

9.1 Introduction

9.1.1 The History of Its Prevalence and Impacts

In March 2009, an outbreak of human infected swine influenza, first name by WHO, occurred in Mexico that rapidly spread worldwide. On Apr 30, 2009, the WHO, the United Nations Food and Agriculture Organization and the World Organization for Animal Health agreed to use the name of influenza A (H1N1), which was then used in bulletins issued by Ministry of Health in China.

In the context of international financial crisis, influenza A (H1N1) spread worldwide, posing new variables to the already bleak world economic outlook. So far, strict measures for prevention and control of influenza A (H1N1) have been adopted in China. To some extent, thanks to the transparent information system on infectious diseases as well as emergency precaution and management mechanism established by the Chinese government, the spread of influenza A (H1N1) in China has been well controlled.

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9.1.2 Etiology and Epidemiology

The first case of influenza A (H1N1) in the United States was confirmatively diagnosed via laboratory diagnosis on Apr 15, 2009. The researchers discovered that influenza A (H1N1) virus is a complex virus composed of 4 variant viruses, including 1 avian influenza virus, 1 common influenza virus and 2 swine influenza viruses that widely spread in pigs. Meanwhile, the researchers proved that rearrangement and recombination of influenza A (H1N1) virus genes commonly and extensively exist. Currently, the pathogenesis of influenza A (H1N1) virus has attracted focused attention by scholars both internationally and within China. It has been demonstrated that apoptosis of host cells induced by influenza A (H1N1) virus contributes to its pathogenesis. Many studies have proved that influenza A (H1N1) virus induces apoptosis that is mediated via Caspase approach, which can be regulated by multiple channels, multiple factors and multiple genes. It has been also proved that influenza A (H1N1) virus induces apoptosis with a complex process.

On Apr 30, 2009, influenza A (H1N1) was listed as a class B infectious disease and an infectious disease receiving frontier health inspection and quarantine, which should then be managed as the class A infectious diseases in China.

For the epidemic of influenza A (H1N1), the patients with influenza A (H1N1) are the source of its infection and the virus spreads from person to person along with respiratory aerosols and droplets. The populations are generally susceptible to the infection, and the young and middle-aged adults are especially vulnerable. Factors influencing its prevalence include host factors, virus factors, social conditions, health care insurance, and other natural and social factors.

9.1.3 Clinical Manifestations and Diagnosis

Human infected influenza A (H1N1) commonly has an incubation period of 1–7 days, which is comparatively longer than common influenza and human infected avian influenza.

The symptoms of influenza A (H1N1) range from normal body temperature and mild upper respiratory infection to severe or fatal pneumonia. Most of the patients experience typical influenza like symptoms and self heal. Severe and death cases are mostly caused by occurrence of complications, including preliminary viral pneumonia and secondary bacterial pneumonia.

The most common symptoms include cough, fever, sore throat, runny nose, nasal obstruction, cough, expectoration, headache, systemic soreness, and fatigue. Some patients may experience vomiting and/or diarrhea. Mild upper respiratory symptoms rarely occur, but no fever. And the physical signs include pharyngeal congestion and enlarged tonsils.

Its diagnosis is mainly based on the epidemiology, clinical manifestations and etiological examination. The inpatients show increased or decreased WBC count; severe cases commonly show decreased total WBC count, lymphocytes, and platelets. Other diagnostic examinations include serological test, virus isolation (such as throat swab as well as specimens of oral gargle, nasopharyngeal or tracheal aspirates, sputum, and lung tissue), reverse transcription polymerase chain reaction (RT-PCR) are also important ways for the diagnosis of influenza A (H1N1). Early detection and early diagnosis are the keys for its prevention, control and effective treatment.

9.1.4 Radiological Demonstrations

9.1.4.1 Nervous System

Japanese researchers have discovered that influenza virus infection in some children may further cause encephalitis due to its invasion into their central nervous system.

By craniocerebral CT scan, the patients with influenza A (H1N1) complicated by encephalitis are mostly demonstrated with symmetric low density in bilateral basal ganglia, bilateral thalamus, and bilateral white matter of frontal brain; and some patients may show no obvious abnormalities. Brain

MR imaging shows symmetric long T1 and long T2 signals at the above mentioned sites. FLAIR and DWI often demonstrate high signal. In some patients, cerebellum is demonstrated with involvement. By contrast scan or imaging, meningeal enhancement may be demonstrated.

9.1.4.2 Chest

The patients with influenza A (H1N1) rarely show chest abnormalities. And the common abnormalities by chest radiology include GGO and consolidation. The lung lesions in adult lungs commonly distribute in the lower fields of both lungs, but no characteristic distribution in pediatric patients. The focal, multiple or diffuse lung lesions may be within one lung or both lungs, resembling to SARS and other viral pneumonia. The radiological signs of pulmonary nodules, interstitial thickening, patches of opacity around bronchus, air trapping sign, and pleural effusion are rare. Severe and critical cases of influenza A (H1N1) can be complicated by pulmonary embolism. Being different from other lung infections, the cases of influenza A (H1N1) rarely show enlarged lymph nodes, with no central lobular nodule and tree-buds sign by MSCT.

Compared to common chest X-ray, CT scan is more sensitive to GGO and shows higher accuracy in detecting the distribution of lesions. However, common chest X-ray has the advantage of simple operational procedures, especially bedside chest X-ray for critical cases of influenza A (H1N1).

9.1.4.3 Abdomen

The patients with severe influenza A (H1N1) may subject to liver damages. By CT scan, the liver is demonstrated with enlargement and plump margin. For those with complicating ascites, ultrasound demonstrates liquid dark area in the abdominal cavity. Some patients with coupled peritonitis is demonstrated with observable peritoneal thickening.

9.2 Typical Cases

Case 1

[Brief Case History]

An 8-year-old boy, a primary school student, complained of fever, cough and wheezing for 2 days. The boy began to experience irregular fever with no known causes 2 days ago, with a body temperature of up to 39 °C. Other symptoms include paroxysmal cough, expectoration of yellowish thick sputum in a small quantity, wheezing, shortness of breath, chest distress, dysphagia, occasional abdominal pain that aggravated when coughing and was more severe around the

navel. By physical examination, body temperature 37.9 °C, heart rate 117/min, breathing rate 33/min, and blood pressure 105/60 mmHg; the lips no cyanosis; pharynx apparently congested, no follicular hyperplasia on the posterior pharyngeal wall, and no white spots; degree II enlargement of both tonsils; by auscultation, a small quantity of fine moist rales and wheezing in both lungs. At the age of 3 years, the patient experienced 2 episodes of asthma, which improved after treatment. His mother had a medical history of asthma. The patient reported a history of contact to

patients with fever like symptoms. Throat swab by CDC showed the M gene of type A influenza virus positive, the NP gene of swine H1N1 influenza virus positive, and the HA gene of type A H1N1 influenza virus positive. Routine blood test revealed WBC count $6.69 \times 10^9/L$, GR% 78.1%, LY% 12.7%, MONO% 7%, and HGB 118 g/L. Blood biochemistry showed AST 52.5U/L, CK 2022.5U/L, CK-MB 35.1U/L, and troponin 1.08 ng/ml; CD3+ T cell count 332/ul, CD4+ T cell count 173/ul, CD8+ T cell count 106/ul, and the ratio of CD4+/CD8+ 1.63. Blood gas analysis revealed pH 7.4, PaCO₂ 35.7 mmHg, PaO₂ 109 mmHg, AB 21.6 mmol/L, and BE -3.7 mmol/L.

[Radiological demonstration] Fig. 9.1.

[Diagnosis] Influenza A (H1N1) pneumonia.

[Discussion]

The early chest radiological signs in pediatric patients with influenza A (H1N1) pneumonia are non-characteristic, resembling to common lung infection. Influenza A (H1N1) pneumonia can be radiologically demonstrated as increased and thickened lung markings in both lungs as well as small patches of opacity. Along with the progression of the disease, the small patches of opacity may fuse into large flakes of opacity and/or ground glass like opacity.

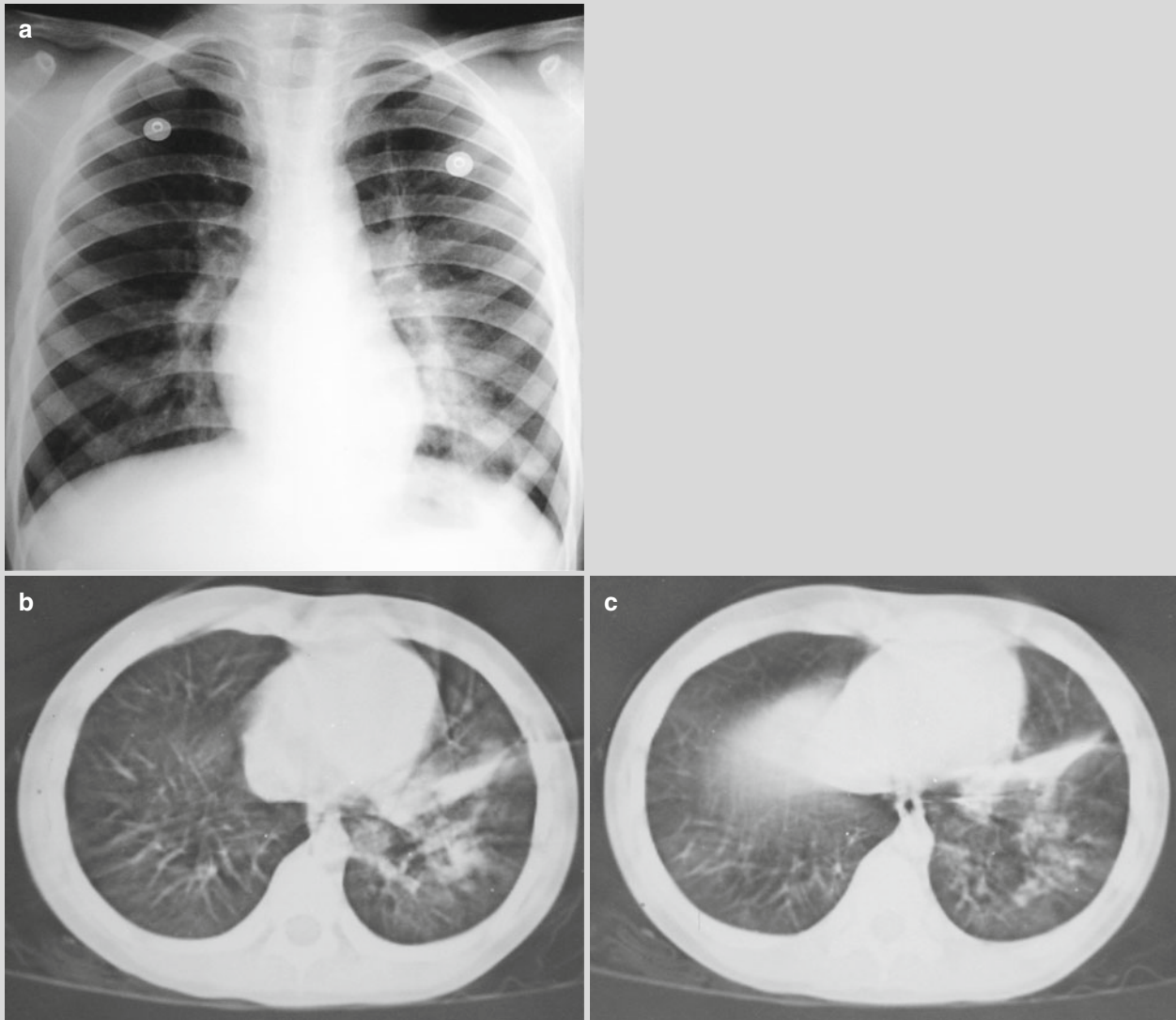


Fig. 9.1 Chest X-ray showed increased lung markings in both lungs, sporadic poorly defined patches of increased density opacity in the left middle and lower lung fields (a). CT scan demonstrated multiple con-

solidations, patches and GGO like opacities in the left lower lung lobe (b, c) (Reprint with permission from Hongjun Li and Ning Li (Eds), Radiology of Influenza A (H1N1), Springer, 2013)

Case 2**[Brief Case History]**

A 4-years-old boy complained of fever and cough for 3 days. He experienced no chills, but poor appetite. By physical examination, bilateral lower lungs showed little moist rales. Chest X-ray and routine blood test immediately after onset showed no abnormality. Throat swab after hospitalization showed the M gene of type A influenza virus positive, the NP gene of swine H1N1 influenza virus positive, and the HA gene of type A H1N1 influenza virus positive.

[Radiological demonstration] Fig. 9.2

[Diagnosis] Pneumonia induced by type A H1N1 influenza virus.

[Discussion]

Radiologically, this case was shown with ground-glass opacity in bilateral lower lungs, which is consistent with the onset location of pneumonia induced by type A influenza. For this case, the symptoms are mild with early onset, which should be differentiated from other lung infections. Lobar pneumonia is demonstrated with consolidation of inflammatory lesion or lung segment. Lobular pneumonia is demonstrated with patchy opacity around bronchi. And the key point for its differentiation from pulmonary tuberculosis is the onset location, and the lower lungs are not the common onset location of pulmonary tuberculosis. For this case, its differential diagnosis from other pathogenic infections with interstitial inflammation is challenging, such as early mycoplasma pneumonia and other viral infections induced viral pneumonia. For this case, due to its early onset and mild symptoms, radiology helps little in the differential diagnosis. And this case is to raise awareness of the clinicians about the prevailing seasons of the disease. The possibility of pneumonia induced by type A influenza virus should be considered if relatively slight ground glass opacity is observed.

Early chest radiography shows no characteristic signs of pediatric pneumonia induced by type A H1N1 influ-

enza virus. During its progressive stage, radiology demonstrates parenchymal lesions in lung, with parenchymal infiltration of singular or multiple small patches of opacity or their fusion into large opacity. In children, the progressive stage is shown with patchy opacity; while in infants, flakes and cotton like opacity.

