Dry-Snuff Induced Chronic Hypersensitivity Pneumonitis: A Case Report

Case study

Abstract

Dry nasal snuff, commonly known as “chhikni” in India,is a smokeless tobacco preparation prepared from dried powdered tobacco which could be inhaled, "sniffed" or "snuffed" into the nasal cavity[1]. The inhalation of nasal snuff (smokeless tobacco) is a common addiction in rural India. In the western world, there is a resurgence of interest in the use of nasal snuff. Very few cases have been reported regarding long-term effects of nasal snuff on lung parenchyma. We hereby present the case of a 67-year-old woman who developed chronic hypersensitivity pneumonitis because of sniffing dry snuff. She sniffed dry snuff for 30 years about four to five times a day .She was successfully treated with corticosteroids and was discharged on home based minimal O_2 support. This case report emphasizes on the respiratory complications associated with snuffing noxious substances and considering hypersensitivity pneumonitis as a possibility.

Introduction

India is the second largest consumer of tobacco globally, and accounts for about one-sixth of the world's total tobacco-related deaths. The tobacco problem in India is complex, with consumption of a variety of smokeless and smoking forms of tobacco[2]. With a gradual decline in cigarette smoking, the tobacco industry has come up with different forms of smokeless tobacco preparations to attract new customers. Such products are being massively promoted and are gaining popularity rapidly. Dry nasal snuff ,one of the oldest known forms of tobacco in Europe ,is one of them.The term ‘smokeless tobacco’ is used to describe a form of tobacco preparation that is consumed without heating or burning at the time of use. Smokeless tobacco can be used orally or nasally. For nasal use, a small quantity of very fine tobacco powder mixed with aromatic substances together called dry snuff is inhaled[3]. The health risks associated with it are different to those related to smoking and oral wet snuff. Also the nicotine contained in the product leads to dependency[4]

Variety of agents that include microbes, animal and plant proteins, organic and inorganic chemicals may cause hypersensitivity pneumonitis in susceptible individuals[5]. Hypersensitivity pneumonitis (HP), also called extrinsic allergic alveolitis, is a respiratory syndrome involving the lung parenchyma ,specifically the alveoli, terminal bronchioles , and alveolar interstitium, due to a delayed allergic reaction. Such reactions are secondary to a repeated and prolonged inhalation of different types of organic dusts or other substances to which the patient is sensitized and hyper responsive[6].

Case Presentation

A 67-year-old female with a past medical history significant for hypertension but on regular medications, presented to our hospital with acute-onset shortness of breath and dry cough. She reported inhaling dry snuff for 30 years four to five times a day.On presentation,the patient was drowsy but conscious. She complained of dyspnea and dry cough but denied presence of any chest pain, palpitations, hemoptysis, fever, dizziness or syncopal episodes. She was tachypneic, tachycardiac, normotensive and hypoxic with an O_2 saturation of 60% on 15L

non-rebreather mask but afebrile. Physical examination was positive for diffuse pulmonary crackles and scattered wheezes over both lung fields but otherwise unremarkable.She was intubated in view of low saturation and was kept on continuous positive airway pressure mode. Labs showed leukocytosis of 20,000/uL.Arterial blood gas analysis was significant for pH-7.25 , pCO_2-68 , pO_2-75 , HCO_3^--16.5 and SPO_2-60% on 15L O_2 via non-rebreather mask .Chest X-ray showed a diffuse micronodular pattern with patchy opacities (Figure 1A). A computed tomography (CT) scan of the chest was done and showed mosaic pattern in bilateral lung fields with few areas of ground-glass opacities bilaterally, suggestive of inflammatory pneumonitis (Figure 1B).Blood culture , endotracheal tube secretion culture and urine culture were negative for any growth.COVID-19 real time polymerase chain reaction was negative.Procalcitonin ,Immunoglobulin E levels ,Rheumatoid arthritis factor levels and angiotensin converting enzyme levels were within normal limits.Antinuclear antibodies were negative.Based on the patient's clinical presentation and the relationship between chronic usage dry snuff and the development of her symptoms, she was diagnosed with chronic hypersensitivity pneumonitis and was given Inj. Methylprednisolone 1 Gram followed by 500 mg once 24 hourly for treatment. After three days, her dyspnea and cough improved significantly, and she was extubated and kept on 2L O_2 via nasal cannula. A repeat chest X-ray showed improvement in the infiltrative process. She was discharged home on daily 60 mg prednisone for a total of two weeks, followed by slow tapering with a recommendation to avoid the use of dry snuff or any other inhalational noxious substance usage.

