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SARCOIDOSIS BETWEEN SUSPICION AND CERTITUDE DIAGNOSIS

NICOLETA ARIESANU, DOINA TODEA

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Classical definition of sarcoidosis is as a multi-system disease of unknown etiology, usually affecting the respiratory tract and other organs, and characterized by the formation of nonnecrotizing epithelioid granulomas in affected tissues.

The first step in diagnosing sarcoidosis is a good systemic evaluation.

Even if the things look very simple, there are no conclusive morphological features that enable the pathologist to make a doubtless diagnosis of sarcoidosis and the expressions as "suggestive of sarcoidosis" are not always helpful. The epithelioid granulomas seen in the affected organ with sarcoidosis are similar to those in other diseases such as tuberculosis, fungal diseases, other autoimune granulomatosis and some malignancy.

The differentiation between each one of them requires a certain combination of multiple diagnostic tests. The major objective of these tests is to exclude or to prove the presence of an infectious, tumoral, or immunogenic entities on the one hand, and to characterize the radioimagistic, imunologic and if is possible genetic profile of the sarcoidosis affected patients on the other.

Only thus may one accurately differentiate between sarcoidosis and sarcoid-like reactions and establish a clear diagnosis.

MANAGEMENT OF PATIENTS WITH COPD AND DIAGNOSTIC STANDARD

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Chronic obstructive pulmonary disease (COPD) is a leading cause of death and disability internationally.Alveolar hypoxemia and consequent hypoxemia increase in prevalence as disease severity increases.Ventilation/perfusion mismatch resulting from progressive airflow limitation and emphysema is the key driver of hypoxia, which may be exacerbated by sleep and exercise.

Uncorrected chronic hypoxemia is associated with the development of adverse sequelae of COPD, including pulmonary hypertension, secondary polycythemia, systemic inflammation and scheletal muscle dysfunction.

A combination of these factors leads to diminished quality of life, reduced exercise tolerance, increased risk of cardiovascular morbidit, and greater risk of death.

Long term oxygen therapy has been shown to improve pulmonary hemodynamics, reduce erythrocytosis, and improve survival in selected patients with sever hypoxemic respiratory failure.

ALPHA-1 ANTITRYPSIN DEFICIENCY: AN UNDER-RECOGNIZED CAUSE OF CHRONIC OBSTRUCTIVE PULMONARY DISEASE

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The main risk factors for the occurrence of COPD include alpha1-antitrypsin deficiency (AAT), which is frequently underdiagnosed. The prevalence of AAT deficiency in patients with COPD is 2-3%. All patients with COPD and incompletely reversible airflow obstruction should be routinely screened for AAT deficiency (ERS/ATS standard).

AAT deficiency is an autosomal, codominant, hereditary disorder characterized by decreased serum level of alpha-1antitripsine. AAT is synthesized in the liver having an essential role in the protection against neutrophil elastase, an enzyme responsible for the massive destruction of pulmonary tissue, with the occurrence of emphysema. There are approximately 100 alleles whose mutations cause mild, moderate or severe AAT deficiency. The most common mutant AAT allele are: Pi*M, Pi*S, Pi*Z, in which Pi*ZZ produces the most severe manifestation of the disease.

Normal serum AAT value: 150-350 mg/dL (20-48 μM)

AAT deficiency is underdiagnosed worldwide: only 5% of individuals with PI*ZZ AATD in the U.S. are diagnosed, which means 5,000 of about 80,000-100,000 PI*ZZ. The average time until diagnosis of COPD with AATD is 7.4 years. AAT deficiency is underdiagnosed because the clinical differentiation between AATD and typical COPD and asthma is difficult, pulmonary symptoms of AAT deficiency being similar to those of typical COPD. Diagnosis of AAT deficiency can be easily achieved using a simple test dosing plasma AAT values, later in the case of low values requiring genotyping and phenotyping.

Management of patients with COPD and AATD is different from the ordinary management of the patient with COPD, involving changes in lifestyle, genetic counseling, augmentation therapy. Augmentation therapy improves long-term evolution of the disease, slowing the annual decline in FEV1, loss of lung tissue, and decreases mortality.

PATHOGENETIC MECHANISM OF NON-STEROIDAL ANTIINFLAMMATORY DRUGS-INTOLERANCE IN BRONCHIAL ASTHMA

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Bronchial asthma with intolerance to aspirin (ASA) was described almost 100 years ago, but still we do not know exactly its pathogenesis. An adverse reaction to ASA and other non-steroidal anti-inflammatory-drug (AINS) may be caused either by type I hypersensitivity with specific IgE, either by the inhibition of cyclo-oxygenase-1 (intolerance mechanism) (Szczeklik's theory) (in the early '70).

IgE mediated hypersensitivity is limited to a single AINS (or to more drugs, but with very similar chemical structure). Intolerance has been noted to many non-related AINSs. Finally, a profound inflammation of the airways and sometimes also in the skin is developed and it may also involve the bradykinin pathway.

A new hypothesis of the intolerance mechanism is that of the complement system with high anaphylatoxins and high C3-convertase (proteomics arguments brought by Lee et al.) (2006). Also the viral hypothesis (human rhinoviruses) is discussed.

Independent of the mechanism, it is possible to induce tolerance to ASA, by the technique of "desensitization", in patients in which ASA is needed as cardio-vascular prophylaxy.

PHYSIOPATHOLOGY OF CENTRAL SLEEP APNEA SYNDROME AND CHEYNE-STOKES RESPIRATION: FROM DIAGNOSIS TO TREATMENT

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Central sleep apnea syndrome (central SAS) is defined by the absence of respiratory flow accompanied by the absence of thoraco-abdominal respiratory movement in more than 50% of the respiratory events recorded, desaturations being less severe or absent compared to obstructive sleep apnea syndrome.

The presentation aims to answer the most important questions regarding central SAS. Central sleep apnea syndrome is divided into two major types regarding the pathophysiological mechanism underlying the syndrome: central SAS in context of hypoventilation/hypocapnia encountered in patients with kyphoscoliosis, narcotic intoxication, neurological and neuromuscular diseases, central alveolar hypoventilation (Ondine syndrome); also central SAS in context of hyperventilation/hypocapnia encountered in patients with idiopathic central SAS, high altitude central SAS, central SAS due to opioid intake, heart failure and neurological diseases, the last two pathologies being frequently associated with Cheyne-Stokes Respiration (periodical crescendo-descrescendo respiratory pattern).

Heart failure (HF) leads to central SAS in~50% of patients. The presentation details the pathophysiological mechanisms, the prevalence and also the risk factors leading to the appearance of central SAS in patients with HF. In the initiation of the treatment several steps must followed, the most important aspect being treating the underlying disease which causes central SAS.

The gold-standard treatment is noninvasive ventilation (BiPAP S/T, Adaptive Sero-Ventilation), assessment of the type of noninvasive ventilation is performed during the manual titration night. At the end of the presentation a series of clinical cases will illustrate the theoretical concepts presented.