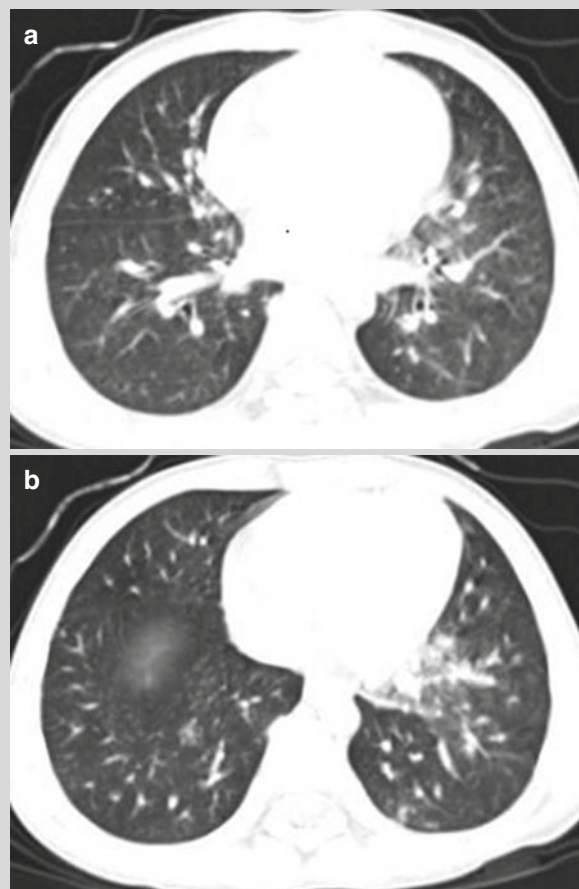


Fig. 9.2 (a, b) Chest CT scan revealed rare poorly defined ground glass opacity in bilateral lower lung lobes, which was predominantly in the left lower lung lobe

Case 3**[Brief Case History]**

A 14-years-old boy complained of sore throat for 3 days; fever, fatigue and muscle soreness for 2 days. He experienced mild cough without sputum. By physical examination, body temperature 38.7 °C, pharyngeal congestion, and I degree tonsillar swelling. By routine blood test, WBC $4.97 \times 10^9/L$, and LY% 23.5%. Chest CT scan on hospitalization showed the both lungs with multiple nodular and patchy high density opacity. Throat swab after hospitalization, the M gene of type A influenza virus was positive, the NP gene of swine H1N1 influenza virus positive, and the HA gene of type A H1N1 influenza virus positive.

[Radiological demonstration] Fig. 9.3

[Diagnosis] Pneumonia induced by type A H1N1 influenza virus.

[Discussion]

The early lesion of pneumonia induced by type A H1N1 influenza virus is an interstitial inflammation, which is mainly demonstrated as increased, thickened and blurred pulmonary vascular markings as well as grid like opacity and nodules in different degrees. For this case, the main radiological signs were multiple scattering nodular and patchy opacities, which should be differentiated from multiple pulmonary true nodules. The patients with multiple pulmonary metastatic nodules always have a medical history of primary disease. For this case, some of the inflammatory nodules dis-

appeared after treatment, and the lesions in the right lower lung evolved into flake of high-density lesion.

The mild cases of pneumonia induced by type A H1N1 influenza virus show diversifying radiological signs, which are commonly non-specific. The radiologi-

cal demonstrations include pulmonary parenchymal and interstitial inflammation, pleural inflammation, mediastinal and axillary lymphadenectasis, with lesions disseminating along bronchi, which resembles to viral pneumonia.

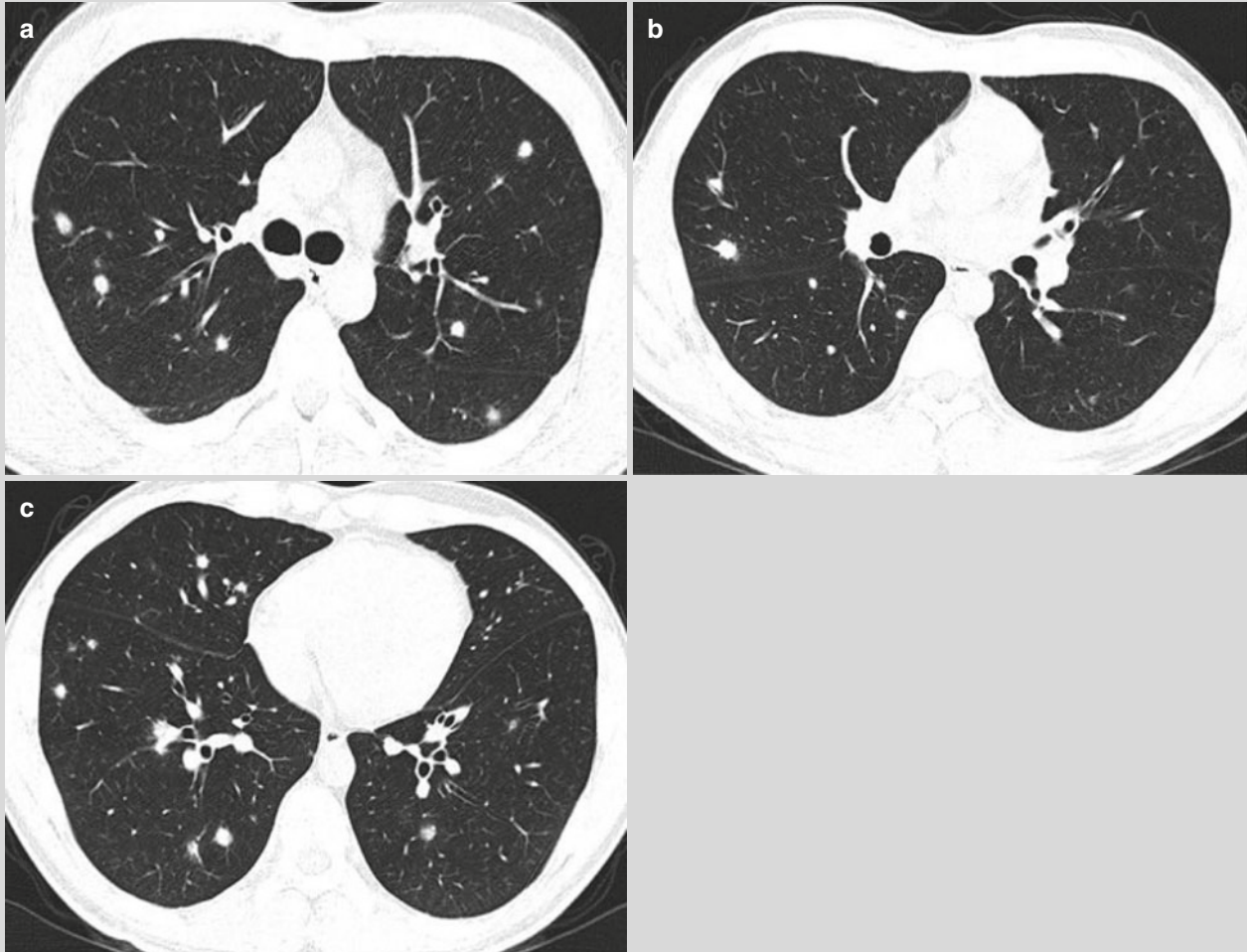


Fig. 9.3 (a–c) The both lungs were shown with multiple nodular and patchy high density opacity, predominantly in the right lower lung lobe

Case 4

[Brief Case History]

A 14-years-old girl was hospitalized due to chief complaints of fever and cough for 4 days. The patient experienced fever with no known cause on Nov. 6, 2009 with a body temperature of 37.5 °C. The fever was irregular with accompanying fatigue, paroxysmal cough, and expectoration of a small quantity yellowish sputum. Three days later, the patient experienced fever again with no known cause with a body temperature of 39.0 °C as well as aversion to cold, chest distress, dizziness, headache, systemic weakness, muscle soreness and other upsets. But the patient experienced no runny nose, no nasal obstruction, no chest pain, no shortness of breath, no palpitation, no

hemoptysis, and no difficulty breathing. However, she had sore throat with sensation of foreign substance when swallowing and nausea. By physical examination, body temperature 37.3 °C, pulse 108/min, respiration 22/min, and blood pressure 102/63 mmHg. Lips showed no cyanosis; pharynx with obvious congestion, slight follicular hyperplasia at the posterior pharyngeal wall but no white spots, and both tonsils with II degree swelling. The both lungs showed coarse breathing sound as well as fine dry and moist rales. She reported a history of contact to a patient with similar fever and a history of contact to a patient with definitively diagnosed type A H1N1 influenza. In addition, her school was affected by type A H1N1 influenza. By CDC throat swab, the M gene of type A influenza virus

was positive, the NP gene of swine H1N1 influenza virus positive, and the HA gene of type A H1N1 influenza virus positive. By routine blood test, WBC $15.31 \times 10^9/L$, GR% 82%, PLT $144.0 \times 10^{12}/L$, and HGB 140.9 g/L.

[Radiological demonstration] Fig. 9.4

[Diagnosis] Pneumonia induced by type A H1N1 influenza virus.

[Discussion]

Type A H1N1 influenza is more common in teenagers and young adults but rare in the elderly aged above 60 years. The patients commonly experience fever, cough, and sore throat; and some patients may also experience

headache, nausea, vomiting, and diarrhea. Pneumonia is the most common complication of type A H1N1 influenza, which is radiologically demonstrated as lung parenchymal infiltration opacity, more commonly bilateral. The parenchymal infiltration is demonstrated as singular or multiple small patches of opacity or their fusion into large opacity and/or ground glass opacity. For this case, chest X-ray demonstrated multiple blurry flakes of opacity, while CT scan revealed multiple consolidations. Such findings indicated that CT scan should be timely ordered for clinical patients with respiratory symptoms in order to detect early lesions for appropriate treatment.

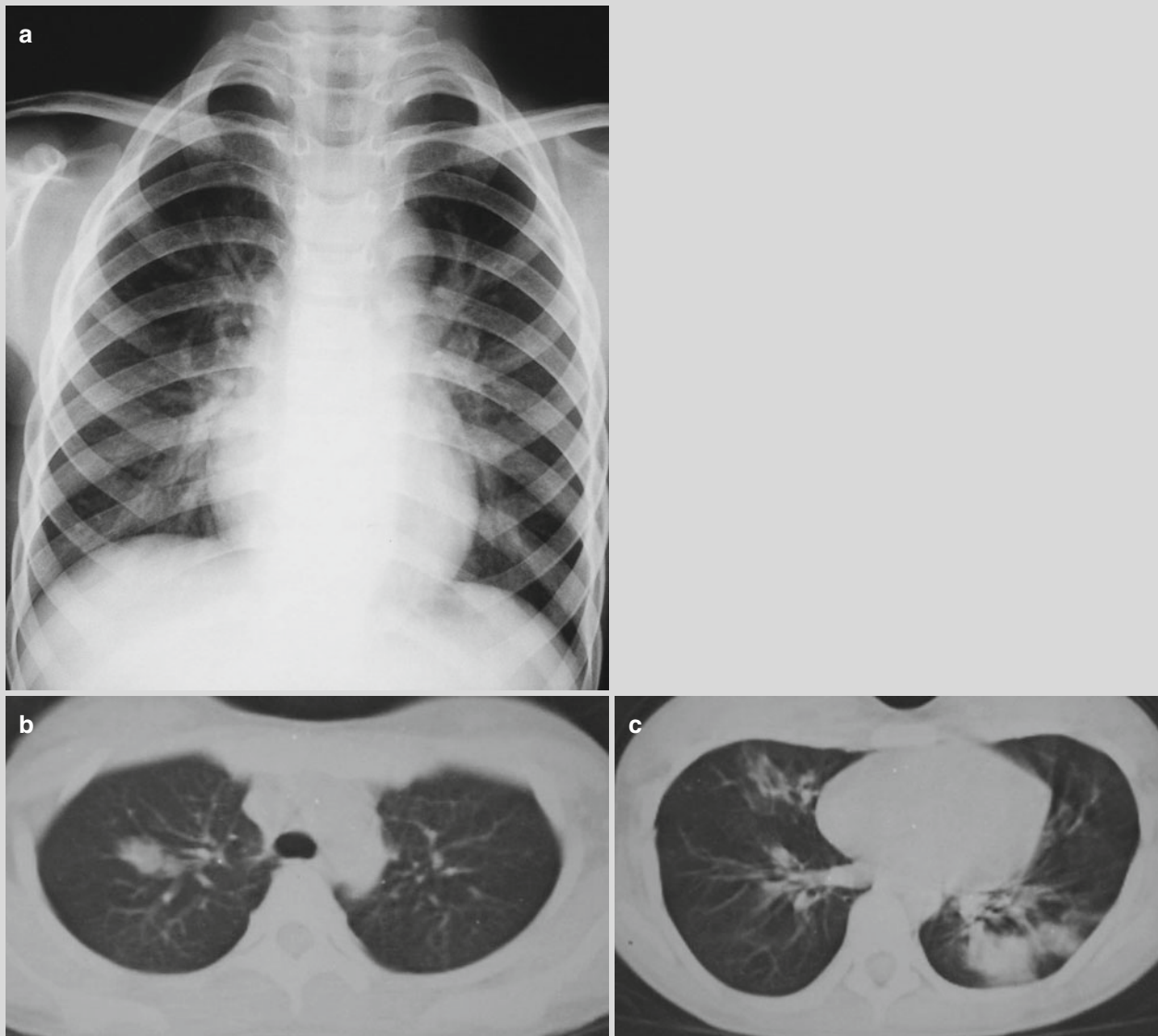


Fig. 9.4 Chest X-ray showed increased lung markings and multiple flakes of poorly defined opacity in both lungs (a); CT scan demonstrated multiple poorly defined consolidation opacity in the right upper lung lobe and the left lower lung lobe; and multiple cords like

and patchy opacity in the right middle lung lobe (b, c) (Reprint with permission from Hongjun Li and Ning Li (Eds), *Radiology of Influenza A (H1N1)*, Springer, 2013)

Case 5**[Brief Case History]**

A 17-years-old teenager girl complained of fever and cough for 2 days with running nose but no aversion to cold, and malaise. She had a history of contact to a patient with type A influenza. By physical examination, body temperature 39.6 °C, pharyngeal congestion, and I degree tonsillar swelling. By throat swab, relevant items (such as FluA and SWH1) were shown positive.

[Radiological demonstration] Fig. 9.5.

[Diagnosis] Pneumonia induced by type A H1N1 influenza virus.

[Discussion]

In this case, CT scan showed focal ground-glass opacity and consolidation opacity with pleural effusion. The condition should be differentiated from bacterial pneumonia. Bacterial pneumonia is commonly demonstrated as lobar or segmental consolidation opacity that is confined and predominantly in one segment or one lobe. Its occurrence in both lungs or with diffuse lesion



Fig. 9.5 Chest CT scan showed patchy high density opacity in the right middle and lower lung lobes, and left pleural effusion in a large quantity

in one lung is rare, with slower progression of the lesions than pneumonia induced by type A H1N1 influenza.

