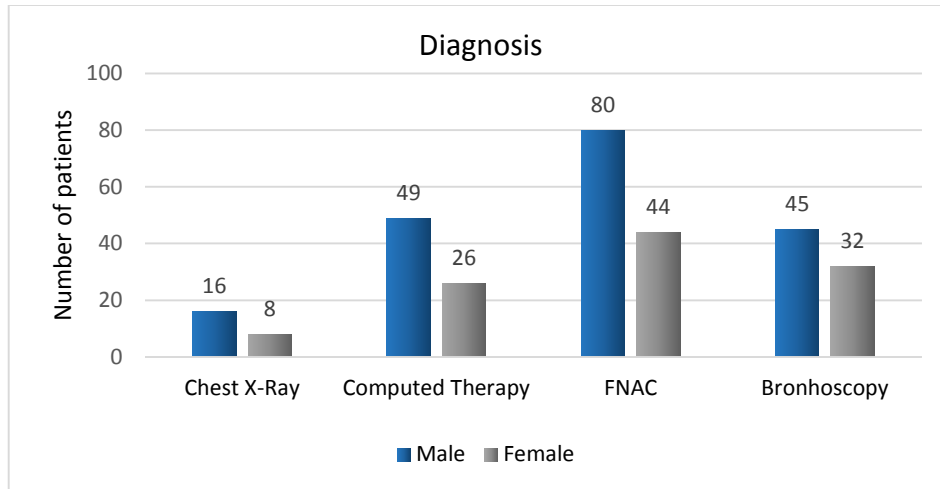


Figure 22: Gender ratio related to diagnosis of lung cancer

Table 29: Cross table analysis between gender and lung cancer related to treatment

Treatment	Male	Female	Total
Surgery	66 (61.7%)	41(38.3%)	107
Radiation Therapy	35(54.6%)	29(45.4%)	64
Chemotherapy	33(62.2%)	20(37.8%)	53
Combined Therapy	45(59.5%)	31(40.5%)	76

Among 300 patients, 107 had surgery from which 66 were male and 41 were female. Out of 64 patients, 35 male and 29 female had radiation therapy. Out of 53, 33 male and 20 female had chemotherapy. And out of 76, 45 male and 31 female had combined therapy.

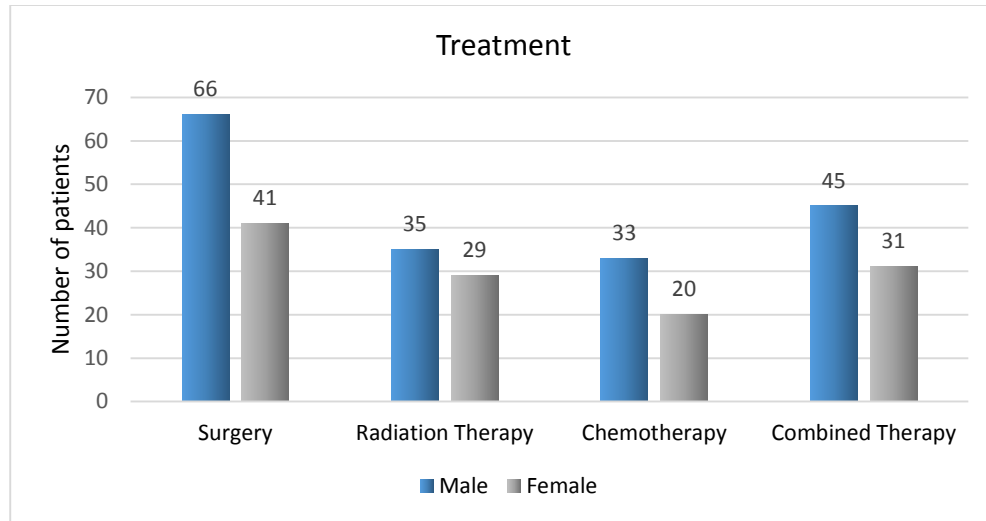


Figure 23: Gender ratio related to treatment of lung cancer

Table 30: Cross table analysis between gender and lung cancer related to histological type

