Contents lists available at SciVerse ScienceDirect

Respiratory Investigation

journal homepage: www.elsevier.com/locate/resinv

Case report

Fatal acute interstitial pneumonia in a worker making chips from wooden debris generated by the Great East Japan earthquake and tsunami

Shinya Ohkouchi[a,*], *Masahito Ebina*[a,1], *Katsuhiko Kamei*[b,1], *Hiroshi Moriyama*[c,1], *Tokiwa Tamai*[a], *Risa Shibuya*[a], *Masakazu Ichinose*[a], *Toshihiro Nukiwa*[a,d]

[a]*Department of Respiratory Medicine, Graduate School of Medicine, Tohoku University, Japan*
[b]*Medical Mycology Research Center, Chiba University, Japan*
[c]*Division of Respiratory Medicine, Graduate School of Medical and Dental Sciences, Niigata University, Japan*
[d]*South Miyagi Medical Center, Japan*

ARTICLE INFO

Article history:
Received 9 May 2012
Received in revised form
31 August 2012
Accepted 4 September 2012
Available online 19 October 2012

Keywords:
Tsunami
Acute interstitial pneumonia
Hypersensitivity pneumonitis
Environmental
Occupational

ABSTRACT

A man was admitted to our hospital with shortness of breath. He was involved in making wood chips from contaminated debris created by the tsunami that occurred after the Great East Japan Earthquake. Fungi detected at his home and workplace were possible inducers of hypersensitivity pneumonitis, but the absence of precipitating antibodies countered this diagnosis. His rapid and progressive clinical course and surgical lung biopsy and bronchoalveolar lavage findings suggested acute interstitial pneumonia. Electron probe X-ray microanalysis revealed the deposition of excessive exogenous substances in bronchiolar regions. Inhalation of harmful materials was suspected to be the cause of acute lung injury.

1. Introduction

The Great East Japan Earthquake hit the East coast of the main island of Japan on March 11, 2011. It was 1 of the most disastrous earthquakes (magnitude 9.0) in world history. Approximately 20,000 people were affected by this event, and almost all of them were killed by the tsunami [1]. Immediately after the disaster, the main problem in the respiratory units in hospitals was a 2–3-fold increase in the incidence of community-acquired pneumonia (CAP) compared with the same period in 2010. Half of all CAP patients came from overcrowded shelters [2].

Currently, a major problem impeding recovery from this tragedy is the impossibility of removing the large amount of

Abbreviations: CAP, community-acquired pneumonia; AIP, acute interstitial pneumonia; BALF, bronchoalveolar lavage fluid; BFS, bronchial fiberscope; EPMA, electron probe X-ray microanalysis

*Correspondence to: Department of Respiratory Medicine, Graduate School of Medicine, Tohoku University, 1-1 Seiryo-machi, Aoba-ku, Sendai, Miyagi 9808574, Japan. Tel.: +81 227 178 539; fax: +81 227 178 549.
E-mail address: ohkouchi@idac.tohoku.ac.jp (S. Ohkouchi).

[1] Contributed equally.

2212-5345/$ - see front matter © 2012 The Japanese Respiratory Society. Published by Elsevier B.V. All rights reserved.
http://dx.doi.org/10.1016/j.resinv.2012.09.001

debris from the disaster area. It will take overs 20 years for the local industrial waste companies to dispose of the debris created by the tsunami (Fig. 1A). Moreover, the tsunami resulted in radioactive contamination of the soil due to the transport of radioactive material released during an accident at the Fukushima-Daiichi atomic power plant. Regrettably, other municipalities refused to receive any debris, even that without excessive radioactive contamination, because of the opposition from residents. Therefore, workers employed by the local industrial waste disposal companies were overworked.

In this study, we report the case of a male construction worker who crushed wooden debris to create wood chips. This man developed acute interstitial pneumonia (AIP) and died within 6 months of the disaster. Immediately after the disaster, massive wooden debris containing huge amounts of unknown impurities was urgently delivered to his construction company. He was forced to work every day without a day off to rest, and he regularly did not use his mask because of discomfort despite the recommendations of his company and the government. Accidental deaths occurring during the disposal of debris generated by the tsunami, such as that which occurred in this case, should be evaluated in the future.

