



## Case report

## “Exercise induced asthma” is not always asthma

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## ABSTRACT

A 25 year old woman was referred to our center for further evaluation of an exercise-induced dyspnea. Moreover, the patient suffered from hoarseness and recurrent sinusitis and otitis.

After initially finding nothing suspicious, a spiro-ergometry was performed. Interestingly, we saw a relevant limitation of the inspiratory flow-volume curve under maximal exercise load. Further evaluation (in particular the bronchoscopy and the resulting biopsies) led us to the final diagnosis of a granulomatosis with polyangiitis.

After 4 weeks of an established therapy regime with prednisone and rituximab the prior detected subglottic stenosis and the inspiratory flow-volume curve limitation could no longer detected.

We describe a rare differential diagnosis of an exercise-induced asthma and we underline the importance of a multimodal therapy concept. We highlight the critical nature of the flow-volume curve in spiro-ergometry under maximal exercise load. We recommend frequent follow-up control visits to monitor the subglottic stenosis.

A 25-year-old Caucasian woman presented with a 3-month history of shortness of breath, wheezing during exercise, hoarseness and post-nasal drip. She had been treated with combined INHALED ICS/LABA (budesonid/formoterol) for several weeks without improvement of these symptoms. She had no known allergies, no family history of asthma or hay fever. The patient suffered from recurrent sinusitis and otitis and reported an increase in dyspnea during her rugby workout. She reported no epistaxis or hemoptysis and denied chest pain or palpitations. She quit smoking in April 2017 (2 pack years). The patient was referred to our hospital because of ongoing symptoms despite an established asthma therapy. The patient was not receiving any other medication.

Physical examination revealed a fully oriented patient with normal vital parameters and normal nutritional condition. Auscultation of the lungs and heart was unremarkable. In particular, no stridor in rest was detected. A correct inhalation technique was observed.

Laboratory examinations are shown in [Table 1](#).

To determine a significant ventilation limitation, a pulmonary function test (PFT) and arterial blood gas analysis (ABGA) were performed. This revealed normal lung volumes, unaffected diffusion capacity and gas exchange and no flow-volume-curve-limitation ([Fig. 1a](#)).

A bronchoprovocation test with methacholine yielded no bronchial hyperactivity.

To further explore the cause of unexplained dyspnea a spiro-ergometry (CPET) was performed. The result showed above average performance (201 Watt, 149% predicted) with a normal maximal oxygen uptake (98% predicted). Besides a normal alveolar-arterial difference (aADO<sub>2</sub>), there were no hints pointing towards a cardiac limitation as the patient had an unremarkable O<sub>2</sub> pulse and a sinus rhythm without alterations of the conduction system. Noteworthy, however, was the finding at the end of the exercise test where the patient developed an evident inspiratory and expiratory stridor. A correlation to the flow-volume-curve during the maximal burden revealed reduced inspiratory and expiratory flows with a typical oval-shaped curve ([Fig. 1b](#)).

A chest x-ray and CT-scan showed sub-pleural masses in the anterior right lobe at the apex and adjacent to the mediastinum, with no pleural effusion and no mediastinal or hilar lymphadenopathy ([Fig. 2](#)).

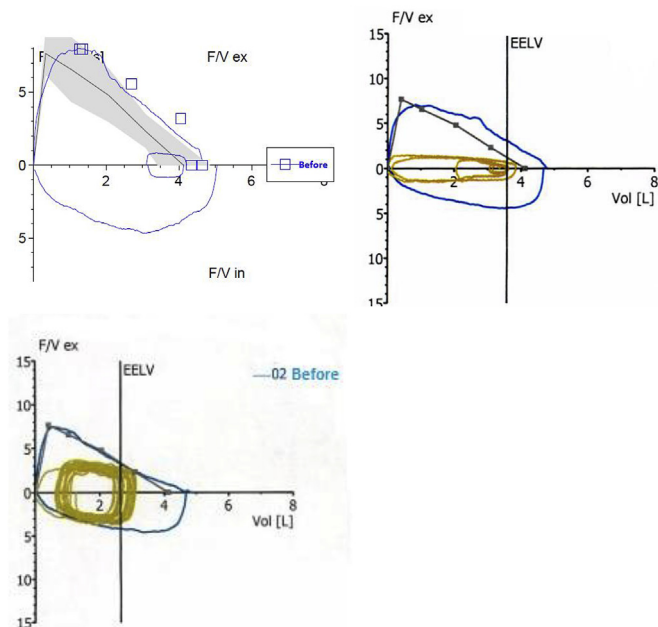
To exclude malignancy, a bronchoscopy was performed revealing a subglottic stenosis ([Fig. 3a](#)). Mucosa biopsies showed basophilic necrosis with epithelioid macrophages and scattered giant cells in the periphery of the necrosis. The adjacent stroma had a mixed inflammation consisting of lymphocytes, plasma cells and some

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**Table 1**  
(Laboratory findings and systemic antibodies).