Case 6**[Brief Case History]**

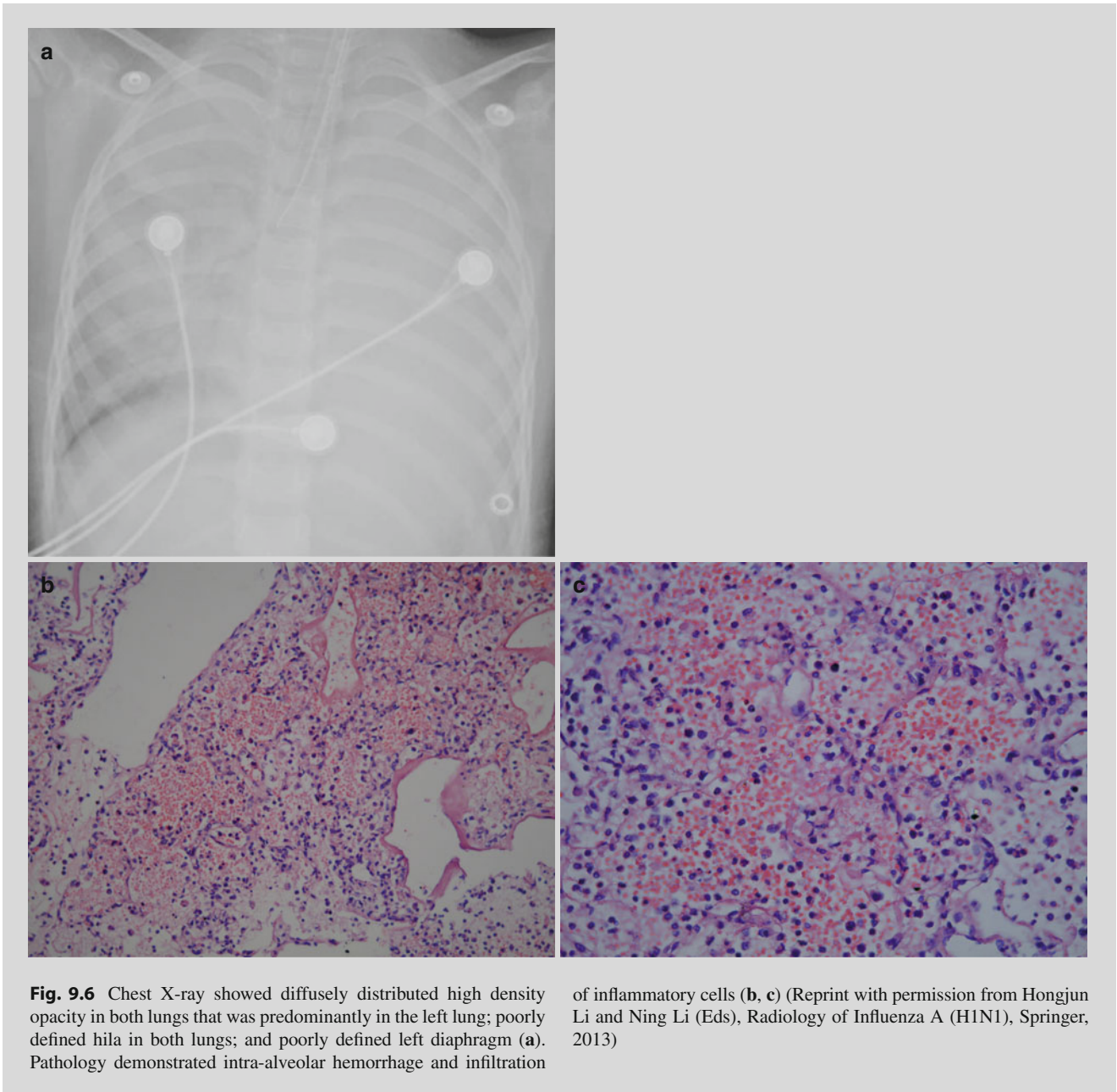
A 5-years-old boy experienced fever, cough with expectoration, no aversion to cold or chills, systemic malaise, muscle soreness, and poor appetite. By physical examination, respiration 40/min, pulse 130/min, and body temperature 39 °C. The boy was conscious but low spirits, with dyspnea, nasal flaring, and a large quantity of rales in both lungs. He denied a history of contact to patient with type A influenza. Throat swab showed the M gene of type A influenza virus positive, the NP gene of swine H1N1 influenza virus positive, and the HA gene of type A H1N1 influenza virus positive. By routine blood test, WBC $1.9 \times 10^9/L$, GR% 69.9%, and PLT $87 \times 10^9/L$. Blood chemistry revealed ALT 13.2U/L, AST 30.9/L, SpO₂ 79-85%.

[Radiological and pathological demonstrations] Fig. 9.6

[Diagnosis] Pneumonia induced by type A H1N1 influenza virus (severe case).

[Discussion]

Children are susceptible to type A H1N1 influenza, with high incidence and mortality. Being different from seasonal severe influenza that tends to affect the elderly aged above 65 years and young children aged under 2 years, 71% of patients with severe pneumonia induced by type A H1N1 influenza virus are from the population aged 5–59 years. By chest X-ray, the lung lesions are characterized by early patches and flakes of high density opacity in local lung lobe or segment, and their rapid progression into large flakes and diffuse lesions, air bronchogram in multiple lobar or segmental consolidations, and compensatory emphysema in the contralateral non-diseased lung. In addition, the pulmonary parenchymal and interstitial lesions concur, with predominantly interstitial lesions in the advanced stage of the disease.



Case 7**[Brief Case History]**

A 29-years-old young woman experienced cough with no sputum and fever. Physical examination and laboratory test showed LDH 505U/L, post delivery, and a history of tonsilectomy.

[Radiological examination] Fig. 9.7

[Diagnosis] Pneumonia induced by type A H1N1 influenza virus.

[Discussion]

This case was radiologically demonstrated with multiple ground glass opacity and consolidation opacity in both lungs with some lesions closely adhering to the

adjacent pleura, and the condition was clinically diagnosed as a severe case. And the clinical diagnosis was firstly considered to be pneumonia. For this case, the radiological signs are non-characteristic, and differential diagnosis from other factors induced pneumonia is necessary. But in this case, the symptoms occurred after delivery. Due to comparatively weak immunity during pregnancy, slight edema of respiratory mucosa tends to develop into respiratory infection. Pneumonia induced by type A H1N1 influenza virus in pregnancy is radiologically demonstrated as multiple ground glass opacity and consolidation opacity, with pleural lesion in some cases.

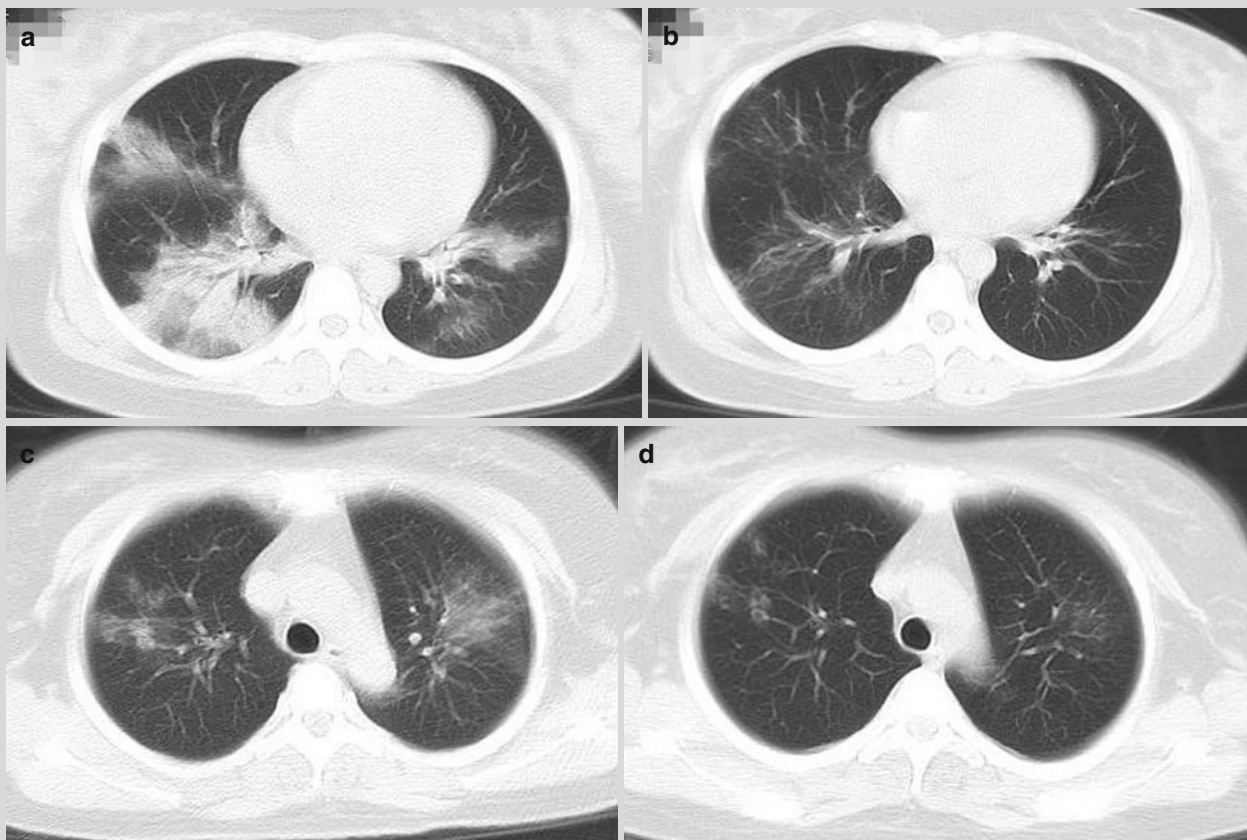


Fig. 9.7 Chest CT scan showed multiple patches and flakes of opacity in both lungs, some of which were ground-glass opacity and consolidation opacity (a, b). After treatment for 22 days and 30 days, CT scan showed absorption of the lesions (c, d)

Case 8**[Brief Case History]**

A 31-years-old woman, 4 days after cesarean section, complained of fever, shortness of breath, and progressive dyspnea for 2 days. She began to cough 1 day ago with pinkish foam like sputum and show progressive dyspnea. Her condition failed to be improved by symptomatic ther-

apy but aggravated. Physical examination showed a body temperature of 40 °C, and heart rate 136/min. The lips showed cyanosis, and breathing sound coarse with moist and dry rales all over the lungs. She reported a history of close contact to a patient with type A influenza. And throat swabs revealed the M gene of type A influenza virus negative, the NP gene of swine H1N1 influenza virus negative,

and the HA gene of type A H1N1 influenza virus positive. Routine blood test showed WBC $12.97 \times 10^9/L$, GR% 91.1%, RBC $3.24 \times 10^{12}/L$, and HGB 83 g/L.

[Radiological demonstration] Fig. 9.8

[Diagnosis] Pneumonia induced by type A H1N1 influenza virus.

[Discussion]

During pregnancy, the assistant total T cell count decreases and the activity of natural killer cells also decreases. As a result, the pregnant women are vulnerable

to virus and fungal pneumonia. In mild cases, the lesions are demonstrated as ground glass opacity and small flakes of cotton like high density opacity in singular or multiple lung lobes. In severe cases, the lesions are demonstrated as masses of cotton like opacity, ground glass opacity, and air bronchogram in multiple lung lobes. Such lesions may progress within 24 h into diffuse high density opacity in both lungs with accompanying atelectasis, which may be complicated by unilateral or bilateral pleurisy, pleural effusion or pericardial effusion.

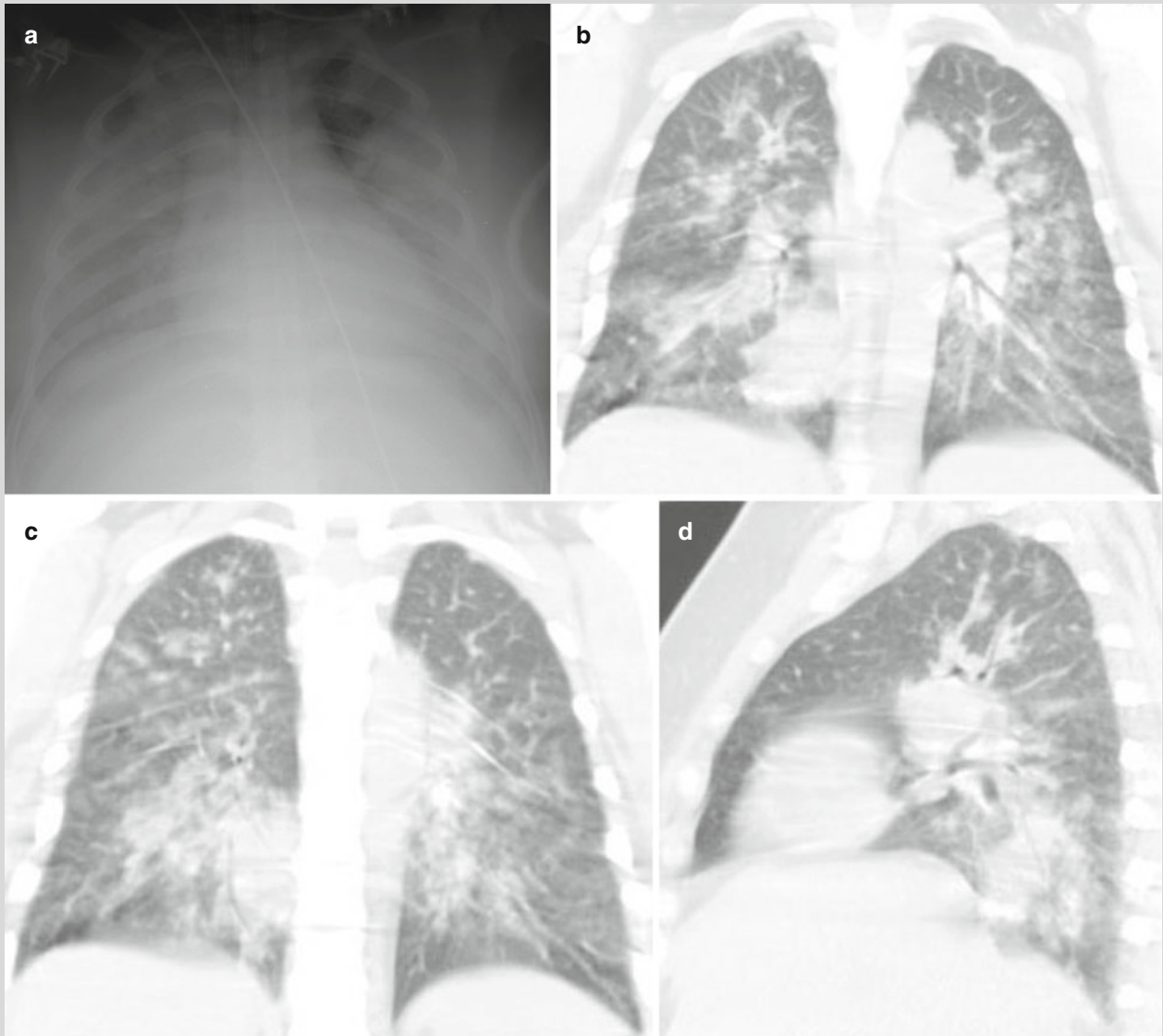


Fig. 9.8 Chest X-ray showed large flakes of high density opacity in both lungs that was predominantly in the right lung, and poorly defined hilum in both lungs (a). CT scan demonstrated multiple consolidation and ground glass opacity in both lungs, air broncho-

gram in the right upper lung lobe as well as thickened and deranged lung markings in both lungs (b-f) (Reprint with permission from Hongjun Li and Ning Li (Eds), *Radiology of Influenza A (H1N1)*, Springer, 2013)

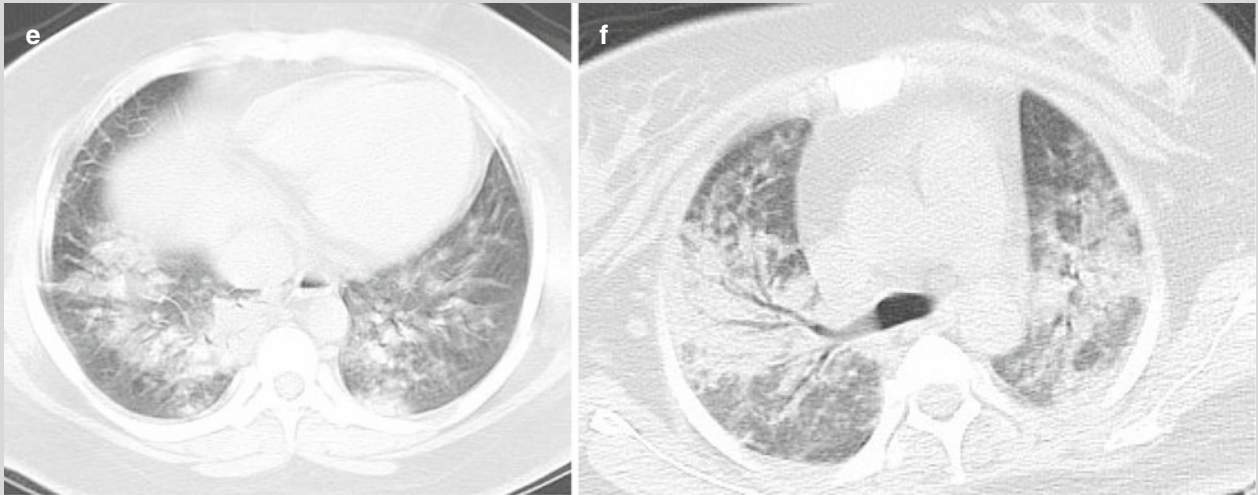


Fig. 9.8 (continued)

Case 9

[Brief Case History]

A 26-years-old young man experienced slight cough, expectoration of a little sputum, and fever with a body temperature of 38 °C. Laboratory test revealed WBC count $4.9 \times 10^9/L$, GR% 55.9%, and LDH 135 U/L.

