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CHRONIC HYPERSENSITIVITY PNEUMONITIS MIMICKING PNEUMONIA : A CASE REPORT

Respiratory Medicine

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ABSTRACT

The hypersensitivity pneumonitis (HP) or extrinsic allergic alveolitis (EAA) is the paradigm of lung response to inhaled organic and inorganic. It is a relatively rare disease, constituting 2% of cases of interstitial lung diseases. The authors describe a case of a 60 years old man, with history of farming, who presented cough, sputum expectoration, fever, dyspnoea, chest pain, generalised swelling and haemoptysis. The study pointed to the initial diagnosis of pneumonia, but further study revealed that it was a case of chronic hypersensitivity pneumonitis. The authors present this case to emphasize the importance of history of exposure to any toxic substances, a lack of specificity of clinical manifestations, the need for invasive diagnostic methods and, sometimes, poor response to therapy.

KEYWORDS

INTRODUCTION

Lung response to inhaled organic material, either in the form of particles or of organic protein, or even of simple chemicals, organic and inorganic is referred as the hypersensitivity pneumonitis (HP) or extrinsic allergic alveolitis (EAA). HP has been considered as highly variable and difficult to assess prevalence.

HP can be present in three forms clinically acute, subacute and chronic - depending on the nature of the aggressor agents, their concentration, their physical characteristics, individual variability and the intensity of individual exposures. After inhalation the acute response is a nonspecific diffuse pneumonitis with inflammatory cell infiltration of the bronchioles, alveoli, and interstitium. In chronic and subacute stages, loosely formed, noncaseating, epithelioid cell granulomas can be dispersed in the interstitium.

In a picture of a single determinant HP, its eviction is mandatory. The treatment is based on corticotherapy, but the evidence for long term benefit is absent [1].

Presentation of case :

The authors present the case of a 60 years old male, from surat, watchman by occupation with a history of farming, and diabetes. He presented with complaints of persistent cough, sputum expectoration, fever, dyspnoea, chest pain, generalised swelling and haemoptysis.

He had no family or personal history of pathological relevance. He denied smoking and alcohol consumption. He was not suffering from Koch's, Asthma or IHD. He was taking metformin for Diabetes mellitus.

On physical examination the patient was alert and oriented but pedal oedema, pleural effusion, and pulmonary oedema and complain of breathlessness so the patient underwent for ECG, in which the findings were normal.

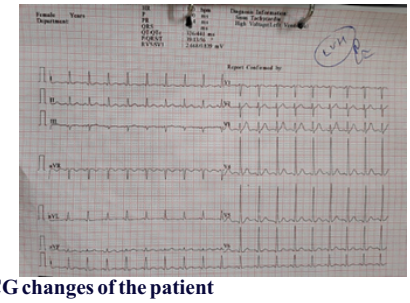


Fig 1: ECG changes of the patient

X-Ray

A chest x-ray was performed in which consolidation on right side was observed so diagnosis of pneumonia was made for which antibiotic was prescribed but no improvement was observed in X –Rays.

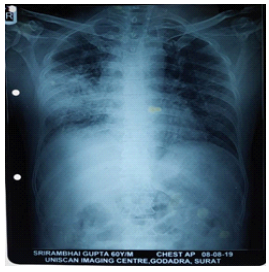
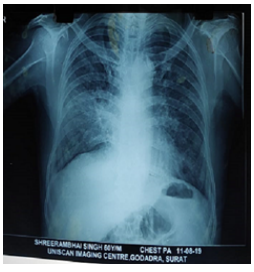


Fig 2



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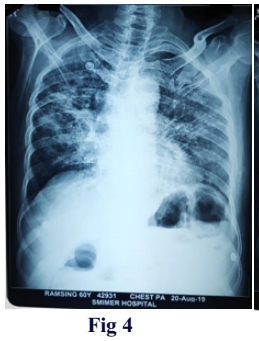

Fig 4

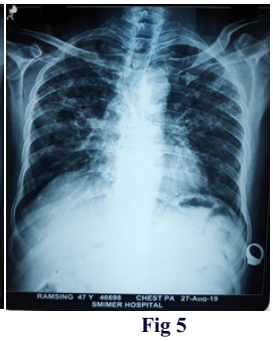

Fig 5

Figure no 2 to 5 showing the X-Ray images during the treatment course, here in fig 5 we can clearly observe improvement after steroid treatment.

After pneumonia treatment, Sputum AFB for tuberculosis was done, which was negative.

2D ECHO was performed to see the changes in heart and it was observed that due to chronicity of the disease patient developed pulmonary artery hypertension (PAH).

