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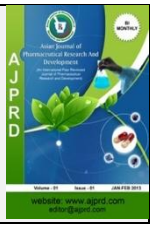

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Review Article

Farmer's Lungs Disease: It's Take A Breath Away!

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A B S T R A C T

Farmers were evaluated for the presence of farmer's lung disease by serologic methods and by clinical histories. Farmer's lung disease (FLD) is a form of hypersensitivity pneumonitis (HP) caused by inhaling microorganisms from hay or grain stored in conditions of high humidity in the agricultural workplace. The epidemiology of the disease is not well known, and is based on studies conducted by Central European and Asian groups. The clinical presentation may vary, differentiating the chronic (exposure to lower concentrations of the antigen over a longer period time) and the acute forms (after exposure to high concentrations of the antigen). It is more common in middle-aged men, although this probably reflects differences in exposure levels. It is also more common in non-smokers, probably because tobacco reduces the IgG response to inhaled antigens, affects cytokine production, and alters macrophage function. The etiology of the disease is clear - the inhalation of mouldy hay dust - and much can be done to prevent it ifthis is borne in mind. Mouldy hay dust is a very complex material consisting of innumerable fungal spores, hyphae and bacteria and fragments of vegetable matter. The treatment of FLD is based mainly on avoiding exposure to the antigen. This is the only measure that has been shown to delay disease progression. Corticosteroids are traditionally recommended in patients with impaired lung function and beta agonist and alpha blockers are also helpful in the treatment of disease.

Keyword: Farmer's lungsdisease(FLD), Hypersensitivity Pneumonitis(HP), Allergy, Dust.

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INTRODUCTION

Farmer's lung disease(FLD) is a form of hypersensitivity pneumonitis(HP). According to previous studies, the most common antigens are usually thermophilic actinomycetes and fungi. In patients with respiratory symptoms and agricultural occupational exposure, radiological, lung function and/or anatomical pathology findings must be compatible with FLD, bronchoalveolar lavage must Show lymphocytosis, and tests must find sensitivity to the antigen. The main treatment is avoidance of the antigen, so it is essential to educate patients on preventive measures. To date, no controlled studies have assessed the role of immunosuppressive therapy in this disease. Corticosteroid treatment has only been shown to accelerate resolution of the acute forms, but there is no evidence that it is effective in preventing disease progression in the long-term or reducing mortality.[1]

History

In 1713 Ramazzini' observed that grain handlers and farmers of the Po valley were troubled by lung diseases. The first modern mention of lung disease in farmers is attributed to Cadham1 in Winnipeg in 1924; he reported three From the respiratory section, department of medicine, University of Manitoba and the clinical investigation unit, St. Boniface General Hospital, Winnipeg Reprint requests to: Dr. C.P.W. Warren, Clinical investigation unit, St. Boniface General Hospital, 409 Tache Ave., Winnipeg, Man. R2H 2A6 cases of asthma after exposure to wheat rust (Puccinia graminis)[2].Bjornsson wrote that his grandfather, an Icelandic farmer, suffered from "heymaedi" (hay shortness of breath) in the early 19th century[3].

1. What is now known as farmer's lung was first described by Campbell' in 1932 in England: five farmers had dyspnea and pulmonary infiltrates after exposure to wet hay from which white mould dust arose. To grain dust asthma and farmer's lung as respiratory hazards of farming, Lowry and Schuman in 1956 added silo-filler's disease. The following review examines lung disease in farmers, with an emphasis on Canadian workers[2].
2. Later termed farmer's lung disease (FLD), this clinical entity has attained considerable importance in respiratory medicine. FLD is a hypersensitivity reaction to organic antigens inhaled from moldy hay. It was the first hypersensitivity pneumonia (HSP) or extrinsic allergic alveolitis described; today it remains the most prevalent and best studied. FLD, unrecognized, can cause severe disability and death[3].
3. Yet is often diagnosed by history alone and can be treated effectively by avoidance of moldy hay. Classically, it is manifested by fever, cough and dyspnea four to six hours after exposure and the pathological correlate is alveolar and interstitial inflammation of the lung[3].

