Smith Seminars
Online Continuing Education
AARC-Approved for 2 CRCE
Air Pollution Effects on the Lungs

Objectives
Identify how pollution affects health and welfare
Be familiar with tools to help the patient decrease the effects of air pollution
Become aware of the impact of environmental pulmonary diseases due to exposure to air pollution.
Learn the current diagnosis and treatment for inhalation pulmonary diseases due to exposure to air pollution.

Exposure to air pollution is associated with numerous effects on human health, including pulmonary, cardiac, vascular, and neurological impairments. The health effects vary greatly from person to person. High-risk groups such as the elderly, infants, pregnant women, and sufferers from chronic heart and lung diseases are more susceptible to air pollution. Children are at greater risk because they are generally more active outdoors and their lungs are still developing. Exposure to air pollution can cause both acute (short-term) and chronic (long-term) health effects. Acute effects are usually immediate and often reversible when exposure to the pollutant ends. Some acute health effects include eye irritation, headaches, and nausea. Chronic effects are usually not immediate and tend not to be reversible when exposure to the pollutant ends. Some chronic health effects include decreased lung capacity and lung cancer resulting from long-term exposure to toxic air pollutants. The scientific techniques for assessing health impacts of air pollution include air pollutant monitoring, exposure assessment, dosimetry, toxicology, and epidemiology.
Although in humans pollutants can affect the skin, eyes and other body systems, they affect primarily the respiratory system. Air is breathed in through the nose, which acts as the primary filtering system of the body. The small hairs and the warm, humid conditions in the nose effectively remove the larger pollutant particles.
Both gaseous and particulate air pollutants can have negative effects on the lungs. Solid particles can settle on the walls of the trachea, bronchi, and bronchioles. Most of these particles are removed from the lungs through the cleansing (sweeping) action of cilia, small hair-like outgrowths of cells, located on the walls of the lungs. This is what occurs when you cough or sneeze.
A cough or sneeze transports the particles to the mouth. The particles are removed subsequently from the body when they are swallowed or expelled. However, extremely small particles may reach the alveoli, where it takes weeks, months, or even years for the body to remove the particles. Gaseous air pollutants may also affect the function of the lungs by slowing the action of the cilia. Continuous breathing of polluted air can slow the normal cleansing action of the lungs and result in more particles reaching the lower portions of the lung. The lungs are the organs responsible for from the air from the blood-stream. Damage to the lungs from air pollution can inhibit the respiratory process of absorbing oxygen and removing carbon dioxide and contribute to the occurrence of respiratory diseases such as bronchitis,

emphysema, and cancer. This can also put an additional burden on the heart and circulatory system.

Hazardous air pollutants may cause other less common but potentially hazardous health effects, including cancer and damage to the immune system, and neurological, reproductive and developmental problems. Acute exposure to some hazardous air pollutants can cause immediate death.

Human health effects associated with indoor air pollution are: headaches, tiredness, dizziness, nausea, and throat irritation. More serious effects include cancer and exacerbation of chronic respiratory diseases, such as asthma. Radon is estimated to be the second leading cause of lung cancer in the U.S. Environmental tobacco smoke causes eye, nose and throat irritation, and is a carcinogen. Asthma, particularly in children, is associated with poor indoor air quality.

Long before pollutant effects become manifest in human health, they are first seen in the environment. Air pollution impacts in the ecosystem form and function are also a serious concern. Damage to ecosystems from air pollution can exact a significant economic as well as an environmental cost. There are many harmful environmental effects of air pollution: acid rain, the greenhouse effect, depletion of stratospheric ozone, smog, and decreased visibility.

Acid rain is a broad term used to describe several ways that acids fall out of the atmosphere. A more precise term is acid deposition, which has two parts: wet and dry. Acid deposition occurs when emissions of sulfur dioxide and nitrogen oxides in the atmosphere react with water, oxygen, and oxidants to form acidic compounds. These compounds fall to the earth in either dry form (gas and particles) or wet form (rain, snow, and fog). In the United States, about 63% of annual SOx emissions and 22% of NOx emissions are produced by burning fossil fuels for electricity generation. Because it typically takes days to weeks for atmospheric SO2 and NOx to be converted to acids and deposited on the earth's surface, acid deposition occurs in a multistate scale hundreds of miles away from its sources.

Acidity is measured in terms of pH on a logarithmic scale from 1.0 to 14.0. A pH of 1.0 indicates high acidity, whereas a pH of 14.0 indicates high alkalinity; a pH of 7.0 indicates a neutral solution. Precipitation falling through a clean atmosphere is normally somewhat acidic, with a pH of about 5.6. Acid rain, however, can have a pH values below 4.0.

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In the environment, acid deposition causes soil and water bodies to acidify (making the water unsuitable for some fish and other wildlife) and damages some trees, particularly at high elevations. It also speeds the decay of buildings, statues, and sculptures that are part of our national heritage. The nitrogen portion of acid deposition contributes to eutrophication (oxygen depletion) of water bodies, the symptoms of which include algal blooms (some of which may be toxic), fish kills, and loss of plant and animal diversity.

These ecological changes impact human populations by changing the availability of seafood and creating a risk of consuming contaminated fish or shellfish, reducing our ability to use and enjoy our coastal ecosystems, and causing economic impact on people who rely on healthy coastal ecosystems, such as fishermen and those who cater to tourists.