Histological Type	Male	Female	Total
Small-Cell Lung Carcinoma	25 (55.5%)	20(44.5%)	45
Squamous-Cell Carcinoma	53(58.8%)	37(41.2%)	90
Adenocarcinoma	70(55.5%)	56(44.5%)	126
Large-Cell Carcinoma	18(60%)	12(40%)	30
Carcinoid Tumor	6(66.7%)	3(33.3%)	9

Among 300 patients, 45 had small-cell lung carcinoma from which 25 were male and 20 were female. Out of 90 patients, 53 male and 37 female had squamous-cell carcinoma. Out of 126, 70 male and 56 female had adenocarcinoma. Out of 30, 18 male and 12 female had large-cell carcinoma. And out of 9, 6 male and 3 female had carcinoid tumor.

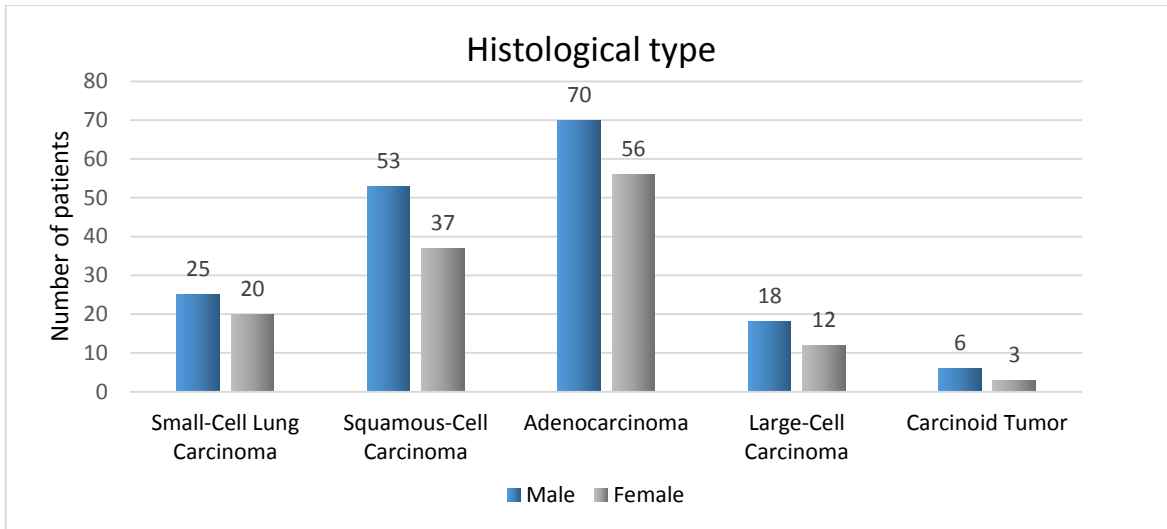


Figure 24: Gender ratio related to histological type of lung cancer

Table 31: Cross table analysis between gender and lung cancer related to smoking habit

Smoking habit	Male	Female	Total
Smoker	111 (61.3%)	70(38.7%)	181
Ex-smoker	14(66.7%)	7(33.3%)	21
Non-smoker	22(56.4%)	17(43.6%)	39
Passive Smoker	35(59.5%)	24(40.5%)	59

Among 300 patients, 181 were smoker from which 111 were male and 70 were female. Out of 21 patients, 14 male and 7 female were ex-smoker. Out of 39, 22 male and 17 female were non-smoker. Out of 59, 35 male and 24 female were passive smoker.