	Results	Range
Eosinophilic count	$0.918 \times 10^9/l$	[0–0.300] [ $\times 10^9/l$ ]
IgE	128 IU/ml	[ < 100] [IU/mL]
proBNP	26 ng/l	[ < 177] [ng/l]
D-Dimers	< 0.30 $\mu\text{g/ml}$	[ < 0.50] [ $\mu\text{g/ml}$ ]
PR3-ANCA	14 U/ml	[ < 3] [U/mL]
ANCA-titer	1:20	[ < 1:20] [Titer]
Anti-MPO antibodies	< 5 U/ml	[ < 5] [U/mL]
C3c and C4	1.48 and 0.37 g/l	[0.8–1.8] [g/L] and [0.1–0.4] [g/L]



**Fig. 1.** a and b: Four weeks after therapy with a rituximab and a prednisone regime.

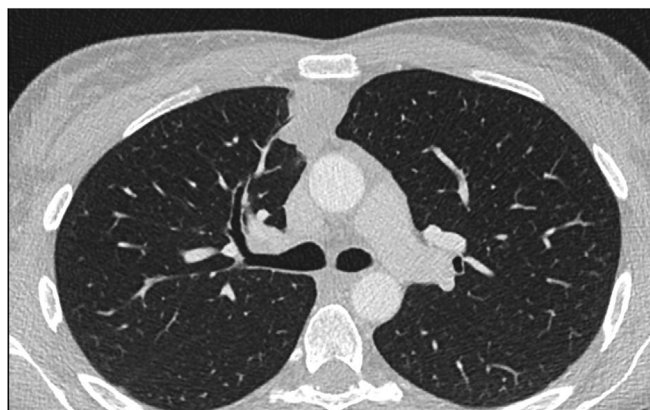
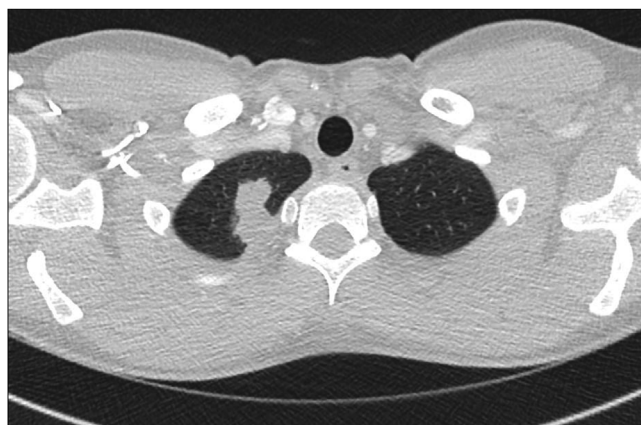
eosinophils. Though the few sampled small vessels had no signs of inflammation, these findings together with the clinical presentation were consistent with granulomatosis with polyangiitis (GPA) (Fig. 4). Special stains for fungi and acid-fast bacteria were negative. Based on the Chapel Hill Consensus Conference Nomenclature of Vasculitides and integrating all findings, we diagnosed GPA. Further findings were diffuse erythema in the bronchi and in the trachea.

Based on the results our working hypothesis was vasculitis, most likely GPA because of the subglottic stenosis.

The results of the systemic antibodies and the complement factors are shown in Table 1.

While evaluating extra-pulmonary manifestations, a kidney involvement with a relevant proteinuria of 1 g daily with glomerular pattern was seen. Active sediment casts could be excluded. A comprehensive otorhinolaryngological examination revealed an otitis media on the left side and a perforation of the septum nasi. A CT-scan of the paranasal sinuses detected a granuloma in the right sinus maxillaris (Fig. 5).

A b-cell-depletion with rituximab and a prednisone regime was established after excluding a relevant immunosuppression, pregnancy or latent mycobacteriosis.



**Fig. 2.** CT-scan.

## 1. Discussion

In our case we describe a rare differential diagnosis of asthma in a young woman. The patient did not respond to asthma treatment prescribed by the general practitioner and was therefore referred for further evaluation. Unremarkable test results, i.e. flow-volume-curves at rest, body plethysmography, exhaled nitrogen oxide and a methacholine provocation test, prompted further investigation with spiro-ergometry.

Patients with shortness of breath can present with different types of airflow-volume-curves depending on whether the ventilation limitation is obstructive or restrictive (Fig. 6).

1. Asthmatic patients typically have a complete or partially reversible obstruction (Fig. 6.1). A complete reversibility, which is more prevalent, can be shown by a normalization of the obstruction after bronchodilation ( $FEV_1/FVC > 70\%$ ). A partial reversibility, found less often, can be shown with a persistent obstruction ( $FEV_1/FVC < 70\%$ ) and an improvement of the  $FEV_1$  of more than 120ml and 20% improvement from baseline.
2. Chronic obstructive pulmonary patients typically have an irreversible/fixed obstruction (Fig. 6.2).
3. A restrictive ventilation limitation is shown with a decreased total lung capacity (TLC) and forced vital capacity (FVC). The  $FEV_1$  is also low because of the reduced lung volume although the  $FEV_1/FVC$  ratio is normal (Fig. 6.3).

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