CARDIOVASCULAR RISK ASSESSMENT AND VARIOUS WAYS TO EXPRESS THE SEVERITY OF OBSTRUCTIVE SLEEP APNEA SYNDROME

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Clinically significant Obstructive Sleep Apnea (OSA) are increasingly recognized as a risk factor for cardiovascular pathology, the main involved mechanisms being represented by intermittent hypoxemia, sympathetic activation and systemic inflammation.

The evaluation of OSAS severity by cardio-respiratory poligraphy/polisomnography (expressed as Apnea Hypopnea Index/Oxygen Desaturation Index) may not always be the best way to assess the future cardiovascular risk, significant predictive factors could be represented by: nocturnal oxyhaemoglobin levels (assessed by overnight continuous pulse-oxymetry), the evaluation of pulse transit time (PTT) or pulse propagation time (PPT), methods still requiring validation in larger clinical trials.

The presentation will briefly analyze the validated methods as well as some new ones utilized (in research purpose yet) for the assessment of cardiovascular risk in patients with OSAS.

RESPIRATORY DISEASES THAT MAY DEVELOP OBSTRUCTIVE SYNDROME

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A number of respiratory diseases may develop obstructive syndrome (on the large or on the distal airways). Often these conditions can be labeled as classical obstructive disorders (asthma or COPD) given the high frequency of atopy and chronic smoking.

The large airways obstruction can evolve with "asthma-like" syndrome in extrinsic compression (thyroid diseases or mediastinal enlarged lymph nodes), intrinsic obstruction (inhaled foreign body, glottic edema, tumor or infectious diseases of the larynx), tracheomalacia. Sleep apnea syndrome causes upper airway obstruction during sleep. Smaller airway obstruction can be produced besides COPD and asthma by other diseases with restrictive dysfunction or mixed ventilation disorder (bronchiolitis, tuberculosis, tumors, foreign bodies, sarcoidosis, pneumoconiosis, diffuse fibrosis, pulmonary embolism, obesity).

Uncontrolled heart failure syndrome may be due to "asthma-like" symptoms predominantly nocturnal or during exercise. In the diagnostic approach several diseases listed above will be excluded by history, clinical examination, ENT, appropriate imaging investigations (radiography, chest CT, ultrasound), biochemistry, sleep investigation, special respiratory functional tests (spirometry, plethysmography, diffusion lung capacity, cardiological examination, ECG or cardiopulmonary exercise test).

PULMONARY DAMAGE IN COLLAGEN DISEASE: MECHANISMS , RADIOLOGICAL PATTERNS, DIAGNOSIS, MONITORING AND PROGNOSIS

MILENA ADINA MAN, RUXANDRA RAJNOVEANU, BIANCA DOMOKOS, VASILE MURESAN, MONICA POP

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Pulmonary damage in collagen disease is common and is responsible for the significant increase in morbidity and mortality of these diseases.The pathogenesis of lung damage is poorly understood, but demonstrates damage of several components of lung structure (interstitium, airways, pleura or vascular disease).

Identification of pathogenetic mechanisms involved is relatively simple when one compartment is affected, but becomes difficult when affected several aspects of lung struture. Interestitial abnormalities are characterized by lower forced vital capacity (FVC), peak expiratory decrease, flow expiratory volume decrease (FEV) and altered diffusing capacity or transfer factor of the lung for carbon monoxide (TLCO). If there is predominant vascular anomalies it is characterised by decreased TLCO and KCO lower transfer constant (DLCO/Vol alveloar) but have normal lung volumes.

Extrapulmonary restrictive syndrome (inspiratory muscle involvement or aiming the chest) is identified with decreased lung volumes but normal TLCO and increased KCO. Emphysema and airway obstruction in smokers and patients with connective tissue disease may mitigate the restrictive abnormalities of lung volumes , but the decline TLCO and KCO is still present. In the absence of emphysema, a disproportionate decline TLCO value compared with lung volumes or increased interstitialdescribed on HRCT may be associated with the presence of vascular disease.

Although there are many possible changes in the lung there is a more common radiologic pattern for each connective tissue disease , although sometimes interstitial syndrome may be the first disease sign. 25% of idiopathic pulmonary fibrosis occurr in the context of an undifferentiated connective tissue disease. Evaluation of lung damage, assessing the severity and progression of disease is a major cause of morbidity, decreased quality of life and an important cause of mortality.

Identifying the subgroup of patients receiving effective treatment improves quality of life and significantly increases their survival.

RECENT ADVANCES IN RESPIRATORY SOUND ANALYSIS

SORIN C. MAN

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The science of (pulmonary) auscultation began with the invention of the stethoscope by Laënnec. However, auscultation is subject to a variety of limitations: terminology problems, observer variability, and inadequate understanding of the pathophysiology of respiratory sounds. Several studies suggest that the level of agreement among physicians for the type of respiratory sound present using a stethoscope is poor.

This inability to identify respiratory sounds accurately can lead to a wrong diagnosis. But now, lung sounds can be studied objectively. Computerised acoustic analysis is a technique used to evaluate the respiratory sounds and guidelines have been published to standardize terminology. The equipment is portable and measurements can be made at the patient bedside.

THE LUNG AND THE HEMATOLOGIC DISEASES–A COMPLEX CONNECTION

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The lung is oftern severely affected during the course of hematologic disorders. Pulmonary complications occur in up to 50% of patients with hematological disorders, with considerably influence on morbidity and mortality. The distinctive anatomical structure and function of the lung parenchyma (interactions between air spaces and capillary bed-gas exchange units) may render localized parenchymal damage clinically relevant due to the injury induced by the pathogens and to their interactions with inflammatory cells. In the lung parenchyma a variety of inflammatory cells whose precursors are in the bone marrow pass through, park in, proliferate, and release microbicidal and cytohistotoxic substances.

Constitutive parenchymal lung cells (bronchiolar and alveolar epithelial cells, endothelial cells, "interstitial" cells) may be a distinctive target for toxic substances or may have an important part in the inflammatory/reactive and reparative processes after an injury event. Polymorphonuclear cells, macrophages and lymphocytes produce cytokines and growth factors, responsible for the majority of the clinical effects in response to infections (Pneumocystis carinii, cytomegalovirus, etc.), and to certain drugs or to radiation. Pathogenic agents are allowed to reach the lung very easily through either or both the airways and the vascular bed and accumulate there in large amounts.

Inflammatory/immunologic reactions may be particularly weak or, on the contrary strong, in the lungs either spontaneously or due to toxic action of drugs and radiation or to the immunodeficiency induced by hematological disorders. Hematological disorders may harbor in lung parenchymal structures at the onset (i.e., lympho-/myeloproliferative disorders primary in the lung) or during the disease course.

Progress has been made in understanding the pathogenesis of lung diseases, mostly in in conditions of severe immunosuppression such as bone marrow transplantion, acquired immunodeficiency syndrome or leukemia. Such a complex interaction needs a practical diagnostic approach to these pulmonary complications.

STABLE COPD AND CURRENT GUIDELINES

FLORIN MIHĂLŢAN

Institute of Pneumology, Bucharest ”Marius Nasta”

In a world where pulmonologists are faced with multiple challenges from the increasing prevalence of COPD, there is a neglect of the real progress regarding the strategic plan, management and treatment of stable COPD.

