Case report

Bullous emphysema — Not always nicotine-related!
Unusual distribution of emphysema in a patient with a rare hobby

Camilla Lüttecke-Hecht*, Tim O. Hirche

Department of Respiratory Medicine, Sleep Medicine and Ventilation (Zentrum für Pneumologie, Allergologie, Schlaf- und Beatmungsmedizin), Deutsche Klinik für Diagnostik, Aukammalle 33, 65191 Wiesbaden, Germany

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A B S T R A C T

In a patient admitted for further investigation of haemoptysis and dyspnoea and known emphysema of the lung, a remarkable distribution of emphysematous bullae could be detected on CT-imaging. Further history, besides smoking, revealed apnoea diving-activity during younger adult age. The distinct appearance of partially septated pleura-based bullae lead to the suspicion of a positive-pressure barotrauma of the lungs in the past, now complicated by infection and bleeding. This case highlights the importance of thorough questioning of the patient and underlines the consideration of differential diagnoses of emphysema.

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1. Introduction

The 67 year-old male patient was referred to our department by a respiratory physician due to persistent haemoptysis, he therefore was treated with oral Moxifloxacin over 10 days during a stay in Andalusia/Spain prior to admission. Present complaint was coughing, increased amount of phlegm and halithosis, no fevers, loss of weight or night sweats. Similar symptoms intermittently occurred over the past three years and were successfully treated with antibiotics. A bullous emphysema was first diagnosed three years ago, the patient then suffered of pneumonia and lung abcess. An actual x-ray of the lung showed a prespecified bulla in the right apical lower lobe with an air-fluid-level. The patient quit smoking 15 years ago (regular smoking was started at the age of 36, sporadic smoking before), about 30 packyears.

Past history: Coronary artery disease (2 vessel-disease), implantation of 2 drug-eluting stents prox. RIA and dist. RCA 06/2013 (CABG was refused by the patient), hypertension, and diabetes type II (Insulin-therapy). Medication: Aspirine and Clopidogrel. The patient’s father died of tuberculosis in 1989, tuberculosis skin-testing in the patient back then was reactive.

1.1. Physical examination

171 cm, 84 kg, decreased breath sounds bilaterally.

1.2. Blood testing results

C-reactive protein 1.13 mg/dl (normal max. 0.5 mg/dl), haemoglobin 12.1 g/dl (normal 13.1 – 16.8 g/dl), GGT 127 U/l (normal max. 61 U/l), results within the range: white blood cell count, platelet count, MCV, MCH, glucose, calcium, phosphate, creatinine, GFR, urea, ALT, AP, magnesium, sodium, potassium, total protein, INR.

1.3. ECG

Sinus rhythm, 93 bpm, no axis deviation, isolated negative T in III.

1.4. Echocardiography

Discrete hypertrophy of the basal septum and impaired diastolic function, slight mitral valve regurgitation and tricuspid valve regurgitation, PAPsys 34mmHg + central venous pressure, estimated PA-pressure of 39 mmHg within the upper normal range.

1.5. Chest X-ray (external report)

Predescribed subpleural emphysematous bulla (6.6 × 5.0 × 3.3 cm) right apical lower lobe with increasing cystwall...
thickness and air-fluid-level, sclerosis of the aorta and degenerative changes of the thoracic spine.

1.6. CT scan of the lungs

Marked subpleural bullous emphysema bilaterally (right 7.9 × 3.5 cm; left 4.9 × 1.8 cm) ([Images 1–3]), partially septated on the right side with a discrete fluid-air-level, slightly enlarged mediastinal lymphnodes (max. 7 mm), no inflammatory consolidation, no pleural effusion.

1.7. Lung function testing

Rtot 0.3 kPa s/l (102%), FEV1 2.5 l (84%), VCin 3.0 l (76%), FEV1% VCmax 82%, TLC 5.7 l (87%), RV 2.7 l (108%), RV%TLC 117%, lung function testing within the normal range.

1.8. Diffusion capacity

TLCOcSB 5.4 mmol/min/kPa (63%), TLCO/VA 1.0 mmol/min/kPa/l (79%), slightly impaired.

1.9. Arterial blood gas-analysis at rest on room air

pO2 76 mmHg, pCO2 35 mmHg, pH 7.40, BE -2.0 mmol/l, HCO3- 22.0 mmol/l, within the normal range.

1.10. Arterial blood gas-analysis on exertion on room air (6 minutes walk-test)

Increase of pO2 from 76 mmHg to 86 mmHg, pCO2 37 mmHg, pH 7.42, walking distance 440 m, no hypoxaemia or hypercapnia on exertion.

1.11. Bronchoscopy

Regular endobronchial anatomy, endobronchial tissue atrophic, signs of chronic bronchitis, substantial pussy mucus in the lower lobes bilaterally, no bleeding.

1.12. Bronchoalveolar lavage

Increased content of cells with normal differential percentage, CD4/CD8 ratio normal, cytologically signs of alveolar haemorrhage, flow cytometry normal.

1.13. Microbiological results of the lavage fluid

No isolation of pathogenic bacteria, no proof of mycobacteria microscopically or in cultural growth.

