Case report

Baron Munchhausen's lung function expertise

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\textbf{ARTICLE INFO}

\textbf{Article history:}
Received 29 September 2012
Accepted 31 October 2012

\textbf{Keywords:}
Asthma
Munchhausen's syndrome
FEV\textsubscript{1} (forced expiratory volume in the first second)
Lung function test
Psychosomatic disorder

\textbf{ABSTRACT}

A young anorectic woman suffering from asthma since her early childhood exacerbated without obvious cause and did not respond to treatment. Body plethysmography showed an isolated huge increase of the expiratory resistance. During inspiration, resistance was not elevated. However, in 1 out of 3 successive manoeuvres, expiratory resistance was completely normal. We believe this to be a Munchhausen's case coupled with a proven asthma. The pattern in lung function can be explained by putting the tongue forward into the spirometer's mouthpiece. In the assessment of asthma refractory to treatment, especially in workers in paramedical professions, Munchhausen's syndrome should be considered.

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1. Introduction

Not all that wheezes is asthma. Vocal cord dysfunction, for example, is a syndrome capable of mimicking and complicating asthma. Some cases have been described as Munchhausen's stridor.\textsuperscript{1} Here we present another variant of Munchhausen's syndrome with symptoms of uncontrolled asthma.

2. Case

A 30 year old woman working in a paramedical profession had manifested allergic asthma in early childhood. A skin prick test revealed hypersensitivity to pollen and animal dander. Her childhood was described as extremely traumatic. Dating back to her adolescence, the BMI had always been around 16 kg/m\textsuperscript{2}, pointing at a possible mental anorexia. With mental distress, associated with exams in her professional career, the asthma exacerbated frequently. In previous years, the response to treatment was often unsatisfactory. Last autumn, suffering again from recurrent attacks of breathlessness with no improvement to a course of systemic corticosteroids over a period of two weeks, she was hospitalised.

When being admitted, FeNO (concentration of nitrite oxide in exhaled air) reflecting eosinophilic airway inflammation was normal. The first night she complained of acute difficulties to breathe. Breathing was shallow without prolongation of expiration and wheezing was absent. She did not respond for hours to oxygen supplementation, beta-agonists, and systemic corticosteroids. Body plethysmography showed an immense and isolated increase of the expiratory resistance. Neither symptoms nor FEV\textsubscript{1} improved after reversibility testing with albuterol. However, in 1 (Fig. 1) out of 3 manoeuvres, the expiratory resistance curve normalised completely, while in the other 2 efforts, resistance curves were the same as prior to albuterol.

An exercise test was performed and had to be halted early due to dyspnoea. The flow volume curve prior to and during the spirometry was not concavely-shaped and the ratio of FEV\textsubscript{1}/VC was normal. All volumes were low, corresponding to shallow breathing. Blood gas analysis at rest was normal. When cycling, she hyperventilated: pCO\textsubscript{2} increased and pO\textsubscript{2} decreased. The alveolar-arterial difference of pO\textsubscript{2} (AaDO\textsubscript{2}) remained within the normal range.

Despite a steady reduction and final stop of oral corticosteroids she felt much better within 4 weeks. At discharge, a FEV\textsubscript{1} of 104% predicted could be measured and the flow--volume curve had normalised totally.

Within the stay at our institution, she experienced three episodes of unexplained fever and the CRP only slightly elevated. The blood culture revealed different microbes at each fever episode. All exams, including CT scan of the thorax and abdominal ultrasound, were negative. Within hours after the first dose of antibiotics, the fever vanished.
3. Discussion

Despite cycling of acute breathlessness and phases with normal lung function, we do not believe this to be a case of brittle asthma. Many details indicated to us that the symptoms were produced intentionally by the patient in a Munchhausen’s manner.

In phases of dyspnoea, expiration was not prolonged. Work of breathing did not seem to be elevated; she did not perform pursed lips breathing, which questioned a typical asthma attack. In addition, at least some improvement from beta-agonists and systemic corticosteroids would have been expected in an asthma attack.

The pre-bronchodilator resistance curve in Fig. 1 showed airway obstruction exclusively during expiration. A totally normal inspiratory resistance is atypical for severe bronchial obstruction. This phenomenon was seen in repeated lung function tests. However, for one test, in 1 out of 3 manoeuvres, the expiratory resistance was completely normal. Despite a relevant increase of intra-thoracic gas volume (ITGV), the crossing of the resistance curve with the x-axis gave no hint for trapped air as it normally occurs with the inspiratory part crossing on the left and the expiratory part crossing on the right of point zero.

All the pathologic changes seen in lung function testing can be explained by the tongue being put forward into the mouthpiece of the Fleisch pneumotach exclusively during exhalation. This manoeuvre was impossible when performing the spiroergometry. There, the spirometer is integrated in the mask and cannot be obstructed by the tongue. As expected, in the later setting, bronchial obstruction was not detectable.

The combined findings, including the episodes of fever of unknown origin, reassured us with our diagnosis of a Munchhausen’s syndrome complicating her known asthma.

4. Conclusion

In asthma refractory to treatment, Munchhausen’s syndrome should be taken into consideration. It may mimic asthma as well as complicate a pre-existing asthma. In this case, the fact that the young women worked in a paramedical profession and that she obviously suffered from mental anorexia could have been a hint to an increased likeliness of Munchhausen’s syndrome.

Conflict of interest statement

There exists no conflict of interest.

References

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