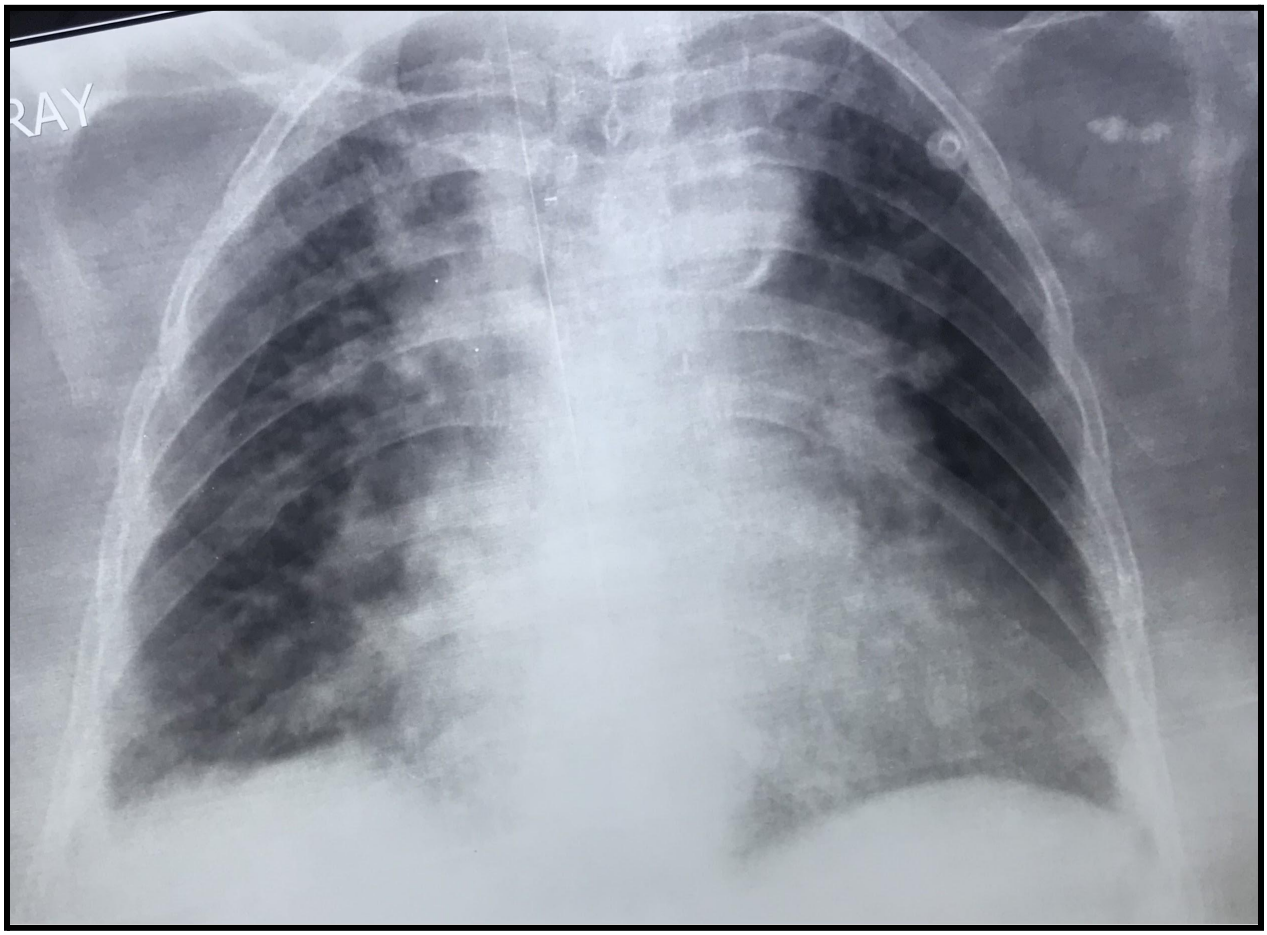

Figure 1A-A chest X-ray showed a diffuse micronodular pattern with patchy opacities