[Radiological demonstration] Fig. 9.9

[Diagnosis] Pneumonia induced by type A H1N1 influenza virus.

[Discussion]

In this case, the patient experienced symptoms of fever and slight cough, but CT scan showed local blurry lung markings in the left upper lung lobe and rare patches of ground glass opacity, which were small and confined. The diagnosis by radiology was considered to be lung inflammation.

In this case, the patient is a young man with rare and confined lesions in lungs by radiological examination. However, it still needs to be differentiated from bacterial and viral pneumonia, both of which may be local interstitial and parenchymal inflammation with lesions of confined ground glass opacity by radiology. The early manifestations of pneumonia induced by type A H1N1 influenza virus resemble to pneumonia induced by other viruses. Some previous studies were performed to stage slight, moderate and severe pneumonia induced by type A

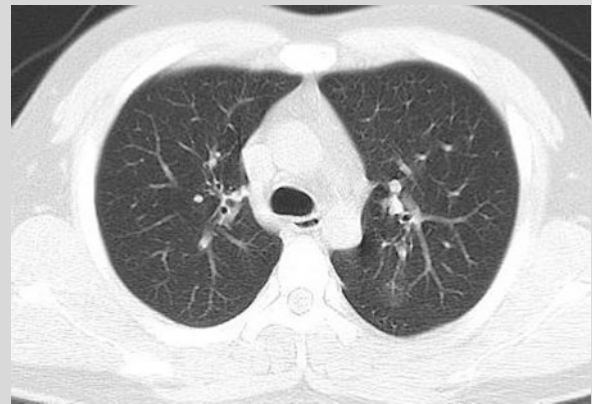


Fig. 9.9 CT scan showed small flakes of ground glass opacity near the oblique fissure in the left upper lung lobe

H1N1 influenza virus based on the radiological findings. Some studies have demonstrated that chest ultrasound in the emergency room can detect interstitial lesions of early pneumonia induced by type A H1N1 influenza virus, while CR fails to detect any lung lesion during the same stage of the disease. However, these studies also indicated that CT scan can more favorably show the lung lesions for the patients with pneumonia induced by type A H1N1 influenza virus, especially during the early stage and for the severe cases.

Case 10**[Brief Case History]**

A 43-years-old woman complained of fever and dry cough.

[Radiological demonstration] Fig. 9.10

[Diagnosis] Pneumonia induced by type A H1N1 influenza virus.

[Discussion]

Radiologically, the patient showed subpleural patchy opacity with lobular distribution in sizes of 1–1.5 cm, with multiple lung lobes involved. As a result, lung inflammation was considered and differentiation is needed to define the diagnosis from bacterial pneumonia, fungal pneumonia, *Pneumocystis jiroveci* pneumonia and other viral pneumonia. Pneumonia caused by bacteria (commonly staphylococcus, pneumonic diplococcus and streptococcus) is pathologically characterized by bronchitis, peribronchitis, alveolitis, peri-alveolitis and multiple lobular pneumonia. And it is radiologically demonstrated as thickened lung marking as well as patchy and nodular opacity that are commonly located in the medial lung and fuse within 2–3 days. Pneumonia caused by fungi (commonly aspergillus and cryptococcus) is pathologically characterized by exudative and purulent inflammation in lungs. And it is radiologically demonstrated as singular or multiple nodules, lumps or cavities. Pneumonia caused by *Pneumocystis jiroveci* is pathologically characterized by serous and exudative alveolitis. And it is radiologically demonstrated as lobar or segmental lesions, which are



Fig. 9.10 CT scan showed subpleural patchy opacity in the right middle lung lobe and the left lower lung lobe

poorly defined patchy opacity along bronchi commonly in bilateral middle and lower lung fields. In some cases, the lesions may show progression and fusion within several hours or several days. Pneumonia caused by other viruses, such as adenovirus, respiratory syncytial virus, measles virus and cytomegalovirus, commonly occurs in young children, which is pathologically characterized by bronchitis and bronchopneumonia with infiltration of inflammatory cells that are predominantly mononuclear cells. And it is radiologically demonstrated as small nodular opacity and patchy opacity that are commonly located in middle and medial parts of bilateral middle and lower lung fields.

Case 11**[Brief Case History]**

A 52-year-old man complained of cough, expectoration of a little sputum, and fever. Laboratory test revealed WBC count $6.5 \times 10^9/L$, GR% 85.5 % and LDH 160U/L.

[Radiological demonstration] Fig. 9.11

[Diagnosis] Pneumonia induced by type A H1N1 influenza virus.

[Discussion]

Radiologically, this case is demonstrated with multiple patchy opacities along bronchovascular bundles with varying density in both lungs, some of which were shown to be ground-glass opacity and blurry small nodular opacity. Lung inflammation should be firstly considered for diagnosis. Pneumonia induced by type A H1N1 influenza virus is radiologically demonstrated as flakes and patches consolidations and ground glass opacity, with predominant lobular distribution.

Pneumonia induced by type A H1N1 influenza virus should be differentiated from bacterial pneumonia,

Pneumocystis jiroveci pneumonia and other viral pneumonia. For this case, its differential diagnosis from bacterial pneumonia is challenging. Bacterial pneumonia is radiologically demonstrated as thickened lung markings and more obvious lobar and segmental distribution. The lesions of pneumonia induced by type A H1N1 influenza virus are distributed in multi-lobes and segments with low density. The lesions of bacterial pneumonia integrate or show morphological changes within 2–3 days. Pneumonia caused by fungi and *Pneumocystis jiroveci* is pathologically alveolitis and is radiologically demonstrated as singular or multiple nodules, lumps or cavities, otherwise, lobar and segmental lesions as well as blurry patchy opacity commonly bilateral middle and lower lung fields along bronchi. Pneumonia caused by other virus is radiologically demonstrated as small nodular opacity and patchy opacity that are commonly located in middle and medial parts of bilateral middle and lower lung fields.

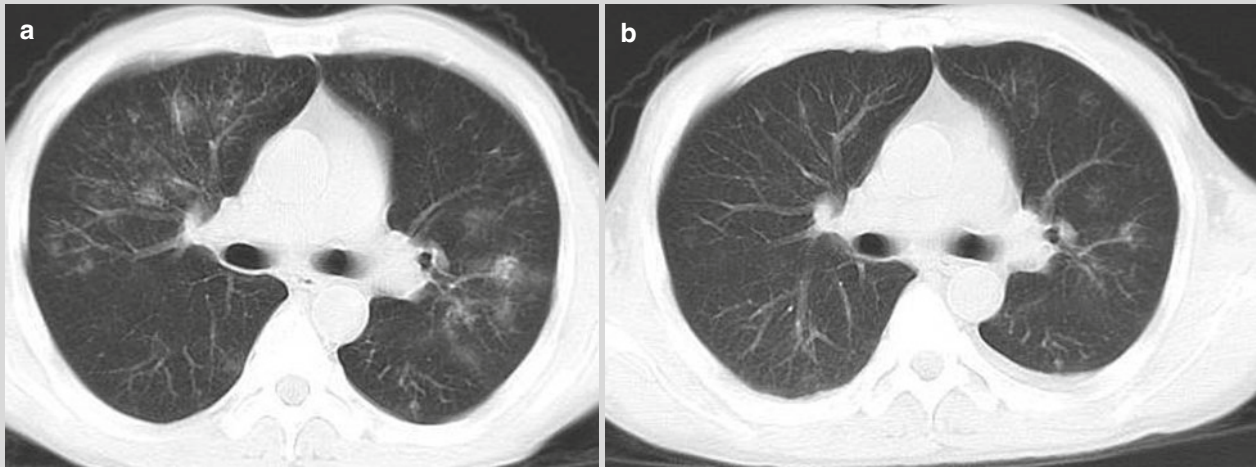


Fig. 9.11 CT scan showed multiple small nodules and blurry patchy opacity distributing along lung markings in both lungs (a). Reexamination by CT scan after treatment for 10 days showed decreased lesions, indicating absorption (b)

Case 12

[Brief Case History]

A 41-year-old man complained of high fever, wheezing and progressive dyspnea for 5 days. He had taken cephalosporin by himself, with no obvious improvement. And 2 days ago, he began to cough up pinkish foamy sputum. The patient reported a history of contact to a patient with type A influenza. By routine blood test 3 days ago, WBC count $12.11 \times 10^9/L$. And routine blood test on admission showed WBC count $17.45 \times 10^9/L$, and GR% 81.1%. Chest X-ray in another hospital revealed inflammation in both lungs, and predominantly in the left lower lung lobe. By throat swab after being hospitalized, the M gene of type A influenza virus positive, the NP gene of swine H1N1 influenza virus positive, and the HA gene of type A H1N1 influenza virus positive. Sputum smear revealed cocci positive.

[Radiological demonstration] Fig. 9.12

[Diagnosis] Pneumonia induced by type A H1N1 influenza virus.

[Discussion]

For this case, CT scan demonstrated patchy opacity and ground glass opacity at lung margin of both lungs, which is consistent to the early radiological sign of pneumonia induced by type A influenza virus. The lesions were predominantly located in the left lower lung lobe. In this case, routine blood test indicated increased WBC count, which continued to increase after hospitalization. Sputum smear revealed cocci positive, indicating bacterial infection that complicated primary viral infection.

For this case, it should be differentiated from simplex bacterial pneumonia, which is commonly demonstrated as lobar or segmental high density opacity that is comparatively confined and is not predominantly located at lung margin. This case also needs to be differentiated from mycoplasmal pneumonia, which is radiologically demonstrated as centrilobular ground glass opacity or consolidation, which may develop into segmental or lobar consolidation and is commonly fan like in shape. Such radiological signs are rarely found in the cases of pneumonia induced by type A influenza virus.

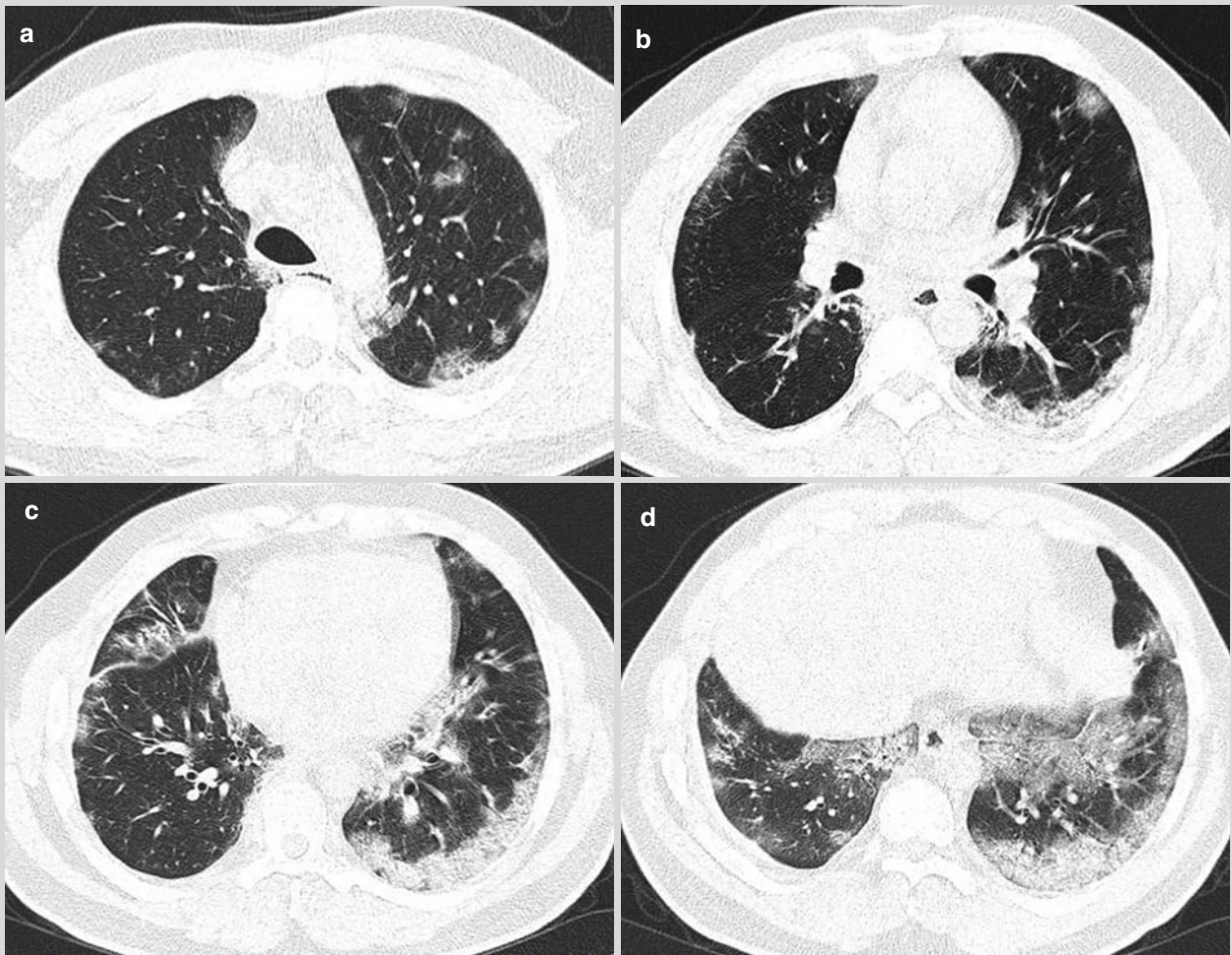


Fig. 9.12 Chest CT scan showed multiple flakes and cords like high density opacity and ground glass opacity in both lungs that was predominantly in the left lower lung lobe. The lesions were revealed to be commonly located at the lung margin (a–d)

Case 13

[Brief Case History]

A 54-year-old woman complained of cough, expectoration of whitish yellow sputum, and fever. She reported medical history of hypertension and diabetes. Laboratory test revealed LDH 383U/L.

