Berzelius symposium 94

International Conference on

Multidisciplinary approach to diffuse parenchymal lung disease

1–3 June 2017 in Stockholm · Sweden

Svenska Läkaresällskapet

The symposium is granted 14 European CME credit (ECMEC) by the European Accreditation Council for Continuing Medical Education, EACCME®

The symposium is arranged by the Swedish Society of Medicine in cooperation with the Swedish Society of Pathology · the Swedish Society of Radiology and the Swedish Respiratory Society.

Berzelius symposium 94

International Conference on

Multidisciplinary approach to diffuse parenchymal lung disease

This international course is designed to give a review of diffuse parenchymal lung diseases with clinical, radiologic and pathological correlations. The course will run for two and half days and will consist of a series of presentations with corresponding multidisciplinary sessions. The speakers are expert thoracic clinicians, radiologists and pulmonary pathologists. The presentations will illustrate the importance of a multidisciplinary approach to solving difficult problems in diffuse parenchymal lung disease.

This multidisciplinary international course could be one of the very few courses offered in the world concerning this type of disease, giving lectures and prac-tical sessions with very well known lecturers.

By the end of this course the participants should have a better understanding of the importance of correlation between clinical, radiologic and pathologic data in diagnosis and management of diffuse parenchymal lung disease.

Welcome to the symposium!

Organizing and/or scientific committee: Cristian Ortiz-Villlalón MD PhD (chair), Magnus Sköld MD PhD, Riitta Kaarteenaho, Sven Nyrén MD PhD, Giovanni Ferrara MD PhD, Jacek Pawlowski MD, Anna Nordgren-Rogberg MD, Göran Elmberger MD PhD

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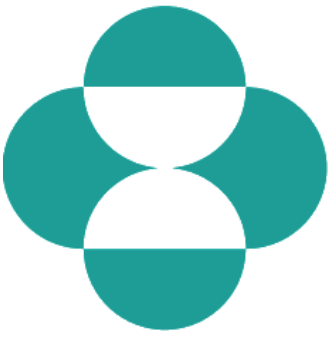

MSD

PROGRAMME

Thursday, 1 June 2017

10.00 – 11.00 Coffee and registration

11.00 – 11.05 **Introduction and Welcome**
Dr. Cristian Ortiz-Villalón, Sweden

Session 1 DPLD: Understanding the approach
Chair: Magnus Sköld, Sweden

11.05 – 11.35 **Early diagnosis: Addressing the clinical suspicion of DPLD**
Dr. Luca Richeldi, Italy

11.35 – 12.05 **Main radiological patterns in DPLD**
Dr. Johny Verschakelen, the Netherlands

12.05 – 12.35 **Identifying histological patterns in DPLD.**
Does the histology matter? Dr. Henry Tazelaar, USA

12.35 – 13.35 Lunch

Session 2 IPF: New diagnostic tools and treatment
Chair: Giovanni Ferrara, Sweden

13.35 – 14.05 **Advances in the diagnosis of idiopathic pulmonary fibrosis**
Dr. Riita Kaarteenaho, Finland

14.05 – 14.35 **Advances in MRI of IPF**
Dr. Hans-Ulrich Kauczor, Germany

14.35 – 15.05 **Advances in the treatment of idiopathic pulmonary fibrosis**
Dr. Luca Richeldi, Italy

15.05 – 15.25 **Proposal of new technique, Spiral Array, and concept of UIP bucket**
Dr. Junya Fukuoka, Japan

15.25 – 15.40 Panel discussion

15.40 – 16.00 Coffee break

Session 3: Workshop 1
16.00 – 17.30 **Multidisciplinary approach (4 cases)**

18.30 – 21.00 Welcome reception

Friday, 2 June 2017

Session 4: Environment-related DPLD
Chair: Cristian Ortiz-Villalón, Sweden

09.00 – 09.45 **Environment-related DPLD: A clinical overview**
Dr. Kjell Toren, Sweden

09.45 – 10.15 **Radiologist's approach to environment-related DPLD**
Dr. Martine Rémy Jardin, France

10.15 – 10.45 **Histopathological update on asbestos related diseases**
Dr. Richard Attanoos, UK

10.45 – 11.15 Coffee break

11.15 – 11.45 **What radiology says on hypersensitivity pneumonia**
Dr. Hans-Ulrich Kauczor, Germany

11.45 – 12.15 **Pathologic differential diagnosis in hypersensitivity pneumonitis**
Dr. Anja Roden, USA

12.15 – 13.00 **DPLP related to catastrophic events: Lessons from 9/11**
Dr. Anna Nolan, USA

13.00 – 14.00 Lunch

14.00 – 16.30 **Session 5: Discussion in separate groups**
(participants may join one of the respective groups)

Saturday, 3 June 2017

Session 6: Chronic rejection post transplantation
Chair: Sven Nyrén, Sweden

09.00 – 09.30 **Chronic rejection after lung transplantation: a clinical dilemma?**
Dr. Gerdt Riise, Sweden

09.30 – 10.00 **Imaging in chronic rejection**
Dr. Johny Verschakelen, the Netherlands

10.00 – 10.30 **Chronic rejection: a pathologists view**
Dr. M. Angeles Montero, UK

10.30 – 11.00 Coffee break

Session 7: Workshop 2
11.00 – 13.00 **4 cases**

13.00 Summary and end of the symposium

General information

The Society's building in Stockholm

The conference Hall

When & Where?

1–3 June 2017 at the Swedish Society of Medicine (SSM),
Klara Östra Kyrkogata 10 in Stockholm, Sweden.

Lunches and coffee are included in the participation cost and will be served at the SSM.

