WHAT CAN HIDE BEHIND AN ASTHMATIC PERSON

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Abstract: We present the case of a male patient, 37 years old, who for two weeks had complained of right stabbing thoracic pain, cough with sero-mucous expectoration, 38°C body temperature and dyspnea at exercise. He had taken temperature-lowering drugs, but without effect. Seven years before, he had been diagnosed with bronchial asthma, for which he was treated with a combination of inhalation corticosteroids and long-term action bronchodilators for about 3 years, with good evolution. He discontinued chronic asthma therapy for 2 years, but he was recommended to take Ventolin whenever necessary; he was free of bronchial obstructive bouts for about 2 years. Chest auscultation evidenced basal crackles (rales), while chest X-ray revealed a tendency of right basal condensation at the level of the posterior segment. The diagnosis was right basal pneumonia and therapy included antibiotics, muccolitic agents, and antipyretics. Evolution was good during the treatment and for another 2 weeks, when suddenly his condition worsened and was readmitted to the hospital in a more severe state than the first time. Differential diagnosis included: pulmonary neoplasm, pulmonary tuberculosis, pulmonary abscess. Complex investigations eventually elucidated the diagnosis.

Keywords: asthma, lung abscess, fever, bronchiectasis

Case Report

We are presenting the case of a patient, aged 37 years, coming from an urban environment, who was first seen in December 2012 in the Medical Clinic I, with the following complaints: chest pain, productive cough, sero-mucous expectoration, dyspnea during exertion, 38°C fever and chills. During adolescence, he had presented repeated acute upper respiratory tract infections and had been diagnosed with asthma back in 2005. He currently works as a salesman and has been smoking around 10 cigarettes per day during a time span of 20 – 30 years. He has not been smoking since 2005 though, and admits using alcoholic beverages occasionally.

Two weeks ago, the patient suddenly accused an episode of chest pains, productive cough with sero-mucous expectoration, 38°C fever, chills and dyspnea during exertion. He later followed antithermic therapy but without any positive results. After having been diagnosed with asthma back in 2005, he had started a 3-year-long therapy with CSI/LABA, with positive evolution. For the past 2 years, though he had not been using any sort of medication for chronic asthma, and, although he was recommended treatment with Ventolin as needed, he did not take it. He had not been accruing asthma broncho – obstructive seizure.

After admission, the physical examination evidenced 38°C fever, BMI 28.65 kg/m², well represented adipose tissue, anatomically correct thorax, rhythmic, symmetrical, synchronous breaths, 16/min, physiological sounds, normal sonority of the chest at percussion, basal crackles on the right hemithorax, BP = 120/70 mmHg, HR = 88 b/min, without any other pathological changes.

The stage diagnosis after the first examination was: febrile syndrome, basal pneumonitis on the right, intrinsically intermittent asthma and overweight.

The most significant laboratory tests evidenced: lymphopenia = 23.2%, mononcitosis = 11.4%, eosinophilia = 5.7%, inflammatory syndrome with ESR = 57 mm/h, fibrinogen = 454.8 mg/dl, CRP – 48, IgG = 2.492 mg/dl, hyposideremia (Fe = 25 mg/dl). No pathogen germs were revealed in the sputum – Gram stain smear: epithelial cells, polymorphic flora.

Consequently, the positive diagnosis included: basal pneumonia on the right, mixed ventilation dysfunction,
hyposideremia of infections cause, intrinsically intermittent asthma, overweight.

The patient received treatment with 500 mg Zinnat, 2x1 tb/day, Bromhexin 3x1 tb/day, Paracetamol 500 mg 3x1 tb/day, Ketocanazole 200 mg 1 tb/day, but after three days there were no positive developments, with persistent symptoms, so the treatment was changed: Fortum 1 g 2x1 fl/day, Bromhexin 3x1 tb/day, Paracetamol 500 mg 3x1 tb/day, Ketocanazole 200 mg 1tb/day for 10 days, with positive evolution. After 3 days of Fortum administration, the patient presented expectoration, in smaller quantities but without fever or chills, overall the general condition was improved. IDR at PPD was conducted = 8 mm at 72 h (07/12/2012) and culture for BK. We asked for advice from the lung diseases clinic (10/12/2012), the diagnosis was: bilateral pneumonia mostly on the right. Tuberculin allergic hypersensitivity. Asthma with mixed ventilation dysfunction, with Vmax of 43%, with the following recommendations: sputum examination, cyto-bacterial + BK, the continuation of hematological and immunological investigations, continuous treatment with Fortum and Ciprinol 1g/day, reevaluation needed.

The patient was discharged with the following recommendations: to avoid cold, to return after the validation vaccination, reevaluation after one month: radiological, biological, immunological and spirometry, Ventolin, 2 puffs as needed (when using > 2 puffs of Ventolin/month, he should return for reassessment).