2. Case report

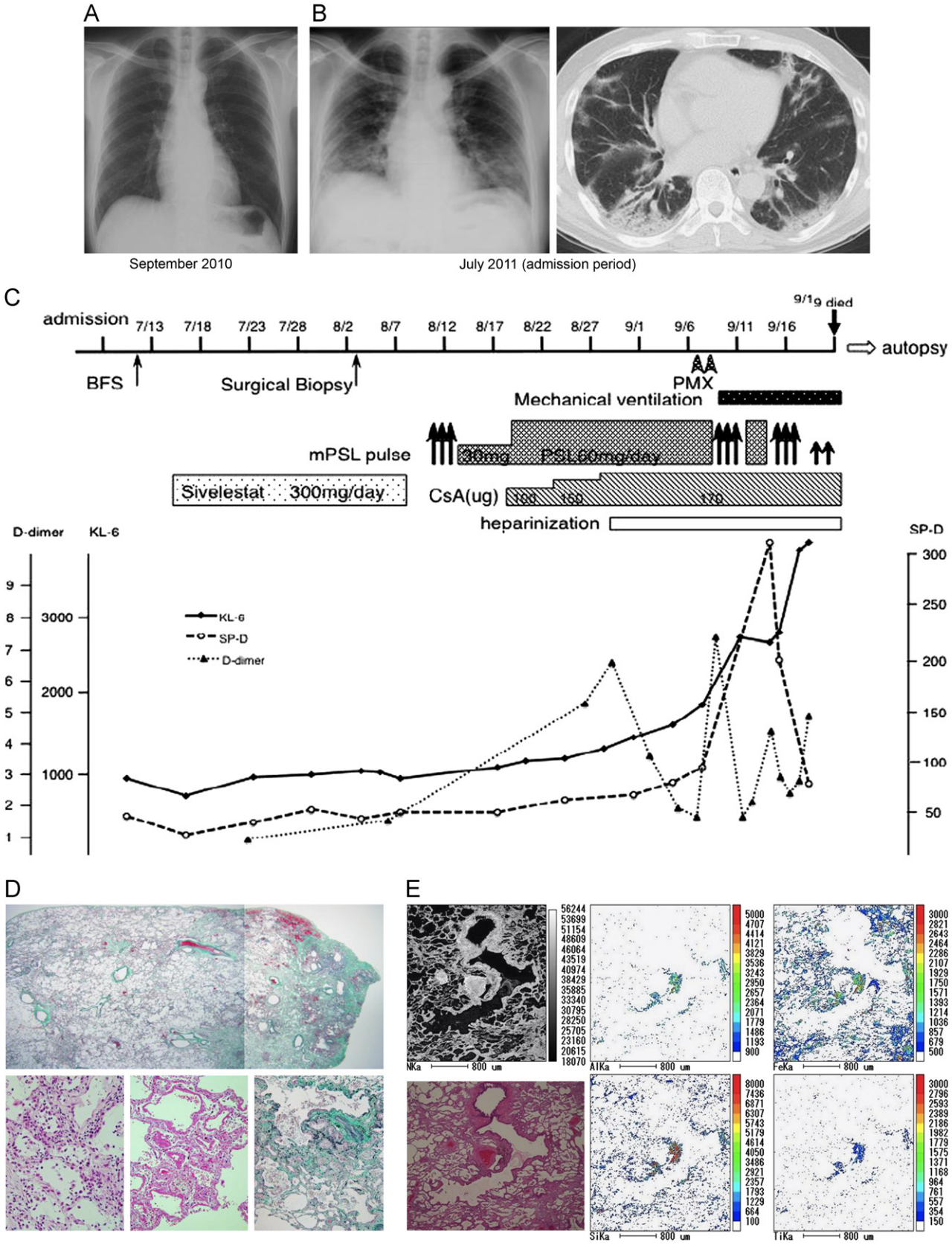
A 61-year-old man began working for an industrial waste disposal company in the Miyagi Prefecture in Japan in November

Fig. 1 – Characteristics of the tsunami caused by the Great East Japan Earthquake and the job of the patient in a waste disposal company. (A) The destructive tsunami that hit the East coast of the main island of Japan (right). Massive debris, including substances derived from human activities, covered the vast area damaged by the tsunami (left). (B) The wooden chips were generated from wooden waste using heavy equipment (right) and a shredder (left). In a typical situation, wooden waste is separated from contaminants. However, immediately after the disaster, the waste included high levels of impurities.