The earth's climate is fueled by the sun. Most of the sun's energy, called solar radiation, is absorbed by the earth, but some is reflected back into space. Clouds and a natural layer of atmospheric gases absorb a portion of earth's heat and prevent it from escaping into space. This keeps our planet warm enough for life and is known as the natural greenhouse effect. Without the natural greenhouse effect, the earth's average temperature would be much colder, and the planet would be uninhabitable. Scientific evidence shows that the greenhouse effect is being increased by the release of certain gases into the atmosphere that cause the earth's temperature to rise. This is called global warming. Carbon dioxide, methane, particulate matter (especially black carbon or soot), nitrous oxide, fluorinated compounds, and ozone are some of the compounds contributing to global warming. Carbon dioxide accounts for about 81% of greenhouse gases released in the United States. Carbon dioxide emissions are largely due to the combustion of fossil fuels in electric power generation, motor vehicles, and industries. Methane emissions, which result from agricultural activities, landfills, and other sources, are the next largest contributors to greenhouse gas emissions in the United States and worldwide.

Continued emissions of greenhouse gases could cause a 2.5° to 10° Fahrenheit rise in temperature by the year 2100. This could lead to more extreme weather events such as droughts and floods, threaten coastal resources and wetlands by raising the sea level, and increase the risk of certain diseases by producing new breeding sites for pests and pathogens. Agricultural regions and woodlands are also susceptible to changes in climate that could result in increased insect populations and plant disease.
This degradation of natural ecosystems could lead to reduced biological diversity.
The stratosphere, located about 6 to 31 miles above the earth, contains a layer of ozone gas that protects living organisms from harmful ultraviolet-B radiation (UV-B) from the Sun. UV-B (280 to 315 nanometer wavelength) has been linked to many harmful effects including various types of skin cancer, cataracts, and harm to some crops, certain materials, and some forms of marine life. In the mid-1970s, it was discovered that some human-produced gases could cause stratospheric ozone depletion. Gases containing chlorine and bromine accumulate in the lower atmosphere, are eventually transported to the stratosphere and then converted to more reactive gases that participate in reactions that destroy ozone. Ozone depletion allows additional UV-B radiation to pass through the atmosphere and reach the earth's surface, leading to increases in UV-related health and environmental effects.
Several substances have been associated with the stratospheric ozone depletion, including chlorofluorocarbons (CFCs), halons, carbon tetrachloride, methyl bromide, and methyl chloroform. One example of ozone depletion is the annual ozone hole over Antarctica that has occurred during the Antarctic spring since the early 1980s. Rather than being a literal hole, the ozone hole is a large area of the stratosphere with extremely low amounts of ozone. Ozone levels fall by over 60% during the worst years. Even over the United States, ozone levels are about 3% below normal in the summer and 5% below normal in the winter.
Smog is a term used in our daily language. It is the mixing of smoke particles from industrial plumes with fog that produces a yellow-black color near ground level. Under the right conditions, the smoke and sulfur dioxide produced from the burning of coal can combine with fog to create industrial smog. The burning of fossil fuels like gasoline can create another atmospheric pollution problem known as photochemical smog.

Photochemical smog is a condition that develops when primary pollutants (oxides of nitrogen and volatile organic compounds created from fossil fuel combustion) interact under the influence of sunlight to produce a mixture of hundreds of different and hazardous chemicals known as secondary pollutants. Smog is the brownish haze that pollutes our air, particularly over cities in the summertime. Smog can make it difficult for some people to breathe and it greatly reduces how far we can see through the air.

Smog is a mixture of pollutants with ground-level ozone being the main culprit.

Increased levels of ground level-ozone are generally harmful to living systems because ozone reacts strongly to destroy or alter many other molecules. Excessive ozone exposure reduces crop yield and forest growth. It interferes with the ability of plants to produce and store food, reducing overall plant health and the ability to grow and reproduce. The weakened plants are more susceptible to harsh weather, disease, and pests. In addition, increases in tropospheric ozone lead to a warming of earth's surface.

Air pollution also has an effect on visibility. Visibility is a measure of aesthetic value and the ability to enjoy scenic vistas, but it also can be an indicator of general air quality. Visibility degradation results when light encounters tiny pollution particles (sulfates, nitrates, organic carbon, soot, and soil dust) and some gases (nitrogen dioxide) in the air. Some light is absorbed by the particles and other light is scattered away before it reaches the observer. More pollutants mean more absorption and scattering of light, resulting in more haze. Haze obscures the clarity, color, texture, and form of what we see. Humidity magnifies the haze problem because some particles, such as sulfates, attract water and grow in size, scattering more light.

In the United States' scenic areas, the visual range has been substantially reduced by air pollution. In eastern parks, average visual range has decreased from 90 miles to 15-25 miles. In the West, average visual range has decreased from 140 miles to 35-90 miles.

Risks

The warnings about risks from hazardous substances are everywhere. Many products now flaunt warning labels or claims about being "all natural" and "chemical free."

Ideally, regulators would like to eliminate all pollution and its risks, but this is usually not a realistic expectation. Regulators must address the most important risks and decrease them to the level at which they believe the risks are smaller than the benefits of the activity causing the pollution.

This is similar to what millions do each day when they balance the risks of an automobile accident with the convenience and necessity of driving. Just as a driver will buckle up and drive defensively to be safer, agencies take regulatory action to eliminate as much risk as is possible without losing the benefit.

Scientific results may show that certain hazardous substances pose a low health risk to people, but the public may still be concerned about these hazardous substances because of different attributes of the risk.