Symposium secretariat

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Symposium website: http://www.sls.se/thrombosis

Social programme

Thursday, 1 June 2017 at 6.30 p.m
Welcome reception at the Swedish Society of Medicine.

EACCME accreditation granted EACCME-15230-G

Berzelius symposium 94
Multidisciplinary approach to diffuse parenchymal lung disease in Stockholm, Sweden on 1–3 June 2017 has been accredited by the European Accreditation Council for Continuing Medical Education (EACCME) to provide the following CME activity for medical specialists.

The symposium is designated for a maximum of, or up to 14 European CME credits (ECMEC).

Speakers abstracts

Early diagnosis: Addressing the clinical suspicion of DPLD

Prof Luca Richeldi, Italy

Brief Overview: Diffuse Parenchymal Lung Diseases are a group of respiratory diseases, often idiopathic, and often affecting older groups of the general population (1). Although all together represent about 15% of all the patients referred to respiratory medicine specialists, their diagnosis remains particularly difficult: very often symptoms at onset are not specific, and often patients receive treatments for other respiratory conditions for years before to receive the right diagnosis (1, 2).

Relevance: Different forms of DPLD have different prognosis, varying from self-limiting to progressive and fatal within a few years. Early diagnosis might give the possibility to treat patients with forms of DPLD, especially idiopathic pulmonary fibrosis, with new drugs, and to affect survival (3-5). This lecture will review the evidence and research questions related to early diagnosis of DPLD and it´s potential impact on the course of the disease in this specific group of patients.

Key Learning Points:
After this Activity, participants should be able to:
1. Address the clinical suspicion of DPLD at an early stage
2. Discuss the clinical findings and diagnostics necessary to address these conditions
3. Evaluate the current literature pertaining to DPLD and diagnosis/treatment at early stage.

References:
1. Travis WD, Costabel U, Hansell DM, King TE, Lynch DA, Nicholson AG, Ryerson CJ, Ryu JH, Selman M, Wells AU, Behr J, Bouros D, Brown KK, Colby TV, Collard HR, Cordeiro CR, Cottin V, Crestani B, Drent M, Dudden RF, Egan J, Flaherty K, Hogaboam C, Inoue Y, Johkoh T, Kim DS, Kitaichi M, Loyd J, Martinez FJ, Myers J, Protzko S, Raghu G, Richeldi L, Sverzellati N, Swigris J, Valeyre D, Pneumonias AECoII. An official American Thoracic Society/European Respiratory Society statement: Update of the international multidisciplinary classification of the idiopathic interstitial pneumonias. *American Journal of Respiratory and Critical Care Medicine* 2013; 188: 733–748.

2. Raghu G, Collard HR, Egan JJ, Martinez FJ, Behr J, Brown KK, Colby TV, Cordier J-F, Flaherty KR, Lasky JA, Lynch DA, Ryu JH, Swigris JJ, Wells AU, Ancochea J, Bouros D, Carvalho C, Costabel U, Ebina M, Hansell DM, Johkoh T, Kim DS, King TE, Kondoh Y, Myers J, Müller NL, Nicholson AG, Richeldi L, Selman M, Dudden RF, Griss BS, Protzko SL, Schünemann HJ, Fibrosis AEJACoIP. An official ATS/ERS/JRS/ALAT statement: idiopathic pulmonary fibrosis: evidence-based guidelines for diagnosis and management. *American Journal of Respiratory and Critical Care Medicine* 2011; 183: 788–824.

3. Loupasakis K, Berman J, Jaber N, Zeig-Owens R, Webber MP, Glaser MS, Moir W, Qayyum B, Weiden MD, Nolan A, Aldrich TK, Kelly KJ, Prezant DJ. Refractory sarcoid arthritis in World Trade Center-exposed New York City firefighters: a case series. *J Clin Rheumatol* 2015; 21: 19–23.

4. Richeldi L, du Bois RM, Raghu G, Azuma A, Brown KK, Costabel U, Cottin V, Flaherty KR, Hansell DM, Inoue Y, Kim DS, Kolb M, Nicholson AG, Noble PW, Selman M, Taniguchi H, Brun M, Le Maulf F, Girard M, Stowasser S, Schlenker-Herceg R, Disse B, Collard HR, Investigators IT. Efficacy and safety of nintedanib in idiopathic pulmonary fibrosis. The New England Journal of Medicine 2014; 370: 2071–2082.

5. King TE, Bradford WZ, Castro-Bernardini S, Fagan EA, Glaspole I, Glassberg MK, Gorina E, Hopkins PM, Kardatzke D, Lancaster L, Lederer DJ, Nathan SD, Pereira CA, Sahn SA, Sussman R, Swigris JJ, Noble PW, Group AS. A phase 3 trial of pirfenidone in patients with idiopathic pulmonary fibrosis. *The New England Journal of Medicine* 2014; 370: 2083–2092.

Main radiological patterns in DPLD

Dr. Johny A Verschakelen, Belgium

DPLD very often shows CT changes that are composed in a repeating arrangement which can be recognized as a pattern. In this way generally 4 patterns are described: the nodular pattern, the linear (reticular) pattern, the increased lung attenuation pattern and the decreased lung attenuation pattern. Defining this *appearance pattern* of disease (classifying the abnormalities in a category that is based on their appearance) is a first step towards the radiological diagnosis or differential diagnosis and should be performed together with the determination of location and distribution of the abnormalities, the *distribution pattern* of disease. A careful study of the "macroscopic" distribution of the disease in the lung and the "sub-macroscopic" relationship of the abnormalities with the different elements of the secondary pulmonary lobule, often allows to suggest if pathology is (predominantly) located in or surrounding the blood vessels, the lymphatics or the airways or if the interstitium is predominantly involved. Although this stepwise analysis may result in a radiological diagnosis or a narrow differential diagnosis, making a definitive radiological diagnosis may be hampered by the fact that disease appearance and/or distribution patterns may not be clear, change over time or that patterns overlap.
The diagnosis of DPLD often requires a multidisciplinary approach. During the multidisciplinary conferences the radiologist may not only suggest a radiological diagnosis or differential diagnosis but may be able to suggest a distribution pathway of disease which can be a valuable addition to the clinical findings, help to suggest and guide additional diagnostic procedures and may be necessary when interpreting the pathological findings if a biopsy is performed.

