CHEST®

Official publication of the American College of Chest Physicians

Pneumothorax Following Transthoracic Fine-Needle Aspiration of the Lung

Ryland P. Byrd, Jr and Thomas M. Roy

Chest 2005;128;1071-1072
DOI 10.1378/chest.128.2.1071

disease with the MAGE expression profile in primary tumor tissue, venous blood, and bone marrow aspirates of cancer patients in several cohorts. Subsequently, a detailed statistical assessment of the data sets has to be done, and the proposed method for summarizing the patterns of MAGE expression may be helpful for the generation of a later decision model. However, far more clinical experience is needed with this approach, and quantitative data will have to be included in the final analysis as relevant carrier of additional information.

Ingo Mecklenburg, MD
Peter Kufer, MD
Ludwig-Maximilians-University
Munich, Germany

Correspondence to: Ingo Mecklenburg, MD, Institut für Immunologie, Goethestrasse 31, 80336 Munich, Germany; e-mail: ingo.mecklenburg@med.uni-muenchen.de.

References

1 Mecklenburg I, Stratakis DF, Huber RM, et al. Detection of melanoma antigen-A expression in sputum and bronchial lavage fluid of patients with lung cancer. Chest 2004; 125: 164S–166S
2 Ohman Forslund K, Nordqvist K. The melanoma antigen genes: any clues to their functions in normal tissues? Exp Cell Res 2001; 265:185–194
3 Xiao J, Chen HS. Biological functions of melanoma-associated antigens. World J Gastroenterol 2004; 10:1849–1853

Pneumothorax Following Transthoracic Fine-Needle Aspiration of the Lung

To the Editor:

We read with interest the article by Choi et al[1] (November 2004) concerning the incidence and risk factors of delayed pneumothorax after transthoracic fine-needle aspiration (FNA) of pulmonary lesions guided by a variety of radiologic techniques. The article is both well written and informative. As the authors indicate in their discussion, it is a common practice to obtain a chest radiograph typically 4 h after percutaneous transthoracic needle aspiration.[2]

The incidences of early and delayed pneumothorax in the study by Choi et al were 18.6% and 3.3%, respectively. However, all of the patients in their study who required tube thoracostomy for delayed pneumothorax were symptomatic. This represents $< 1\%$ of their study population. Interestingly, none of the patients in this study with delayed pneumothorax had undergone CT-guided FNA. We have previously reported that immediate pneumothorax develops in 17.2 to 24.1% of patients undergoing CT-guided FNA.[3,4] In our studies, delayed pneumothorax occurs in 2.5 to 3.1% of patients who underwent this procedure. Moreover, delayed pneumothorax requiring intervention with pleural space evacuation occurred in 1.3 to 1.6% of patients.

While we agree the conclusion of Choi et al that late pneumothorax is clinically important, the practice of obtaining a delayed postprocedure chest radiograph after CT-guided FNA does not appear to be an efficient use of resources. Based on our research, chest radiography appears to add little information regarding lung expansion to that obtained by CT at the end of CT-guided FNA. Instruction to seek medical attention in the event of symptoms of pneumothorax appears be a more effective method of addressing this potential complication.

Ryland P. Byrd, Jr, MD
Quillen Veterans Affairs Medical Center
Mountain Home, TN
Thomas M. Roy, MD
Quillen College of Medicine
East Tennessee State University
Johnson City, TN

Correspondence to: Ryland P. Byrd, Jr, MD, Veterans Affairs Medical Center 111-B, PO Box 4000, Mountain Home, TN 37684-4000; e-mail: Ryland.Byrd@med.va.gov

References

1 Choi CM, Um SW, Yoo CG, et al. Incidence and risk factors of delayed pneumothorax after transthoracic needle biopsy of the lung. Chest 2004; 126:1516–1521
2 Perlmutt LM, Braun SD, Newman GE, et al. Timing of chest film follow-up after transthoracic needle aspiration. AJR Am J Roentgenol 1986; 146:1049–1050
3 Byrd RP Jr, Fields-Ossirio C, Roy TM. Delayed chest radiographs and the diagnosis of pneumothorax following CT-guided fine needle aspiration of pulmonary lesions. Respir Med 1999; 93:379–381
4 Shantazeerappa HN, Mathai MG, Byrd RP Jr, et al. Intervention in patients with pneumothorax immediately following CT-guided fine needle aspiration of pulmonary nodules. Med Sci Monit 2002; 8:401–404

To the Editor:

In our study (November 2004),[1] most transthoracic needle biopsies were performed by fluoroscopic and ultrasonography guidance. Only 4.5% of the transthoracic needle biopsies (21 of 458) were performed with CT scan guidance. Therefore, it is difficult to compare our data to the data of Byrd et al[2] and Shantaveerappa et al.[3]

Definitely, CT scanning is more sensitive than a posteroanterior chest radiograph for the detection of pneumothorax. After the analysis of our data, we had a suspicion that a delayed pneumothorax was simply so small and localized that it would go undetected by a posteroanterior chest radiograph at 4 h but would be detectable by CT scan if one were performed.