Air Pollution and Respiratory Health

The Air Pollution and Respiratory Health Branch (APRHB) leads CDC’s fight against environmental-related respiratory illnesses, including asthma, and studies indoor and outdoor air pollution.

Smoke from wildfires or burning debris after natural disasters is a mixture of gases and fine particles that can cause breathing difficulties or coughing and can harm your eyes.

Natural disasters, such as hurricanes and floods, can leave a lot of debris. Some of this debris may be burned during cleanup. Smoke from these outdoor fires is unhealthy to breathe, causing coughing and shortness of breath or tightness in the chest. It also can irritate the eyes, nose, or throat.

These problems can begin a very short time after the smoke is breathed in. The patient may have little warning, especially if they have lung or heart disease. Infants, children, pregnant women, older adults, and people with chronic diseases such as asthma are at greater risk from smoke.

Tell your patient that if they smell or see smoke, or know that fires are nearby, they can take the following steps to protect themselves and their family:

- Leave the area if you are at greater risk from breathing smoke.
- Limit exposure to smoke outdoors and indoors.
- Stay inside and use an air conditioner. If they do not have an air conditioner or smoke is likely to get inside the house, leave the area until the smoke is completely gone.
- Avoid activities that put extra demands on the lungs and heart. These include exercising or physical chores, both outdoors and indoors.
- Make sure they take all their medications according to the doctor's directions.
- They need to contact their doctor if their health gets worse.
- Dust masks, bandanas, or other cloths (even if wet) will not protect them from smoke.

Asthma is a serious environmental health threat, but it can be controlled by taking medication and by avoiding contact with environmental triggers such as dust mites, furry pets, mold, tobacco smoke, and certain chemicals.

Dust mites are in almost everybody's homes, but they don't cause everybody to have asthma attacks. To help prevent asthma attacks, they may use mattress covers and pillowcase covers to make a barrier between dust mites and the patient. They should not use down-filled pillows, quilts, or comforters. They should remove stuffed animals and clutter from your bedroom.

Furry pets may trigger an asthma attack. When a furry pet is suspected of causing asthma attacks, the simplest solution is to find the pet another home. If pet owners are too attached to their pets or are unable to locate a safe, new home for the pet, they should keep the pet out of the bedroom of the person with asthma.

Pets should be bathed weekly and kept outside as much as possible. People with asthma are not allergic to their pet's fur, so trimming your pet's fur will not help your asthma. They should vacuum often to clean up anything that could cause an asthma attack. If the floors have a hard surface, such as wood or tile, and are not carpeted, they can be damp mopped every week.

Mold grows anywhere there is moisture but can be prevented. Exposure to damp and moldy environments may cause nasal stuffiness, throat irritation, coughing or wheezing, eye irritation, or skin irritation.

Inhaling or breathing in mold can cause an asthma attack. They should get rid of mold in all parts of their home to help control the asthma attacks. Keep the humidity level in the home between 35% and 50%. In hot, humid climates, they may need to use an air conditioner or a dehumidifier or both. Water leaks need to be fixed, which allow mold to grow behind walls and under floors.

Environmental tobacco smoke is often called secondhand smoke because the smoke created by a smoker is breathed in by a second person nearby. Parents, friends, and relatives of children with asthma should try to stop smoking and should never smoke around a person with asthma. They should only smoke outdoors and not in the family home or car. They should not allow others to smoke in the home, and they should make sure their child's school is smoke-free.
Carbon monoxide (CO), an odorless, colorless gas that can cause sudden illness and death, is found in combustion fumes produced by cars and trucks, generators, stoves, lanterns, burning charcoal and wood, gas ranges, and heating systems.

Tell the patient they can prevent carbon monoxide exposure:

- Have the heating system, water heater and any other gas, oil, or coal burning appliances serviced by a qualified technician every year.
- Install a battery-operated CO detector in the home and check or replace the battery when they change the time on the clocks each spring and fall. If the detector sounds leave your home immediately and call 911.
- Seek prompt medical attention if they suspect CO poisoning and are feeling dizzy, light-headed, or nauseous.
- Don't use a generator, charcoal grill, camp stove, or other gasoline or charcoal-burning device inside the home, basement, or garage or near a window.
- Don't run a car or truck inside a garage attached to the house, even if they leave the door open.
- Don't burn anything in a stove or fireplace that isn't vented.
- Don't heat the house with a gas oven.

Air pollution can affect our health in many ways with both short-term and long-term effects. Different groups of individuals are affected by air pollution in different ways. Some individuals are much more sensitive to pollutants than are others. Young children and elderly people often suffer more from the effects of air pollution. People with health problems such as asthma, heart and lung disease may also suffer more when the air is polluted. The extent to which an individual is harmed by air pollution usually depends on the total exposure to the damaging chemicals, such as the duration of exposure and the concentration of the chemicals must be taken into account.
Examples of short-term effects include irritation to the eyes, nose and throat, and upper respiratory infections such as bronchitis and pneumonia. Other symptoms can include headaches, nausea, and allergic reactions. Short-term air pollution can aggravate the medical conditions of individuals with asthma and emphysema.
Long-term health effects can include chronic respiratory disease, lung cancer, heart disease, and even damage to the brain, nerves, liver, or kidneys. Continual exposure to air pollution affects the lungs of growing children and may aggravate or complicate medical conditions in the elderly. It is estimated that half a million people die prematurely every year in the United States as a result of smoking cigarettes.