[Radiological demonstration] Fig. 9.13

[Diagnosis] Pneumonia induced by type A H1N1 influenza virus.

[Discussion]

The patient was radiologically revealed with multiple patchy/flaky consolidations in both lungs, which closely adhered to the adjacent pleura, and pneumonia should be firstly considered for clinical diagnosis. The patient had a medical history of diabetes and was vulnerable to viral and/or bacterial infection.

Radiologically, differential diagnosis should be made from bacterial pneumonia, fungal pneumonia, Pneumocystis

jiroveci pneumonia and other viral pneumonia. Pneumonia induced by type A H1N1 influenza virus is radiologically demonstrated as ground glass opacity, possibly consolidation opacity and centrilobular nodular opacity, and accompanying pleural response. The lesions of severe pneumonia induced by type A influenza virus are pathologically revealed as diffuse alveolar damage. And chest CT scan shows severe pneumonia induced by type A influenza virus with diversifying lesions, with no definitive specificity. Both lungs are shown to be involved with multiple flakes of ground glass opacity as well as consolidation and nodules that are more commonly located in the bilateral peripheral middle and lower lung lobes. In this case, more consolidations were revealed, which may be related to the underlying disease of diabetes. After appropriate therapy was given, the lesions were shown to be obviously absorbed.

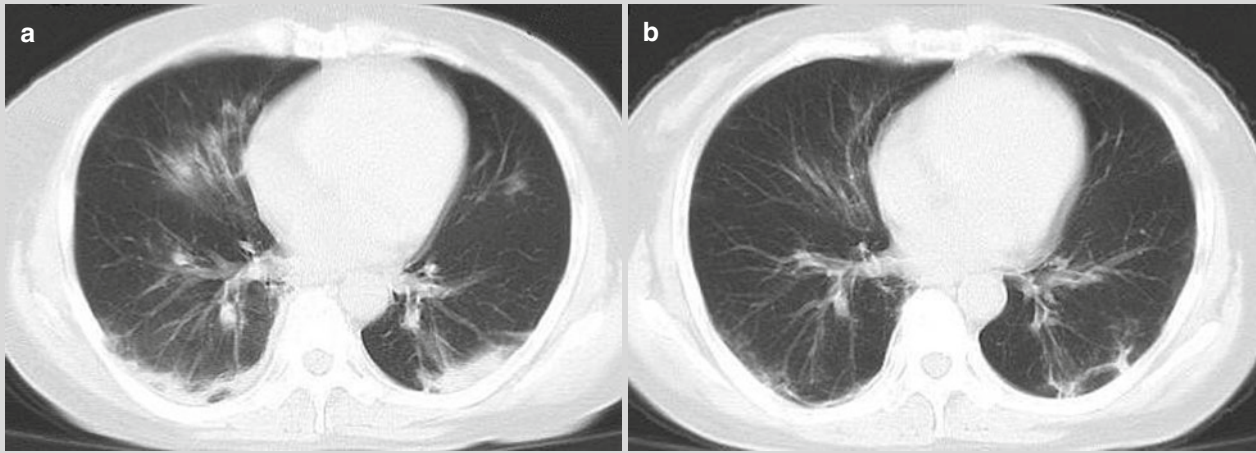


Fig. 9.13 CT scan showed patchy and curved flake like opacity in the right middle lung lobe, lingular segment of the left lung, and subpleural areas in bilateral lower lung lobes, some of which were

dense (a). Reexamination after treatment for 9 days showed absorption of the lesions and residual curved linear opacity in subpleural areas (b)

Case 14

[Brief Case History]

A 58-year-old man complained of slight cough, expectoration of whitish thick sputum, and fever. Laboratory test revealed WBC count $9.7 \times 10^9/L$, GR% 92.8%, and LDH 427U/L. He experienced respiratory failure of degree I.

[Radiological demonstration] Fig. 9.14

[Diagnosis] Pneumonia induced by type A H1N1 influenza virus.

[Discussion]

The patient was radiologically showed with multiple flakes of ground glass opacity in upper and lower lobes of both lungs that were multiple and closely adhered to the adjacent pleura. Pneumonia should be firstly considered for clinical diagnosis. Pneumonia induced by type A H1N1 influenza virus is commonly demonstrated as unilateral or bilateral diffuse or multiple patches of ground glass opacity with or with no consolidation that is mostly located around bronchovascular bundles or in the subpleural area. However, such radiological signs are non-specific, and differential diagnosis should be made from *Pneumocystis jiroveci* pneumonia, other viral pneumonia, and mycoplasmal pneumonia. Pneumonia caused by *Pneumocystis jiroveci* is pathologically characterized by serous and exudative alveoli-

tis. And it is radiologically demonstrated as lobar or segmental ground glass opacity. In some cases, the lesions may show progression and fusion within several hours or several days. Pneumonia caused by other virus is radiologically demonstrated as patches/flakes of ground glass opacity with/without consolidation, no mediastinal lymphadenectasis, and rarely pleural effusion. Compared to pneumonia caused by type A H1N1 influenza virus, other viral pneumonia shows more lesions of minor airways, which are demonstrated as centrilobular nodules that are more common in young children. And other viral pneumonia is pathologically characterized by bronchitis and bronchopneumonia with infiltration of inflammatory cells that are predominantly mononuclear cells. In addition, it is radiologically demonstrated as small nodular opacity and patchy opacity that are commonly located in middle and medial parts of bilateral middle and lower lung fields. Mycoplasmal pneumonia is radiologically demonstrated as ground glass opacities with/without consolidation and sporadic small nodules. Compared to pneumonia caused by type A H1N1 influenza virus, mycoplasmal pneumonia commonly involves one lung, with mediastinal lymphadenectasis and pleural effusion.

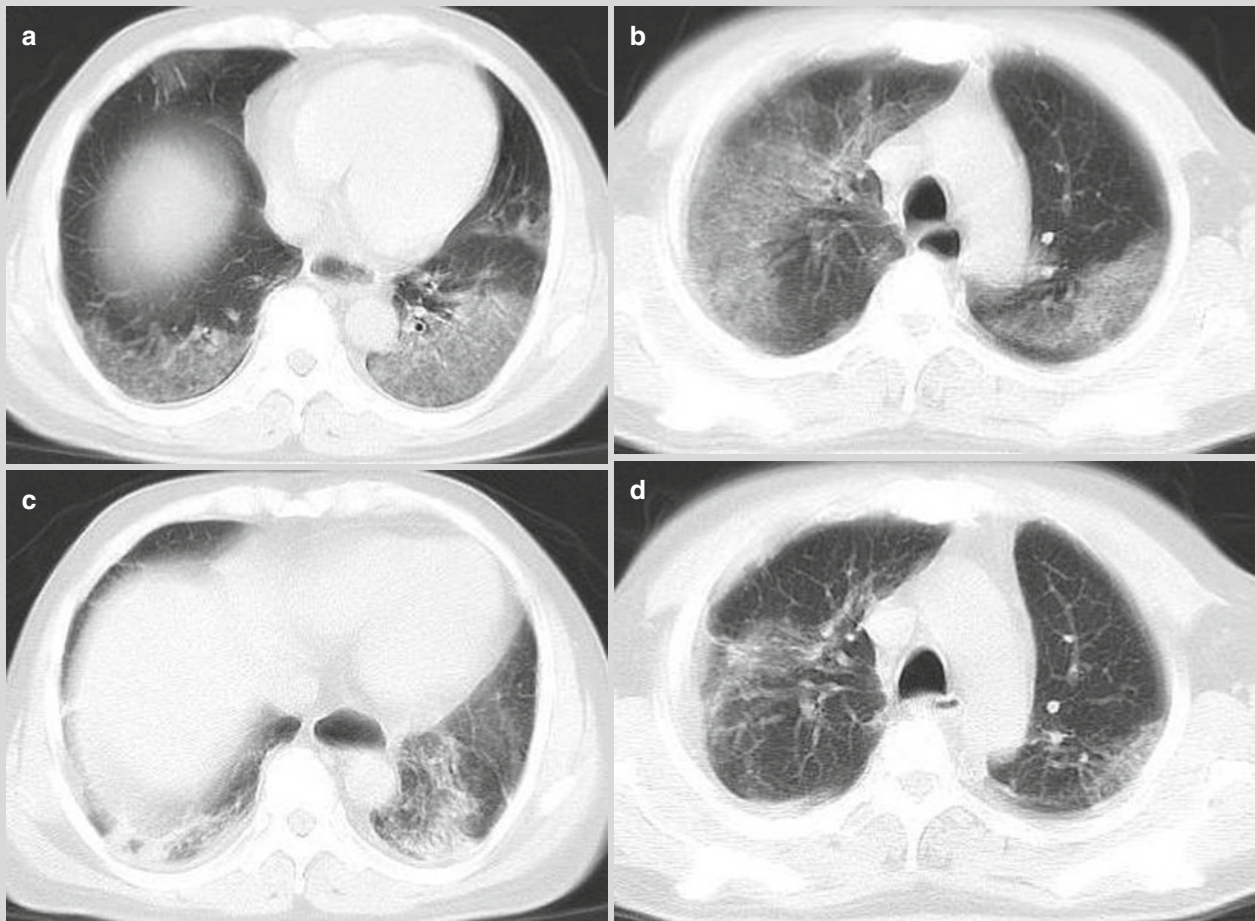


Fig. 9.14 CT scan showed multiple flakes of ground glass opacity in both lungs that closely adhered to the pleura (**a, b**). Reexaminations after treatment for 27 days (**c**) and 36 days (**d**), the lesions were

shown with absorption. But curved linear or strips and flakes of opacity was revealed, with local pleural thickening

Case 15

[Brief Case History]

A 38-year-old man complained of cough rarely with sputum and fever. Laboratory test revealed WBC count $9.2 \times 10^9/L$, GR% 69.6%, and LDH 182U/L.

[Radiological demonstration] Fig. 9.15

[Diagnosis] Pneumonia induced by type A H1N1 influenza virus.

[Discussion]

The patient was radiologically showed with flakes of ground glass opacity in the right upper lung lobe, with thick-wall cavity inside and partly with consolidation. Pneumonia should be firstly considered for clinical diagnosis. In literature reports, pneumonia induced by type A H1N1 influenza virus is rarely reported with lesions of cavity, and this case is categorized into a rare case. The cavity might be one of the radiological signs of pneumo-

nia induced by type A H1N1 influenza virus itself, and may also be a radiological sign of the complication. And it should be differentiated from bacterial pneumonia and fungal pneumonia. Pneumonia caused by bacteria may show cavity during its progression, which is commonly found in the cases of streptococcal and gram-negative bacterial infections. Clinically, pus or sputum with unpleasant odor is common, and the cavity is commonly revealed in consolidation opacity with poorly defined boundary and sometimes with air-fluid level. Pneumonia caused by fungi is radiologically demonstrated as singular or multiple nodules, lumps or cavities. Typical pneumocystis pneumonia is radiologically demonstrated as thin-wall cavity with no or rare surrounding lesions. Aspergillosis is radiologically demonstrated as cavity in poorly defined flakes of consolidation opacity with varying thickness of the wall. The typical cases are shown

with nodules or lumps in the cavity and observable gas containing crescent sign, and aspergilloma is mobile along with posture changes. Pneumonia caused by

Pneumocystis jiroveci, if complicated by bacterial or fungal infection, is radiologically demonstrated as cavities or cavities in lobar or segmental consolidation.

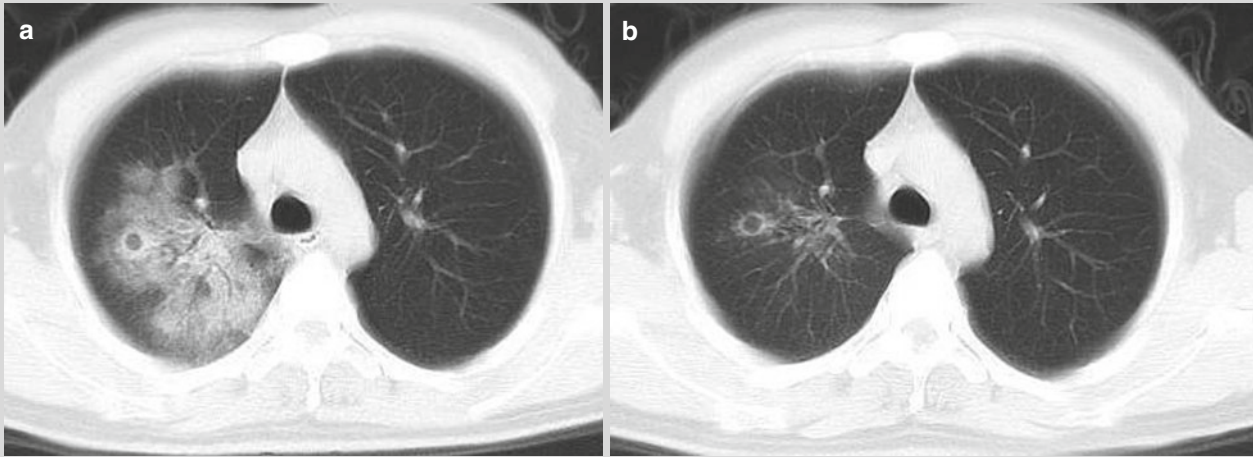


Fig. 9.15 CT scan showed flakes of poorly defined opacity in the right upper lung lobe, with bronchi sign and thick-wall translucent opacity (a); Reexamination after treatment showed thin-wall translucent opacity (b)

Case 16

[Brief Case History]

A 37-year-old man complained of cough, expectoration, and a sore throat for 7 days. He experienced expectoration of whitish foamy sputum and a fever for 4 days with the highest body temperature of 39 °C. Other symptoms included aversion to cold, wheezing and difficulty breathing for 2 days. By physical examination, the pharynx was shown with obvious congestion, and the right tonsil swollen in degree I. Both lungs were found with moist rales. By routine blood test, WBC count $8.9 \times 10^9/L$, GR% 55%, and LY% 34%. By throat swab after hospitalization, the M gene of type A influenza virus positive, the NP gene of swine H1N1 influenza virus positive, and the HA gene of type A H1N1 influenza virus negative.