In this presentation:
1) the pattern approach in the diagnosis of DPLD will be explained and demonstrated.

2) the basic anatomical and physiological knowledge that is required to recognize the appearance and distribution patterns of lung disease will be emphasized.

3) it will be shown that recognizing patterns is not only helpful to suggest a radiological diagnosis or differential diagnosis but can also be a valuable addition to the multidisciplinary discussion.

References:
1) Verschakelen JA, De Wever W. Computed Tomography of the Lung: A Pattern Approach. 2nd ed , Springer due September 2017

2) Zompatori M, Bnà C, Poletti V, Spaggiari E, Ormitti F, Calabro E, Tognini G, Sverzellati N. Diagnostic Imaging of Diffuse Infiltrative Disease of the Lung. Respiration 2004; 71:4

3) Nishino M, Itoh H, Hatabu H. A practical approach to high-resolution CT of diffuse lung disease. Eur J Radiol 2014; 83: 6

Histological patterns in DPLD. Does Histology matter?

Dr. Henry Tazelaar, USA

Patterns represent consistent and recurring characteristics and traits that help in identification and serve as an indication for predicting future behavior. Six patterns of diffuse lung disease have been described by Dr. Kevin Leslie: acute injury, fibrosis, cellular infiltrates, alveolar filling, nodules, and minimal change. Fibrotic patterns have some of the worst prognoses. Fibrotic patterns can be characterized histologically as heterogeneous (usual interstitial pneumonia) or homogeneous (fibrotic nonspecific interstitial pneumonia and airway centered). Histology is extremely important in determining underlying etiology, potential treatment options as well as prognosis. Radiologic and pathologic patterns have some overlap, but the ATS/JRS/ALAT criteria for a radiologic diagnosis of IPF likely need improvement so there is better correlation with a pathology diagnosis of usual interstitial pneumonia/idiopathic pulmonary fibrosis. Despite the success of multidisciplinary diagnoses, pathologic diagnoses still carry the most weight.

Key learning points

At the end of the lecture participants should be able to

1. Name six patterns of diffuse lung disease
2. Describe fibrotic patterns of lung disease which are heterogeneous and homogeneous
3. Be able to integrate radiologic and pathologic diagnoses

References:

1. Yagihashi K, Huckleberry J, Colby TV, Tazelaar HD, Zach J, Sundaram B, Pipavath S, Schwarz MI, Lynch DA for the Idiopathic Pulmonary Fibrosis Clinical Research Network (IPFnet). Radiologic-pathologic discordance in biopsy-proven usual interstitial pneumonia. Eur Respir J. 2016;47:1189–97.

2. Flaherty KR, Thwaite EL, Kazerooni EA, Gross BH, Toews GB, Colby TV, Travis WD, Mumford JA, Murray S, Flint A, Lynch JP 3rd, Martinez FJ. Radiological versus histological diagnosis in UIP and NSIP: survival implications. Thorax. 2003;58:143–8.

3. Walsh SLF, Calandriello L, Sverzellati N, Wells AU, Hansell DM, on behalf of The UIP Observer Consort. Interobserver agreement for the ATS/ERS/JRS/ALAT criteria for a UIP pattern on CT. Thorax. 2016;71:45–51.

4. Flaherty KR, King TE Jr, Raghu G, Lynch JP III, Colby TV, Travis WD, Gross BH, Kazerooni EA, Toews GB, Long QI, Murray S, Lama VN, Gay SE, Martinez FJ. Idiopathic interstitial pneumonia: what is the effect of a multidisciplinary approach to diagnosis? Am J Respir Crit Care Med. 2004;170:904–10.

5. Leslie KO. My approach to interstitial lung disease using clinical, radiological and histopathological patterns. J Clin Pathol. 2009;62:387–401.

Advances in the diagnosis of idiopathic pulmonary fibrosis

Dr. Riita Kaarteenaho, Finland

Brief overview

The latest international statement of idiopathic pulmonary fibrosis (IPF) has instructed diagnostic of IPF more systematic than previously. IPF can be diagnosed based on clinical and radiological investigations when a high resolution computed tomography (HRCT) is categorized as typical usual interstitial pneumonia (UIP) pattern with honeycombing. Otherwise the diagnosis of IPF is needed to be confirmed by histological investigation of a surgical lung biopsy (SLB). The ultimate diagnosis is made in a multidisciplinary team meeting by pulmonologists, radiologists and pathologists. Although this approach has produced benefits, some weaknesses have emerged since there are number of patients who do not meet the HRCT criteria for typical UIP. Often these patients are referred to as possible IPF or unclassifiable ILD. All patients with possible HRCT pattern cannot undergo SLB operation due to the risks of which an acute exacerbation of IPF is the most severe. Other characteristic HRCT features, such as traction bronchiectasis and heterogeneity, may increase the accuracy of imaging in the future. Further, combining other information e.g. clinical data and biomarkers into the current diagnostic approach may be beneficial. Transbronchial cryobiopsy technique may substitute in some cases SLB.

The lecture contain information of the current diagnostic approach of IPF.