Research into the health effects of air pollution is ongoing. Medical conditions arising from air pollution can be very expensive. Healthcare costs, lost productivity in the workplace, and human welfare impacts cost billions of dollars each year.

Environmental Pulmonary Disease

Environmental pulmonary diseases result from inhalation of dusts, allergens, chemicals, gases, and environmental pollutants. The lungs are continually exposed to the external environment and are susceptible to a host of environmental diseases. Pathologic processes can involve any part of the lungs, including the airways (occupational asthma, reactive airways dysfunction syndrome, or toxic inhalations), interstitium (pneumoconioses or hypersensitivity pneumonitis), and pleura (asbestos-related diseases).
Prevention of occupational and environmental pulmonary diseases centers on reducing exposure (primary prevention).

Exposure can be limited by the use of:

- Administrative controls by limiting the number of people exposed to hazardous conditions
- Engineering controls by using enclosures, ventilation systems, and safe clean-up procedures
- Product substitution by using safer, less toxic materials
- Respiratory protection devices such as respirator, dust mask, and gas mask

Many erroneously assume that a patient who has used a respirator or another respiratory protection device has been well protected. Although respirators do offer a degree of protection, especially when fresh air is provided by tank or air hose, the benefit is limited and idiosyncratic. When recommending use of a respirator, clinicians should consider several factors. Workers with cardiovascular disease may be unable to carry out jobs that require strenuous work, especially if they must wear a self-contained breathing apparatus (tank). Respirators that are tight-fitting and that require the wearer to draw air through filter cartridges can increase the work of breathing, which can be especially difficult for patients with asthma, COPD, or interstitial lung diseases.
Medical surveillance is a form of secondary prevention. Workers can be offered medical tests that identify disorders early when treatment might help reduce long-term consequences.

Air Pollution–Related Illness

Asbestosis

Alveolar macrophages attempting to engulf inhaled fibers release cytokines and growth factors that stimulate inflammation, oxidative injury, collagen deposition, and ultimately fibrosis. Asbestos fibers may also be directly toxic to lung tissue. Risk of disease is generally related to duration and intensity of exposure and type, length, and thickness of inhaled fibers.

Symptoms and Signs

Asbestosis is initially asymptomatic but can cause progressive dyspnea, nonproductive cough, and fatigue. The disorder progresses in > 10% of patients even after cessation of exposure.

Advanced asbestosis may cause clubbing, dry bibasilar crackles, and, in severe cases, symptoms and signs of right ventricular failure (cor pulmonale).

Diagnosis

Diagnosis is based on history of exposure and chest x-ray or chest CT. Chest x-ray shows linear reticular opacities signifying fibrosis, usually in the peripheral lower lobes. Opacities are often bilateral and are often accompanied by pleural changes.

Honeycombing signifies more advanced disease, which may involve the mid and lower lung fields. As with silicosis, severity is graded on the International Labor Organization scale (International Classification of Radiographs of Pneumoconioses) based on size, shape, location, and profusion of opacities. In contrast to silicosis, asbestosis produces reticular opacities with a lower lobe predominance. Hilar and mediastinal adenopathy and nodular opacities are uncharacteristic and suggest a different diagnosis. Chest x-ray is insensitive; thin-section chest CT is useful when asbestosis is a likely diagnosis. CT is also superior to chest x-ray in identifying pleural abnormalities.

Pulmonary function tests, which may show reduced lung volumes and diffusing capacity for carbon monoxide (DLco), are nonspecific but help characterize changes in lung function over time. Pulse oximetry measured at rest and with exertion is nonspecific but sensitive for detecting asbestos-induced impairment.

Bronchoalveolar lavage or lung biopsy is indicated only when noninvasive measures fail to provide conclusive diagnosis; demonstration of asbestos fibers indicates asbestosis in people with pulmonary fibrosis, although such fibers can occasionally be found in lungs of exposed people without disease and may not be present in specimens from patients with asbestosis. Thus, demonstration of asbestos fibers may be helpful but is not necessary for diagnosis.

Prognosis

Prognosis varies; many patients have no or mild symptoms and do well, whereas some develop progressive dyspnea and a few develop respiratory failure, right ventricular failure, and cancer. Lung cancer (usually non–small cell lung carcinoma) develops in patients with asbestosis at 8 to 10 times the rate of those without asbestosis and is especially common among workers exposed to amphibole fibers, although all forms of inhaled asbestos have been associated with an elevated cancer risk. Asbestos and smoking have a synergistic effect on lung cancer risk.

Treatment

No specific treatment exists. Early detection of hypoxemia and right ventricular failure leads to use of supplemental O_2 and treatment of heart failure. Pulmonary rehabilitation can be helpful for patients with impairment.

Prevention

Preventive measures include eliminating exposure, asbestos abatement in occupational and nonoccupational settings, smoking cessation, and pneumococcal and influenza vaccination. Smoking cessation is particularly important in light of the multiplicative risk of lung cancer in patients who have both tobacco smoke and asbestos exposures.

Mesothelioma

Pleural mesothelioma is the only known pleural cancer and is caused by asbestos exposure in nearly all cases.
Asbestos workers have up to a 10% lifetime risk of developing the disorder, with an average latency of 30 yr. Risk is independent of smoking. Mesothelioma can spread locally, or it can metastasize to the hilar and mediastinal lymph nodes, pericardium, diaphragm, peritoneum, liver, adrenals, or kidneys and, rarely, the tunica vaginalis of the testis.