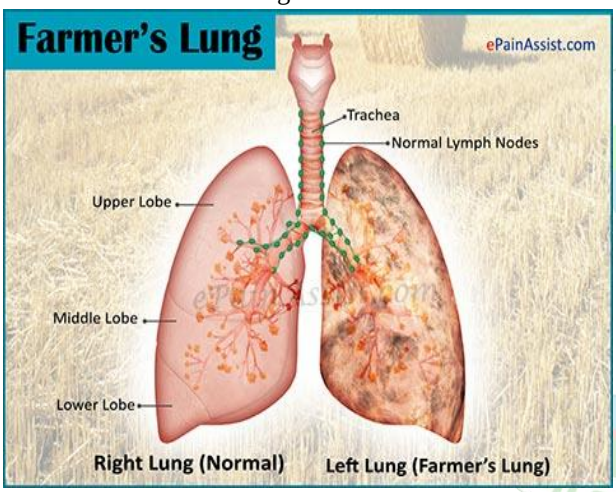

Figure: 1 Figure showing FL

Epidemiology

To understand the epidemiology of FLD, one has to know the antigen characteristics, since the antigen is the source of the disease. Improperly dried hay (greater than 30% moisture content) when stored in silos or stacked allows growth of micro-organisms and heat production. This warm, damp environment causes thermophilic fungi to proliferate and sporulate beginning within one week and continuing for months to years.

The lowest layers of hay contain the highest concentration of spores. Agitation may release white clouds of these respirable particles (0.5 u-1.3 u) into the air and cause disease in a susceptible host.[2]. The prevalence of FLD, established by questionnaire, is 9% among southern Manitoba cattle farmers .In the United States rates among farming populations vary from 0.42% in Wisconsin to 3% in Wyoming. In Scotland 2.3-8.6% of farmers in varying regions have a history consistent with the disease.[2]

Etiology

The disease is more common in wetter climates, where damp hay ferments and heats, and moulds grow. The spores of these moulds may be inhaled in prodigious numbers: 10'/m' was the count in the air of one barn. In the 1960s Williams1' showed that the reaction in the lungs occurred some hours after inhalation of the mouldy hay dust, that it occurred in the peripheral parts of the lung and that it was accompanied by a systemic febrile reaction.

Pepys observed an association with serum antibodies precipitated by extracts of mouldy hay. He then showed that thermophilic actinomycetes - in particular Micropolyspora faeni and sometimes Thermoactinomyces vulgaris- were the responsible moulds. Antibodies against these moulds were found in 80% Gf patients with farmer's lung but, since they were also found in 20% of healthy farmers, they indicated only exposure to the mould.

Detection of such antibodies should be considered diagnostic only in the appropriate clinical context. Pepys believed that the disease is the result of an antigen (the mould) antibody reaction that activates complement, producing an Arthus reaction. However, the presence of granulomas in lung biopsy specimens and of precipitins in healthy people led others to suspect that cellular lymphocyte-mediated hypersensitivity is involved.[2]

The etiology of the disease is clear - the inhalation of mouldy hay dust - and much can be done to prevent it ifthis is borne in mind. Mouldy hay dust is a very complex material consisting of innumerable fungal spores, hyphae and bacteria and fragments of vegetable matter. It is not surprising, therefore, that we have this small group of patients who complain of permanent exertional dyspncea as the result of irreversible lung damage. In this group the radiological changes are non-specific and it is likely that some are regarded as 'pulmonary fibrosis of unknown etiology', 'healed tubercle' or 'honeycomb lung'. Occasionally this chronic type may follow a single severe attack.

Many years later he presented with severe exertional dyspncea; clinically and radiologically the appeared to have pulmonary fibrosis of unknown etiology. Lung biopsy revealed an extensive diffuse fibrosis more marked in the centre of lung lobules.[4]

Dusts

Organic Dusts

- Due to the nature of Wisconsin farming, organic dusts are the most common cause of agricultural respiratory disease on most Wisconsin farms. Silos, dairy and poultry barns, and grain bins are sources of high levels of organic dusts.
- Organic dust is a complex mixture of vegetable matter, pollens, animal dander, insect, rodent and bird feces, feathers, microorganisms, bacterial and fungal cell wall toxins, pesticides, and antibiotics and can be thought of as a chemical soup.