However they are and guidelines for the practitioner in the attempt to discipline thinking. We will review the real progress made by the teams that created these guidelines and emphasize small but sometimes eminent steps taken on all the continents. Gold was historically the protocol that has changed the concept and went from classifications based on spirometry to those based on the risk of exacerbation.

Other guides have launched other adjustments COPD therapy on symptoms , on the problems of differential diagnosis and the therapeutic novelties that we will review.

I will speak about NICE guideline, the Catalonia guideline, the Japanese, Canadian, Australian one, etc. . At the end of the presentation there will be some conclusions about the direction, the future that will build on the approach of stable COPD.

TYPICAL AND ATYPICAL FOUNDING OF PULMONARY FUNCTION TESTING FOR DIFFUSE INTERSTITIAL LUNG DISEASES

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Diffuse interstitial lung diseases (ILD) are usually characterized by a restrictive ventilatory defect, i.e. a reduction in lung volumes with preserved respiratory flows, together with a similar decrease in the gas diffusion through the alveolar-capillary membrane, expressed by transfer factor of the lung for carbon monoxide (DLco). However, sometimes in early stages of ILD, lung volumes and DLco may be within the normal range. Also, not infrequently it was noticed a discrepancy between little/no modified spirometry parameters and much lower lung gas transfer, a situation that allows us the assumption of a pathogenic hypotheses and accordingly, the guidance of the final diagnosis. So DLco represents the most useful tool for detection or exclusion of the early interstitial lung damage in connective tissue diseases. The occurrence of pulmonary hypertension in PID evolution can induce a disproportionate reduction in DLco relative to lung volumes. Not least, lung volumes can be relatively preserved in smokers with idiopathic pulmonary fibrosis, possibly due to coexisting emphysema, although the Tiffeneau index remains normal. Moreover, in sarcoidosis and in histiocytosis X evidence of airflow obstruction is seen in more than a quarter of the patients. These data suggest that using restrictive lung function as an exclusive diagnostic biomarker for ILD is neither sensitive, nor specific diagnostic tool.

All patients with ILD must be monitored for disease progression, respectively for identification of possible complications at 3-6 months intervals, or even shorter, if clinical evolution indicates it. Measurements of forced vital capacity (FVC) and of DLco, and also of 6 minute walk distance, are clinical tests easy to perform, the results are measured immediately and they require a minimal effort from the patient, therefore they represent functional parameters used mainly in monitoring the evolution of patients with ILD. It seems that longitudinal changes of these functional parameters present a better prognostic value than baseline values. Nevertheless, since pathological processes coexist in ILD, complex functional patterns can be identified, thus, monitoring only few parameters could often be misleading. Therefore, a stronger predictor of mortality than individual measurements of lung functions appears to be the physiological composite index.

In conclusion, a complex and serial pulmonary function testing represents a useful tool to support the diagnosis, but especially in predicting and monitoring the spontaneous and/or under treatment evolution of ILD.

PULMONARY INFLAMMATION, AN UNDERESTIMATED CARDIOVASCULAR RISK FACTOR

IOANA MUNTEANU, ANA MARIA SALARY, VIRGINIA BOBOC, FLORIN MIHALTAN

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Cardiovascular and chronic lung disease represent 60 % of all deaths worldwide. The common risk factors identified are: smoking, air polution, infectious agents. Epidemiological data have shown that patients with COPD have an increased risk of heart attack or stroke in the first 5 days of infectious exacerbation (especially viral).

Studies in recent years have tried to identify a causal relationship in this respect or just the presence of an epiphenomenon. Chronic inhalation of small particles that penetrate the alveoli accelerates coronary atherosclerosis dyslipidemia regardless of the topic.

Maintaining neutrophilic inflammation in the lungs can cause accidents that are secondary such as acute coronary plaque rupture. It is therefore necessary to establish a collaboration between specialties and patients exposed to smoking, particles resulting from combustion etc. be screened for both diseases.

Achieving targeted therapies in this subset of patients remains an open challenge.

PULMONARY FUNCTION TESTING AND ITS ROLE IN THE MANAGEMENT OF GRANULOMATOSIS WEGENER

ROXANA MARIA NEMES, FLORIN MIHALTAN

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Pulmonary function testing it is very important in the clinical management of patients with Wegener's granulomatosis,. Reduced lung volumes and diffusing capacity occurred frequently, also abnormality in obstruction to airflow can find.

Spirometry, bodyplethysmography, diffusing capacity, oximetry should be performed as soon as possible to identify abnormalities of functional respiratory parameters.

Decreased diffusing capacity for CO (DLCO) is a common finding in Wegener, but in alveolar hemorrhage, the single-breath diffusing capacity is increased. Monitoring of lung function during treatment using serial measurements revealed decrease in lung volumes, airflow obstruction improved or stabilized, a reduction of diffusing capacity was common.

Routine pulmonary function tests may be a useful tool in the staging of patients with Wegener's granulomatosis and in following responses to therapy.

RESPIRATORY DISEASES ASSOCIATED WITH INFLAMMATORY INTESTINAL DISEASES

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The respiratory and gastrointestinal systems share several functional similarities. In health, the two systems remain structurally distinct and functionally integrated, so as to maintain physiological homeostasis. However, in conditions of disease, pathophysiological alterations in one system may be reflected in the other.

This article focuses on the respiratory involvement in inflammatory bowel diseases.

Extraintestinal manifestations of both Crohn's disease and ulcerative colitis (UC) have been well described in the last 40 years, although pulmonary findings are often overlooked because they are thought to be rare.

Respiratory involvement in IBD is seen with some pathophysiological mechanisms: both the colonic and respiratory epithelia share embryonic origin, both types of epithelial cells include goblet cells and submucosal glands, and both the lungs and gastrointestinal tract contain submucosal lymphoid tissue.

This similarity of the immune system causes similar pathogenic changes. Pulmonary involvement is often asymptomatic and detectable only by lung function investigation and/or imaging findings.

Using bronchoalveolar lavage we can find early signs of lymphocytic pulmonary alveolitis, and early signs of bronchiectasia can be found using radiological exams/CTs especially after bowel resections. Respiratory diseases in patients affected by IBD can be often classified as a) -"airways disease" such as tracheobronchial stenosis, bronchiectasia, chronic bronchial suppuration, chronic bronchitis, COPD, asthma and bronchiolitis and b)- "parenchymal disease" such as interstitial lung disease, localized interstitial fibrosis, necrotic nodule and eosinofilic pneumonitis.

The airway inflammation may not always be detectable by routine PFT and only an increase in bronchial hyper-responsiveness together with high IgE levels may be found associated with chronic bowel inflammation in the absence of the atopic symptoms.

As a conclusion the association between inflammatory intestinal diseases and respiratory diseases is not enough investigated and can be seen as a future challenge.