Key learning points:

1) Current diagnostic approach for IPF
2) Weaknesses in the current diagnostics
3) Advances of the diagnostics of IPF

Key references

1. Raghu G, Collard HR, Egan JJ et al; ATS/ERS/JRS/ALAT Committee on Idiopathic Pulmonary. An official ATS/ERS/JRS/ALAT statement: idiopathic pulmonary fibrosis: evidence-based guidelines for diagnosis and management. Am J Respir Crit Care Med 2011 15;183(6):788–824

2. Hutchinson JP, Fogarty AW, McKeever TM, Hubbard RB. In-hospital mortality after surgical lung biopsy for interstitial lung disease in the United States. 2000 to 2011. Am J Respir Crit Care Med 2016 15;193(10):1161–7.

3. Sharp C, McCabe M, Adamali H, Medford AR. Use of transbronchial cryobiopsy in the diagnosis of interstitial lung disease-a systematic review and cost analysis. QMJ 2017 Apr 1;110(4):207–214.

4. Brownell R, Moua T, Henry TS, et al. The use of pretest probability increases the value of high-resolution CT in diagnosing usual interstitial pneumonia. Thorax 2017 May; 72(5):424–429.

5. Walsh SL, Wells AU, Desai SR et al. Multicentre evaluation of multidisciplinary team meeting agreement on diagnosis in diffuse parenchymal lung disease: a case-cohort study. Lancet Respir Med 2016;4(7):557–65.

Advances in MRI of IPF

Dr. Hans-Ulrich Kauczor, Germany

Brief overview
Imaging – namely CT – is important in the differential diagnosis as well as in the assessment of functional constraints and therapy response in interstitial lung disease. Due to novel technical refinements MRI is currently entering the field of diffuse parenchymal lung disease and aims to provide reliable contributions to:
(1) visualization of structural changes and their patterns: ultrashort TE sequences might be capable to visualize the typical patterns, such as reticular and reticulonodular changes, ground glass, consolidation and honey-combing with "high" resolution
(2) assessment of pulmonary function, i.e. restrictive ventilatory impairment, reduction of perfusion and gas exchange using contrast-enhanced perfusion MRI, ventilation MRI with hyperpolarized gases or oxygen and MR elastography
(3) assessment of inflammatory activity using routine contrast-enhanced MRI and T2 weighted sequences, which is a clear advantage over CT which has limitations in this regard.

Relevance to the congress concept
The development and implementation of MRI for IPF is a multidisciplinary effort by medical physicists, radiologists, biologists, pneumologists, physiologists and pathologists covering along the whole translational chain from bench to beside and respective feedback loops. The focus is on visualization and quantitation of several pathological and pathophysiological conditions, e.g. inflammation, ventilation, perfusion, fibrosis, compliance, and remodeling

Key learning objectives
1) to learn about recent technical refinements in pulmonary MRI
2) to understand the potential and limitations of MRI of IPF
3) to consolidate knowledge about the current "evidence" of MRI of IPF

Key references
1) Mariappan-YK et al. Estimation of the Absolute Shear Stiffness of Human Lung Parenchyma Using 1H Spin Echo, Echo Planar MR Elastography; JMRI 2014;40:1230–1237

2) Buzan-MTA et al. T2 mapping of CT remodelling patterns in interstitial lung disease; Eur Radiol 2015;25:3167-3174

3) Ohno-Y et al. Pulmonary high-resolution ultrashort TE MRI: comparison with thin-section standard and low-dose CT for the assessment of pulmonary parenchymal disease. JMRI 2016;43:512-532

4) Cleveland-ZI et al. MRI of disease progression and resolution in a transgenic mouse model of pulmonary fibrosis; Am J Physiol Lung Cell Mol Physiol 2017;312:L488-L499

5) Lavalle-LP et al. Pulmonary fibrosis: tissue characterization using late enhanced MRI compared with unenhanced anatomic high-resolution CT; Diagn Interv Radiol 2017; 23:106–111

Advances in the treatment of idiopathic pulmonary fibrosis

Prof Luca Richeldi, Italy

Brief Overview: Idiopathic pulmonary fibrosis is a form of fibrosis limited to the lung, of unknown ethiology and often leading to respiratory failure and death within 5 years from diagnosis (1, 2). The treatment of IPF has radically changed in the last 5 years, thanks to the introduction, for the first time, of 2 drugs able to slow down the progression of the disease (3, 4).
Relevance: This lecture will review the evidence and research questions related to the use of nintedanib and pirfenidone in the treatment of IPF. It will address also the new and current clinical trials in different subgroups of IPF-patients, and will give some insight in the development and clinical testing of new compounds.

Key Learning Points:
After this Activity, participants should be able to:
1. Address the use of new drugs in patients with IPF
2. Discuss the use of new drugs in particular subgroups (early and advanced stages)
3. Evaluate critically the current literature pertaining to pirfenidone, nintedanib and potential new drugs for IPF.

References:
1. Travis WD, Costabel U, Hansell DM, King TE, Lynch DA, Nicholson AG, Ryerson CJ, Ryu JH, Selman M, Wells AU, Behr J, Bouros D, Brown KK, Colby TV, Collard HR, Cordeiro CR, Cottin V, Crestani B, Drent M, Dudden RF, Egan J, Flaherty K, Hogaboam C, Inoue Y, Johkoh T, Kim DS, Kitaichi M, Loyd J, Martinez FJ, Myers J, Protzko S, Raghu G, Richeldi L, Sverzellati N, Swigris J, Valeyre D, Pneumonias AECoII. An official American Thoracic Society/European Respiratory Society statement: Update of the international multidisciplinary classification of the idiopathic interstitial pneumonias. *American Journal of Respiratory and Critical Care Medicine* 2013; 188: 733–748.