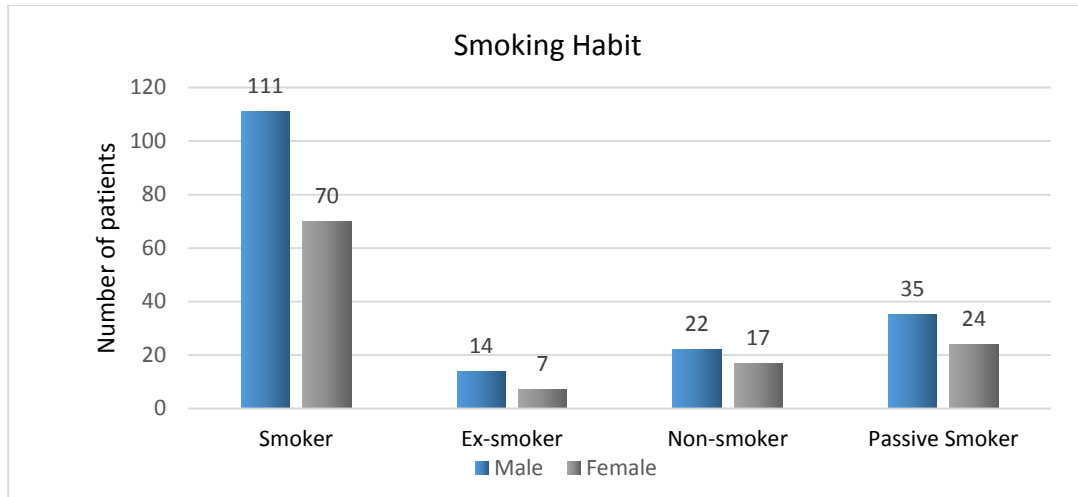


Figure 25: Gender ratio related to smoking habit of lung cancer

Table 32: Cross table analysis between gender and lung cancer related to stages

Stages	Male	Female	Total
IA	50 (61.7%)	31(38.3%)	81
IB	33 (52.4%)	30(47.6%)	63
IIA	36 (52.9%)	32(47.1%)	68
IIB	25 (62.5%)	15(37.5%)	40
IIIA	18 (60%)	12(40%)	30
IIIB	10 (66.2%)	5(33.8%)	15
IV	2 (66.7%)	1(33.3%)	3

Among 300 patients, 81 had IA from which 50 were male and 31 were female. Out of 63 patients, 33 male and 30 female had IB, out of 68, 36 male and 32 female had IIA. Out of 40, 25 male and 15 female had IIB. Out of 30, 18 male and 12 female had IIIA. Out of 15, 10 male and 5 female had IIIB. And out of 3, 2 male and 1 female had IV.