However, according to his previous work, delayed pneumothorax still occurred in 4 of 158 patients (2.53%) even though a CT scan was performed after the CT scan/fine-needle aspiration.[3] This rate is not so different from that in our study[1] (3.3%; 15 of 458). The study by Shantaveerappa et al[3] helps us to resolve our suspicion. Furthermore, the rate of intervention was even higher (all patients, 1.7% [2 of 158]; patients with delayed pneumothorax, 50% [2 of 4]) in their study[3] than that in ours (all patients, 0.65% [3 of 458]; patients with delayed pneumothorax, 20% [3 of 15]).[1] I think that the two studies showed quite similar data but different interpretations.

Choon-Taek Lee, MD, PhD
Seoul National University Bundang Hospital
Gyeonggi-Do, Korea

Correspondence to: Choon-Taek Lee, MD, PhD, Seoul National University Bundang Hospital, 300 Gumi-Dong, Bundang-Gu, Seongnam, Gyeonggi-Do 463-707, Korea; e-mail: ctlee@snu.ac.kr.

References

1 Choi CM, Um SW, Yoo CG, et al. Incidence and risk factors of delayed pneumothorax after transthoracic needle biopsy of the lung. Chest 2004; 126:1516–1521
2 Byrd RP Jr, Fields-Ossorio C, Roy TM. Delayed chest radiographs and the diagnosis of pneumothorax following CT-guided fine needle aspiration of pulmonary lesions. Respir Med 1999; 93:379–381
3 Shantaveerappa HN, Mathai MG, Byrd RP Jr, et al. Intervention in patients with pneumothorax immediately after CT-guided fine needle aspiration of pulmonary nodules. Med Sci Monit 2002; 8:401–404

Interleukin-1β Gene Polymorphisms Associated With COPD

To the Editor:

We read with interest the article by Hegab et al.[1] Although interleukin (IL)-1β is also thought to be one of the important cytokines in COPD, Joos et al[2] failed to show the relationship between functional single nucleotide polymorphisms at the IL-1β promotor gene at position −511. However, since their reports were published, there was an explosion of reports revealing the association between *IL-1β* −511 polymorphisms and a variety of diseases ranging from psychological[3] to dental disease.[4] Therefore, completely healthy elderly people were recruited from a large health survey in the Sendai area.

To elucidate the genotype of *IL-1β* polymorphisms at positions −31 and −511, polymerase chain reaction and restriction enzyme fragment length polymorphisms were performed on blood samples.[2,3] Table 1 shows the characteristics of 85 COPD patients and 68 healthy subjects and the distribution of *IL-1β* −511 and −31 genotypes in these two groups. The T allele at −511 SNP ($p = 0.02$) was overrepresented in the healthy control subjects. The homozygote subjects were particularly at lower risk for COPD, with an odds ratio of 0.43 for −511 C/C and T/C. Although *IL-1β* −31 and −511 loci had linkage disequilibrium, there were no differences on the C allele −31 and genotyped frequency of *IL-1β* between the COPD patients and the control subjects.

Different from the report Joos et al,[2] we showed a significant association between only the T allele at −511 SNP and susceptibility to COPD. First, because both the COPD patients and the control subjects were elderly, we suspect that the *IL-1β* −511 loci strongly affects susceptibility to COPD with chronic inflammation for a long time. Second, since there is much evidence linking *IL-1β* −511 polymorphisms and other diseases,[3–5] healthy elderly smokers who have not had these diseases may have the T allele at −511 SNP. Since there were no differences in the distribution of *IL-1β* polymorphisms at position −31 between the COPD patients and the control subjects, we believe that our sample size was not large enough to detect small differences between the two groups.

Table 1—*IL-1β Genotypes in Healthy Control Subjects and COPD Patients

Characteristics/Polymorphism	Control Subjects (n = 68)	Patients (n = 85)	p Value	Odds Ratio	95% Confidence Interval
Age, yr	65.6 ± 10.6	66 ± 8.6	0.80		
Pack-years	44 ± 17	47 ± 14	0.67		
Gender					
Male	66 (97.1)	82 (95.3)			
Female	2 (2.9)	3 (4.7)	0.83		
Pulmonary function					
FVC (% of predicted)	98 ± 5	84 ± 7	0.11		
FEV_1/FVC, %	95 ± 5	47 ± 2	0.01		
IL-1β -511 (T-C)					
Genotypes					
T/T	25 (36.8)	17 (20.0)	0.02	0.43	0.20–0.89
T/C	29 (42.6)	42 (49.4)	0.40	1.31	0.69–2.50
C/C	14 (20.6)	26 (30.6)	0.16	1.70	0.80–3.59
Alleles					
C	57 (41.9)	94 (55.3)			
T	79 (58.1)	76 (44.7)	0.02	0.58	0.37–0.92
IL-1β -31 (T-C)					
Genotypes					
C/C	20 (29.4)	20 (23.5)	0.41	0.74	0.36–1.52
T/C	32 (47.1)	40 (47.1)	0.79	0.92	0.48–1.75
T/T	16 (23.5)	25 (29.4)	0.41	1.35	0.65–2.81
Alleles					
C	72 (52.9)	80 (47.1)			
T	64 (47.1)	90 (52.9)	0.31	1.27	0.81–1.99

*Data are presented as mean ± SEM or No. (%) unless otherwise indicated.

Pneumothorax Following Transthoracic Fine-Needle Aspiration of the Lung

Ryland P. Byrd, Jr and Thomas M. Roy

Chest 2005;128; 1071-1072
DOI 10.1378/chest.128.2.1071