2. Raghu G, Collard HR, Egan JJ, Martinez FJ, Behr J, Brown KK, Colby TV, Cordier J-F, Flaherty KR, Lasky JA, Lynch DA, Ryu JH, Swigris JJ, Wells AU, Ancochea J, Bouros D, Carvalho C, Costabel U, Ebina M, Hansell DM, Johkoh T, Kim DS, King TE, Kondoh Y, Myers J, Müller NL, Nicholson AG, Richeldi L, Selman M, Dudden RF, Griss BS, Protzko SL, Schünemann HJ, Fibrosis AEJACoIP. An official ATS/ERS/JRS/ALAT statement: idiopathic pulmonary fibrosis: evidence-based guidelines for diagnosis and management. *American Journal of Respiratory and Critical Care Medicine* 2011; 183: 788–824.

3. King TE, Bradford WZ, Castro-Bernardini S, Fagan EA, Glaspole I, Glassberg MK, Gorina E, Hopkins PM, Kardatzke D, Lancaster L, Lederer DJ, Nathan SD, Pereira CA, Sahn SA, Sussman R, Swigris JJ, Noble PW, Group AS. A phase 3 trial of pirfenidone in patients with idiopathic pulmonary fibrosis. *The New England Journal of Medicine* 2014; 370: 2083–2092.

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Introduction of recent technology of Spiral Array and hypothesis of UIP bucket

Dr. Junya Fukuoka, Japan

1) Spiral Array is a newly proposed method of tissue microarray where sections of paraffin donor block are applied instead of cored tissue cylinder. The created array can skip the transportation of donor blocks and creation of painful holes to important blocks and provide coverage of tissue heterogeneity. Lung pathology shows tremendous tissue heterogeneity especially for cases of non-neoplastic disease. The creation of tissue microarray has been challenging due to its coverage. Usage of Spiral Array may be suggested for this occasion. Benefits and shortcoming using examples of lung cases will be presented at the session.

2) Histological usual interstitial pneumonia is an important unit of idiopathic pulmonary fibrosis, however, indistinguishable UIP pattern can be seen in other conditions such as connective tissue disease (CTD) associated lung disease and hypersensitivity pneumonitis. For clinical management, separation of etiological background is considered as important, however, the reproducibility and/or interobserver agreements of the etiological separation for histologic UIP cases is questionable and true importance of such separation may be uncertain. We came up with a hypothetical idea of "UIP bucket" where UIP is a, in fact, IPF, but each case may have different accelerator for the disease. The accelerators may be CTD related inflammation inside the lung, hypersensitivity reaction inside the lung, or gastro-esophageal reflux related aspiration pneumonia. In the session, the idea will be proposed with example cases and discussions will be made based on the review of literatures.

Environment-related DPLD: A clinical overview

Kjell Toren, MD, PhD, Sweden

The lecture will address the importance of occupational and environmental exposures as risk factors for idiopathic pulmonary fibrosis and other interstitial pneumonias. Regarding idiopathic pulmonary fibroisis the importance of exposure to inorganic dust, wood dust, agricultural dust and metal dust will be reviewed. Further, other interstitial pneumonias like pulmonary alveolar proteinosis, Ardystil syndrome, Chorean humidifier disease and flock workers' lung will be presented.

This topic is of importance as the occupational burden of diffuse pulmonary lung disease may be substantial.

Learning points:
Keep searching for possible environmental and occupatiuonal causes of diffuse pulmonary lung disease
Ask about the patients' occupations
Old and new causes of disease may occur even in modern industry

Key references:
Moya C, Antá JM, Newman taylior AJ, et al. Outbreak of organising pneumonia in textile priniting sprayers. Lancet 1994 ;343 :498–502

Gustafsson T, Dahlman-Höglund A, Nilsson K, Ström K, Tornling G, Torén K. Occupational exposure and severe pulmonary fibrosis.
Respir Med 2007 ;101 :2207–2212.

Kern DG, Kuhn C, Ely EW, et al. Flock worker's lung. Chest 2000 ;117 :251–259.

Kim K-W, Ahn K, Yang HJ, et al. Humidifier disinfectant-associated children's interstitial lung disease. Am J Respir Crit Care Med 2014 ;189 :48–56.

Radiologist's approach to environment-related DPLD

Dr. Martine Rémy Jardin, France

1. Brief overview of the topic of the lecture
The radiologist's task is quite straightforward when the clinician has already linked the patient's respiratory disease and a specific occupational or environmental exposure. In this situation, the radiologist describes the underlying changes on HRCT images, thus participating in the confirmation of the suspected diagnosis. Because HRCT is a powerful imaging tool, the examination can also be helpful in identifying associated abnormalities that can participate in the patient's respiratory impairment. When the patient is simply referred for evaluation of respiratory symptoms, the radiologist will focus on a pattern-based approach as recommended for any infiltrative lung disease. Despite the broad spectrum of imaging appearances of environment-related diseases, the CT patterns of the most frequent occupational and environmental lung diseases can be recognized, subsequently allowing the radiologist's to make suggestions on a potential environmental or occupational exposure. However, one should point out that the recognition of the cause of lung disease will rely on a multidisciplinary approach and none of the CT patterns can be considered as pathognomonic of a single environment-related DLPD. In addition to the knowledge of the major environment-related DLPD, radiologists should be aware of newly described occupational exposure risks and follow regular updates of environmental exposures existing in residential and recreational settings. This lecture will review these different aspects of the radiologist's involvement in exposure-related diffuse lung disease with special interest in the recent role of medical imaging in screening and surveillance of occupational and environmental lung diseases.