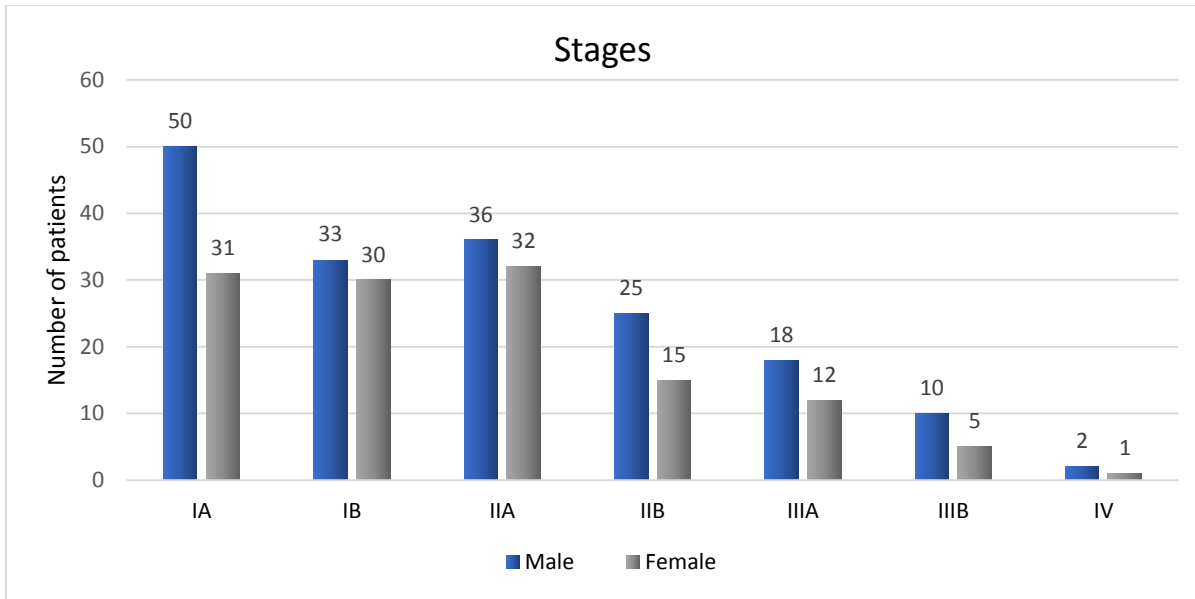


Figure 26: Gender ratio related to stages of lung cancer

It is seen that patients having the propensity for smoking (78%) are the casualties of lung growth than the non-smokers (22%). Tobacco smoke contains more than sixty cancer-causing agents. A channel cigarette can channel some of them, so a non-channel cigarette is more destructive than a sifted one. The frequency of lung growth is exceptionally connected with smoking. (Source: NIH.) Smoking, especially of cigarettes, is by a long shot the fundamental supporter of lung disease. (Biesalski et al., 1998)

Over the created world, just about 90% of lung cancer are caused by smoking, (Peto et al., 2006) In the Unified States, smoking is assessed to represent 87% of lung disease cases where 90% in men and 85% in ladies. (Samet et al., 1988) Among male smokers, the lifetime danger of creating lung cancer is 17.2%; among female smokers, the hazard is 11.6%. This hazard is altogether lower in nonsmokers: 1.3% in men and 1.4% in ladies. (Villeneuve et al., 1994) Tobacco smoke contains more than 60 known cancer-causing agents, including radioisotopes from the radon rot arrangement, nitrosamine, and benzopyrene. Also, nicotine seems to discourage the invulnerable reaction to threatening developments in uncovered tissue.

The time span a man smokes and also rate of smoking builds the individual's possibility of creating lung growth. In the event that a man quits smoking, this possibility relentlessly diminishes as harm to the lungs is repaired and contaminant particles are slowly evacuated. Moreover, there is confirm that lung tumor in never-smokers has a superior forecast than in smokers and that patients who smoke at the season of determination have shorter survival times than the individuals who have stopped. (Tammemagi et al., 2004)

The inward breath of smoke from another's smoking is a reason for lung growth in nonsmokers. An inactive smoker can be delegated somebody living or working with a smoker too. Ongoing examination of side stream smoke recommends that it is more perilous than coordinate smoke inward breath.

CONCLUSION

5. CONCLUSION

At early stage, treatment of cancer is one of the best ways to stop spreading of cancer. As well as by leading a healthy life style, we can minimize our exposure to certain risk factors and might lower our risks to develop cancer. In future there should be research done among the all classes of cancer patients from diverse groups, so that identification of these factors will be more meaningful and give more generalizable conclusions.

In the present circumstance, growth rate is expanding step by step. Distinctive hazard factors such as age (53% above 64 years old), sex (63% male and 37% female) and 45% hereditary so forth add to cancer improvement. Some vitals like 58.7% low blood pressure, 65% high pulse rate, 51% moderate respiratory rate, 50% moderate temperature patients and maximum 49.3% is less than 10 g/dL hemoglobin.

Individuals ought to be more cognizant about their environmental factors like 23% asbestos exposure, 11.7% radon gas, 8.3% chromium, 16% arsenic present in nature and other 5% was the reason for lung cancer like pesticides, harmful chemical and polluted air. Environmental pollution cause many respiratory disease which leads to lung cancer such as 27% pneumonia, 39% tuberculosis, 16% bronchitis, 18% asthma, 7% diffuse pulmonary fibrosis, 60% chronic infection and 33% chronic obstructive pulmonary disease. Besides respiratory disease there are some other diseases which are found for example 38.3% heart problem, 33.3% diabetes and 28.4% hypertension.

The main reason behind lung cancer is smoking which is determined because 60.3% are smokers, 7% ex-smokers, 13% non-smokers and 19.7% passive smokers. Maximum men are addicted to smoking and in our country mostly women are interested in chewing tobacco but now-a-days women are also involved in smoking too. From overall observation it is seen that 44% chew tobacco, 78% cigarette, 25% cannabis and 7% take other drugs including both men and women. The long one consumes tobacco or cigarette the more one has the tendency to have lung cancer. From that it is known that the one who smokes more than 8 years has 40.3% risk for cancer. If anyone wants to be in safe zone then one has to quit smoking for more than 5 years because after that the probability is less to those who quit less than 5 years.