Two weeks after completion of therapy, the patient showed a good general status, without complaints, but later on he accused fever and rare productive cough. He self-administered 500 mg Ciprinol, 2x1 tb/day, 5 days without complaints during therapy, but at the end the symptoms reappeared: mild fever and cough with muco-purulent expectoration.

The patient came for a second hospitalization, accusing cough with muco–purulent expectoration, fever 37.6 to 38.4° C, affected general condition, marked asthenia, dyspnea during longer walks. During physical examination, the following observations were made: basal crackles, right basal bronchophonia, AV = 88 b/min, BP = 125/75 mmHg. Chest X-ray revealed widened lung interstitium. A right basal tissue formation of 54/52 mm was highlighted with well defined uneven structure, which raises the suspicion of a lung abscess (negative evolution aspect) (figure no. 1).

**Figure no. 1.Chest x-ray – suspicion of a lung abscess**

We asked for a native thoracic MDCT examination on continual sections (19/2013) which showed: pulmonary condensation area at the medium right pulmonary lobe, with air leakage bronchograms, bronchial dilatation with bilateral basal sacciform aspect, thickened wall and low parathuid content, pretracheal mediastinal lymphadenopathy, infracarinal location and in the aorto-pulmonary window, measuring up to 2.7 cm, without pleural effusion, no sub-diaphragmatic pathological changes (figure no. 2).

**Figure no. 2. Native thoracic MDCT examination**

*Conclusion:* bilateral posterior basal bronchiectasis, medium right pulmonary lobe condensation and mediastinal lymphadenopathy. Other laboratory examinations (10/01/2013) showed: monocytosis (1.14 10³/ml), lymphopenia (24.1%), hypoalbuminemia (40.8%), high alpha1-globulin (4.2%), hypergammaglobulinemia (29.9%), inflammatory syndrome (fibrinogen = 475.4 mg/dl, ESR = 57 mm/h, CRP = 63.8 mg/dl), C3 > 248 mg/dl, hyposideremia (Fe= 27.3 mg/dl). The pharyngeal exudate and sputum did not isolate any pathogens, in the Gram smear sputum, integral and destroyed leukocytes were found, rare red blood cells, mixed flora associated. The urine analysis revealed leucocyturia (WBC 25), but urine culture was found negative < 1000 bacteria/ml.

Causes of pulmonary cavity lesions may be:

1. Lung abscess: - with anaerobic bacteria: Gram-negative bacilli (Fusobacterium, Prevotella, Bacteroides), Gram-positive cocci (Peptostreptococcus), Gram-positive bacilli (Clostridium, Actinomyces).
   - aerobic bacteria: Gram-positive cocci (Streptococcus milleri, Staphylococcus aureua), Gram-negative bacilli (Klebsiella pneumoniae, Pseudomonas aeruginosa, Burkholderia pseudomallei), Gram-positive bacilli (Nocardia).
   - mycobacteria (M. tuberculosis, Avium cellular kansi),
   - fungal (histoplasmosis, aspergillosis, coccidioidomycosis, sporotrichosis, blastomycosis, cryptococcal infection, muccormycosis, sporotrichosis, infection with Pneumocystis carinii)
   - parasitic (Paragonimiasia, hydatic infection, amoeba infection)
2. Bronchiectasis

We also asked for advice from the Clinic of Infectious Diseases (10/01/2013) Dg: Right basal lung abscess. Recommendations Imipenem 4x1 fl/day, Vancomycin 2x1g, Fluconazole 150 mg 1/day.

We determined the pulmonary tumour markers:

- squamous cell cancer antigen that increases in squamous cell lung cancer, the prevalence of elevated SCC varies between 39 and 78%, SCC = 0.66 ng/ml (NV<1.5)
CLINICAL ASPECTS

- CYFRA 21-1 = 0.99 ng/ml (NV<3.3), cytokertatin fragment increases in non-small cell lung cancer
- Neuron – specific enolase NSE = 20.5 ng/ml (NV<16.3), characteristic for small cell lung cancer, is an important prognostic marker, the positive predictive value is 92%, but searching the literature, patients with this type of cancer have very high values >400 ng/ml.

The positive diagnosis was: Right basal lung abscess. Bilateral bronchiectasis. Hyposideremia of infectious cause. Intrinsically intermittent asthma. Overweight.