Inorganic Dusts

- Inorganic dusts are primarily an issue in field activities associated with plowing, tilling, haying, and harvesting.
- Grain handling, manual harvesting of tree fruit and grapes, Christmas tree farms, potato harvesting, and small vegetable harvesting by hand can also cause an exposure to inorganic dust that may be higher than OSHA regulatory

levels for nuisance dust (Schenker, 1998). Inorganic dust is much less of an issue in Wisconsin as compared to the Great Plains and the major fruit producing areas. Silicates, including primarily the noncrystalline diatomite silica but also crystalline silica or quartz, make up the bulk of inorganic dust (Neiuwenhuijsen and Schenker, 1999).

Animal Confinement Gases

The primary animal confinement gases of human health concern are hydrogen sulfide (H_2S) and ammonia (NH_3). Carbon dioxide (CO_2) and methane (CH_4) are also formed and are considered simple asphyxiants and are of secondary concern. CO_2 is produced from animal respiration and is of concern if 5000 ppm or greater. CH_4 may be a risk for explosion at higher concentrations. Bacterial decomposition of animal manure and urine results in the gas production.

Noninfectious Respiratory Diseases and Syndromes

Many respiratory conditions may initially present as a viral-like syndrome and may be misdiagnosed and treated inappropriately as bacterial sinusitis and bronchitis with antibiotics.

Farmer's Hypersensitivity Pneumonitis

"Doc, I think I have Farmer's Lung". "Well, you farm and have a cough and shortness of breath so you must be right. You might as well stop farming"

Farmer's Hypersensitivity Pneumonitis (FHP) is what was previously referred to as Farmer's Lung Disease. It is a form of hypersensitivity pneumonitis, or allergic alveolitis, that is specific to sensitization to thermophillic actinomycetes (Gram+ filamentous bacteria) or Aspergillus fungal species found in organic dust in agricultural operations. Occupations at risk include dairy farmers, poultry workers, zoo keepers, and nursery workers.

Asthma and Occupational Asthma

Asthma is a classic IgE antigen-antibody mediated sensitization to an environmental antigen and is defined as a chronic inflammatory pulmonary disorder with reversible obstruction of the lungs as a result of exposure to variable stimuli. The obstruction may reverse either spontaneously or with treatment. The clinical hallmarks are wheezing, cough, and dyspnea (air hunger).

The most common cause is from environmental allergens. Generally, farmers and agricultural workers have a lower prevalence of asthma than the general population. This may be because of the healthy worker effect in which those who do not tolerate the dusty work conditions leave that occupation.

There is a recent body of literature from Europe and Australia that suggests that children growing up on farms have a lower prevalence of asthma, hay fever, respiratory, and allergic, or atopic, diseases compared to children not raised on farms.

Chronic Bronchitis

Chronic bronchitis defined as a daily productive cough for three months a year for at least two years. Chronic bronchitis is estimated to have a prevalence of 25-50% in animal and grain production workers, and grain elevator workers. Swine confinement workers have the highest prevalence. Cigarette smoking by itself is a significant risk in developing chronic bronchitis but it also additive and probably synergistic with the agricultural exposures, particularly endotoxin. Prevention involves adequate respiratory protection, decreasing levels of dusts and gases in agricultural operations, and smoking cessation.

Sinus Conditions

Sinusitis is common and occurs in up to 25% of swine confinement workers. Rhinitis symptoms are reported to occur in 20-50% of animal confinement workers. A recently reported syndrome, mucous membrane inflammation syndrome consisting of eye, nasal, and throat symptoms, has been recently described. This complex of symptoms is an irritant reaction and not IgE mediated and is the most commonly reported syndrome in animal confinement workers.

Other Toxic Gases, Fumes, And Chemicals

Nitrogen Oxides

Nitrogen dioxide (NO_2) is a severe respiratory irritant and is associated with Silo filler's disease (SFD). Silos are can be hazardous confined spaces during the fall when being filled with grain (generally corn but also oats) silage and haylage.

Anhydrous Ammonia

Anhydrous ammonia (NH_3) is a commonly used nitrogen fertilizer. It is a liquid under pressure but a gas under atmospheric conditions. The toxic property of concern is the extremely high hydroscopic property, or extreme affinity for water. Anhydrous ammonia avidly draws out water from tissues and causes a severe caustic burn, freezing and dehydration of tissue, particularly of mucous membranes, including the eyes, sinuses, nose, and upper respiratory tract. An extremely pungent odor is very noticeable. Corneal burns and laryngeal edema can result.