[Radiological demonstration] Fig. 9.16

[Diagnosis] Pneumonia induced by type A influenza virus.

[Discussion]

Early pneumonia induced by type A H1N1 influenza virus is radiologically demonstrated as roughly symmetrical ground glass opacity in both lungs. Initially, the virus mainly invades the lung interstitium and the early lesions are mainly changes of interstitial pneumonia, which are radiologically demonstrated as multiple ground glass

density opacity in both lungs. Further progression of the lesions causes alveolar exudation or secondary bacterial/fungal infection, which is shown as consolidation of lungs tissue. The consolidation opacity is commonly located at the lung margin, with fusion of some lesions and air bronchogram. The progression of lesions may also cause fibrosis of lung tissue, which is radiologically demonstrated as widened interlobular septum and honeycomb like opacity in one or both lungs.

For this case, it needs to be differentiated from other viral pneumonia, such as SARS. Both are viral inflammation, with extremely similar radiological signs during their early stage. The early radiological signs include fibrin exudation, consolidation, and their further progression into pulmonary fibrosis. Severe pneumonia caused by type A influenza virus is radiologically demonstrated as flakes of exudation and fusion as well as reticular or nodular high density opacity in both lungs that fuse into poorly defined cloudy opacity. And the lesions may further develop into extensive consolidation opacity, which needs to differentiate from consolidation of lung tissue in the cases of lobar pneumonia. In combination to the case history and the illness course, the differential diagnosis can be made.

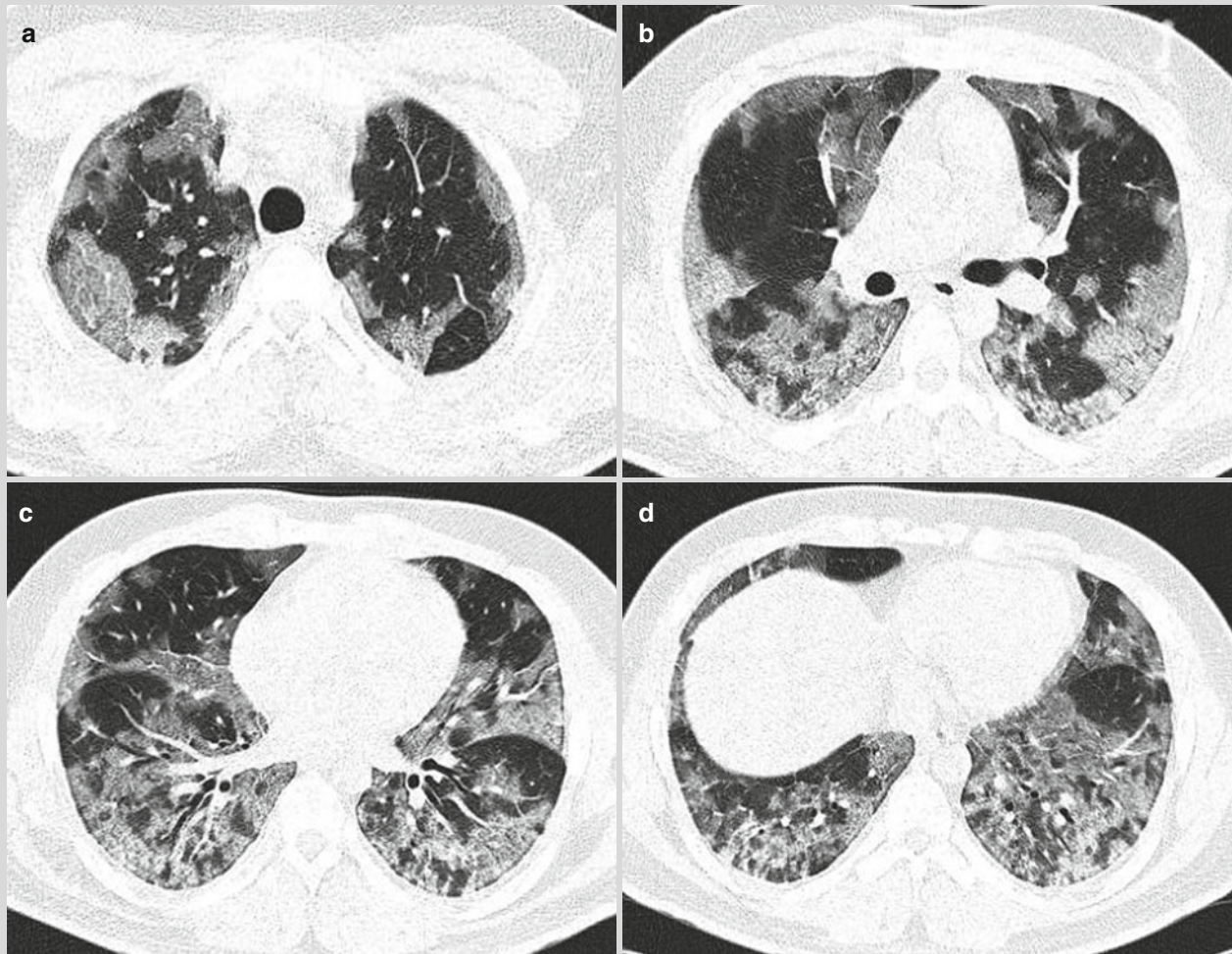


Fig. 9.16 CT scan showed sporadic flakes of high density opacity and ground glass opacity that were predominantly in bilateral lower lungs lobes as well as consolidation of partial lung tissues with gas containing bronchi (a–d)

Case 17

[Brief Case History]

A 41-year-old man experienced fever and cough for 10 days with accompanying sore throat, systemic malaise, and muscle soreness. He began to experience high fever 5 days ago with the highest body temperature of 39.6 °C. Chest X-ray in another hospital showed inflammation in both lungs. After intravenous infusion of cephalosporin, the symptom of fever was slightly improved. Recently, the patient began to experience symptoms of orthopnea and bloody sputum. By physical examination, the pharynx congested, and the bilateral tonsils swollen in degree I. Moist rales in both lungs, predominantly in lower lungs. By routine blood test on admission, WBC count $10.6 \times 10^9/L$, and LY% 7.5%. Sputum smear revealed cocci positive and bacillus negative. By throat swab after hospitalization, the M gene of type A influenza virus positive,

the NP gene of swine H1N1 influenza virus positive, and the HA gene of type A H1N1 influenza virus negative.

[Radiological demonstration] Fig. 9.17.

[Diagnosis] Influenza A (H1N1) pneumonia; Subcutaneous emphysema in the left thoracic wall.

[Discussion]

In this case, lung consolidation opacities were predominantly located in the left lower lung lobe a long period of development. Ground glass opacities sporadically distributed in both lungs, indicating virus infection. After treatment by anti-inflammatory therapy, his condition was improved, indicating a complication of bacterial infection. The patient experienced respiratory failure and was given several times of CPR, which resulted in ribs fracture and subcutaneous emphysema of the chest wall. The key point for differential diagnosis from lobar pneumonia is that the lesion of consolidation in the cases of

lobar pneumonia is located within one lung lobe or one lung segment. However, in this case, the lesion of consolidation was concurrent with sporadic GGO in the left

lower lung. Therefore, their differential diagnosis can be made in combination to clinical manifestations and the absorption stage of lobar pneumonia.

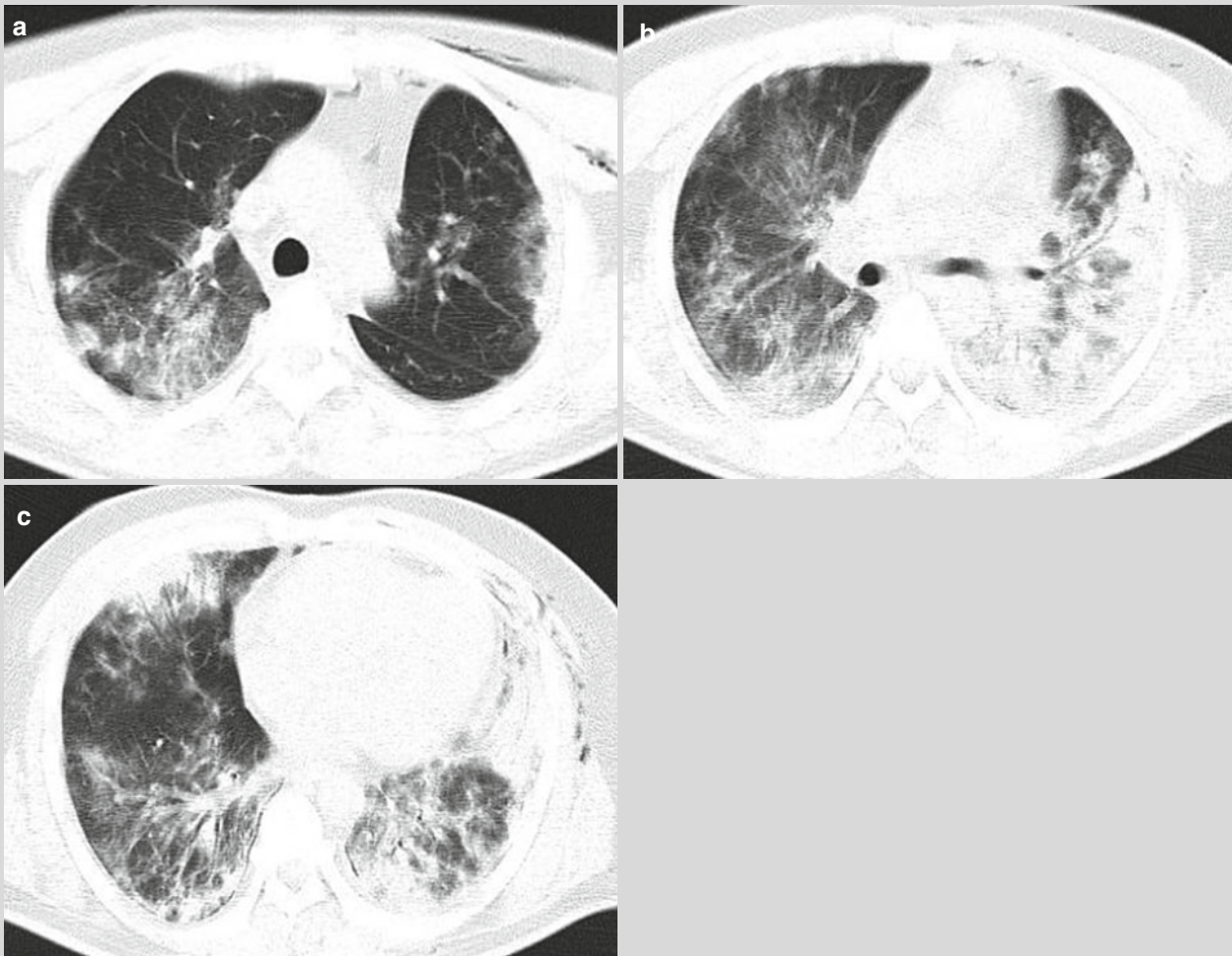


Fig. 9.17 Chest CT scan showed sporadic flakes of high density opacity and ground glass opacity, predominantly in bilateral lower lung lobes; consolidation of partial lung tissues in the left lower lung lobe; beads like air density opacity in the left thoracic wall (a–c)

Case 18

[Brief Case History]

A 54-year-old man complained of fever for 7 days, cough and expectoration for 6 days, as well as difficulty breathing for 2 days which aggravated during nights. He denied a history of contact to patient with type A influenza. And his physical signs included throat congested, no tonsil swollen, and moist rales in both lungs. Laboratory throat swab test showed etiological factors related to influenza A (H1N1), including FluA and SWH1, positive.

[Radiological demonstration] Fig. 9.18.

[Diagnosis] Pneumonia induced by type A H1N1 influenza virus.

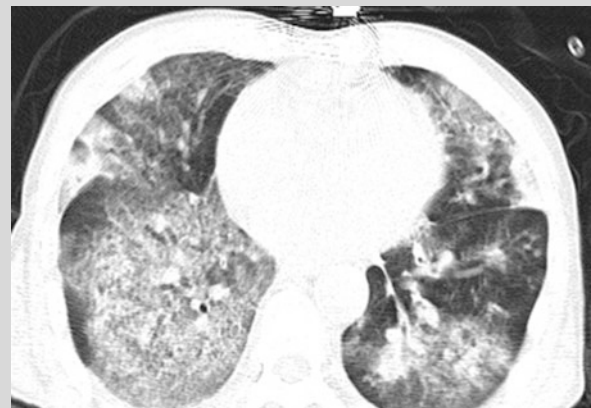


Fig. 9.18 Chest CT scan showed ground glass opacity and patches of high density opacity in both lungs, with local consolidations

[Discussion]

In this case, the lesions were diffusely distributed in the right lower lung field, accompanied by interstitial thickening. And the condition should be differentiated from SARS. The early manifestations of pneumonia induced by type A H1N1 influenza virus resemble to SARS, demonstrated as fibrinous exudation and consolidation that further progress into pulmonary interstitial

fibrosis. Severe and critical pneumonia induced by type A H1N1 influenza virus are demonstrated as flakes of alveolar exudation with fusion, network like or nodular opacity and inflammatory consolidation in both lungs, rarely with pleural effusion. The lesions are commonly located in both lower lungs, which are radiologically demonstrated as large flakes of dense consolidation opacity along with their progression.

Case 19

[Brief Case History]

A 42-year-old man complaint of fever and cough for 5 days. By physical examination, body temperature 39.5 °C, the tonsils swollen in degree I, and the breathing sound of both lungs coarse. He reported a non-definitive history of contact to patient with type A influenza. By laboratory throat swab, the M gene of type A H1N1 influenza virus positive, the NP gene of swine H1N1 influenza virus positive, and the HA gene of type A H1N1 influenza virus positive. By routine blood test, WBC count $3.24 \times 10^9/L$, GR% 57.74%, LY% 32.74%. Blood biochemistry revealed TP 55 g/L, AST 100U/L, ALT 69U/L, ALB 27 g/L, CK 621U/L, and LDH 511U/L. He experienced persistent fever during hospitalization with body temperature of 37.4–39.2 °C, cough, expectoration and chest distress. By auscultation, moist rales in both lungs that was more extensive in the right lung. Laboratory test on d 4 after hospitalization, blood routine test revealed WBC count $11.47 \times 10^9/L$, GR% 83.01%, LY% 8.82%; blood

biochemistry revealed TP 55 g/L, A 24 g/L, AST 64U/L, ALT 54U/L, and BUN 3.4 mmol/L.

[Radiological demonstration] Fig. 9.19.

[Diagnosis] Pneumonia induced by type A H1N1 influenza virus.