Fig. 2 – Clinical course and pathologic findings of the patient. A. His chest radiographs taken before the onset of disease were mostly normal. B. The chest radiograph (left) and computed tomographic scan (right) taken at admission revealed reduced lung volume, predominantly dorsal consolidation in both lungs, and traction bronchiectasis. C. Changes in serum levels of SP-D, KL-6, and D-dimer in the clinical course after admission. The various treatments applied were ineffective. D. The tissues obtained by surgical lung biopsy revealed focal distribution of organizing fibrosis in subpleural lesions (Upper panel, Elastica-Masson stain ×12.5). Proliferative-phase diffuse alveolar damage with thickening of the alveolar walls was uniformly observed in the fibrotic lesions in the biopsied tissues (lower left panel, H&E stain ×400). Hyaline membrane in the exudative phase of diffuse alveolar damage was broadly distributed throughout the autopsied lung tissues (lower middle panel, H&E stain ×400). Subepithelial fibrosis and shedding of airway epithelial cells in bronchioles were observed in both biopsies (lower right panel, Elastica-Masson stain ×200) and autopsied lung tissues and suggested chronic bronchiolitis. E. Electron probe X-ray microanalysis revealed excessive silica, aluminum, iron (ferrous oxide), and titan deposition. These findings suggest that the patient inhaled large amounts of contaminated materials while working.

2010. He was a smoker for 42 pack-years. He worked as a heavy equipment operator who crushed wood received from house-wrecking companies into wood chips to generate biomass fuel for boilers in regional plants (Fig. 1B). He had no previous respiratory illness before the earthquake, and his chest radiograph taken in September 2010 was normal (Fig. 2A).

The Great East Japan Earthquake damaged his house and forced him to reside in a storage shed that was extremely



musty and humid. Although his company usually separated wooden waste from impurities in typical situations, the large amount of wooden debris created by the tsunami was contaminated with unknown materials. Because of this emergency, he was obligated to work daily without a day off for rest. He used sealed masks, such as an N95; however, he occasionally removed the mask because of unpleasantness. On several occasions, he was subjected to irritating odors when he accidentally crashed into contaminated mold-infested cob walls and unknown chemical cans in farmhouses.

In the middle of May 2011, he experienced shortness of breath and underwent a medical examination at another hospital. He was found with bacterial or atypical pneumonia and treated with antibiotics, including a second-generation cephem, a quinolone, and a macrolide; however, his condition gradually worsened. At the beginning of July, he was admitted to our hospital. Physical examination showed that his breathing rate was slightly elevated and weak fine crackles were audible in his lower back area. His laboratory findings revealed mild hypoalbuminemia, hypoxia, hypercapnia, and his serum levels of CRP and KL-6 were elevated. His physical and serological test findings were not indicative of collagen disorders. The bronchoalveolar lavage fluid (BALF) collected using a bronchial fiberscope (BFS) did not contain any pathogenic microorganisms, and only a mild increase in neutrophils was detected. Significant restrictive impairment (vital capacity) was observed in his pulmonary function test (Table 1).

His chest radiographic and computed tomographic findings revealed decreased lung volume and traction bronchiectasis with consolidation primarily in the bilateral lower dorsal lung field (Fig. 2B).

A transbronchial lung biopsy using a BFS revealed mild alveolitis (data not shown). First, we suspected subacute hypersensitivity pneumonitis and lung injury caused by exposure to nonspecific antigens or infectious microorganisms contaminating the wooden debris he worked with. Therefore, a carbapenem and a neutrophil elastase inhibitor (sivelestat) were administered, but his condition did not improve (Fig. 2C). An environmental survey was performed of his shed and workplace, and we detected *Aspergillus niger* and *Mucor* species, both of which have been reported as latent causes of hypersensitivity pneumonia due to wood chip exposure [3–8]. However, we did not detect any precipitated antibodies against these pathogens (data not shown).

At the beginning of August, a surgical lung biopsy of three segments (S2, S4, and S9) of his right lung was performed via video-assisted thoracic surgery. Low-magnification views revealed localized organizing fibrosis chiefly in subpleural lesions and mild emphysematous changes in each of the three biopsied lung tissues (Fig. 2D, upper). The localized fibrotic lesions contained various phases of diffuse alveolar damage, most of which were organizing phase (Fig. 2D, lower left), without apparent hyaline membrane formation, which was frequently observed in the autopsied lung tissue of this patient (Fig. 2D, lower middle). The shedding of epithelial cells and subepithelial fibrosis in the bronchioles were observed in both the biopsies (Fig. 2D, lower right) and in the autopsied lung tissues of this patient. Although bronchiolitis with mild lymphocyte infiltration was observed, no firm granulomas indicative of acute hypersensitivity pneumonitis were observed. Finally, we diagnosed these pathologic findings as AIP and administered additional clinical treatment against AIP (Fig. 2C). Despite steroid therapy (methylprednisolone pulse therapy for three days followed by a high dose of oral prednisolone), cyclosporine A, anticoagulants (heparin), endotoxin hemodiafiltration using polymixin B-immobilized fiber columns, and mechanical ventilation, his condition worsened rapidly and irreversibly. He died 2 months after admission (Fig. 2C). After his death, the analysis of his biopsied lungs by electron probe X-ray microanalysis (EPMA) revealed excessive silica and metal deposits in the bronchiolar lesions (Fig. 2E). Excessive deposits of cobalt and tungsten suggestive of hard

Table 1 – Laboratory findings at admission period.