THE NEW PULMONARY REHABILITATION GUIDELINES AND THEIR APPLICABILITY

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Pulmonary rehabilitation is the central component of the management of chronic respiratory diseases, being in continuous development. In 2006 the American Thoracic Society (ATS) and the European Respiratory Society (ERS) developed the first Guide of Pulmonary Rehabilitation valuing the international scientific expertise and the clinical experience.

The 2006 Statement provided a reference point for the pulmonary rehabilitation, the document being complementary to the clinical practice guidelines based on evidence of the American College of Chest Physicians (ACCP) and the American Association of Cardiovascular and Pulmonary Rehabilitation (AACVPR), which formally graded the quality of the scientific evidence, and the AACVPR guidelines for pulmonary rehabilitation programs which provided practical recommendations.

The significant developments made in the field in the recent years justified the reconsideration of the current position; in June 2010 the development of a new statement was initiated, completed in 2013 with the latest update of the guideline for pulmonary rehabilitation which highlights the new definition, the key concepts and the major benefits of pulmonary rehabilitation.

These guides certify the effectiveness of the pulmonary rehabilitation and they bring to our attention the most effective concepts with practical applicability. Pulmonary rehabilitation includes patient education and the adoption of an as healthy as possible lifestyle, representing a standard of care for patients with chronic respiratory pathology, so that they become more active and more independent, through a multidisciplinary approach made by team work.

Updated international guidelines are also applied in our country with a high rate of success on the quality of life in patients included in pulmonary rehabilitation programs, by increasing the independence, increasing the exercise capacity, reducing the psycho-emotional stress and improving the symptoms.

MANAGEMENT OF SARCOIDOSIS-AN UPDATE OF THE MAIN TREATMENT RECOMMENDATIONS

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Sarcoidosis is a systemic disease, with unknown causes. It affects young or middle-aged people, frequently presenting with bilateral hilum lymphadenopathy, pulmonary infiltration, ocular lesions, skin lesions. Sarcoidosis mainly affects adults younger than 40 years with the highest incidence in people aged 20-29. Most studies report a higher prevalence in females. Because the etiology is unknown, the diagnosis of sarcoidosis is established by histopathological demonstration of epithelioid granulomas without caseification, after exclusion of other diseases with similar histological and clinical picture. Sarcoidosis remains a diagnosis of exclusion, there are no definitive tests, specific and sensitive for diagnosis. Treatment of sarcoidosis has as main objectives the suppression of symptoms and the prevention of the development of pulmonary fibrosis. Most patients require only symptomatic treatment with non-steroidal anti-inflammatory agents (>75%), while 15% cases require continuous treatment. Corticosteroids are the basis of treatment. The old recommendation was prednisone 1mg/kg/body, but new studies have shown efficacy in 20 mg prednisone for 21 days, with improvement of lung function tests and symptom reduction. Patients requiring long-term treatment with corticoisteroids may be given 10 to 15 mg of prednisone every other day. Administration of oral corticosteroid in patients with stage II for 3 months, followed by 15 months of inhaled corticosteroid (budesonide) has shown to improve lung function and reduce exacerbations. Non corticosteroid therapy began to be increasingly widely used. Methotrexate is a good alternative to prednisone. Chloroquine and Hidroxicloroquine, antimalarial drugs, are successfully used in skin lesions, hypercalcemia, neurologic sarcoidosis and bone lesions. Azathioprine is another second-line treatment used together with corticosteroids, with good results. Treatment with infliximab and thalidomide has been applied in cases of refractory sarcoidosis, especially for skin lesions, with promising results and good tolerance. For asymptomatic pulmonary disease, without affecting respiratory parameters, periodical reassessment is required, without starting treatment. Corticosteroid therapy in severe cases stage II and III, can improve vital capacity and radiological changes. For cases of extrapulmonary sarcoidosis (heart, liver, eyes, kidneys and central nervous system), drug treatment is indicated. Topical corticosteroids are also used in ocular lesions. In conclusion, sarcoidosis, a disease of unknown etiology, still attracts considerable research, being a rare condition with relatively good prognosis, which sometimes can evolve unfavorably with the development of irreversible debilitating injuries.

MUCOVISCIDOSIS-A MISTERIOUS DISEASE

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Mucoviscidosis, also known as cystic fibrosis is the most frequent monogenic autosomal recessive disorder with chronic progressive evolution. It can involve several organs and systems, lung affectation is present in 90% of the patients. The most frequent cause leading to death is the irreversible damage of the respiratory function.

Early diagnosis and correct treatment can decrease morbidity and mortality by reducing the complications. The diagnosis of cystic fibrosis is based on determining the serum trypsin level, sweat test (considered “gold standard” for diagnosing cystic fibrosis) and genetic testing to discover the exact form of mutation.

The primary goals of the management are early diagnosis, prevention and treatment of the respiratory infections and associated manifestations, as well as maintaining adequate nutrition.

PULMONARY INVOLVEMENT IN SYSTEMIC AUTOIMMUNE DISEASES

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Systemic autoimmune diseases are a heterogeneous group of immunologically mediated inflammatory disorders including multiorgan involvement. Normally the immune system recognizes the tissues in the body that are not ”foreign” and does not attack them.

Autoimmune disorders are diseases caused by the body producing an immune response against its own tissues. The cause of autoimmune diseases is unknown, but it appears that there is a genetic predisposition to develop autoimmune disease in many cases (i.e. they are passed down through families).

Autoimmune disorders fall into two general types: those that damage many organs (”systemic”), and those where only a single organ or tissue is directly damaged by the autoimmune process (”localised”). As expected in a multisystem disease, the entire pulmonary system is vulnerable to injury.

Any of its compartments may be independently or simultaneously affected. It is difficult to assess the true prevalence of lung disease in cases of systemic autoimmune diseases. In this presentation, we will review the pulmonary manifestations caused by rheumatoid arthritis, systemic lupus erythematosus, systemic sclerosis, polymyositis/dermatomyositis, mixed connective tissue disease, Sjögren’s syndrome, Wegener’s granulomatosis, Churg-Strauss syndrome, Goodpasture’s syndrome and ankylosing spondylitis.

PHYSIOPATHOLOGICAL AND DIAGNOSTIC CORRELATIONS IN DIFFICULT ASTHMA

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Exercise induced asthma and exercise induced bronchial constriction are characterized by the transient narrowing of the airways following an intense effort. The majority of asthmatic patients will develop asthma symptoms after physical exercise (80-90%), exercise induced broncho-constriction occurs in approximately 10-15% of general population and elite athletes show an increased prevalence of exercise-induced broncho-constriction (21-50%).

Symptoms could be specific: dyspnea, increased respiratory effort, chest tightness, wheezing, cough, hypersecretion of mucus; symptoms occur 5-30 minutes after vigorous exercise, sometimes during the exercise. Also, these patients could develop non-specific symptoms: low performance, abdominal pain, headache, muscle cramps, fatigue, and malaise. Exercise induced broncho-constriction from a physiopathological point of view can be produced through several mechanisms.

A first mechanism could be increased water loss during cooling in inspiration, which causes dehydration of the airway lining. One acute effect of dehydration is the release of mediators such as prostaglandins, leukotrienes and histamine, which can stimulate smooth muscle, causing contraction and changes in vascular permeability.