Lung cancer disease shows signs and symptoms like 60% cachexia, 70% anorexia, 49% loss of body weight, 90% coughing, 52% hemoptysis, 18% dyspnea, 17% hoarseness and 15% dysphagia. Lung cancer is identified with symptoms such as 91% chest pain, 66% anemia, 21% bone pain, 7% liver enlargement, 75% cervical lymph nodes, 9% supraclavicular, 10% axillary and 6% other types of lymph nodes enlargement.

Among lung cancer different types is scattered for example 15% small-cell lung carcinoma, 30% had squamous-cell carcinoma, 42% adenocarcinoma, 10% large-cell carcinoma and 3% carcinoid tumor. Through various way lung cancer is diagnosed among them 8% chest X-ray, 25% computed therapy, 41.3% FNAC and 25.7% bronchoscopy. People of different stages got admitted in the hospital, among them 27% IA, 21% IB, 22.7% IIA, 13.3% IB, 10% IIIA, 5% IIIB and 1% IV stage. Most got cured in first and second stages but very less amount of people survived at the very last stages. Lung cancer survival rate is maximum 5 years in life. After proper diagnosis and determining the stages patients need treatment such as 35.7% surgery, 21.3% radiation therapy, 17.7% chemotherapy and 25.3% combined therapy.

Taking everything into account, attention to the inferable division of lung cancer causes among the Bangladeshi growth persistent for tobacco smoking, biting, cleanliness, tumor causing bacterial and viral contamination, age air contamination, water containing arsenic than significant way of life factors, for example, dietary elements, passive smoking is more conspicuous than the other factor like word related introduction and dietary factor in Bangladesh. Our examination recommends the hazard factor of tumor found is like reports from other piece of the nation and the world. We have to be more careful about what we eat and drink, what we work with and for, what we should avoid and accept from nature is totally in our hand. First we have to educate ourselves then we spread awareness around us.

REFERENCES

6. REFERENCES

1. Aberle DR, Abtin F, Brown K (March 2013). "Computed tomography screening for lung cancer: has it finally arrived? Implications of the national lung screening trial". *Journal of Clinical Oncology*. 31 (8): 1002–8.
2. Arriagada R, Goldstraw P, Le Chevalier T (2002). *Oxford Textbook of Oncology* (2nd ed.). Oxford University Press. p. 2094. ISBN 0-19-262926-3.
3. Barbone, F; Bovenzi M, Cavallieri F, Stanta G (December 1997). "Cigarette smoking and histologic type of lung cancer in men". *Chest (American College of Chest Physicians)* 112(6):1474–1479.
4. Biesalski, HK; Bueno de Mesquita B, Chesson A et al. (1998). "European Consensus Statement on Lung Cancer: risk factors and prevention. Lung Cancer Panel". *CA Cancer J Clin* 48 (3): 167–176; discussion 164–166. doi:10.3322/canjclin.48.3.167. PMID 9594919.
5. Brentnall T. Management strategies for patients with hereditary pancreatic cancer. *Curr Treat Options Oncol*. 2005; 6:437–445. doi: 10.1007/s11864-005-0046-6.
6. Catelinois O, Rogel A, Laurier D, et al (September 2006). "Lung cancer attributable to indoor radon exposure in France: impact of the risk models and uncertainty analysis". *Environ. Health Perspect*. 114 (9): 1361–6. doi:0.1289/ehp.9070. PMID 16966089. PMC: 1570096.
7. Chiu, HF; Cheng MH, Tsai SS et al. (December 2006). "Outdoor air pollution and female lung cancer in Taiwan." *Inhalation Toxicology* 18 (13): 1025–1031.
8. Collins, LG; Haines C, Perkel R, Enck RE (January 2007). "Lung cancer: diagnosis and management". *American Family Physician*
9. D'Antonio C, Passaro A, Gori B, Del Signore E, Migliorino MR, Ricciardi S, Fulvi A, de Marinis F (May 2014). "Bone and brain metastasis in lung cancer: recent advances in therapeutic strategies". *Therapeutic Advances in Medical Oncology*. 6 (3): 101–14.
10. Definition of Carcinoma. Retrieved 27 January 2014.
11. Dela Cruz CS, Tanoue LT, Matthay RA (2015). "Chapter 109: Epidemiology of lung cancer". In Grippi MA, Elias JA, Fishman JA, Kotloff RM, Pack AI, Senior RM. *Fishman's Pulmonary Diseases and Disorders* (5th ed.) McGraw-Hill. p. 1673.