Hematology		**Serology**	
WBC	6000/μl	CRP	3.1 mg/dl
Neut	68%	PCT	0.39 ng/ml
Eos	2%	KL-6	752 U/ml
Baso	0%	SP-D	20.7 ng/ml
Lymh	17%	SP-A	73.6 ng/ml
Mono	12%	β-D-glucan	<4 pg/ml
RBC	405×10^3/μl	M. Pneumonia	1.57 (INDEX)
Hb	12.7g/dl	M. Pneumonia	2.07 (INDEX)
Hct	37.7%	Aspergillus EIA	(-)
PLT	174×10^3/μl	Mycoplasma Ab	(-)
		BNP	<5.8 pg/ml
		RA	(-)
		ANA	(-)
Biochemistry		IgG	858mg/dl
TP	5.3 g/dl	IgA	262 mg/dl
Alb	2.8 g/dl	IgM	94 mg/dl
AST	36 IU/L	Ds DNA	<10.0 IU/ml
ALT	36 IU/L	RNA Ab	(-)
LDH	208 IU/L	Sm Ab	(-)
ALP	397 IU/L	SS-A Ab	(-)
T-Bil	0.8 mg/dl	SS-B Ab	(-)
BUN	11 mg/dl	Scl-70 Ab	(-)
Cr	0.7 mg/dl	P-ANCA	<1.3 U/ml
Na	142 mEq/L	C-ANCA	<3.4 U/ml
K	3.5 mEq/L	C3	121 mg/dl
Cl	106 mEq/L	C4	44.2 mg/dl
		CH50	75.0 U/ml
		Blood gas analysis (room air)	
BALF		pH	7.440
Cell no.	3.4×10^7	PaO_2	76.0 Torr
Cell no./ml	4.5×10^5	$PaCO_2$	34.1 Torr
BALF			
AM	83%	HCO^{3-}	22.8 mmol/L
Neu	12%	SaO_2	95.6 %
Lym	3%	P/F ratio	362
Eo	2		
CD4/8 ratio	1.55		
		Pulmonry function	
		VC	2.08 L
		VC (%)	58.1%
		$FEV_{1.0}$	1.88 L
		$FEV_{1.0}$ (%)	91.7%
Sputum culture		RV/TLC	45.72%
Normal flora		DLCO (%)	65.7%

metal lung were not observed (data not shown). Autopsy also showed a mosaic pattern with less and severe damaged area containing uniform widening of the alveolar septa with hyaline membrane (Fig. 2D, lower middle). We concluded that the inhalation of harmful materials from the wooden debris might have induced acute lung injury that subsequently led to acute interstitial pneumonia.

3. Discussion

In this case report, we introduced a man who produced wood chips from wooden waste generated by the tsunami. He developed AIP and died within 6 months of the first occurrence of symptoms. No pathogens responsible for the lung injury were detected in our survey; however, we believe that the cause of the disease was alveolar damage induced by harmful contaminating substances present in the wood chips based on the BALF, pathological, pulmonary function, and EPMA findings and his clinical course. In particular, titan deposits in the patient's lung suggested that he had inhaled large amounts of contaminated materials while working because this type of deposit is rarely detected in normal subjects in an ordinary environment. Although hypersensitivity pneumonitis caused by wood chips contaminated with fungi was suspected, the diagnosis was disproven by the lack of precipitating antibodies in addition to the findings of firm granuloma and bronchiolitis with lymphocyte infiltration in the pathological analysis. However, fungi have been reported as a cause of hypersensitivity pneumonitis in woodworkers.

In major saltwater disasters such as the 2004 Sumatra–Andaman Earthquake, Hurricane Katrina in New Orleans, and the Great East Japan Earthquake, the incidence of common respiratory diseases, including CAP, exacerbation of bronchial asthma, and chronic obstructive disease, increased, but that of interstitial lung diseases and malignancies did not according to some reports [9–11]. Increases in the incidence of these diseases was also not observed after the World Trade Center attacks, despite the atmospheric pollution caused by the huge dust clouds [12–14]. Although the reasons for these findings are unknown, the use of masks may have prevented parenchymal diseases or the observation time (<12 years) may be too short to accurately survey the occurrence of these diseases [15].