2. Relevance of the lecture to the congress concept:
This lecture will describe the most frequent imaging patterns and discuss the importance of a multidiscipline approach to environment-related DPLD.

3. Key learning points:
- Environmental exposures occur most commonly at work but also exist in residential and recreational settings.
- Chest radiographs continue to be the first-line imaging modality, completed by HRCT for recognition of more precise features of lung infiltration and/or destruction.
- Social and preventive interventions are important consequences of the diagnosis made by the multidisciplinary team.

4. Key references:
-Rose CS, Lynch DA, Cool CD. Exposure-related diffuse lung disease. Sem Respir Crit Care Med 2008; 29: 620–630

-Muzaffar SA, Christiani DC. Frontiers in occupational and environmental lung disease research. Chest 2012; 141: 772–781

-Weissman DN. Role of chest computed tomography in prevention of occupational respiratory disease: review of recent literature.

-Jun JS, Jung JI, Kim HR et al. Complications of pneumoconiosis: radiologic overview. Eur J Radiol 2013; 82: 1819–1830

Histopathological update on asbestos related diseases

Dr. Richard Attanoos, United Kingdom

Asbestos is a high-profile health hazard that has been extensively used in several thousand products including textiles, insulation, cement, and friction materials. Asbestos comprises two distinct mineralogical groups (serpentine and amphiboles) with different physical, chemical and biological properties. Exposures to asbestos may result in a variety of non-neoplastic conditions (effusions, pleural plaques, diffuse pleural fibrosis and asbestosis) and neoplasia (malignant mesothelioma and lung cancer).

It is essential to accurately diagnose asbestos induced disease as many cases will be subject to medicolegal scrutiny and personal injury claims for compensation. It is important to appreciate that clinical, radiological and pathological criteria for compensatable disease may vary between experts, disciplines and locations (subject to Country or State legislation). In most cases, there is an important role in the multidisciplinary approach to diagnosing asbestos related disease.

The focus of the presentation is to illustrate the spectrum of non-neoplastic asbestos related disease with emphasis on the evolving diagnostic criteria, interpretive challenges and pitfalls.

The role, advantages and limitations of mineral analysis will be discussed.

Learning Points

• Fibre toxicity is related to cumulative dose, fibre size and fibre bio-persistence (fibre type). Asbestos fibres are ubiquitous in the environment and there is no evidence that low-level ambient exposures represent any health hazard. Industrial workplace exposures to asbestos in which disease is recorded are typically orders of magnitude above ambient exposures. It is recognised that asbestos related pleural disease (plaques, fibrosis, mesothelioma) may arise at far lower cumulative exposure than those required to induce parenchymal diseases such as asbestosis and lung cancer.

• Pathology has a pivotal role in the diagnosis of asbestosis and it is essential to adhere to criterion standards – the present 'State of the Art' criteria are those published by the College of American Pathologist – Pulmonary Pathology Society (CAP-PPS) Asbestosis Committee. This has been adopted by the updated 2014 Helsinki conference

• Exposure assessments, critical to the evaluation of asbestos related disease for compensation, are complex and difficult to determine. They may be undertaken by clinical, hygienist dose reconstruction or pathological means. None is perfect. Mineral analysis in suitable samples undertaken by electron microscopy is the best method of determining an individual's past exposure to respirable amphibole asbestos. Past exposures to chrysotile are better determined by a substantiated occupational history.

References

1. Roggli VL, Gibbs AR, Attanoos RL et al. Pathology of Asbestosis – An Update of the Diagnostic Criteria. Report of the Asbestosis Committee of the College of American Pathologists and Pulmonary Pathology Society. Arch Path Lab Med 2010; 134: 462–480

2. De Vuyst P, Karjalainen A, Dumortier P, et al. European Respiratory Society. Guidelines for Mineral Fibre Analysis In Biological Samples: Report of the ERS working group. ERJ 1998 (11); 1416–1426.

3. Attanoos RL, Alchami FS, Pooley FD Usual Interstitial Pneumonia in asbestos exposed cohorts – concurrent idiopathic pulmonary fibrosis or atypical asbestosis? Histopathology, 2016 Sep;69(3):492-8.

4. Attanoos RL. Asbestos related Lung Disease Surg. Pathol Clin.2010; 3 (1); 109 – 128.

What radiology says on hypersensitivity pneumonitis

Dr. Hans-Ulrich Kauczor, Germany

Brief overview

Hypersensitivity pneumonitis (HP) is a common interstitial lung disease (ILD) resulting from the inhalation of a large variety of antigens by susceptible individuals. The disease is best classified as acute and chronic (fibrosing or not). The broad range of presenting symptoms, radiologic, and lung biopsy findings, and lack of validated diagnostic criteria makes diagnosis challenging. Key radiological criteria include diffuse and/or centrilobular ground-glass opacities, mosaic perfusion, air trapping, fibrosis, lung cysts, and emphysema. The radiological extent of fibrosis, represented by reticulations and honeycombing, is associated with worse outcome. The histologic and radiologic features in some cases may resemble those of usual interstitial pneumonia or nonspecific interstitial pneumonia. Software-based quantitative imaging biomarkers might provide a good basis for longitudinal surveillance and therapy response imaging as well as outcome predications, in particular when used as input of complex disease models.

Relevance to the congress concept

The diagnosis of HP often is extremely challenging. The multidisciplinary establishment of specific guidelines with sets of minimum necessary "criteria" by which a patient could attain a diagnosis of acute/chronic/fibrotic HP is required. Disease models integrating the all the patient data including quantitative imaging biomarkers might improve the accuracy for a diagnosis of chronic HP, stratify different disease phenotypes and predict clinical outcome.