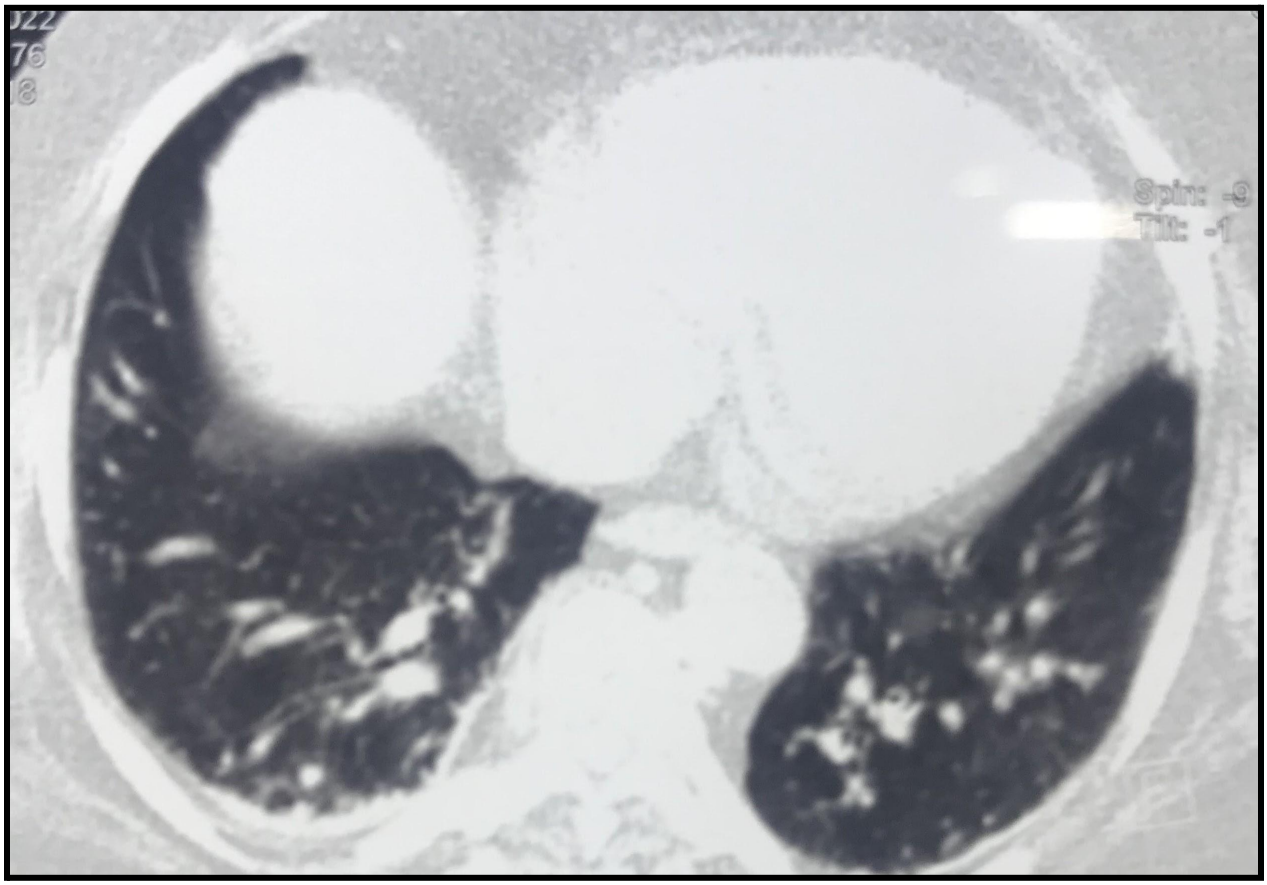

Figure 1B-HRCT thorax suggestive of mosaic pattern in bilateral lung fields with few areas of ground-glass opacities bilaterally.

Discussion

Hypersensitivity pneumonitis (HP), also known as extrinsic allergic pneumonitis, has been classified as an interstitial lung disease which is characterized by a complex process of immunological reaction of the lung parenchyma in response to repetitive inhalation of a sensitized allergen by a susceptible individual. The term HP defines the disease process more appropriately than the previous name ,extrinsic allergic alveolitis, as the inflammation doesn't involve the alveoli exclusively but the bronchioles as well. The severity of the disease and clinical presentation varies greatly depending on the type inhaled antigen and quantity. Traditionally HP has been classified into acute, sub-acute and chronic forms based on the time course and the presentation. But more recently it has been classified as Acute or Inflammatory HP (symptoms less than six months) and Chronic or fibrotic HP, this classification has been proposed based on clinical, radiologic and pathologic characteristics.[7]

The pathophysiology behind being, inhaled antigens which are less than 5 μm in diameter may reach the lung parenchyma, and move to the lymphatic channels and thus get deposited at the level of respiratory bronchioles. Pathogens and allergens involved in HP cause similar spectrum of clinical features, with an almost exclusive involvement of distal airways and alveoli along with interstitial infiltration by inflammatory cells. Also the high levels of serum precipitating antibodies against the antigens are responsible for the alveolar inflammation (precipitins). The levels of IgE

and eosinophils stay within normal limits. The pathogenesis of HP is quite similar regardless of the causative agent: the inflammatory response of the alveolar mucosa is a hypersensitivity reaction of type 3 (immune-complex-mediated) or type 4 (T lymphocytes mediated).[8]