Predisposing factors for bronchiectasis are:
- Bacterial infections (Pseudomonas aeruginosa, Haemophilus influenzae, Moraxella catarrhalis, Klebsiella, Staphylococcus aureus), fungal (Histoplasma capsulatum), mycobacterial (non tubercular mycobacteria), viral (adenovirus, influenza, herpes simplex, measles)
- Congenital: alfl-antitrypsin deficiency, defects in the cilia (bronchiectasis, sinusitis ± infertility ± situs inversus), cystic fibrosis (defect in the transport of Na and Cl)
- Immunodeficiency: primary (hypogam, IgG subclass deficiency [IgG2, IgG4] chronic granulomatose diseases, C deficits), secondary (immunosuppressants, HIV infection)
- Airways obstruction: extrinsic compression (tumoral mass or lymphadenopathy), foreign body (aspirated), neoplasia (endobronchial lesions), mucoid impact (allergic bronchopulmonary aspergillosis), postoperative (after lobular resection)
- Structural congenital defects: lymphatic (yellow nail syndrome), tracheo – bronchial (tracheo – broncho megaliths, cartilage deficiency), vascular (pulmonary hypertension, thrombosis, syndrome), tracheo – broncho (tracheo – broncho megaducts, septa, ring, polyp), congenital (allergic bronchopulmonary aspergillosis)
- Inhalation of toxic substances (direct injury alters the structure of mucous, or sequestration)
- Inhalation of toxic substances (indirect injury alters the structure of mucous, or sequestration)
- Miscellaneous: inflammatory bowel disease (intestinal resection may exacerbate pulmonary disease), transplant (may be secondary to frequent infections due to immunosuppression).

The patient followed a therapy with Imipenem 4x1 tb/day, Vancomycin 2x1 tb/day, Fluconazole 150 mg 1 tb/day 7 days, with favorable evolution, with indications of performing bronchoscopy with broncho-alveolar lavage and biopsy at the Pneumo-Phthisiology Clinic in Cluj. The examination described free trachea, normal bronchial tree, tracheal spur, with moderate mucous-purulent secretions. Free BPS, LSS, rounded spurs with LIS inflammatory bronchial aspect and moderate mucopurulent secretions. Dg: Overinfected bilateral bronchiectasis. Round LID outbreak or exobronchial tumor, for example peribronchonecrotic fibrosis, post-pneumonia. Then the patient took Erdomed 2x1 tb/day 10 days, Clindamycin 4x1 f/d/day 10 days, then 600-mg tb 20 days, and Metronidazole 2x1 f/d/day 10 days then 100 mg 3x1 tb/day 20 days, Fluconazole 1 tb/day, a month with continuous monitoring of the intestinal transit. Lung biopsy (01/18/2013) described pseudostratified cylindrical epithelium on the bronchial wall, with hyperplastic areas covering a rich stroma and mucoscatent glands heavily infiltrated by leukocytes, which permeates the surface epithelium, concluding a chronic bronchitis; no fungi were isolated and the bacteriological examination was negative. We made a biological review at the end of therapy to see if there were changes; we highlighted hyperuricemia (uric acid = 8.6 mg/dl), hypertriglyceridemia (triglycerides 200 mg/dl). We then performed a CT reassessment, at the end of therapy, which evidenced bronchial bilateral hilobasal dilatation, with saciform aspect, with thickened wall and decline fluid, an area of consolidation on the right LM, in resorption, with significant DML bronchial dilatation, pretracheal mediastinal lymph nodes in the aorto-pulmonary window and infracarinal, with sizes of up to 2 cm (in moderate dimensional reduction). No pleural effusion. No pathological subdiafragmatic changes. Conclusion: LMD level and basal posterior bilateral bronchiectasis, LMD pulmonary caudensation in resorption, moderate sized mediastinal lymph nodes.

We advised the patient to follow treatment with Clindamycin 1 tb 600 mg/d, Metronidazole 100 mg tb 3x1 tb/day, Fluconazole 1 tb/day, Isoprinosine 1 tb/day 10 days/month for 3 consecutive months, and to carry out a CT examination after 3 months, anti-influenza vaccination, anti-pneumococcal vaccination, anti-Haemophylus influenzae vaccination, to make prevention of exacerbations (oral antibiotics: Ciprofloxacina 2x500 mg/day or Gentamicin in aerosols). In case of exacerbation, to follow the aggressive management of exacerbations with antibiotics active against H. influenzae, P. aeruginosa, M. catharallis, S. aureus and S. pneumoniae.

We also drew attention on the efforts to facilitate the removal of phlegm from the airways, via postural drainage, chest percussion, pneumatic vests, auto-drainage, positive expiratory pressure devices, intrapulmonary percussive ventilator that can cause the spread of infection to other bronchi, leading to outbreaks or other acute obstructions.

Short – term evolution will be positive, under specific antibiotic treatment, risk of bowel dysbiosis with diarrhea, and possible, long-term, total resorption of the lung abscesses, bronchiectasis exacerbation with the recurrence of the lung abscess or multiple abscesses, possible segmental lobectomy or pneumonectomy.

We presented this case because of the presence of bronchiectasis in the case of a young patient, with no immune deficiency that required multidisciplinary collaboration.

REFERENCES