12. Detterbeck FC, Postmus PE, Tanoue LT. The stage classification of lung cancer: Diagnosis and management of lung cancer, 3rd ed: American college of chest physicians evidence-based clinical practice guidelines. *Chest*. 2013;143
13. Fairchild A, Harris K, Barnes E, et al. (August 2008). "Palliative thoracic radiotherapy for lung cancer: a systematic review". *Journal of Clinical Oncology*. 26 (24): 4001–4011.
14. Goldstein SD, Yang SC (October 2011). "Role of surgery in small cell lung cancer". *Surgical Oncology Clinics of North America*. 20 (4): 769–77.
15. Goodman MT, Kolonel LN, Wilkens LR, Yoshizawa CN, Le Marchand L. Smoking history and survival among lung cancer patients. *Cancer Causes Control* 1990; 1:155–63.
16. Gorlova, OY; Weng SF, Zhang Y et al. (July 2007). "Aggregation of cancer among relatives of never-smoking lung cancer patients". *International Journal of Cancer* 121 (1): 111–118
17. Health Research Ethics Board of the University of Alberta, 2008
18. Herbst RS, Heymach JV, Lippman SM (September 2008). "Lung cancer". *The New England Journal of Medicine*. 359 (13): 1367–80.
19. Honnorat J, Antoine JC, Orphanet J Rare Dis (2007). Paraneoplastic neurological syndromes.
20. Horn L, Lovly CM, Johnson DH (2015). "Chapter 107: Neoplasms of the lung". In Kasper DL, Hauser SL, Jameson JL, Fauci AS, Longo DL, Loscalzo J. *Harrison's Principles of Internal Medicine* (19th ed.). McGraw-Hill. ISBN 978-0-07-180216-1.
21. International Adjuvant Lung Cancer Trial, 2004
22. International Agency for Research on Cancer, 150 Cours Albert Thomas, 2015
23. Jakopovic M, Thomas A, Balasubramaniam S, Schrupp D, Giaccone G, Bates SE (October 2013). "Targeting the epigenome in lung cancer: expanding approaches to epigenetic therapy". *Frontiers in Oncology*. 3 (261): 261.
24. Jemal A, Siegel R, Ward E, et al. Cancer statistics, 2006. *CA Cancer J Clin* 2006; 56:106130.
25. *Journal of Clinical Oncology*, February 2000
26. Kenfield, S. A.; Wei, E. K.; Stampfer, M. J.; Rosner, B. A.; Colditz, G. A. 2008.01.07
27. Kumar V, Abbas AK, Fausto N, Robbins SL, Cotran RS (2005). *Robbins and Cotran pathologic basis of disease* (7th ed.). Philadelphia: Elsevier Saunders. ISBN 978-7216-0187
28. Manser R, Lethaby A, Irving LB, Stone C, Byrnes G, Abramson MJ, Campbell D (June 2013). "Screening for lung cancer". *The Cochrane Database of Systematic*.