Since we were not getting any clinical findings for hypersensitivity pneumonitis so a CT scan was performed.

Fig 6

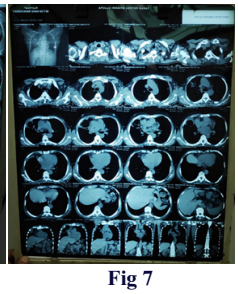

Fig 7

Chest CT findings can observe in fig 6,7 which revealed patchy areas of reticulations with fibrosis and traction bronchiectasis were seen in both the lung fields predominantly involving the both upper lobe and right middle lobe with few areas of honeycombing in the both upper lobe, irregular cavity was observed in the right middle lobe with loss of lung volume, also few prominent lymph nodes were observed in the prevascular, pretracheal, aortopulmonary window and subcarinal region. Findings were indicating the chronic hypersensitivity pneumonitis. Steroids were prescribed and the response was good and patient showed improvement within 4 to 5 days, we can observe improvements after taking steroid in fig 5.

The study pointed to the initial diagnosis of tuberculosis, but further study revealed that it was of hypersensitivity pneumonitis.

Differential diagnosis

The differential diagnoses of hypersensitivity pneumonitis include hot tub lung, usual interstitial pneumonia, cellular chronic interstitial pneumonias and nonspecific interstitial pneumonia, caused by drug reactions and collagen vascular diseases.

The differential diagnoses of hot tub lung include nontuberculous mycobacterial infection unrelated to hot tubs and hypersensitivity pneumonitis due to other causes [4].

Differential diagnoses includes usual interstitial pneumonia if other features of usual interstitial pneumonia like honeycombing and scarring also if fibrosis supervenes. In such conditions differentiating usual interstitial pneumonia from chronic hypersensitivity pneumonitis can be challenging. In this situation the histologic clue for the diagnosis of hypersensitivity pneumonitis is the presence of a cellular chronic interstitial pneumonia typical of chronic hypersensitivity pneumonitis also away from the areas of honeycomb lung [2]. In cases of usual interstitial pneumonia, granulomas may be related to aspiration of gastric contents (food/pill fragments) or superimposed infection. Therefore, granulomas present in usual interstitial pneumonia does not indicates a diagnosis of chronic hypersensitivity pneumonitis.

In most of the cases interstitial chronic inflammation is the most striking feature of hypersensitivity pneumonitis, granulomas dominate the histologic picture in hot tub lung. The granulomas of classic hot tub lung are well-formed, large and present within airspaces, while in case of hypersensitivity pneumonitis they are poorly formed, tiny and present in the interstitium [5]. A clinical history of hot tub use is essential for a diagnosis of hot tub lung apart from histologic findings.

One other entity which is difficult to separate from hypersensitivity pneumonitis is nonspecific interstitial pneumonia. The two conditions share the presence of chronic interstitial pneumonia primarily composed of lymphocytes. Therefore, pathologists encountering nonspecific interstitial pneumonia biopsy samples, should carefully search for tiny loose granulomas or multinucleated giant cells in the interstitium. Also the cases without these observations may still diagnosed as hypersensitivity pneumonitis [3].

Therefore, from the clinical standpoint, descriptive diagnosis of "cellular chronic interstitial pneumonia or a pathologic diagnosis of nonspecific interstitial pneumonia" should always prompt a thorough search for a potential source of antigen exposure. A diagnosis of idiopathic nonspecific interstitial pneumonia should be made only after a meticulous clinical search for antigen exposures, underlying collagen vascular diseases, and drug toxicities.

Pathologically, common features of hypersensitivity pneumonitis and hot tub lung include granulomas, interstitial inflammation, and foci of organizing pneumonia. The distinction between the two entities lies in the relative prominence of interstitial inflammation and granulomas, the morphologic characteristics of the granulomas, and a history of hot tub use.

Clinical diagnosis:

Clinically hypersensitivity pneumonitis is classified as per the duration and onset of symptoms, although cut-offs vary widely [5,6]. Acute hypersensitivity pneumonitis patients develop symptoms within 2-9 hours of exposure to the antigen. Typically they present with systemic or influenza like symptoms like fever, chills, myalgia's, sweating, headache, body aches, and nausea. Dyspnoea and cough and may also occur. Recent data suggest that this classification is less than ideal [7].

It has been suggested that symptoms take longer to resolve in sub-acute hypersensitivity pneumonitis which is a variant of acute hypersensitivity pneumonitis [7]. Chronic hypersensitivity pneumonitis patients present predominantly with dyspnoea and cough [6,7]. Fever, weight loss, fatigue, flulike symptoms and chest discomfort may also occur but are less common. There may be a history of exacerbation of symptoms in certain situations or locations and decreased severity in others. This typical waxing and waning of symptoms is a reflects the exposure to and removal from the source of antigen.