Carbon Monoxide

Carbon monoxide (CO) is a toxic odorless and colorless gas that kills. CO produced by internal combustion engines. Agricultural exposures can occur from kerosene heaters, gasoline-powered pressure washers in animal confinement operations, and running engines in shops or barns. Extremely toxic concentrations can rapidly accumulate in poorly ventilated buildings, as quickly as within 3-5 minutes (NIOSH, 1993).

Fetuses of pregnant women and those with ischemic heart disease and angina are at particular risk for toxic effects at lower levels than healthy adults. Symptoms may initially consist of headache, fatigue, difficulty concentrating and dizziness progressing to chest pain, shortness of breath, visual abnormalities and eventually confusion, weakness, and coma at higher levels or prolonged exposure.

Loss of consciousness can rapidly develop without warning signs in environments with high concentrations. Pulmonary edema and respiratory arrest may occur. Delayed neurotoxicity can occur after significant poisoning but cannot be predicted by the initial presentation or carboxyhemoglobin.

Pesticides

Acute exposure to organophosphates or carbamates resulting in poisoning can result in pulmonary symptoms. This can occur in applicators or in field workers entering a field before the safe re-entry interval guidelines. A concern could be in ginseng production due to the canopy covering the plants and reported high use of pesticides. Excessive bronchial secretions and bronchoconstriction can cause acute respiratory distress, wheezing, chest pain, cough and hypoxia. Hemoptysis and pulmonary edema may occur. The treatment consists protecting the airway, adequate oxygenation, and administration of large doses of atropine to reverse the muscarinic effects of the pesticides. Cardiorespiratory arrest is the usual cause of death in acute poisoning.

Disinfectants

Exposure to high concentrations of disinfectants such as chlorine gas, quaternary ammonium compounds, or mixing bleach with ammonia in poorly ventilated indoor settings may cause acute pulmonary irritation. If the concentration is high enough to cause acute pleuritic chest pain and significant shortness of breath or dyspnea, reactive airway dysfunction syndrome (RADS), a condition clinically identical similar to asthma, may occur. This condition is characterized by a non-immunologic reactive airways response and may last six months or even causes permanent wheezing

Microorganism Related Respiratory Illnesses And Diseases

Ilnesses Related to Environmental Exposures

Storage Mites Storage mites, including, Acarus siro, Lepidoglyphus destructor, and Gypcyphagus domesticus, are found in barns and grain and are antigenetically different from house mites.

Hantavirus

Hantaviruses are members of the single stranded RNA bunyavirus family. Sin Nombre Virus (SNV) is the hantavirus that is considered to be causative organism resulting in hantavirus pulmonary syndrome (HPS). The Center for Disease Control (CDC) defines HPS as febrile illness characterized by bilateral interstitial pulmonary infiltrates and respiratory compromise usually requiring supplemental oxygen and resembling acute respiratory disease syndrome (ARDS).

Exposure occurs from typical agricultural activities such as cleaning animal sheds and grain bins, and seasonally closed buildings such as lake cabins(Zeitz et al, 1995). The greatest risk occurs in buildings with increased rodent populations.

Blastomycosis

Blastomycosis in caused by fungal microorganism, Blastomyces dermatitidis. It is endemic in Wisconsin, particularly along the Mississippi River Valley watershed and other areas near moist soil with decomposing vegetation

Zoonotic Respiratory Diseases

Besides the bacterial source of inflammatory endotoxins, bacteria are also associated with infectious disease. According to the World Health Organization (WHO), zoonoses are those diseases naturally occurring between vertebrate animals and humans. It is critical for the rural practitioner to know where to obtain information about potential zoonotic diseases in his or her service area

Anthrax Inhalational

Anthrax is a disease that has been rooted in agricultural and occupational exposures but has been transformed into a primary disease of bioterrorism.

Tularemia

The Gram (-) coccobacilli, Francisella tularensis, both Types A and B, is the causative organism of tularemia, or "rabbit fever". It is considered to be both under recognized and under reported. Exposure routes include ingestion, inoculation by bites, inhalation, and tick- borne exposures.