When inspiring cold air at high flow, the epithelia of the small airways would become susceptible to dehydration injury, andthe response to this injury involves epithelial plasma exudation as part of the restorative process.

Environmental factors influence the prevalence of these diseases (cold air for winter sports, chlorine derivatives in swimmers, dust for cyclists). The diagnosis is established by highlighting exercise-induced broncho-constriction and bronchial hyper-reactivity. It is important to monitor athletes with increased risk of asthma by regular checks for an early initiation of therapy. Exercise-induced asthma is not a condition separate from asthma.

EVOLVING CONCEPTS IN THE PATHOGENESIS OF INTERSTITIAL LUNG DISEASE

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Pulmonary fibrosis may result from any pathological process that accompanies the association, in various degrees, of inflammatory-fibrotic injuries of the alveolar septa with endothelial dysfunction and impaired alveolar epithelium function.

Initially, the inflammatory process was considered to be the generator of pulmonary fibrosis. The inflammatory process could be triggered by a variety of factors: infection, pollutants, smoking, drugs, autoimmunity. The ineffectiveness of anti-inflammatory drugs has oriented the research toward the fibrotic process (supported by histological arguments: advanced fibrosis, focal fibrosis, myofibroblastic foci). The ineffectiveness of anti-fibrotic treatment led to the exploration of new pathogenic tracks, including endothelial dysfunction, pro-coagulant status, genetic factors (telomere shortening), chemical agents (gastroesophageal reflux and microaspiration). Today it is considered that pulmonary fibrosis occurs after repeated injury (multiple hits) to the alveolar epithelium in individuals genetically predisposed to a dysfunctional tissue repair and architectural alteration of the lung parenchyma. The interaction between genetic and environmental factors generates the pathogenic pathways (inflammation, thrombosis/hemorrhage, fibrosis, immune response, cell proliferation, apoptosis) that will set up a clinical phenotype with a particular evolution. The three major phenotypes (combined emphysema and pulmonary fibrosis, the association with pulmonary hypertension and rapidly progressing forms) highlight idiopathic pulmonary fibrosis (IPF-UIP) as one of the most severe pathologies (mean survival less than 3 years), more severe than many cancers and also disappointing in terms of treatment.

The development of fibrosis in the context of other diseases (collagen vascular diseases, granulomatous disorders, histiocytosis etc.) generates a wide spectrum (over two hundred) of entities. These forms of fibrosis are included under the name of nonspecific interstitial lung pneumonia (NSIP). Among the forms of idiopathic UIP and NSIP, multiple variants are inserted, classified as probably UIP, possible UIP or unclassifiable.

The complexity of pathophysiological processes, multiple radiological aspects, and the diversity of clinical phenotypes impose a multidisciplinary approach to the diagnosis and monitoring: pulmonologist, radiologist, pathologist, immunologist etc. Understanding the pathophysiological mechanisms characteristic of IPF could lead to the emergence of new therapeutic approaches for this disease which currently has an unfortunate prognosis.

COPD EXACERBATIONS IN 2014-A NEW PARADIGM FOR PREVENTING THE FUTURE RISK?

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COPD exacerbations are a major problem for the health status in respiratory medicine. At a global level, it is difficult to determine the real impact of COPD exacerbations in the context of a chronic suboptimal treatment, secondary bacterial infections, under-diagnosis of COPD.

The main objective is to determine the future risk of exacerbation, assessed by the history of exacerbations in the last 12 months.

Setting phenotype-frequent or infrequent exacerbator - becomes a priority for further personalization of treatment and prognosis of the disease.

Asthma-COPD overlap phenotype requires special attention, patients are highly symptomatic, with a low quality of life, frequent and severe exacerbations.

Frequent exacerbator phenotype and chronic bronchitis frequently associates *bronchiectasia* and often require particular management.

The combination of two ultralong bronchodilators with different mechanisms of action significantly reduces future risk of exacerbation in certain patient populations.

There emerge frequent exacerbator subcategories, and reducing the future risk of exacerbation will be made by ultralong BADLA+LAMA combination, while others will require further classical association BADLA/LAMA+ICS.

Preventing future risk of exacerbation means increasing the time to the first exacerbation, shortening recovery after exacerbations, avoidance of severe exacerbations requiring hospitalization, ultimately increasing survival and improving the quality of life of patients with COPD.

THE PARTICULAR FEATURES OF SYSTEMIC INFLAMMATION IN COPD

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COPD is the condition in which pulmonary inflammation is associated with an enhanced systemic inflammatory response, whose consequences are the extrapulmonary manifestations.

The systemic inflammation in COPD has a distinctive pattern, it is augmented in severe diseases and during exacerbations, and the factors of persistence are smoking, advanced age, low FEV1 and low BMI.

The markers of systemic inflammation are represented by cytokines (IL-6, TNF-α, IL-1β), chemokines (IL-8), neutrophils, acute phase proteins (CRP, fibrinogen, serum amyloid D, surfactant protein D).

An essential role in the development and progression of COPD has the „inflamaging” (the association of progressive chronic inflammation with accelerated cellular senescence). This process is controlled by Sirtuines (SIRT 1-7) which have very low levels in patients with COPD. Activation of Sirtuines (specifically SIRT1) induced effective antioxidant and anti-inflammatory mechanisms with potentially favorable effects in the treatment of COPD multimorbidities.

Current pulmonary therapy in COPD reduce pulmonary and systemic inflammation by decreasing the frequency of exacerbations. It was found that beneficial effects on inflammation and systemic manifestations of COPD have also other therapies such as statins, ACE inhibitors, ARBs (angiotensin II receptor blockers), β-blockers and PDE-4 inhibitors.

INTERFACES OF THE OBSTRUCTIVE SYNDROME IN PNEUMOLOGY

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Pneumology is a specialty that often requires rethinking of the case-revision of the initial diagnosis for a better therapeutic management. The bronchial obstructive syndrome can hide either simple diseases or complex neoplastic pathologies with a risk of imminent death if we do not timely intervene.

In the light of these assumptions, we present the case of a 46-year-old woman, initially treated for asthma, in whom the wheezing, stridor and the inspiratory dyspnea were aggravating. The chest X-ray appearance was without modifications, but because of the risk of asphyxia, the patient underwent emergency fibro-bronchoscopy which showed tracheal vegetation formations extending up to approximately 2-3 cm above the tracheal spur and obstructing the trachea almost totally. The final histopathological examination showed squamous papilloma. Polymerase chain reaction (PCR) detected human papiloma virus (HPV) type 6, thought to be the cause of respiratory papilloma. Bronchoscopy desobstructive treatment-electroresection and electrocautery-offered a real benefit, with no bronchial obstructive syndrome after intervention and also improved quality of life. The evolution in time was good, but the case requires careful attention because of the risk of recurrence or malignant transformation.

This case report highlights the importance of correct diagnosis and also the risks involved by unrevised diagnosis in case of unfavorable clinical course and looks into the current management of benign tracheal tumours.