[Discussion]

Pneumonia is the most common complication of influenza A (H1N1), which is caused by primary viral pneumonia or secondary bacterial infection. Chest HRCT mainly demonstrates diffuse or multiple patches of ground glass opacity in lungs with or with no consolidation, which commonly distribute around bronchovascular tree or in the subpleural area. Pathologically, the lesions are demonstrated as bronchial and peribronchial alveolar congestion, inflammatory exudation, and transparent membrane formation. With the progression of the disease, ground glass opacities rapidly fuse and extend with increased density, with flakes of consolidation in or out of the ground glass opacity; only consolidation but no GGO in some cases.

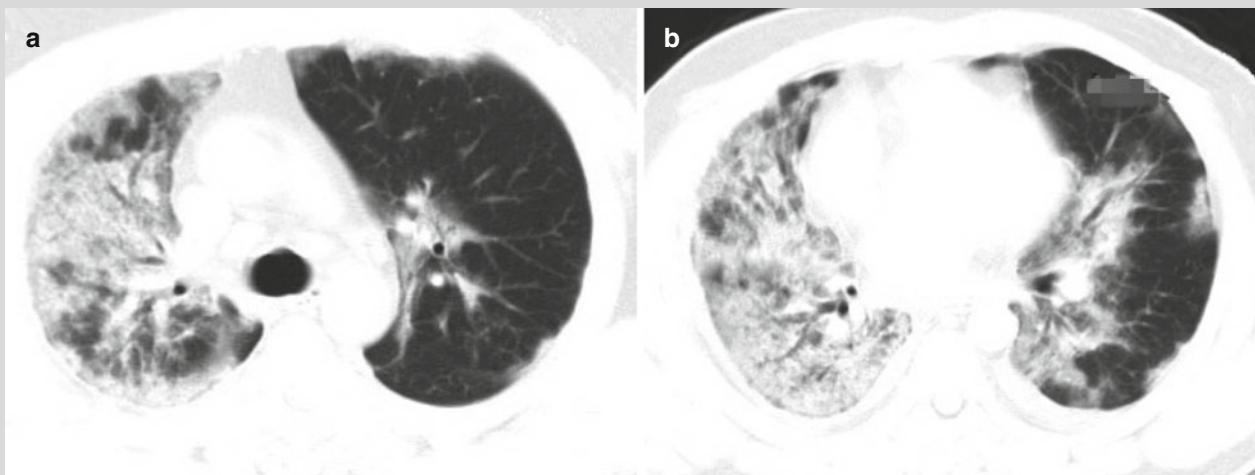


Fig. 9.19 CT scan showed consolidation and ground glass opacity in both lungs, predominantly in the right lung, with air bronchogram inside (a–d). Reexaminations after treatment for 4 days showed pro-

gression of the lesions in both lungs (e–h) (Reprint with permission from Hongjun Li and Ning Li (Eds), *Radiology of Influenza A (H1N1)*, Springer, 2013)

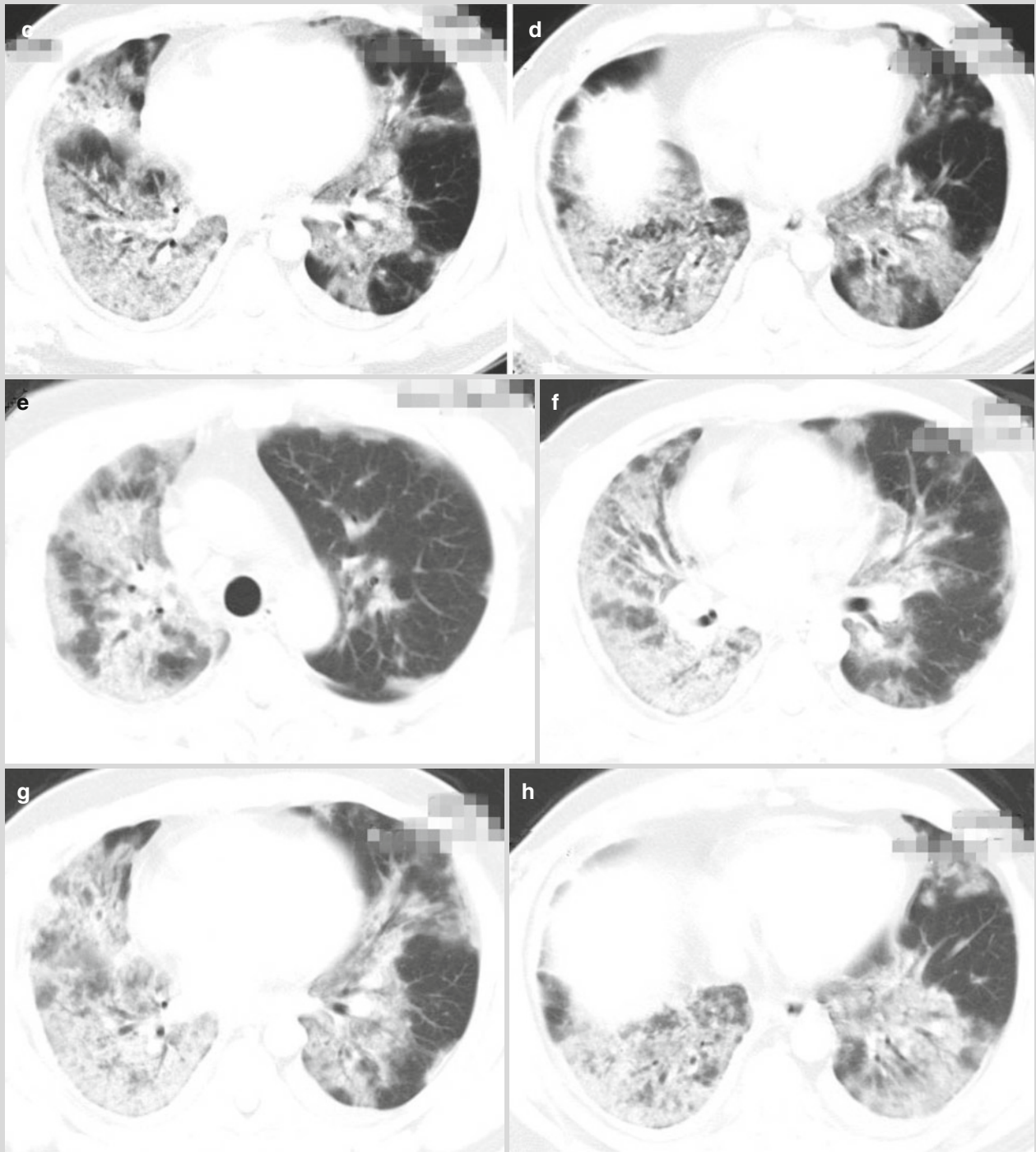


Fig. 9.19 (continued)

Case 20**[Brief Case History]**

A 34-year-old woman complained of fever for 7 days and difficulty breathing for 3 days. She experienced cough and expectoration of whitish or yellowish phlegm. The patient denied a history of contact to patient with type A influenza. By physical examination, body temperature 39 °C, pharynx congested, no tonsillar swelling; coarse breathing sound in both lungs but no moist rales. By laboratory test, the throat swab showed influenza A (H1N1) related etiological factors, such as FluA and SWH1, positive.

[Radiological demonstration] Fig. 9.20

[Diagnosis] Pneumonia induced by type A H1N1 influenza virus.

[Discussion]

Radiologically, the lesions in this case were demonstrated as diffuse ground glass opacity, poorly defined hilum

in both lungs, and accompanying air trapping. The condition should be differentiated from the following diseases:

1. Mycoplasmal pneumonia

The lesions are always demonstrated as centri-lobular ground glass density opacity or consolidation, which may develop into lobar or segmental consolidation in a fan like shape. But these lesions are rare in the cases of pneumonia induced by type A H1N1 influenza virus. Mycoplasmal pneumonia in children is radiologically demonstrated as grid like and spots of interstitial lesions, commonly with enlarged hilum and possibly pleural effusion.

2. Chlamydial pneumonia

The radiological signs of chlamydial pneumonia are non-specific, with flakes of and grid like opacity in unilateral or bilateral lower lung lobes, and the lesions are commonly constrained with slow progression, good mobility and favorable prognosis.

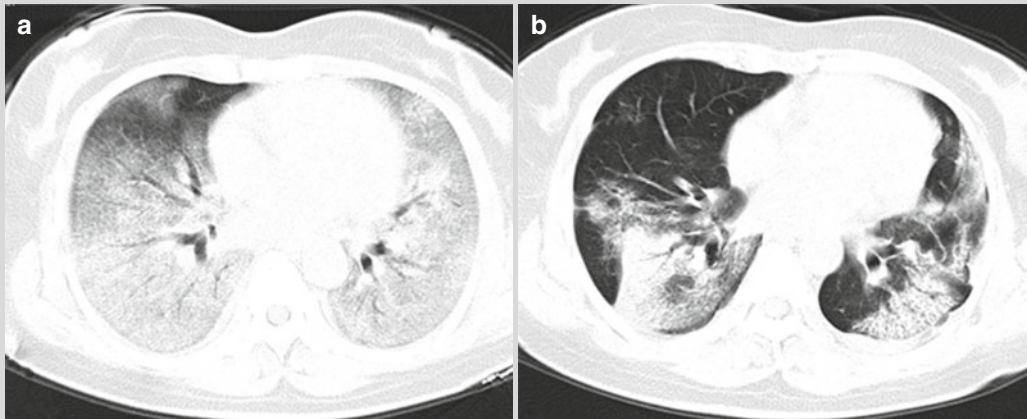


Fig. 9.20 Chest CT scan revealed diffuse ground glass like opacity with high density in both lungs, with air bronchogram inside (a). Reexamination after treatment for 18 days revealed cloud and

cotton like poorly defined opacity in the left upper lung lobe and bilateral lower lungs lobes; obvious absorption of the lesions (b)

Case 21**[Brief Case History]**

A 29-year-old man complained of cough for 7 days and fever for 6 days. He also showed wheezing and expectoration of pinkish foamy sputum. By physical examination, the body temperature was 38.6 °C; the pharynx congested; and moist rales in both lungs. He denied a history of contact to patient

with type A influenza. By laboratory test, the throat swab showed the M gene of type A influenza virus was positive, the NP gene of swine H1N1 influenza virus positive, and the HA gene of type A H1N1 influenza virus positive. By routine blood test, WBC $2.54 \times 10^9/L$, GR% 75.1 %, LY% 15.4 %. Blood gas analysis revealed pH 7.33, PCO_2 54 mmHg, and PO_2 85 mmHg. Blood chemistry revealed ALT

40U/L, AST 32U/L, Cr 115.9 $\mu\text{mol/L}$, and UREA 16.73 mmol/L. Reexamination after treatment for 9 days, routine blood test showed WBC $7.8 \times 10^9/\text{L}$, GR% 88.4%, and LY% 8.2%; by blood gas analysis, pH 7.512, and PO_2 48.8 mmHg, PCO_2 32.16 mmHg; liver function examination revealed ALT 166.8 IU/L, and AST 270.5 IU/L.

[Radiological and pathological demonstrations]

Fig. 9.21

[Diagnosis] Critical pneumonia induced by type A H1N1 influenza virus.

[Discussion]

The patient with critical pneumonia induced by type A H1N1 influenza virus is radiologically demonstrated with multiple large fused consolidations in both lung fields and extensive ground glass opacity around bronchi, which rapidly progressed. The lesions showed great changes even within 1 day and the condition may be complicated by ARDS, pneumothorax, mediastinal and subcutaneous emphysema, even retroperitoneal gas trapping.

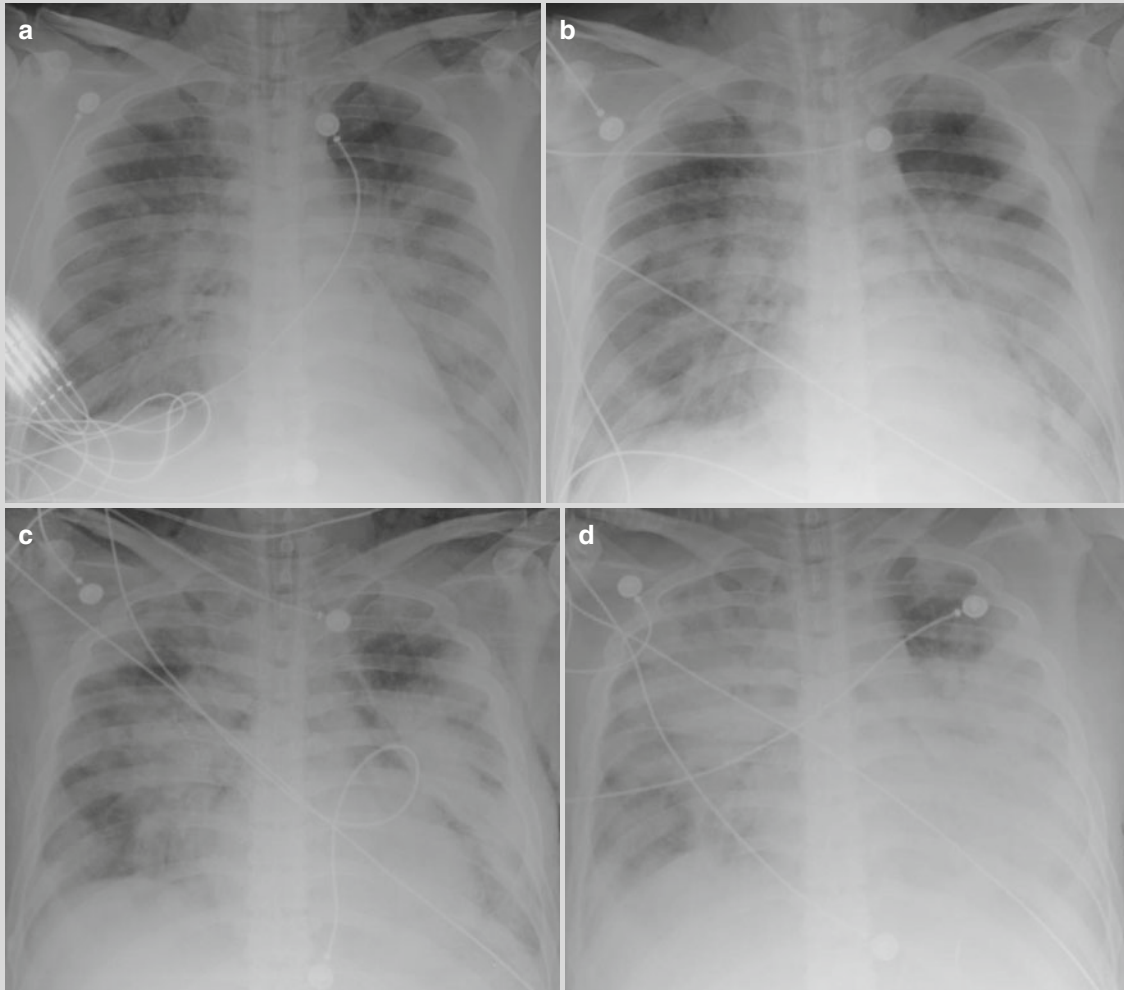


Fig. 9.21 Chest X-ray showed multiple poorly defined flakes of opacity in the bilateral middle and lower lung fields; enlarged and poorly defined hilum in both lungs; poorly defined bilateral costophrenic angles (a). Reexamination after treatment for 1 day revealed progression of the lesions. Chest X-ray showed enlarged range with multiple poorly defined flakes of opacity in the bilateral middle and lower lungs, which showed increased density (b). Reexamination after treatments for 4 days, chest X-ray showed enlarged range with consolidations in both lungs (c). Reexamination after treatments for 5 days, the lesions were shown with progression (d). Reexamination

after treatments for 11 days, the lesions were improved and chest X-ray showed poorly defined flakes and cotton like opacity and increased transparency of both lungs (e). Pathologically, the alveolus was shown with interlobular septal thickening and alveolar wall congestion; infiltration of neutrophils and plasmocytes, predominantly mononuclear cells; exudation of intra-alveolar edema fluid and fibrin (H&E staining, $\times 20$) (f, g). Pathologically, the myocardial space was shown with a large quantity of inflammatory cells (H&E staining, $\times 20$) (h, i) (Reprint with permission from Hongjun Li and Ning Li (Eds), *Radiology of Influenza A (H1N1)*, Springer, 2013)