Symptoms and Signs

Patients most often present with dyspnea and nonpleuritic chest pain. Constitutional symptoms are uncommon at presentation. Invasion of the chest wall and other adjacent structures may cause severe pain, hoarseness, dysphagia, Horner's syndrome, brachial plexopathy, or ascites.

Diagnosis

Chest x-ray
Pleural fluid cytology or pleural biopsy
Sometimes video-assisted thorascopic surgery (VATS) or thoracotomy
Staging with chest CT, mediastinoscopy, and MRI or sometimes with PET and bronchoscopy

The pleural form of mesothelioma, which represents > 90% of all cases (the other 10% include pericardial and peritoneal mesotheliomas), appears on x-ray as diffuse unilateral or bilateral pleural thickening that appears to encase the lungs, usually producing blunting of the costophrenic angles. Pleural effusions are present in 95% of cases and are typically unilateral, large, and hemorrhagic. Diagnosis is based on pleural fluid cytology or pleural biopsy. If diagnosis is uncertain after these procedures, biopsy by VATS or thoracotomy is done.
Staging is done with chest CT, mediastinoscopy, and MRI. Sensitivity and specificity of MRI and CT are comparable, although MRI is helpful in determining tumor extension into the spine or spinal cord. PET may have better sensitivity and specificity for distinguishing benign from malignant pleural thickening. Bronchoscopy should be done to exclude coexisting endobronchial lung cancers. Increased levels of hyaluronidase in pleural fluid are suggestive but not diagnostic of mesothelioma. Soluble mesothelin-related proteins released into the serum by mesothelial cells are being studied as possible tumor markers for disease detection and monitoring, but the false-positive rate may limit their effectiveness.

Prognosis

Mesothelioma remains an incurable cancer, and long-term survival is uncommon. Surgery to remove the pleura, ipsilateral lung, phrenic nerve, hemidiaphragm, and pericardium combined with chemotherapy or radiation therapy may be considered, although it does not substantially change prognosis or survival time. No treatment substantially prolongs survival. Survival from time of diagnosis averages 8 to 15 months, depending on the location and cell type. A few patients, usually younger patients with shorter duration of symptoms, have a more favorable prognosis, sometimes surviving for several years after diagnosis.

Treatment

Supportive care
Pleurodesis or pleurectomy for pleural effusions and relief of dyspnea
Analgesia with opioids and sometimes radiation therapy
Chemotherapy for tumor shrinkage and symptom relief
Experimental therapies

Complete surgical resection usually is not feasible. Combination pemetrexed (an antifolate antimetabolite) and cisplatin shows promise but warrants further study.
The major focus of treatment is supportive care and relief of pain and dyspnea. Given the diffuse nature of the disorder, radiation therapy is usually unsuitable except to treat localized pain or needle-tract metastases. It is not generally used for treatment of nerve root pain. Pleurodesis or pleurectomy can be used to help reduce dyspnea caused by pleural effusions. Adequate analgesia is important but difficult to achieve. Usually, opioids, both transdermal and delivered via indwelling epidural catheters, are used. Chemotherapy using cisplatin and gemcitabine relieves symptoms in most cases and sometimes decreases tumor size.

Additional Asbestos-Related Pleural Disease
Pleural disease, a hallmark of asbestos exposure, includes formation of pleural plaques, calcification, thickening, rounded atelectasis, adhesions, effusion, and mesothelioma.
Pleural disease causes effusion but few symptoms. All pleural changes are diagnosed by chest x-ray or CT, though chest CT is more sensitive than chest x-ray for detecting pleural disorders. Treatment is rarely needed except for cancer.

Discrete Plaques
Discrete plaques, which occur in up to 60% of workers exposed to asbestos, typically affect the bilateral parietal pleurae between the 5th and 9th ribs and adjacent to the diaphragm. Plaque calcification is common and can lead to misdiagnosis of severe pulmonary disease when radiographically superimposed on lung fields. CT can distinguish pleural from parenchymal disease in this setting. Fat stripes may be mistaken for pleural plaques on chest x-ray. CT can distinguish pleural disease from fat.

Diffuse Thickening
Diffuse thickening affects visceral as well as parietal pleurae. It may be an extension of pulmonary fibrosis from parenchyma to the pleurae or a nonspecific reaction to pleural effusion. With or without calcification, pleural thickening can cause a restrictive defect.

Rounded Atelectasis
Rounded atelectasis is a benign manifestation of pleural thickening in which invagination of pleura into the parenchyma can entrap lung tissue, causing atelectasis. On chest x-ray and CT, it typically appears as a curvilinear cicatricial mass, often in the lower lung zones, and can be confused with a pulmonary cancer.

Pleural Effusions

Pleural effusions occur but are less common than the other pleural changes they accompany. These benign effusions are usually bilateral, exudative, and often hemorrhagic. They typically resolve spontaneously.

Beryllium Disease (Berylliosis)

Acute beryllium disease and chronic beryllium disease are caused by inhalation of dust or fumes from beryllium compounds and products. Acute beryllium disease is now rare; chronic beryllium disease is characterized by formation of granulomas throughout the body, especially in the lungs, intrathoracic lymph nodes, and skin. Chronic beryllium disease causes progressive dyspnea, cough, and fatigue. Diagnosis is by history, beryllium lymphocyte proliferation test, and biopsy. Treatment is with corticosteroids.