Influenza

The first human case of Swine influenza was first identified in Wisconsin in 1976. A recent study in Wisconsin identified higher seroprevalence evidence of swine influenza infection was found to be associated with being a farmer or farm family member, or entering the barn greater than four days a week compared to nonfarmers (Olsen et al, 2002). Swine can be a source of zoonotic transmission of swine influenza (most commonly classic swine virus of the H1N1 strain) to humans.

Tuberculosis

Mycobacterium bovis can result in a pulmonary form of tuberculosis in veterinarians, farm workers, abattoir workers, and zookeepers but has become uncommon. Infection occurs through ingestion of contaminated raw milk or milk products and inhalation.

Mycobacterium tuberculosis, the main cause of tuberculosis in humans, is not a zoonotic disease but should be mentioned as there is an increased prevalence found in migrant and seasonal agricultural workers.

The highest rates for both latent tuberculosis infection and tuberculosis disease are found in Mexican and Central American workers in U.S.-Mexican border communities (Lobala and Cegielski, 2001). Clinical suspicion of symptoms consisting of productive cough of over two weeks, chills and fever, weight loss, anorexia, and hemoptysis in individuals of susceptible populations living in substandard housing with lack of access to health care services should include the possibility of M. tuberculosis infection.[5]

Pathogenesis

Antigens Associated With HP

There are over 200 antigens known to be involved in various forms of HP. The antigens can be proteins or glycoproteins from animals, plants, bacteria, protozoa, and viral origins or small molecular weight chemicals and drugs.

Proteins derived from birds and animals constitute a significant source of antigens in causing diseases such as bird fancier's lung, furrier's lung, animal handler's lung, and gerbil keeper's lung, among others.

Pigeons are the principal source of avian allergen in HP, but several other birds have been cited as causative agents such as parakeets, parrots, doves, chickens, and turkeys. The avian antigens may be from the droppings, serum or from the bloom and are inhaled as dust particles.[1]

Clinical Types

FLD is conventionally classified into 3 groups (acute, subacute and chronic), depending mainly on clinical and radiological findings at the time of diagnosis.[7]

Acute

Acute disease occurs after exposure to high concentration of antigen over a short period. Symptoms appear 4–8 h after exposure, and tend to resolve quickly. It is characterized by non-specific symptoms, such as general malaise, low-grade fever or fever, and dry cough.

Subacute

Subacute disease occurs after continuous, but not massive, inhalation of antigens. Symptoms develop more insidiously. It is characterized by general malaise, low-grade fever, asthenia and anorexia, progressive development of dyspnea and non productive cough longer periods.

Chronic

Chronic disease occurs after exposure to lower antigen levels, but over longer periods. It is also described as a progression of untreated acute or subacute disease. It usually occurs with symptoms of progressive dyspnea on exertion and dry cough. Physical examination reveals digital clubbing and dry crackles on auscultation. Chronic obstructive pulmonary disease with centrilobular emphysema, rather than fibrosis, has been described in patients with recurrent acute episodes.[8]

Diagnosis

Diagnosis Several conventional diagnostic criteria have been proposed for FLD, but none have been validated. Like other diffuse interstitial pulmonary diseases (DIPD), the point of departure is clinical suspicion, backed up by a detailed history of workplace and environmental exposure, sug gestive clinical symptoms and lung function values, radiological findings, cytological evidence on bronchoalveolar lavage (BAL), and consistent pathology results. The following additional testing is then performed:

Clinical Laboratory Testing

Specific IgG antibodies or precipitins (precipitating IgG antibodies) against the different suspected antibodies should be determined in the patient's serum, in order to confirm that the individual has been exposed and is sensitized to the causative agent. A negative result for plasma precipitins does not rule out the diagnosis, particularly in the chronic forms of the disease, since antibody titers and levels of exposure are correlated, and may become negative if there has been no contact with the causative antigen for some time. The determination of precipitins may also be limited, firstly by the lack of standardized analytical methods, and secondly because the panel of precipitins will vary among the different regions. Main Criteria Proposed for the Diagnosis of Hypersensitivity Pneumonitis.[9] Consistent clinical history, physical examination and lung function Consistent radiology Exposure identified Anti-antigen antibodies.[10]

