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Case Report

# First Reported Case of Reactive Airway Dysfunction Syndrome in a Laborer Due to Porcelain Tile Dust

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## Abstract

Reactive airway dysfunction syndrome (RADS) is a type of non-immunologically mediated asthma-like disease. It usually occurs after a massive exposure to an irritating substance in the atmosphere in the form of smoke, fumes, gases, and vapor. Unlike bronchial asthma, there is no latency to the symptoms seen in RADS. A number of agents are known to cause RADS, but tile dust, as an etiological agent, has not been previously reported. We report a 45-year-old male laborer, who presented with an acute onset of cough, chest tightness, breathlessness, and audible wheeze after his first time exposure to porcelain tile dust within 5 hours of exposure. Lab tests, including, chest X-ray, electrocardiogram, air blood gas analysis, and serum IgE, were unremarkable. Spirometry showed a mild obstruction [forced expiratory volume in 1 second (FEV1)=72% of predicted], while the bronchodilator reversibility test was significant(14% increase in FEV1 above the baseline).Bronchial biopsy revealed a chronic inflammatory reaction with lymphocytic and plasma cell infiltration and more importantly a striking absence of eosinophils. To the best of our knowledge, this is the first reported case of RADS as a result of exposure to tile dust (porcelain ceramics).

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## Introduction

Reactive airways dysfunction syndrome (RADS) is a non-immune mediated type of asthma-like disease characterized by the immediate onset of symptoms of cough, chest tightness, audible wheeze, and breathlessness after a toxic single exposure to an agent with irritating properties in the atmosphere in the form of dust, vapor, fume, or smoke.[1] More than 30 different agents are known to cause RADS.[2,3] The most common agents implicated in the causation of RADS include chlorine, toluene diisocyanate, and oxides of nitrogen.[2]

The Brazilian standard describes porcelain tile as any ceramic tile made of clay, feldspar, and other inorganic raw materials that is either pressed or extruded and contains water absorption of less than 0.5%. However, porcelain tile dust as a cause of RADS has not been previously reported.

We present a 45-year-old male laborer with an acute onset of asthma after his first time heavy exposure to porcelain tile dust

within 5 hours of exposure whose symptoms persisted for 5 months after the incident. To the best of our knowledge, this is the first reported case of RADS as a result of porcelain tile dust exposure.

## Case Report

A 45-year-old man, non-smoking laborer presented with an acute onset of cough, chest tightness, audible wheeze, and breathlessness within 5 hours of heavy exposure to tile dust. The laborer was engaged in some construction work in our hospital associated with fitting of tiles. He had neither worked with a tile cutter nor had been exposed to a high concentration of tile dust in the past. He never had any such symptoms in the past or any allergic history like seasonal allergic rhinitis, hay fever, or atopic dermatitis. Chest examination revealed bilateral diffuse polyphonic wheeze. Laryngoscopic examination did not reveal any vocal cord dysfunction. Laboratory tests, including blood gas analysis, chest radiography, and ECG, were unremarkable. Serum total IgE level was 40 IU/ml (Normal level 10-179 IU/ml). Spirometry revealed mild obstruction ([forced expiratory volume in 1 second] FEV1=72% of predicted) with significant bronchodilator reversibility (14%). However, his spirometry improved with an FEV1 of 88% of predicted at 4 months from the incident (table1). Due to the persistence of the symptoms, bronchoscopy, performed 4 months after the initial episode, revealed diffuse hyperemia and bronchial biopsy revealed a chronic inflammatory response with lymphocytic and plasma cell infiltration and absence of eosinophils.

The patient was managed similar to acute bronchial asthma. He received intravenous hydrocortisone (100 mg) every 8 hours for the first few days along with salbutamol inhalations via a nebulizer. Once his symptoms improved, he was put on formoterol and budesonide rotacaps. On follow-up, the patient's symptoms had persisted for 5 months, after which he had only occasional cough, not of a magnitude to impair his routine activities.