Etiology

Beryllium exposure is a common but underrecognized cause of illness in many industries, including beryllium mining and extraction, alloy production, metal alloy machining, electronics, telecommunications, nuclear weapon manufacture, defense, aircraft, automotive, aerospace, and metal scrap, computer, and electronics recycling. Because small amounts of beryllium are toxic and are added to many copper, aluminum, nickel, and magnesium alloys, workers are often unaware of their exposure and its risks.

Pathophysiology

Acute beryllium disease is a chemical pneumonitis causing diffuse parenchymal inflammatory infiltrates and nonspecific intra-alveolar edema. Other tissues (skin and conjunctivae) may be affected. Acute beryllium disease is now rare because most industries have reduced exposure levels, but cases were common between 1940 and 1970, and many cases progressed from acute to chronic beryllium disease.

Chronic beryllium disease remains a common illness in industries that use beryllium and beryllium alloy. It differs from most pneumoconioses in that it is a cell-mediated hypersensitivity disease. Beryllium is presented to CD4+ T lymphocytes by antigen-presenting cells, principally in HLA-DP molecules. T lymphocytes in the blood, lungs, or other organs, in turn, recognize the beryllium, proliferate, and form T-lymphocyte clones. These clones then release proinflammatory cytokines, such as tumor necrosis factor-α, IL-2, and interferon- γ. These cytokines amplify the immune response, resulting in formation of mononuclear cell infiltrates and noncaseating granulomas in target organs where beryllium has deposited. On average, about 2% to 6% of beryllium-exposed people develop beryllium sensitization (defined by positive blood lymphocyte proliferation to beryllium salts in vitro), with most progressing to disease. In certain high-risk groups, such as beryllium metal and alloy machinists, chronic beryllium disease prevalence is > 17%. Workers with bystander exposures, such as secretaries and security guards, also develop sensitization and disease but at lower rates. The typical pathologic consequence is a diffuse pulmonary, hilar, and mediastinal lymph node granulomatous reaction that is histologically indistinguishable from sarcoidosis. Early granuloma formation with

mononuclear and giant cells can also occur. Many lymphocytes are found when cells are washed from the lungs (bronchoalveolar lavage) during bronchoscopy. These T lymphocytes proliferate when exposed to beryllium in vitro, much as the blood cells do.

Symptoms and Signs

Patients with chronic beryllium disease often have dyspnea, cough, weight loss, and a variable chest x-ray pattern, typically showing nodular opacities in the mid and upper lung zones, frequently with hilar and mediastinal adenopathy. Patients complain of insidious and progressive exertional dyspnea, cough, chest pain, weight loss, night sweats, and fatigue. Symptoms may develop within months of first exposure or > 40 years after exposure has ceased. Some people remain asymptomatic.

Diagnosis

Diagnosis depends on a history of exposure, the appropriate clinical manifestations, and an abnormal blood or bronchoalveolar lavage (BAL)/beryllium lymphocyte proliferation test (BeLPT) or both. BAL/BeLPT is highly sensitive and specific, helping to distinguish chronic beryllium disease from sarcoidosis and other forms of diffuse pulmonary disease. Chest x-ray may be normal or show diffuse infiltrates that can be nodular, reticular, or have a hazy ground-glass appearance, often with hilar adenopathy resembling the pattern seen in sarcoidosis. A miliary (resembling seeds) pattern also occurs. High-resolution CT is more sensitive than x-ray, although cases of biopsy-proven disease occur even in people with normal imaging tests.

Prognosis

Acute beryllium disease can be fatal, but prognosis is usually excellent unless progression to chronic beryllium disease occurs. Chronic beryllium disease often results in progressive loss of respiratory function. Early abnormalities include air flow obstruction and decreased oxygenation on ABG at rest and during exercise testing. Decreased diffusing capacity for carbon monoxide (DLco) and restriction appear later. Pulmonary hypertension and right ventricular failure develop in about 10% of cases, with death due to cor pulmonale. Beryllium sensitization progresses to chronic beryllium disease at a rate of about 6%/year after initial detection through workplace medical surveillance programs. Subcutaneous granulomatous nodules caused by inoculation with beryllium splinters or dust usually persist until excised.

Treatment

Corticosteroids

In acute beryllium disease, sometimes mechanical ventilation

In chronic beryllium disease, sometimes supplemental O_2, pulmonary rehabilitation, and treatment for right ventricular failure

In end-stage chronic beryllium disease, sometimes lung transplantation

In acute disease, the lungs often become edematous and hemorrhagic. Mechanical ventilation is necessary in severely affected patients.

Some patients with chronic beryllium disease never require treatment because the disease progresses relatively slowly. When needed, treatment is with corticosteroids, which decrease

symptoms and improve oxygenation. Treatment is generally started only in patients with significant symptoms and evidence of abnormal gas exchange or evidence of an accelerated decline in lung function or oxygenation. In symptomatic patients with abnormal pulmonary function, prednisone 40 to 60 mg PO once/day or every other day is given for 3 to 6 months. Then, measures of pulmonary physiology and gas exchange are repeated to document a response to therapy, and the dose is gradually tapered to the lowest dose that maintains symptomatic and objective improvement (usually about 10 to 15 mg once/day or every other day). Lifelong treatment with corticosteroids is usually required. There is anecdotal evidence that the addition of methotrexate (10 to 25 mg PO once/week) reduces the need for corticosteroids as it does in sarcoidosis.