Exposure to antigen Inspiratory crackles Dyspnea Predominantly lymphocytic BAL Consistent radiology Recurrent fever Reduced DLco Anti-antigen antibodies Lung biopsy consistent Improvement after avoidance Schuyler et al.[11] Consistent clinical picture Evidence of exposure from history or antibody detection in BAL and/or serum Consistent radiology Lymphocytosis on BAL Positive challenge test Consistent biopsy Bibasal crackles Hypoxemia at rest or during exercise Reduced DLco.[12]

Exposure to antigen from history, antibodies or microbiological identification Consistent clinical picture Consistent radiology Reduced DLco Bibasal crackles Hypoxemia at rest or during exercise Restrictive ventilatory changes Consistent lung biopsy Positive bronchial challenge test will be necessary to determine which are the most common anti gens in each site.[5,13]

Skin Prick Tests

Skin prick tests for delayed sensitivity to antigens are considered very unspecific, but some studies have demonstrated diagnos tic benefit, particularly when read immediately (10–15 min), both in FLD, with a sensitivity of 83% and a specificity of 72%,[14]and in bird fancier's lung, with a sensitivity of 90% and a specificity of 85%.[15]

Radiology

In acute phases, the chest X-ray can be normal or show diffuse pulmonary infiltrates. In the chronic phases, a bilateral reticular pattern may be observed. In acute phases, typical chest HCRT findings are diffuse ground glass infiltrates and hyperlucent images (as a result of concomitant bronchiolitis), which together form a pattern of mosaic attenuation.Centrilobular nodules are also typical in some disease phases, but they are more common in the acute phases.

Honeycombing is an important feature of chronic forms. Emphysema is observed in 20% of non-smokers with FLD. Involvement is typically limited to the middle and upper lung fields, although lower fields cannot be ruled out, which means that at times disease appears on CT primarily in a honeycombing pattern and the radiological image is indistinguishable from that of usualinterstitial pneumonia (UIP).

Figure:2 radiology of farmers lungs

Respiratory Function

Patients have a restrictive ventilatory pattern, with altered gas exchange (reduced DLco and desaturation on exertion), also observed with other DIPDs. In chronic forms of FLDan obstructive ventilatory pattern may occur along with emphysema.[16]

Specific Inhalation Challenge

In unclear cases, the diagnosis must be confirmed with a specific inhalation challenge (SIC) conducted with suspected antigens – a range of molds and/or actinomycetes, depending on the region. SIC can now be considered as validated, after the publication of studies in other types of HP, particularly in bird fancier's lung, and the more recent paper from Mu˜noz et al., in both bird fancier's lung and HP. Thus, before performing a surgical lung biopsy, an SIC must be conducted with the appropriate antigens, in hospitals where it is available.[17]

Bronchoscopy

In the study of FLD and other types of DPID, BAL can be used for guiding the differential diagnosis. BAL cytology in FLD generally reveals raised CD8 lymphocyte levels (lymphocyte predominance >20% and usually, but not always, an inverted CD4/CD8 lymphocyte ratio).[18]

Transbronchial Biopsy, Cryobiopsy, and Surgical Lung Biopsy

Transbronchial biopsy (TBB) can be useful in the early stages of the disease.[19]TBB by cryoprobe (or cryobiopsy) is a less invasive endoscopic technique than surgical lung biopsy, and may play an important role in the diagnosis of FLD. Recently published studies report a diagnostic yield in DPID of over 70%, with relatively few complications.[13] Surgical lung biopsy is not usually required to establish a diag nosis of FLD, but it is often vital in differentiating a chronic form of FLD from other interstitial entities, particularly in cases in which no antigen can be identified.[20]

Differential Diagnosis

Even if a surgical lung biopsy is available, differential diagnoses suggested by that specimen can include other entities, ranging as widely as sarcoidosis, drug-induced DIPDs, lymphocytic interstitial pneumonia, and DIPDs due to connective tissue diseases. In advanced chronic forms, it may be difficult to differ entiate the histological pattern of UIP from IPF, or from the NSIP pattern, although fibrosis spreading toward the center of the lob ule in biopsies with a UIP pattern can indicate the possibility of chronic HP.[19] Autopsy series have found that fibrosis is more common than granulomas in the chronic forms of the disease.[21]