A manual explaining the procedures for disposing of waste and debris generated by tsunamis was issued by the Ministry of the Environment of Japan in May 2011. Rough classification of garbage by type, removal of dangerous materials from garbage, and the use of masks to prevent lung diseases caused by dust and gas are recommended in the manual (http://www.env.go.jp) [15–17]. It also notes that low-grade wood chips will not be accepted for use as biomass fuel in factories and recommends the temporary storage of wooden garbage in other prefectures until final disposal. The unnecessary production of wood chips from contaminated debris can be prevented if large amounts of debris can be stored safely away from residential areas. Disease would have been prevented in this case if the patient had continued to wear his mask. The careful handling of debris in accordance with the manuals released by the government should be rigidly performed in the future.

Conflict of interest

Toshihiro Nukiwa received lecture fees from Boehringer Ingelheim.

Shinya Ohkouchi, Masahito Ebina, Katsuhiko Kamei, Hiroshi Moriyama, Tokiwa Tamai, Risa Shibuya, Masakazu Ichinose, they have no potential conflict of interest.

Acknowledgments

The authors thank the patient's wish donating his body for medical research. The authors also thank his family and colleagues for assistance of survey of pathogens.

REFERENCES

[1] Tamura Y, Fukuda K. Earthquake in Japan. Lancet 2011;377:1652.
[2] Suzuki M, Uwano C, Ohrui T, et al. Shelter-acquired pneumonia after a catastrophic earthquake in Japan. J Am Geriatr Soc 2011;59:1968–70.
[3] Furumiya J, Nishimura H, Nakanishi A, et al. Postmortem endogenous ethanol production and diffusion from the lung due to aspiration of wood chip dust in the work place. Leg Med 2011;13:210–2.
[4] Dykewicz MS, Laufer P, Patterson R, et al. Woodman's disease: hypersensitivity pneumonitis from cutting live trees. J Allergy Clin Immunol 1988;81:455–60.
[5] van Assendelft AH, Raitio M, Turkia V, et al. Fuel chip-induced hypersensitivity pneumonitis caused by penicillium species. Chest 1985;87:394–6.
[6] Wintermeyer SF, Kuschner WG, Wong H, et al. Pulmonary responses after wood chip mulch exposure. J Occup Environ Med 1997;39:308–14.
[7] Alwis KU, Mandryk J, Hocking AD, et al. Exposure to biohazards in wood dust: bacteria, fungi, endotoxins, and (1–3)-beta-D-glucans. Appl Occup Environ Hyg 1999;14:598–608.
[8] Mandryk J, Alwis KU, Hocking AD, et al. Work-related symptoms and dose-response relationships for personal exposures and pulmonary function among woodworkers. Am J Ind Med 1999;35:481–90.
[9] Wattanawaitunechai C, Peacock SJ, Jitpratoom P, et al. Tsunami in Thailand-disaster management in a district hospital. N Engl J Med 2005;352:962–4.
[10] Guha-Sapir D, van Panhuis WG. Health impact of the 2004 Andaman Nicobar earthquake and tsunami in Indonesia. Prehosp Disaster Med 2009;24:493–9.
[11] Riggs MA, Rao CY, Brown CM, et al. Resident cleanup activities, characteristics of flood-damaged homes and airborne microbial concentrations in New Orleans, Louisiana, October 2005. Environ Res 2008;106:401–9.
[12] Perlman SE, Friedman S, Galea S, et al. Short-term and medium-term health effects of 9/11. Lancet 2011;378:925–34.
[13] Jordan HT, Brackbill RM, Cone JE, et al. Mortality among survivors of the Sept 11, 2001, World Trade Center disaster: results from the World Trade Center Health Registry cohort. Lancet 2011;378:879–87.

[14] Wisnivesky JP, Teitelbaum SL, Todd AC, et al. Persistence of multiple illnesses in World Trade Center rescue and recovery workers: a cohort study. Lancet 2011;378:888–97.
[15] Antao VC, Pallos LL, Shim YK, et al. Respiratory protective equipment, mask use, and respiratory outcomes among World Trade Center rescue and recovery workers. Am J Ind Med 2011;54:897–905.
[16] Wu M, Gordon RE, Herbert R, et al. Case report: lung disease in World Trade Center responders exposed to dust and smoke: carbon nanotubes found in the lungs of World Trade Center patients and dust samples. Environ Health Perspect 2010;118: 499–504.
[17] Rom WN, Reibman J, Rogers L, et al. Emerging exposures and respiratory health: World Trade Center dust. Proc Am Thorac Soc 2010;7:142–5.