Spontaneous remission of chronic beryllium disease is rare. In patients with end-stage disease, lung transplantation can be lifesaving. Other supportive measures, such as supplemental O_2 therapy, pulmonary rehabilitation, and drugs for treatment of right ventricular failure, are used as needed.

Prevention

Industrial dust suppression is the basis for preventing beryllium exposure. Exposures must be reduced to levels that are as low as reasonably achievable to reduce the risk of sensitization and chronic beryllium disease. More than 50-fold below current Occupational Safety and Health Administration (OSHA) standards is preferred. Medical surveillance, using blood BeLPT and chest x-ray, is recommended for all exposed workers, including those with indirect contact. Both acute and chronic disease must be promptly recognized and affected workers removed from further beryllium exposure.

Building-Related Illnesses

Building-related illnesses (BRIs) are a heterogeneous group of disorders whose etiology is linked to the environment of modern airtight buildings. Such buildings are characterized by sealed windows and dependence on heating, ventilation, and air conditioning systems for circulation of air. Most cases occur in nonindustrial office buildings, but cases can occur in apartment buildings, single-family homes, schools, museums, and libraries. BRIs can be specific or nonspecific.

Specific BRIs are those for which a link between building-related exposure and illness is proved. Examples include legionella infection, occupational asthma, hypersensitivity pneumonitis, and inhalational fever.

Inhalational Fever

Inhalational fever is a febrile reaction caused by exposure to organic aerosols or dusts. Names used to describe this type of BRI include humidifier fever, grain fever, swine confinement fever, and mycotoxicosis, depending on the causative agent. Metal fumes and polymer fumes can also cause febrile illness. The term organic dust toxic syndrome (ODTS) has been used to encompass the subacute febrile and respiratory reaction to organic dust that is typically highly contaminated with bacterial endotoxin. Toxic pneumonitis is a commonly used but less specific term.

Humidifier Fever

Humidifier fever occurs in nonindustrial buildings as a consequence of humidifiers or other types of ventilation units serving as a reservoir for the growth of bacteria or fungi and as a method of aerosolizing these contaminants. The disorder usually manifests as low-grade fever, malaise, cough, and dyspnea. Improvement after removal from exposure, such as a weekend away from the office building, is often one of the first indications of etiology. Humidifier fever has an acute onset and is self-limiting, usually 2 to 3 days. Physical signs may be absent or subtle. Clusters of cases are common.

Unlike immunologically mediated conditions (such as hypersensitivity pneumonitis or building-related asthma) inhalational fevers do not require a period of sensitization. The disorder can occur after initial exposure. Acute episodes do not generally require treatment apart from antipyretics and removal from the contaminated environment. If symptoms persist, evaluation may be required to rule out infection, hypersensitivity pneumonitis, or other conditions. Biologic sampling to detect airborne microbials in the work environment can be costly and time consuming but is sometimes necessary to document the source of contaminated air.

Inhalational fevers of all types are usually prevented by good maintenance of ventilation systems.

Nonspecific BRIs are those for which a link between building-related exposure and illness is more difficult to prove. The term sick building syndrome has been used to refer to illnesses that occur in clusters within a building and that cause often nonspecific symptoms.

Nonspecific BRIs symptoms include:

- Itchy, irritated, dry or watery eyes
- Rhinorrhea or nasal congestion
- Throat soreness or tightness
- Dry itchy skin or unexplained rashes
- Headache, lethargy, or difficulty concentrating

Some building-related factors appear to account for symptoms in some instances. These factors include higher building temperature, higher humidity, and poor ventilation, typically with a failure to incorporate sufficient fresh air from outdoors. Patient factors, including female sex, history of atopy, increased attention to body sensations, worry about the meaning of symptoms, anxiety, depression, and occasionally mass hysteria, also seem to underlie experience of symptoms.

Byssinosis

Byssinosis is a form of reactive airways disease characterized by bronchoconstriction in cotton, flax, and hemp workers. The etiologic agent is unknown. Symptoms are chest tightness and dyspnea that worsen on the first day of the work week and subside as the week progresses. Diagnosis is based on history and pulmonary function test findings. Treatment includes avoidance of exposure and use of asthma drugs.

Etiology

Byssinosis occurs almost entirely in workers who contact unprocessed, raw cotton, especially those who are exposed to open bales or who work in cotton spinning or in the card room. Byssinosis can occur after acute exposure but usually occurs in workers with a history of chronic exposure. Evidence suggests that some agent in the cotton bract leads to bronchoconstriction. Although bacterial endotoxin is a likely cause, the absence of similar symptoms in other settings in which workers are exposed to endotoxin leaves some uncertainty. Prolonged exposure to cotton dust was once thought to cause emphysema, a theory now disproved. Chronic bronchitis symptoms are common among people exposed to cotton dust.

Symptoms and Signs

Symptoms are chest tightness and dyspnea that lessen with repeated exposure. Symptoms develop on the first day of work after a weekend or vacation and diminish or disappear by the end of the week. With repeated exposure over a period of years, chest tightness tends to return and persist through midweek and occasionally to the end of the week or as long as the person continues to work. This typical temporal pattern distinguishes byssinosis from asthma.
Signs of acute exposure are tachypnea and wheezing. Patients with more chronic exposure may have crackles.

Diagnosis

Diagnosis is based on history and pulmonary function tests that show typical airflow obstruction and a reduction in ventilatory capacity, especially if measured at the start and end of a first work shift. Hyperresponsiveness to methacholine is also often observed. Surveillance measures, including symptom reporting and spirometry in textile workers, can aid in early detection.