Hypersensitivity pneumonitis has the most common physical finding is the presence of inspiratory crackles, which can be auscultated in most of the patients with this condition [5,6]. In minority of cases wheezing, digital clubbing, and cyanosis, are demonstrable in only a minority of cases.

Due to the lack of well-defined diagnostic criterion standard the diagnosis of hypersensitivity pneumonitis is mostly difficult [8]. Key investigations for diagnosis include pulmonary function tests, bronchioloalveolar lavage (BAL), and high-resolution computed tomography (HRCT).

Increase in lymphocytes and a predominance of CD8-positive T lymphocytes over CD4-positive T lymphocytes, which is a reversal of the normal CD4:CD8 ratio, in which CD4-positive cells predominate demonstrated by BAL typically. Pulmonary function tests mostly show restrictive changes with a decrease in the diffusing capacity of carbon monoxide (DLCO), but in some patients obstructive changes may be observed [5,6]. Further investigations may include skin hypersensitivity tests, serologic testing for antibodies to various antigens, or antigen challenge tests.

Pathological discussion:

The pathological features of chronic hypersensitivity pneumonitis comprise overlapping usual interstitial pneumonia-like pattern with sub pleural patchy fibrosis, fibroblastic foci, and alternating normal alveoli a nonspecific interstitial pneumonia-like pattern, and centrilobular fibrosis. Epithelioid cell granulomas are sparse or absent, in contrast to pathological features of acute and subacute hypersensitivity pneumonitis, but giant cells are seen in the interstitium. There is an outstanding feature of chronic hypersensitivity pneumonitis i.e. bridging fibrosis between peribronchiolar area and perilobular areas. In chronic hypersensitivity pneumonitis the autopsy cases demonstrated not only upper lobe contraction but also lower lobe contraction, which mimicks usual interstitial pneumonia pattern and diffuse alveolar damage [9].

Discussion of management:

Avoiding the dust that causes the disease is most important for preventing chronic hypersensitivity pneumonitis. Since the disease is completely reversible in the early stages avoiding dust could help lungs to return to normal function. Sometimes it's not possible to avoid dust completely, unless a patient can remove him/herself from the dust-causing environment. Dust can be avoided completely by relocating to a new job or home. It is possible to give up the pet bird for a bird fancier's lung.

In patients with severe cases, treatment may include steroids, such as prednisone. It may be recommended to take this medication for up to 3 months and sometimes longer. Steroids will not cure the disease but may help with your symptoms. There are certain side effects of steroids like weight gain, cataracts, thinning of the bones, increased pressure in your eyes and abnormal blood sugar levels. Immunity suppressing medications such as azahtioprine and mycophenolate show promise as steroid-sparing agents in some individuals to eliminate or reduce steroids. In end stage lung disease with advanced scarring, lung transplantation may be a consideration.

Steps to limit exposure to certain dusts.

- Be sure to remove any standing water inside and outside your home since Allergy-causing fungus and bacteria and can thrive in stagnant, or still water.
- Maintain humid condition at home and work below 60 percent.
- Immediately repair any water damage inside your home or work which includes removing drywall, water-damaged carpeting, and furnishings.

- Properly maintain your heating, air conditioning and ventilation systems.
- Make sure that water in heating, air conditioning and ventilation systems is not recirculated.
- Store farm products properly dry, if you work with them.

Dust cannot be avoided completely, so there are certain protective devices that can reduce the chances of breathing in the dust. Acute attacks of farmer's lung can be prevented by air purifying respirators. Dust respirators are not found to be very helpful since wearing such respirators for long periods can be a challenge. To reduce dust exposure, electrostatic dust filter in the return ducts of central air conditioning systems can be used [10].

Final diagnosis:

To see early stages of the disease chest x-ray and CT (computerized tomography) scan can be used, if there is any scarring.

Lung function tests can be done to see how well your lungs are working.

To find out antibodies against the dust the blood test can be done, these blood tests helps in understanding if the patient has exposed to certain dust.

In bronchoscopy a bronchoscope is passed either through your mouth or nose then it passed into your vocal cords, windpipe and the air passages. This tool also help in collecting specimens from your lung for further testing.

Cardiothoracic surgeon performs video-assisted thoracic surgery (VATS) or open lung biopsy under general anesthesia, this process can also help in getting lung tissue for further testing [10].

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