Treatment

The treatment of FLD is based mainly on avoiding exposure to the antigen: we will address this approach in more detail in the section on prevention. This is the only measure that has been shown to delay disease progression. Treatment with glucocorticosteroids accelerates recovery in the acute forms, but it has not been shown to affect the progress of the disease in the long term, and some authors question their role in the chronic forms.[24]

Corticosteroids are traditionally recommended in patients with impaired lung function. The doses are similar to those given for other DIPDs, such as sarcoidosis (30 mg/day for 1 month, then 20 mg/day, followed by tapering to a mainte nance dose of 7.5–15 mg/day). No scientific evidence is available for recommending either the use or avoidance of inhaled glucocor ticosteroids. Immunosuppressive treatments have been tested in refractory forms of other HPs, such as bird fancier's lung, but not in FLD.

In these patients, the few published cases have reported improved functional progress with rituximab.[10,11] Lung transplantation is indicated in cases of progressive dis ease with respiratory failure, despite the treatments mentioned above. A recent study found improved 5-year survival in patients transplanted due to HP, compared to those transplanted for IPF.[12]

Pharmacologic therapy with supplemental oxygen and parenteral corticosteroids is indicated for ill patients with abnormalities on lung function testing, radiographs or with hypoxemia, For acute symptoms, oral prednisolone at 40-80 mg daily for 1-2 weeks may be sufficient while subacute and chronic HP may require 40-80 mg daily with a taper over several months depending on the response to improvement in symptoms and functional abnormalities100. Other treatments have only been described in isolated cases.

Management

As with other allergic lung diseases, the most important aspect of management is identification of the inciting antigen so that avoidance measures can be implemented. Frequently, avoidance alone is sufficient intervention. In occupational diseases this may include changing

professions and job retraining, changing Working materials, adding fungicides to water-based products, and/or instituting personal respiratory protection. In an effort to reduce farmer's lung, treatment of hay with a buffered propionic acid significantly decreased the concentration of fungal species and thermophilic actinomyces without the deleterious effects on farm machinery or cattle.

Prevention

There are 3 ways of reducing repeated inhalation of organic par ticles by FLD patients, in order to prevent the disease progressing to a fibrosing, and hence irreversible, process:

- definitive with drawal of the patient from the farming environment, which in most cases is unfeasible for financial reasons.
- development of new techniques for drying the hay, and ventilation and mechanization of stock feeding; and
- use of respiratory protective equipment to prevent the antigens entering the respiratory tree. One of the main preventive measures consists of improving hay storage methods. Drying or heating the fodder during storage has proven to be beneficial in reducing antigen levels.[13] Keeping the fodder in loosely packed square bales also hinders the growth microorganisms, but these methods are disappearing, due to the high cost and heavy labor burden, and are being replaced by round bales. These bales are easy to store and handle, but they con tain high humidity levels due to greater compaction of the hay.[30]

Prognosis

The progress of FLD varies widely and depends fundamentally on time and antigen load. Even so, in some patients, the disease continues to progress for as yet unknown reasons, despite the use of appropriate avoidance techniques. The acute phase of FLD is generally reversible, but contin uous exposure or several subacute episodes of hypersensitivity to the allergen can cause fibrotic forms to develop, causing irreversible changes in lung structure and function.Up to 20% of acute forms progress to chronicity. A Finnish study esti mated mean 17-year survival after the appearance of symptoms at 9%–17%.[6]

CONCLUSIONS

In summary, FLD is a disease that may possibly be underdiagnosed in the north of Spain, although no precise epidemiological studies are available. Data on the long-term efficacy of corticos teroide treatment and other immunosuppressants is also scant, and no regional antigen panels are available for diagnostic purposes. More awareness of a suspected diagnosis is needed, taking into account that presentation may not fulfill the conventional criteria for DIPD (e.g., obstructive ventilatory changes and emphysema on chest HRCT). Patients with a diagnosis of FLD should be encour aged to follow a strict strategy for preventing antigen exposure, including, in particular, the proper use of protective respiratory equipment.

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