Treatment

Treatment includes avoidance or reduction of exposure and use of asthma drugs.

Irritant Gas Inhalation Injury

Irritant gases are those which, when inhaled, dissolve in the water of the respiratory tract mucosa and cause an inflammatory response, usually from the release of acidic or alkaline radicals. Irritant gas exposures predominantly affect the airways, causing tracheitis, bronchitis, and bronchiolitis. Other inhaled agents may be directly toxic (cyanide, carbon monoxide) or cause harm simply by displacing O_2 and producing asphyxia (methane, carbon dioxide).
The effect of inhaling irritant gases depends on the extent and duration of exposure and on the specific agent. Chlorine, phosgene, sulfur dioxide, hydrogen chloride or sulfide, nitrogen dioxide, ozone, and ammonia are among the most important irritant gases. Hydrogen sulfide is also a potent cellular toxin, blocking the cytochrome system and inhibiting cellular respiration. A common exposure involves mixing household ammonia with cleansers containing bleach; the irritant gas chloramine is released.

Acute Exposure

Acute exposure to high concentrations of toxic gas over a short time is characteristic of industrial accidents resulting from a faulty valve or pump in a gas tank or occurring during gas

transport. Many people may be exposed and affected. Respiratory damage is related to the concentration of the gas and its solubility.

More water-soluble gases (chlorine, ammonia, sulfur dioxide, hydrogen chloride) dissolve in the upper airway and immediately cause mucous membrane irritation, which may alert people to the need to escape the exposure. Permanent damage to the upper respiratory tract, distal airways, and lung parenchyma occurs only if escape from the gas source is impeded.

Less soluble gases (nitrogen dioxide, phosgene, ozone) may not dissolve until they are well into the respiratory tract, often reaching the lower airways. These agents are less likely to produce early warning signs (phosgene in low concentrations has a pleasant odor), are more likely to cause severe bronchiolitis, and often have a lag of ≥ 12 hours before symptoms of pulmonary edema develop.

The most serious immediate complication is ARDS, which usually occurs within 24 hours. Patients with significant lower airway involvement may develop bacterial infection.

Ten to 14 days after acute exposure to some agents (ammonia, nitrogen oxides, sulfur dioxide, mercury), some patients develop bronchiolitis obliterans progressing to ARDS. Bronchiolitis obliterans with organized pneumonia can ensue when granulation tissue accumulates in the terminal airways and alveolar ducts during the body's reparative process. A minority of these patients develop late pulmonary fibrosis.

Symptoms and Signs

Soluble irritant gases cause severe burning and other manifestations of irritation of the eyes, nose, throat, trachea, and major bronchi. Marked cough, hemoptysis, wheezing, retching, and dyspnea are common. The upper airway may be obstructed by edema, secretions, or laryngospasm. Severity is generally dose-related. Nonsoluble gases cause fewer immediate symptoms but can cause dyspnea or cough.

Patients who develop ARDS have worsening dyspnea and increasing O_2 requirements.

Diagnosis

Diagnosis is usually obvious from the history. Patients should have a chest x-ray and pulse oximetry. Chest x-ray findings of patchy or confluent alveolar consolidation usually indicate pulmonary edema.

CT is used to evaluate patients with late-developing symptoms. Those with bronchiolitis obliterans that progresses to respiratory failure manifest a pattern of bronchiolar thickening and a patchy mosaic of hyperinflation.

Prognosis

Most people recover fully, but some have persistent lung injury with reversible airway obstruction (reactive airways dysfunction syndrome) or pulmonary fibrosis; smokers may be at greater risk.

Treatment

- Removal from exposure and 24-h observation
- Bronchodilators and supplemental O_2
- Sometimes inhaled racemic epinephrine, endotracheal intubation, and mechanical ventilation

Management does not differ by specific inhaled agent but rather by symptoms. Patients should be moved into fresh air and given supplemental O_2. Treatment is directed toward ensuring adequate oxygenation and alveolar ventilation. Bronchodilators and O_2 therapy may suffice in less severe cases. Severe airflow obstruction is managed with inhaled racemic epinephrine, endotracheal intubation or tracheostomy, and mechanical ventilation. The efficacy of corticosteroid therapy (prednisone 45 to 60 mg once/day for 1 to 2 weeks) is unproved, but it is frequently used.

Because of the risk of ARDS, any patient with respiratory tract symptoms after toxic inhalation should be observed for 24 hours.

After the acute phase has been managed, physicians must remain alert to the development of reactive airways dysfunction syndrome, bronchiolitis obliterans with or without organized pneumonia, pulmonary fibrosis, and delayed-onset ARDS.

Prevention

Care in handling gases and chemicals is the most important preventive measure. The availability of adequate respiratory protection (gas masks with a self-contained air supply) for rescuers is also very important. Rescuers without protective gear who rush in to extricate a victim often succumb themselves.

Chronic Exposure

Low-level continuous or intermittent exposure to irritant gases or chemical vapors may lead to chronic bronchitis, although the role of such exposure is especially difficult to substantiate in smokers.

Chronic inhalational exposure to some agents, such as bischloromethyl ether or certain metals, causes lung and other cancers.

In conclusion, exposure to air pollution can cause both acute (short-term) and chronic (long-term) health effects that are associated with many diseases. Gaseous and particulate air pollutants can have negative effects on the lungs and need to be avoided whenever possible.

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