NONINVASIVE VENTILATION ROLE IN THE PHYSIOPATHOLOGICAL MECHANISMS OF RESPIRATORY FAILURE IN PATIENTS WITH COPD AND OBESITY/ HYPOVENTILATION SYNDROME

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Introduction. Noninvasive ventilation (NIV) is increasingly used both in the treatment of acute and chronic respiratory failure. Both COPD and obesity hypoventilation syndrome (OHS) represent an important health issue. Although not identical it is imperative to understand the pathophysiological mechanism that contribute to the development of respiratory failure. The knowledge of common mechanisms may help identify these patients and allow appropriate therapy.

Case presentation. male patient, age 56, ex smoker, obese (BMI=39.24), with a history of COPD stage III GOLD, pulmonary fibrosis, basal bronchiectasis, obstructive sleep apnoea (OSA), chronic pulmonary heart disease, pulmonary hypertension, arterial hypertension, obesity, arrives at the hospital with severe dyspnea, frequent nocturnal apnoea. Clinical and paraclinical outcome reveals cyanosis, basal bilateral crepitation rales, mixt ventilatory dysfunction with severe obstruction of the peripheric airways (FEV1=50.8%), inflamatory syndrome, polyglobulia, hypercapnia (PCO2=53 mmHg), severe hypoxemia (PO2=35 mmHg), oxygen saturation=73.1%.

Management and outcomes. The treatment of this patient consisted in terapy with antibiotics, mucolytics, bronchodilatators, oral anticoagulant, diuretics and also noninvasive ventilation (CPAP)-pressure=10 mmHg, with great outcomes, the correction of hypoxemia and hypercapnia.

Discussions. Altough they are independent entities, the obesity hypoventilation and COPD sometimes occur at the same time, and the complex interactions between ventilatory control, respiratory mechanics, sleep-disordered breathing and neurohormonal disturbances contribues to a severe status of the patient; this is the reason why we must reach a complete diagnosis and also apply correctly the newest terapies. NIV has the same efficacy and outcomes when used for COPD patiens or OHS patiens.

CORRELATION BETWEEN THE NUTRITION STATUS, LUNG FUNCTION AND COMORBIDITIES IN PATIENTS WITH SEVERE AND VERY SEVERE COPD ADMITTED TO THE PNEUMOLOGY CLINIC TG. MURES

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Aim of the study. Evaluating the correlation between BMI, lung function and some comorbidities in patients with severe and very severe COPD hospitalized in the Pneumology Clinical Hospital Tg. Mureș.

Method. We analyzed a group of 79 patients (28 women, 51 men) with severe and very severe COPD. Followed parameters: sex, age, smoking history, BMI, FVC, FEV1, FEV/FVC%, associated cardiovascular diseases (ischemic heart disease, hypertension), diabetes, depression or lung cancer, hematological tests. Patients were divided into 4 groups according to the BMI: normal weight (BMI 18.5-24.9), underweight (BMI <18.5), overweight (BMI 25.0-29.9), obese (BMI>30).

Results. 87% of COPD patients were chronic smokers (71% current smokers). Obese and overweight patients had FEV1 (average 48.9% and 49.4%) and Tiffneau Index (68.7% and 60.3%) with significantly higher values compared to the normal weight and underweight patients: FEV1 (30.5 % and 30.6%), Tiffneau index (54.3% and 64.8%). Overweight and obese patients had more frequently diabetes (55.5% and 15%) and cardiovascular diseases (83% and 55%) than normal weight and underweight patients (0% to 9.5%), (30% and 33%) respectively. 10.1% of patients in the whole group had concomitant lung cancer. The high percentage can be explained both by the increased cancer risk in patients with COPD as well by smoking. Overweight and obese patients had more frequently polycythemia. Depression was more common in normal weight and underweight patients.

Conclusions. BMI influences lung function and the frequent association of comorbidities in patients with severe and very severe COPD. Pulmonary rehabilitation will involve advice on diet, physical activity and normal nutritional status.

INTRICATE MECHANISMS INVOLVED IN ALTERED LUNG FUNCTION IN A YOUNG PATIENT. POSSIBLE SOLUTIONS.

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Background. Severe lung function impairment can have multiple causes and is especially important when dealing with a young patient.

Case Presentation. We present the case of a 41 years old man, admitted for the first time in our clinic 2.5 years ago, accusing rest dyspnea, marked fatigue and cough with purulent expectoration. He is non-smoker, without exposure to respiratory hazards, having a history of multiple episodes of pulmonary and extrapulmonary tuberculosis (ganglionar, pericardial), known for 10 years with Gaucher disease, currently in substitution treatment. The patient is underweight, with cyanotic extremities, finger clubbing, with bilateral basal crackles and hepatosplenomegaly. Chest radiography detects multiple reticular and micronodular opacities disposed predominantly in the basal area. Sputum tests (for ordinary flora and Koch bacillus) are negative. Mild thrombocytopenia, severe hypoxemia and hypercapnia with insufficient response to oxygen are revealed. Computed tomography (CT) identifies important pulmonary reshuffle with large areas of fibrosis, cystic bronchiectasis, interstitial changes. Complex functional tests can detect severe mixed ventilatory dysfunction with severe lowering of transfer constant. Pulmonary changes were classified as secondary to tuberculosis, and also in the context of Gaucher disease. Noninvasive ventilation (NIV) was initiated with improvement of the gas constants and clinical status. Evolution initially consisted of clinical and laboratory significant improvement, then flattening, the absence of exacerbations over a period of two years, then 2 infectious exacerbations that required hospitalization. Currently the patient is stable and continues NIV.

Discussion. pulmonary tuberculosis sequelae may take different aspects, increasing in severity as the number of episodes of tuberculosis and expanding lesion. Gaucher disease can affect lung interstitium, the combination of the two can lend extra gravity of a case.

Conclusions. Regardless of the main reason of damage to the lungs, the two diseases are mutually reinforcing and have limited therapeutic intervention. In this case, any therapeutic solution is worth taking into consideration.

Peculiarity of the case. We present this case because of the rare combination of mutilating tuberculosis sequelae and Gaucher disease with pulmonary involvement, also because of the extreme solution found as therapy in this case.

PULMONARY FUNCTION TESTS IN ASTHMA FOR ADULT AND CHILD

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The diagnosis of asthma is based upon clinical history and characteristic findings from a series of pulmonary function tests. These tests most often include different measures of airflow, bronchodilator responses, lung volumes, and the diffusing lung capacity for carbonmonoxide (CO). Spirometry is the most accurate breathing test for asthma.

The FEV1, from spirometry, is the most reproducible pulmonary function parameter and is related to the severity of airways obstruction, this the reason that it is necessary a very good spirometry regarding technical aspects.

Even in children less than 6 years old the reproducibility criteria could obtain but there are some special features that will be addressed.

Complex functional respiratory tests will also be the subject of the presentation with an explanation in the adult and child and particular aspects.