Key learning objectives

1) to consolidate knowledge about the challenges to diagnose HP
2) to learn about the potential and limitations of radiology (CT) in HP
3) to understand the requirement to define the necessary criteria to diagnose HP

Key references

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Pathologic differential diagnosis in hypersensitivity pneumonitis

Anja C. Roden, MD, USA

Hypersensitivity pneumonitis (HP) is an immunologically-mediated interstitial lung disease that is induced by inhaled organic antigens. HP can occur as an acute, subacute or chronic form. While patients with acute HP are usually not biopsied given the close relationship between the exposure to the inciting antigen and the occurrence of symptoms, subacute and chronic HP might be a diagnostic challenge necessitating tissue to aid in the diagnosis in some cases. The classic morphological triad of HP includes poorly formed non-necrotizing granulomas, cellular interstitial pneumonia and chronic bronchiolitis. However, these findings are not specific to HP and they are not always present making a histologic diagnosis of HP often challenging. In addition, in chronic HP, fibrosis and usual interstitial pneumonia-like features might also be encountered. This lecture will present morphologic features of HP based on cases of subacute and chronic disease. The current literature on the morphologic-clinical correlation of HP will be reviewed and the differential diagnoses of the disease including infection, aspiration, sarcoidosis, usual interstitial pneumonia, lymphoid interstitial pneumonia and non-specific interstitial pneumonia will be discussed.
The presentation of cases including a brief presentation of clinical and radiologic findings, a discussion of morphologic features and clinical follow up will emphasize the importance of the multidisciplinary approach to the diagnosis of HP and its differential diagnoses.

Key learning points
- To become familiar with the morphologic features of subacute and chronic HP
- To correlate morphologic features of HP with outcome
- To get a better understanding of the differential diagnosis of morphologic features of HP

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DPLP related to catastrophic events: Lessons from 9/11

Dr. Anna Nolan, USA

Brief Overview:
The Destruction of the World Trade Center (WTC) complex exposed thousands of first responder and residents of New York City (NYC) to toxins and particulates.(1)
A majority of the WTC related lung diseases are due to airflow obstruction but some exposed workers developed diffuse parenchymal lung disease (DPLD) such as sarcoid and fibrosis.(1-3) Furthermore, WTC exposed Fire department of New York (FDNY) first responders develop systemic autoimmune diseases which could be associated with the development of DPLD. (4)
Relevance: DPLD caused by catastrophic events such as the WTC destruction will be reviewed. Phenotypic overview will be provided and contrasted to DPLD commonly seen in practice. In addition, radiologic and pathologic correlations will also be provided.(5)

Key Learning Points:

After this Activity, participants should be able to:
1. Review the types of exposure that occurred at the WTC site and the forms of DPLD that occurred as a consequence to this exposure.
2. Discuss the clinical manifestations seen in those affected
3. Evaluate the current literature pertaining to DPLD due to catastrophic environmental exposures such as the destruction of the WTC complex

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Chronic rejection after lung transplantation

Dr. Gerdt Riise, Sweden

Background
Lung transplantation is an established treatment for end-stage lung disease. Short-term patient survival has increased in recent years, but long-term outcome is still poor with only 54% of the patients surviving more than 5 years.
Chronic rejection in the form of bronchiolitis obliterans syndrome (BOS) is the main hindrance for long-term survival. BOS is a fibro-proliferative process in the small airways leading to airflow limitation and progressive loss of lung function.
A restrictive form of chronic rejection (RAS) has also been identified with rapidly progressing loss of lung function due to fibrotic interstial and pleural pathology.
The term chronic lung allograft dysfunction (CLAD) has been introduced to describe any chronic decline in lung function, irrespective of its cause.
Despite extensive research efforts there is limited understanding of the immunological mechanisms underlying BOS. So far, no clinically useful biomarker has been identified.

Relevance
CLAD is a diffuse chronic inflammatory and fibroproliferative process in the allograft. Much of the ongoing research in the field of CLAD can give information on other chronic lung diseases, and vice versa.

Learning points
1. CLAD is the major hindrance for long-term survival after lung transplantation
2. Chronic rejection can give info on pathological inflammatory processes in the lung.
3. BOS and RAS mirror pathological processes behind COPD and ILS respectively.

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Imaging in chronic rejection

Dr. Johny A Verschakelen, Belgium

Initially the term chronic rejection was used when a lung transplantation patient developed a persistent and obstructive pulmonary function defect (> 20 % decline in forced expiratory volume in 1 second (FEV1)) for which no other cause could be found and was clinically identified as bronchiolitis obliterans syndrome (BOS). In recent years it became clear that some patients do not qualify for this definition because not every decline in FEV1 was obstructive nor irreversible and a more overarching and descriptive term (although without a true consensus) was introduced describing any chronic decline in FEV1 and/or FVC as chronic lung allograft dysfunction (CLAD). Based on a combination of physiological and radiological features different (often co-existing) phenotypes of CLAD have now been described, including obstructive CLAD (BOS), restrictive CLAD (restrictive allograft syndrome (RAS)) and graft dysfunction not related to chronic rejection. An initially as subtype of BOS considered phenotype which was called neutrophilic reversible allograft dysfunction (NRAD) and recently renamed as azithromycin-responsive allograft dysfunction (ARAD) because patients respond to azithromycin treatment with an increase of FEV1, is now considered as a potential confounder of BOS that should be identified in patients with suspected CLAD.

In this presentation:
1) a review will be given of the CT changes that may be seen in patients with (suspected) CLAD.
2) the potential role of CT to differentiate between the different CLAD-phenotypes will be discussed.
3) it will be shown how CT may be helpful to study pathogenesis and evolution of CLAD related lung changes.