29. Merck Manual Professional Edition, Online edition. "Lung Carcinoma: Tumors of the Lungs". Retrieved on 2007-08-15.
30. Minna, JD (2004). Harrison's Principles of Internal Medicine. McGraw-Hill. pp. 506–516.
31. Morandi, U; Casali C, Rossi G (2006). "Bronchial typical carcinoid tumors". *Seminars in Thoracic and Cardiovascular Surgery* 18 (3): 191–198. doi: 10.1053/j.semtevs.2006.08.005.
32. O'Reilly, KM; Mclaughlin AM, Beckett WS, Sime PJ (March 2007). "Asbestos-related lung disease". *American Family Physician* 75 (5): 683–688. PMID 17375514.
33. Peto, R; Lopez AD, Boreham J et al. (2006). Mortality from smoking in developed countries 1950–2000: Indirect estimates from National Vital Statistics. Oxford University Press. ISBN 0-19-262535-7.
34. Powell CA, Halmos B, Nana-Sinkam SP (July 2013). "Update in lung cancer and mesothelioma 2012". *American Journal of Respiratory and Critical Care Medicine*. 188 (2): 157–66
35. Rami-Porta R, Crowley JJ, Goldstraw P (February 2009). "The revised TNM staging system for lung cancer" (PDF). *Annals of Thoracic and Cardiovascular Surgery*. 15 (1): 4–9. PMID 19262443. Archived (PDF) from the original on 9 May 2012.
36. Raz, DJ; He B, Rosell R, Jablons DM (March 2006). "Bronchioloalveolar carcinoma: a review". *Clinical Lung Cancer (Cancer Information Group)* 7 (5): 313–322.
37. Reveiz L, Rueda JR, Cardona AF (December 2012). "Palliative endobronchial brachytherapy for non-small cell lung cancer". *The Cochrane Database of Systematic Reviews*. 12: CD004284.
38. Riaz SP, Linklater KM, Page R, Peake MD, Møller H, Lüchtenborg M. Recent trends in resection rates among non-small cell lung cancer patients in England. *Thorax*. 2012; 67
39. Rueth NM, Andrade RS (June 2010). "Is VATS lobectomy better: perioperatively, biologically and oncologically?". *The Annals of Thoracic Surgery*. 89 (6): S2107–11.
40. Samet, JM; Wiggins CL, Humble CG, Pathak DR (May 1988). "Cigarette smoking and lung cancer in New Mexico". *American Review of Respiratory Disease* 137
41. Schmid K, Kuwert T, Drexler H, Walk 2010. Mineral waters in rheumatoid arthritis.
42. Subramanian, J; Govindan R (February 2007). "Lung cancer in never smokers: a review". *Journal of Clinical Oncology (American Society of Clinical Oncology)* 25 (5): 561–570.

43. Sun Y, Li Z, Li J, Li Z, Han J (March 2016). "A Healthy Dietary Pattern Reduces Lung Cancer Risk: A Systematic Review and Meta-Analysis". *Nutrients*. 8 (3): 134.
44. Tammemagi CM, Neslund-Dudas C, Simoff M, Kvale P: Smoking and lung cancer survival: the role of comorbidity and treatment. *Chest* 2004, 125 (1):27–37.
45. Thun, MJ; Hannan LM, Adams-Campbell LL et al. (2008). "Lung Cancer Occurrence in Never-Smokers: An Analysis of 13 Cohorts and 22 Cancer Registry Studies". *PLoS Medicine* 5 (9): e185. doi: 10.1371/journal.pmed.005018510.1002/ijc.22615.
46. Tobias J, Hochhauser D (2010). "Chapter 12". *Cancer and its Management* (6th ed.). Wiley-Blackwell. p. 200. ISBN 978-1-4051-7015-4.
47. UN health agency calls for total ban on tobacco advertising to protect young. United Nations News service. 30 May 2008
48. Vaporciyan Ara, Ritsuko Komaki, Jack A Roth, Garrett L Walsh, Jin S Lee Frank V Fossella, Marvin Chasen. Outcome predictors for 143 patients with superior sulcus tumors treated by multidisciplinary approach at the University of Texas, Monaco, April 1998.
49. Villeneuve, PJ; Mao Y (November 1994). "Lifetime probability of developing lung cancer, by smoking status, Canada". *Canadian Journal of Public Health* 85.
50. World Health Organization, 2017
51. World Health Organization. WHO Report on the global tobacco epidemic. 2015.
52. Zlotnik A, Burkhardt AM, Homey B (2011). "Homeostatic chemokine receptors and organ-specific metastasis". *Nature Reviews. Immunology*. 11 (9): 597–606.