BODYPLETISMOGRAPHY AND DIFFUSION THROUGH ALVEOLO-CAPILLARY MEMBRANE IN PRACTICE FOR PULMONOLOGIST

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Bodypletismography and diffusion through alveolo-capillary membrane in a unique breathing using Helium are two methods of complex evaluation of lung function indispensable in practice for pulmonologist.

They allow measurement of residual functional capacity and total lung capacity (TLC) using high-performance equipment.

Calibration of equipment, the technique of performing, relative contraindications, the possible errors that may occur, interpretation of results and their integration in certain respiratory diseases will be subject to the presentation

RESPIRATORY MUSCLES AND PULMONARY REHABILITATION-PRACTICAL ASPECTS

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The respiratory muscles contribute substantially to the realization of the pulmonary gas exchange and ventilation, impairment of its function being clinically manifested by dyspnea, cough and exercise intolerance.

Pulmonary rehabilitation, according to the guides, also addresses to the training of the affected respiratory muscles (maximum inspiratory pressure, PIMax, under 60 cm H2O and the presence of these symptoms: dyspnea, hypercapnia and reduced exercise capacity). The specific training of the respiratory muscles is the endurance training (forced ventilation maintained several minutes) and it can be continuous or interval training (better tolerated by the patients).

In COPD, respiratory muscle training is used alone or in combination with generalized physical training, especially in patients with respiratory muscle weakness, studies showing increasing endurance and strength of these muscles (improving PIMax), increasing exercise tolerance (shown by the 6-minute walk test), improvement of dyspnea (Borg scale and Transition Dyspnea Index, TDI) and improvement in the quality of life, with the reduction of the period of hospitalization, effects that persist if training is long-term.

THE IMPORTANCE OF CARDIORESPIRATORY POLIGRAPHY IN THE DIAGNOSIS OF SLEEP-RELATED BREATHING DISORDERS-PRACTICAL ISSUES

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Over the last twenty years, the management of sleep disorders has become recognized as a field of study in its own right. Due to the fact that clinical practice in a sleep laboratory, of all sleep related breathing disorders, is focused especially on obstructive sleep apnea syndrome (OSAS), it′s important to highlight the practical aspects of diagnosis of sleep apnea by cardio-respiratory poligraphy. Diagnostic criteria for OSAS are based on clinical signs and symptoms determined during a comprehensive sleep evaluation, which includes a sleep oriented history and physical examination and findings identified by sleep recording.

Even if, polisomnography represents the gold standard for diagnosis, when the clinical suspicion of OSAS is high, there was designed a portable device (cardio-respiratory poligraph) which recorder nasal airflow, thoracic-abdominal movements, body position, heart rate, oxygen saturation by pulse oximetry and snoring .

The Apnea is defined as the absence of airflow lasting ≥10 seconds (s). Hypopnea is defined as any airflow reduction lasting ≥10 s associated with the nasal pressure signal excursions drop by ≥ 30% of baseline, with oxygen desaturation 3%.

The diagnosis of OSAS is established using the apnea/hypopnea index (AHI), calculated as the average of apneic and hypopneic events per hour of sleep. Poligraphy is important in clinical practice because requires less technical expertise, is less labor intensive and time consuming, and is easier for patients to access.

TESTS AND PROCEDURES IN SLEEP-RELATED BREATHING DISORDERS IN A SLEEP SERVICE FROM ROMANIA!

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Sleep medicine and sleep services are dedicated to the recognition, diagnosis and treatment of syndromes and diseases that affect sleep. Many of these syndromes and diseases are caused by, or linked to respiratory conditions, in this way Sleep-related Breathing Disorders are actually more frequent and common.

The standard approach to diagnosing Sleep-related Breathing Disorders is in-laboratory, technician-attended, polysomnography. Portable monitoring (PM) has been proposed as a substitute for polysomnography in the diagnostic assessment of patients with suspected OSA.

The addressability of a Sleep Service from Romania, is especially for the diagnosis of OSA, and the patient is brought in the Sleep Laboratory by the family, by himself, or sent by other medical services like cardiology, internal medicine, general practice, diabetology, ORL, neurology, occupational medicine in order to establish the diagnosis.

After the final diagnosis was made will be establish the proper treatment, which is especially therapy with continuous positive airway pressure (CPAP). Diagnostic criteria are based on clinical signs and symptoms determined during a comprehensive sleep evaluation, which includes a sleep oriented history and physical examination and findings identified by sleep recording.

The patient with Sleep-related Breathing Disorders which is taken into evaluation in a sleep laboratory in Romania, will be follow up according to local protocol. There is need for more Sleep Servicies in Romania for a better management of Sleep-related Breathing Disorders.

HEMOPHTHISIS AS THE FIRST SYMPTOM OF BRONCHIECTASIS

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Traction bronchiectasis represents a relatively new pathology, often being the result of an infectious process leading to abnormal and permanent distortion of one or several bronchi.

Bronchiectasis can be classified as an obstructive lung disease occurring as a generalized inflammation of the airways that can easily collapse, leading to airflow obstruction, causing dyspnea, inadequate secretion clearance (often accompanied by coughing) and occasionally hemophthisis. Severe cases may lead to progressive disability and respiratory insufficiency.

The highest frequency of the disease is encountered in the sixth to the eighth decade of life, its most probable cause being atypical mycobacterial infections.

In the pre-antibiotic era the mortality rate was high and patients would die within the first 5 years after the first occurrence of the symptoms. Nowadays it is difficult to establish the mortality rate especially because of the difficulty in determining prevalence and the lack of concrete studies. In general, the prognosis of patients with bronchiectasis is good.

We are presenting the case of a 52-year-old male patient, smoker, IPA=40, with known class CHILD B toxic cirrhosis of the liver, second-degree esophageal varices, Los Angeles grade A esophagitis, third-degree arterial hypertension with very high additional risk, hypertensive nephropathy, insulin-dependent type II diabetes mellitus in his medical history, who came to the “Leon Daniello” Pneumophthisiology Clinical Hospital of Cluj-Napoca presenting cough and moderate amount of hemoptoic expectoration, which had started suddenly approximately three days before hospitalization. Considering the associated digestive pathology, hematemesis could be excluded with an upper digestive endoscopy, which indicated no active bleeding.

The native thoracic CT examination highlighted extensive areas of bilateral subpleural pulmonary fibrosis, associated with traction bronchiectasis localized in the upper right lobe. The fibro-bronchoscopy confirmed the existence of traction bronchiectasis, which also represented the source of the hemophthisis.

Thus the diagnosis of bilateral subpleural pulmonary fibrosis associated with traction bronchiectasis superinfected with Klebsiella spp., distinguished in the bronchoalveolar washing fluid, was established and it resulted in a suddenly onset hemophthisis restored through endoscopic packing with adrenalin, hemostatics associated with long-term antibiotic therapy.

The diagnosis of traction bronchiectasis is often coincidental, its symptoms being minimal. The first symptom in the mentioned medical case was a moderate amount of hemophthisis.

DIAGNOSTIC DIFFICULTIES IN A CASE OF MEDIASTINAL AND PULMONARY TUMOR

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Background. Hodgkin's lymphoma is a potentially curable cancer characterized by the presence of Reed-Sternberg cells in a cellular reactive context, with a higher frequency among male patients, characterized by variability of symptoms and possible Ebstein Barr virus involvement in its etiology.