The pathology is characterized by a bronchiolocentric interstitial mononuclear cell infiltration, small non-necrotizing epithelioid cell granulomas poorly formed, diffuse cellular pneumonitis and variable degrees of pulmonary fibrosis. Granulomas are commonly observed in the bronchiolar wall and alveolar ducts in subacute hypersensitivity pneumonitis; these granulomas are less than 150 μm in diameter, smaller than those observed in sarcoidosis[9].
The vessel wall also shows the deposition of precipitins against causative antigens, immunoglobulins and complement proteins.[10]
T-lymphocyte mediated hypersensitivity reaction is the most important type 4 immune response in the pathogenesis of HP. The TH1-cytokine network plays an important role in the development of HP, later in the chronic form a TH2-like immune response develops.In fact, features associated with chronic HP includes a progressive increase in CD4+ T cell count [11] and the CD4+/CD8+ ratio,the transformation of the response towards TH2 T cell and cytokine profiles, and a decrease in CD8+ T cell count. In acute HP, inflammation of the lung parenchyma appears to be mediated primarily by a type 3 response, as evidenced by high titers of antigen-specific IgG precipitates in serum and an increase in the number of neutrophils in the lungs. The subacute and chronic forms of HP are characterized by T cell-mediated immune responses with increased T cell migration and the development of the characteristic T lymphocytic alveolitis.[12]

Conclusions

Dry snuff, earlier considered harmless, is widely being used in rural India and is slowly gaining popularity in the western world as well. The popularity of smokeless tobacco preparation in rural women and disbelief of it being harmless needs to be abolished amongst the population. This article emphasises on the respiratory complications associated with snuffing noxious substances and considering hypersensitivity pneumonitis as a possibility while treating such patients.

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