2. Diagnosis

Haemoptysis due to therapy with dual platelet-aggregation inhibitor and superinfected emphysematous bulla.

The patient was treated with Piperacillin/Tazobactam 4.5 g intravenously tds over 7 days. Bronchoscopy with thorough clearance of mucus and secretion was performed, also therapy with nebulised saline and Salbutamol qid, marked improvement hereunder.

Due to the remarkable subpleural distribution of emphysema, the patient again was interrogated. He indicated that he practiced apnoea diving over 15 years in the past up to the age of 35, he spent about five weeks per year on this activity doing harpoon fishing. He declared the maximum diving depth 15 m. Without structured exercise, he was able to hold his breath after initial hyperventilation.
longer than 2 min for his dives. He never had a professional physical assessment for diving-fitness, a diving accident could not be recalled. During childhood and adolescence he practiced endurance running (half-marathon and marathon distance), during regular visits to the public pool he was able to dive distances greater than 60 m.

3. Discussion

Apnoea-diving or free breath-hold diving is long practiced, historical proof exists, reaching back longer than 2000 years (wreck,-sponge,-seashell,- or pearl divers) [1]. In the past years, the popularity of this leisure-time-activity increased, nowadays it is also carried out in professional competitions, including several disciplines regarding speed, depth or distance, and underwater harpoon-fishing is still very common [2]. With regular exercise, the diver is able to increase his tolerance level for high CO2 values, and thereby can extend the time under water. By augmenting the TLC at the expense of reducing the RV, the diver can increase the diving depth, as the negative effects of increasing surrounding water pressure can be partially antagonised by this technique. Depending on the diving discipline, record depths of greater than 200 m and diving-time without breathing support exceeding several minutes can be realised [7]. Three phases in diving can physiologically be distinguished: compression-phase during descent, the surrounding water pressure increases (about 1 mbar every 10 m); isopression-phase with a constant water pressure acting on the body by having reached the desired depth (although the depth during that phase often varies due to investigation of the underwater-environment), followed by the decompression-phase on ascent with decreasing surrounding water pressure. Each phase involves specific health risks resulting from the pathophysiologic effects on the human body [8]. Our patient indicated a maximum diving depth of 15 m, which makes a decompression-illness or negative-pressure barotrauma unlikely. However, a positive-pressure barotrauma of the lungs is also possible in lower diving depths. According to the Boyle-Marriott law, the product of pressure and volume is constant (p \times V = \text{const.}), so with constant temperature, given a defined gas-volume, the relation of pressure and volume is reverse [3]. Therefore, the breathing gas in the lungs, that had been compressed by the surrounding water pressure during the dive, expands again on ascent. A positive-pressure barotrauma can occur, if the air in the lungs can not disperse sufficiently and quick enough due to breathholding on ascent, resulting in an increased positive pressure in the lungs, that can lead to central (mediastinum), or peripheral (pleura) tears causing a pneumomediastinum or pneumomothorax with regards to the lung. As a matter of course, all air-filled organs of the human body could potentially be affected by a barotrauma [4]. To prevent this complication, adequate time on ascent must be allowed for the air to flow off the lungs, therefore a barotrauma often occurs in very fast panic-ascents. An over-extension of the lungs with the above named resulting complications is suspected to be the second most cause of death in divers after drowning [5]. With regards to our patient, pressure-related partial pleural tears with consecutive forming of emphysematous bullae must be assumed (in absence of a recalled diving trauma or incidence), located on that part of the lungs, that is highly exposed to shear stress and pressure variation in the thorax: the outer sheathing of the lungs. Fast ascent, even out of lower water depth, or fast changes in diving depth due to hunting after a fish, could have caused the marked lung changes in our patient. The intermittent cigarette smoking at this time must be taken into account as a contributing factor to the described mechanism, as it causes air trapping (as do bronchial infections with partial or complete mucus plugging in the distal bronchioles), that prevents airflow out of the airways [4]. The distinct distribution of the bullae and the septated appearance support the assumption, that the underlying cause in origin is trauma, rather than emphysematous changes resulting from cigarette-smoking. Apposedly, lung function testing showed normal values except slight impaired diffusion capacity, no further emphysematous changes throughout the lungs could be detected in the CT-scan (as should be expected in long-term cigarette smoking). No medical investigation or x-ray of the lungs took place in the past, so that the existence of the emphysematous bullae at an earlier stage (longer than three years) can only be hypothesised. In the actual context, the origin of the emphysea has no therapeutic consequence, but shows — once again — how important thorough questioning of the patient with regards to the past history could be. Due to recurrent infection of the bullae and preserved lung-function, surgical resection has to be considered intermediate-term, at this stage impossible though, as the patient is under dual platelet aggregation-inhibition and the conservative treatment results were fully satisfactory. Future aim should be close surveillance of the patient and detecting eventual corresponding changes to prevent further complications.

4. Conclusion

- Unusual distribution of emphysea in lung imaging should lead to consideration of differential diagnoses
- Critical consideration and matching of investigational and imaging results, as well as symptoms, should be of paramount importance
- Every respiratory physician should be familiar with diving physiology and come to terms with the particularities of this increasing popular sparetime-activity
- Surgical resection of localised emphysea as a therapeutic option should be critically discussed

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