## Discussion

Our case report met all the criteria of RADS as laid classically by Brooks et al.[4] which include a documented absence of preceding respiratory complaints; onset of symptoms occurring after a single specific exposure incident or accident; exposure to a gas, smoke, fume, or vapor, present in very high concentrations and with irritant qualities to its nature; onset of symptoms occurring within 24 hours after the exposure and persisting for at least 3 months; symptoms simulating asthma with cough, wheezing, and dyspnea predominating; pulmonary function tests probably showing airflow obstruction; positive Methacholine Challenge Test [an indication of non-specific bronchial hyperresponsiveness]; and other types of pulmonary diseases being ruled out.

More than 30 different agents are known to cause RADS.[2,3] Chlorine, toluene diisocyanate, and oxides of nitrogen are the most commonly implicated ones.[2] Others include hydrofluoric acid, thermal degradation products of fluorocarbons, ozone, etc., and many more are yet to be discovered.[5-7] In 2002, an outbreak of RADS/irritant-induced asthma (IIA) was reported among firefighters exposed to irritants in the atmosphere during and after the World Trade Center disaster. This has since led to the concept of "World Trade Center Cough", which is characterized by a persistent cough that develops among firefighters after such exposure.[8]

The pathogenesis of RADS is not fully understood. The acute pathological changes of RADS have been studied by subjecting mice to a high concentration of chlorine in the atmosphere. The findings include flattening of bronchial epithelium, necrosis, and evidence of epithelial regeneration, while bronchoalveolar lavage reveals an increased number of neutrophils.[9] Due to the persistence of the symptoms, the bronchial biopsy in our patient was done after 4 months and it revealed a chronic inflammatory response with lymphocytic and plasma cell infiltration and the absence of eosinophils.

There is no single gold standard for the diagnosis of RADS. The diagnosis is likely when there is

**Table 1:** Spirometry parameters at one and four months after the incident

| Parameter | At one month from the incident | | BDRT % increase | At 4 months from the incident[α] | |
|---|---|---|---|---|---|
| | Observed | % predicted | | Observed | % predicted |
| FEV1 | 2.57 (Lit.) | 72 | 14 | 3.17 (lit.) | 88 |
| FVC | 3.74 (Lit.) | 86 | - | 3.96 (lit.) | 90 |
| FEV1/FVC | 68.72 | 83.70 | - | 80.05 | 92.29 |

BDRT=Bronchodilator Reversibility Test, showing an increase in FEV1 over the baseline (14% here) done 15 minutes after inhalation of 400 µg of salbutamol through a metered dose inhaler (MDI). FEV1=Forced expiratory volume in 1 s; FVC=Forced vital capacity; α=Spirometry was done at 4 months from the incident just before performing bronchoscopy. BDRT was not carried out at that time.

acute onset of respiratory tract symptoms such as cough, breathlessness, chest tightness, etc., within 24 hours of exposure to an agent with irritating properties in the atmosphere. However, the symptoms should persist for at least 3 months. Clinical examination may show hyperinflation of lungs, use of accessory respiratory muscles, and wheeze. Lung function may reveal mild obstruction or a significant bronchodilator reversibility response or a positive bronchoprovocation test such as positive methacholine test. However, our patient showed a mild obstruction with an FEV1 of 72% of predicted and a significant bronchodilator reversibility test (14% increase in FEV1 above the baseline) and his spirometry showed an improvement in FEV1 to 88% at 4 months from the incident.

The management of RADS is the same as that for patients suffering from asthma from any other cause.[10] Our patient was managed similar to bronchial asthma. For the first few days, he received intravenous hydrocortisone (100 mg) every 8 hours along with oxygen and salbutamol nebulization. Once his symptoms improved, he was switched to inhaled rotacaps, containing formoterol fumarate (6 mcg) and budesonide (400 mcg).

The prognosis of RADS is highly variable. In some cases, the symptoms may persist for months or even years.[11] In our case, however, the prognosis was better. Our patient's symptoms persisted for 5 months, after which he had only occasional cough, which did not affect his routine work. We have herein described a classic case of RADS due to exposure to porcelain tile dust, the like of which has not been previously reported to the best of our knowledge.

## Conclusion

The present case was RADS as a result of first time heavy exposure to porcelain tile dust, which was diagnosed according to the criteria laid by Brooks et al.[4] Our case report draws attention towards the recognition of this entity; otherwise, most of these patients are wrongly labeled as bronchial asthma by the majority of general physicians.

**Conflict of Interest:** None declared.

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