Case description. Patient of 33 years, with a family history of lung cancer is presented in pulmonology service accusing intermittent dry cough, then haemorrhagic, right anterior and posterior chest pain, chest tightness, low grade fever, fatigue and weight loss. Pulmonary x-ray shows right upper lobe congestion with partial resorbtion and symptom relief following antibiotic and cortisone treatment. Because of the recurrence of symptoms and the computed tomography imaging describing a mediastinal tumor in the right upper pulmonary lobe as well as multiple mediastinal lymph nodes, patient underwent a series of exhaustive investigations within two years following, including ultrasound guided transthoracic puncture, two bronchoscopies with transbronchial biopsy, mediastinoscopy with lymph node biopsy and sternal puncture with equivocal histopathological results.

The need for exploratory thoracotomy was established and surgical right upper lung lobe biopsy was performed, histology and immunohistochemical profile advocating for Hodgkin lymphoma, nodular sclerosis type, mixed cellularity variant.

Discussion. The present case illustrates the need to resort to an increased number of invasive investigations to obtain a positive diagnosis. Diagnostic difficulties were caused both by technical limitations, as well as low patient compliance. Another feature of this case of Hodgkin lymphoma is the atypical presentation and slow natural evolution of the disease in the absence of specific therapy.

PULMONARY AND MEDIASTINAL METASTASIS OF MALIGN MELANOMA, CASE REPORT

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Introduction. Melanoma represents the most severe skin disease, with growing incidence in white people, because of the intense UV exposure. Metastasis can affect any organ, and in approximative 5% of the cases patients can present melanoma metastasis without knowing the location of the primary tumor.

Case description. A 55 years old female patient, ex-smoker, accuses graveyard cough, left hemitorax pain, shiver, asthenia, weight loss of 5-6 kg in two month. The blood tests reveal an inflammatory syndrome. The chest CT scan reveals a 60 mm pulmonary tumor, that is centrated on the left lung bronchus, including left pulmonary artery, and a pulmonary nodule in the left lower lobe. Fibrobronchoscopy reveals an exterior compression, with minimum culminal bronchic stenosis. The patient is transferred in the thoracic surgery department, for rezection of the pulmonary tumor, and biopsy of the mediastinal tumor. The imunohistopthological tests confirm the diagnosis of pulmonary and mediastinal metastasis of melanoma.

Discussion. This case illustrates the importance of a thoroughly anamnesis led by young doctors, avoiding the omission of medical history data that might seem insignificant from the patient's point of view. A retrospective analysis showed that 3 years before the respiratory symptoms occurred the patient was diagnosed with left thigh melanoma, that was resected apparently with safety margins. Another specific feature of the case consists in the long period of time during the diagnosis of the melanoma and the detection of its' metastases.

SOLITARY FIBROUS TUMOR OF THE LUNG, CASE REPORT

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Introduction. Solitary fibrous tumors of pleura and lung are rare primary tumors that arise from the submesothelial tissue and usually show a benign clinical course. Immuno-histochemical analysis is used to make the diagnosis. Most of them behave as slow-growing, painless masses.

Large tumors may give rise to compression symptoms, or cause hypertrophic pulmonary osteoarthropathy and refractory hypoglycemia, due to the production of a growth hormone-like substance and an insulin-like growth factor respectively.Usually they are found incidentally on chest radiography.Benign solitary fibrous tumors can be cured by complete surgical resection.

Case description. A 27 year old woman, assymptomatic, was found to have a right pulmonary opacity in the chest film, made for tuberculosis contact. Bacteriological examination is negative for Mycobacterium Tuberculosis. The computer tomography scan of the chest reveald a right upper lobe lung mass, 37 mm diameter, well defined with a lobate contour, without infiltration of the adjacent lung. Bronchoscopy shows,scarring stenosis on the dorsal segment bronxhus of right upper lobe with secondary suppuration.

Given the radiological and endoscopic findings, we think at a pulmonary tuberculoma, but we can't exclude other pathologies.

At right thoracotomy, the tumour is removed at right upper lobectomy and also some mediastinal lymphadenopathy were removed. Histopathology shows a mesenchymal tumor.

Discussion. Complete resection of solitary fibrous tumor is usually curative. Morphological and pathological features are important in distinguishing them from other tumors and in predicting clinical behaviour.

ALVEOLAR PROTEINOSIS A CASE PRESENTATION

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Pulmonary alveolar proteinosis (PAP) is a rare lung disorder characterised by alveolar filling with phospholipidic material that stains positive using the periodic acid-Schiff (PAS) method, leading to alterations in the gas exchange process. There are two forms: primary (idiopathic) and secondary due to various conditions (imunodepression, malignant hematologic affections, silicosis, or even diffuse interstitial lung diseases).

We present the case of a 42-year old male patient, ex smoker, with nonspecific symptoms: nonproductive cough, progressive dyspnea, respiratory failure with hypoxemia 69-75%.

Chest X-ray and CT scan showed alveolitic pattern with air bronchogram on both pulmonary fields. PAS staining of BAL fluid and transbronchial lung biopsy established the diagnosis of pulmonary alveolar proteinosis.

Conclusion. The fibrobronchoscopy and its associated techniques: bronchoalveolar lavage and transbronchial lung biopsy play a decisive part in diagnosing alveolar proteinosis.

TUBERCULOSIS DESEASE DIAGNOSED ON A PACIENT WITH 2 CAUSES OF IMUNOSUPPRESSION

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Numerous types of cancer undergoing chemotherapy have been incriminated as a risk factor for pulmonary tuberculosis, although the literature provides inconsistent information about this. Most studies designates extrapulmonary tuberculosis as beeing the common form among immunocompromised patients. We present the case of a 58 years old pacient diagnosed with colon cancer with right hemicolectomy in July 2013, currently under chemotherapy, cardiac comorbidities (Atrial Fibrillation , Hypertension stage IIIC), Diabetes with insulin treatment, Hyperthyroidism under drug control, presenting chronic cough syndrome that started three months ago accompanied by night sweating for witch he performed a chest CT scan in September that reveals two nodular formations 24mm and 18mm respectively, with central cavity, that raise the suspicion of a specific pulmonary syndrome without being able to exclude a possible pulmonary metastases.

Because of the decompensation of his comorbidities the general condition of the pacient deteriorates that beeing the reason for wich he postpones the pulmonary investigations until November when he repeats the chest CT scan. The second exam highlights changes suggestive for disseminated active cavitary tuberculosis. Specific bacteriological tests turn out positive and Haemophilus influenzae is also isolated. This confirmes the diagnosis of secondary pulmonary Tuberculosis fibro-caseous cavitary form located in the left upper lobe with bronchogenic dissemination in a patient with two causes of immunosuppression (diabetes undergoing insulin treatment and colon cancer undergoing chemotherapy).

Rapid diagnosis of Tuberculosis disease and prompt initiation of treatment are essential for evolution and prognosis.