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Chronic rejection: a pathologist view

M. Angeles Montero M.D., Ph.D, Spain

Lung transplantation has become the main treatment for terminal respiratory conditions, however chronic lung allograft dysfunction (CLAD) is a major limitation for its long-term success. Until recently, bronchiolitis obliterans syndrome (BOS) was considered the only form of CLAD, which comprised irreversible obstructive pulmonary tests and obliterative bronchiolitis on histology, but CLAD is now regarded as a heterogeneous condition. Restrictive physiology is now viewed as an explanation for the decreasing of the forced vital capacity, with Restrictive allograft syndrome (RAS) comprising what is considered an identical histological picture of pleuroparenchymal fibroelastosis (PPFE) as seen in non-transplanted patients.

PPFE in non-transplanted patients is a rare condition that is typically an idiopathic disorder and is now recognised as such in the revised ATS/ERS classification of idiopathic interstitial pneumonias. The histopathological features are subpleural and intra-alveolar fibrosis and elastosis of the alveolar walls, along with variable fibrous thickening of the pleura.

Despite many efforts, the aetiology of RAS still remains elusive, however the identification of PPFE in the setting of autoantibodies in some patients raises the possibility of an immunologic mechanism. The presence of fibrointimal thickening in medium size arteries and veins, in the bronchovascular bundles and lobular septa respectively, as well as some capillary loss and dilated capillaries with intravascular mononuclear cells, support the evidence of antibody mediated rejection (AMR) as a possible physiopathological mechanism of RAS/PPFE.

Key learning points:
1. CLAD comprises BOS, obliterative bronchiolitis on histology, and RAS, with PPFE as its histological picture.
2. PPFE is an idiopathic disorder now recognised in the revised ATS/ERS classification of idiopathic interstitial pneumonias.
3. Recent evidence supports AMR as a possible explanation for RAS/PPFE in transplanted patients.

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Who was Berzelius?

Jöns Jacob Berzelius, one of the most prominent natural scientists of the 19th century, was born in 1779 in Väversunda, in the county of Östergötland in southern Sweden, a region with rich cultural traditions.
Orphaned at an early age, he went to several foster-homes and received his schooling in nearby Linköping. After graduating in medicine at the University of Uppsala, he moved to Stockholm, where he became assistent master without pay at the so-called »Surgical School«, and worked as a doctor for poor people. At the age of 28 he became professor of medicine and pharmacy.

In 1808 Berzelius was one of the seven men who founded The Swedish Society of Medicine »For the perfection of science through mutual mediation of knowledge and collective experience, for the promotion of friendly confidence between doctors«.

Berzelius have enriched our knowledge of nature of life phenomena, established the atomic weights of most of the known elements, presented his electrochemical theory for the understanding of the nature of chemical compounds and laid the foundation for the sciences of the chemistry of rock types. He also found that elements combine with each other according to fixed numerical relationships. In addition to this, in his striving for order and method, with his talent for simplicity and clarity in expression, he created the chemical symbolic language in 1813, which since that time has been an essential instrument of chemistry.
With time he became a practised lecturer but preferred to express himself in writing and this he did superbly. Impressive are the great scientific works where he also demonstrated his interest and ability to spread knowledge about the latest advances of natural sciences.

Berzelius delight in research and debate was united with a great humility before the great scientific questions. Both his attitude and artistry of formulation is illustrated by the following passage in his Manual of Cheamistry (vol 3, 1818):

»All our theory is but a means of conistently conceptualizing the inward processes of phenomena, and it is presumable and adequate when all scientifically known facts can be deduced from it. This mode of conceptualization can equally well be false and, unfortunately, presumable is so frequently. Even though, at a certain period in the development of science, it may match the purpose just as well as a true theory. Experience is augmented, facts appear which do not agree with it, and one is forced to go in search of a new mode of conceptualization within which these facts can also be accomodated; and in this manner, no doubt, modes of conceptualization will be altered from age to age, as experience is broadened, and the complete truth may perhaps never be attained. But even if the goal can never be reached, let us never abondon our endeavor to get closer to it.«

Parts of this text is found in: Berzelius – Creator of the chemical language, by Carl Gustaf Bernhard, the Royal Swedish Academy of Sciences

History of the SSM building

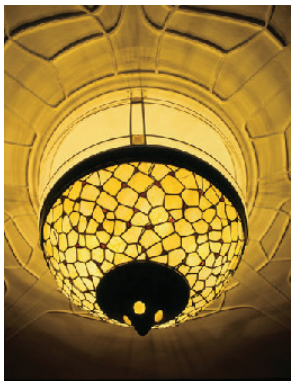
In 1879, the Swedish Society of Medicine moved from what was then the home of Karolinska Institutet at Norr Mälarstrand to its own premises in Jakobsgatan in Stockholm. It soon outgrew this location and a search for new premises was resumed. On Walpurgis night in 1889, six men were inside the Katarina lift at Slussen in Stockholm.

A fault developed in the machinery, causing the lift cage to fall. One of the passengers, Carl Westman, was injured, but a fellow passenger, Johan Rissler, a surgeon and member of the building committee of the Society of Medicine, immediately assisted him.

In 1904, the Society announced an architectural competition for a building on a site it had purchased in Klara Östra Kyrkogata.
The winner was Carl Westman, and the building was finished two years later.

The Society's building which dates from 1906, was a breakthrough for the architect Carl Westman and the national romantic style architecture he favoured.

The building itself is work of art – from its facade of handmade brick and Christian Eriksson's granite reliefs in the entrance to its mosaic floors, carved balustrades, chandeliers, and ventilation grilles – all Westman signatures. The building today is a Swedish, turn of the century architectural treasure.



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