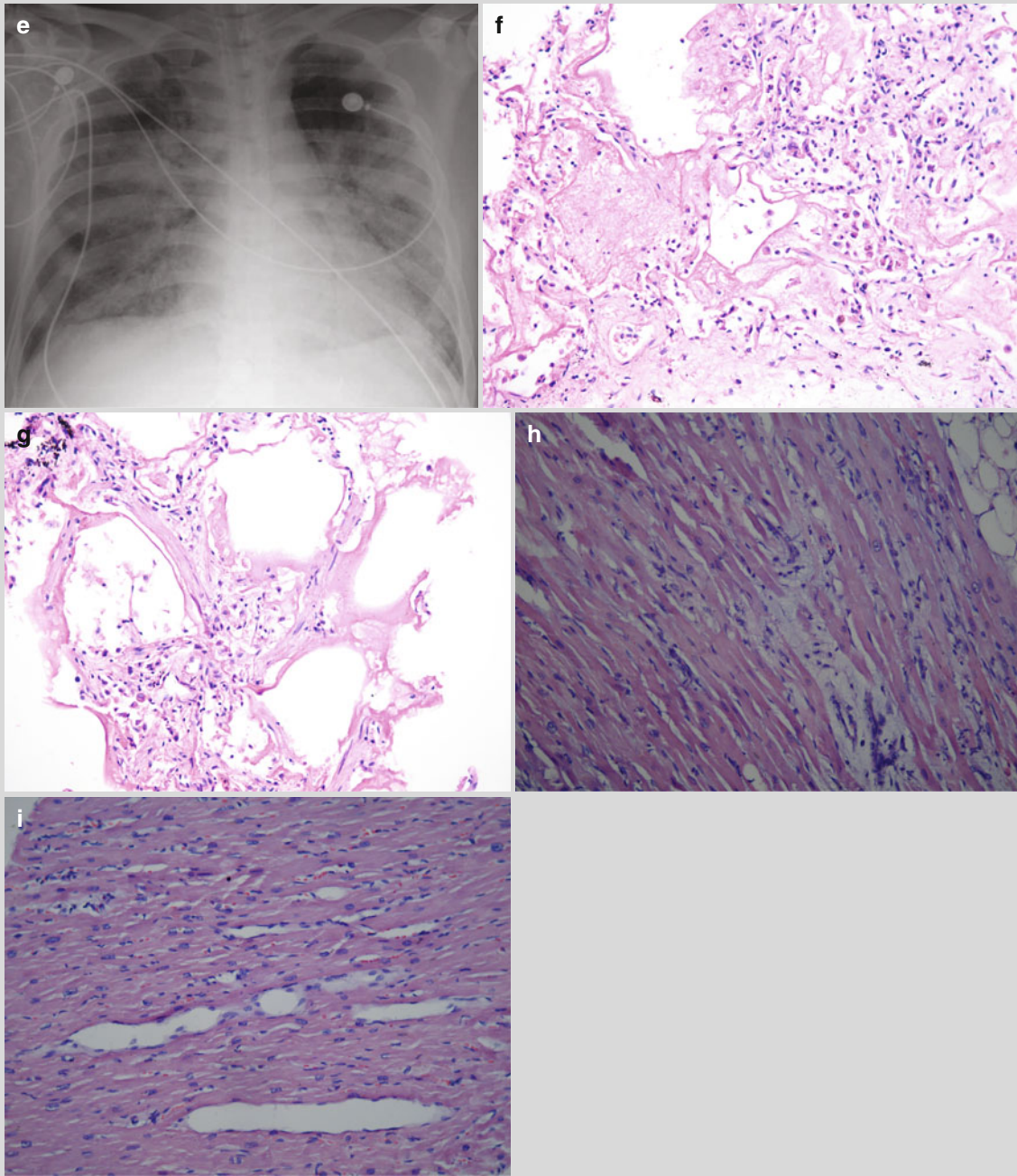


Fig. 9.21 (continued)

Case 22**[Brief Case History]**

A 21-year-old young woman complained of fever and cough for 9 days. She also experienced cough and expectoration of yellowish sputum, which failed to be improved by anti-inflammatory therapy, with a body temperature fluctuating between 38 °C and 38.7 °C, chest distress, shortness of breath, and expectoration of yellowish sputum. By physical examination, the body temperature 39.7 °C; the pharynx congested; the tonsil swollen in degree I; moist rales in both lungs that were absent after coughs. She reported a non-definitive history of contact to patient with type A influenza. By laboratory test, the throat swab showed that the M gene of type A influenza virus positive, the NP gene of swine H1N1 influenza virus negative, and the HA gene of type A H1N1 influenza

virus positive. By routine blood test, WBC $8.64 \times 10^9/L$, GR% 76.14%, LY% 19.34%; by blood chemistry, CK 140U/L, and LDH 547U/L.

[Radiological demonstration] Fig. 9.22

[Diagnosis] Pneumonia induced by type A H1N1 influenza virus and bronchiectasis.

[Discussion]

Radiologically, severe pneumonia induced by type A H1N1 influenza virus is demonstrated as the followings: (1) multifocal or diffuse ground glass opacity; (2) multifocal ground glass opacity and consolidation opacity; (3) multifocal or diffuse consolidation; (4) distribution of the lesions in the subpleural lung periphery or around bronchovascular bundles; (5) rapid progression of the lesions into diffuse ground glass opacity or consolidation within 1–3 days.

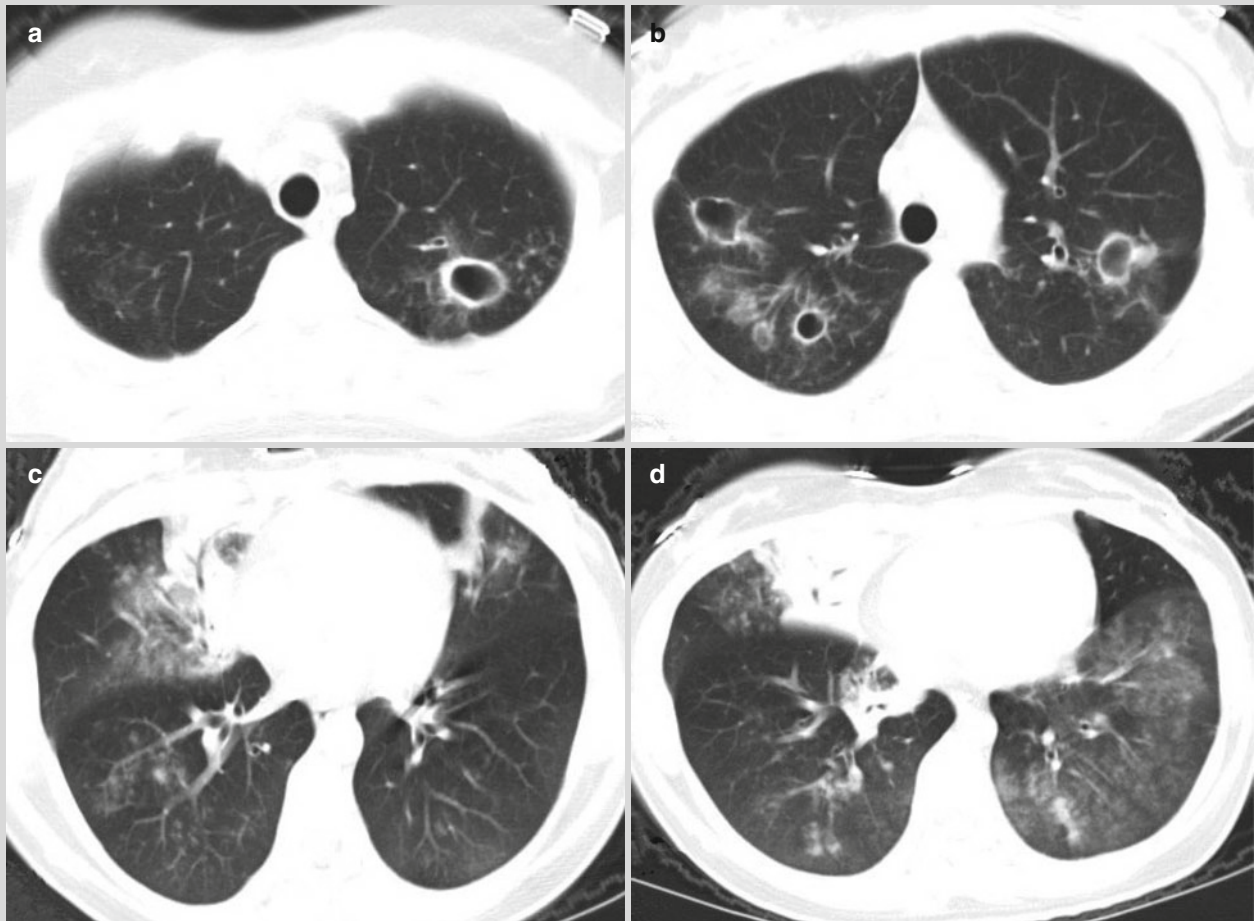


Fig. 9.22 CT scan showed multiple irregular thin-wall cavities in the bilateral upper lung lobes, which was surrounded by sporadic patches of opacity; bronchial wall thickening in the left upper lung lobe, with widened bronchial lumen (a, b). CT scan showed multiple

consolidations and ground glass opacities in both lungs; air bronchogram in consolidation opacity of the medial segment in the right upper lung lobe (c, d)

Case 23**[Brief Case History]**

A 57-year-old woman complained of right upper abdominal pain for 3 days, whose aggravation, chest distress and shortness of breath for 2 days, with disturbance of consciousness. The pain in the right upper quadrant was paroxysmal migratory. She had a medical history of chronic bronchitis for more than 20 years, with long-term use of hormone and edema of lower limbs. She denied medical histories of hypertension and diabetes, but did experienced symptoms of polyphagia, polydipsia and polyuria. The patient reported a history of contact to patient with type A influenza. By physical examination, she was in coma, overweight, ecchymosis all over the body; severe edema of bulbar conjunctiva, conjunctiva hemorrhage in the left eyelid, both pupils in equal size and round being sensitive to light; overwhelming moist rales in both lungs with occasional dry rales; heart rate 102/min, no pathological murmur in each auscultatory valvular regions; abdominal circumference 100 cm, abdominal tension increased; pitting edema inferior to elbow joints of upper limbs and inferior to knees of lower limbs. By laboratory test, the throat swab showed that the M gene of type A influenza virus positive, the NP gene of swine H1N1 influenza virus positive, and the HA gene of type A H1N1 influenza virus positive. By routine blood test, WBC $21.52 \times 10^9/L$, GR% 93%, HGB 139 g/L, PLT $88 \times 10^9/L$. Blood gas analysis revealed pH 7.141, PCO_2 19.1 mmHg, PO_2 60 mmHg, SO_2 92%, BE -22 mmol/L, K^+ 5.2 mmol/L. Blood chemistry revealed ALB 26.7 g/L, GLOB 46 g/L, GLU 42.8 mmol/L, BUN 14.12 mmol/L, osmotic pressure 327 mmol/L. By routine urine test, KET strongly positive (++++), GLU strongly positive (++++). Reexamination after treatment for 1 day, blood gas analysis revealed pH 7.417, PCO_2 48 mmHg, PO_2 100 mmHg, BE 6 mmol/L. Reexamination after treatment for 4 days, by routine blood test, WBC $15.9 \times 10^9/L$, GR% 87.5%, HGB 117 g/L, PLT $113 \times 10^9/L$; blood gas analysis revealed pH 7.567, PCO_2 45.5 mmHg, PO_2 74 mmHg, BE 19 mmol/L, HCO_3^- 41.4 mmol/L, SPO_2

96%; blood chemistry revealed ALT 50U/L, AST 48U/L, ALB 33.2 g/L, GGT 883U/L, PA 136 mg/L, GLU 12.57 mmol/L, GRF 56 ml/min, LACT 2.3 mmol/L. The erythrocyte sedimentation rate 78 mm/h, PT within normal limites, D-dimer positive. Sputum smear revealed negative bacilli in a small quantity, no fungal spore or acid-fast bacillus. By sputum culture, acinetobacter baumannii positive. Reexamination after treatment for 6 days, by routine blood test, WBC $15.5 \times 10^9/L$, GR% 88.8%, PLT $164 \times 10^9/L$, HGB 109 g/L; by blood chemistry, ALB 34.4 g/L, PA 158 mg/L, CRP 87.6 mg/L, GLU 10.46 mmol/L, HBDH 255U/L, LDH 357U/L; by blood gas analysis, pH 7.43, PCO_2 64 mmHg, PO_2 67 mmHg, BE 14.9 mmol/L, HCO_3^- 41.4 mmol/L, FiO_2 60%, Na^+ 144 mmol/L, and K^+ 3.5 mmol/L. By sputum culture, gram negative bacilli grew in a small quantity. Sputum smear revealed a small amount of positive cocci and negative cocci; a great amount of negative bacilli and fungal spore; D-dimer weakly positive (\pm). By thoracocentesis, 200 ml dark red bloody pleural fluid was drained.

[Radiological demonstration] Fig. 9.23

[Diagnosis] Pneumonia induced by type A H1N1 influenza virus; right hydropneumothorax; a small quantity of pleural effusion in the left thoracic cavity.

[Discussion]

Severe pneumonia induced by type A H1N1 influenza virus is radiologically characterized by diffuse ground glass opacity and consolidation in the lung periphery of both lungs and their rapid progression. The lesions often progress into diffuse ground glass opacity or consolidation in both lungs within 1–3 days. And the condition is commonly complicated by pneumothorax and mediastinal emphysema. In this case, the lesions in both lungs alternatively aggravated and alleviated, with changes by reexaminations each day. Therefore, the patients with pneumonia induced by type A H1N1 influenza virus should receive chest plain X-ray or CT scan each day during the following-ups for immediate understandings about the progression of the lesions and appropriate treatment.

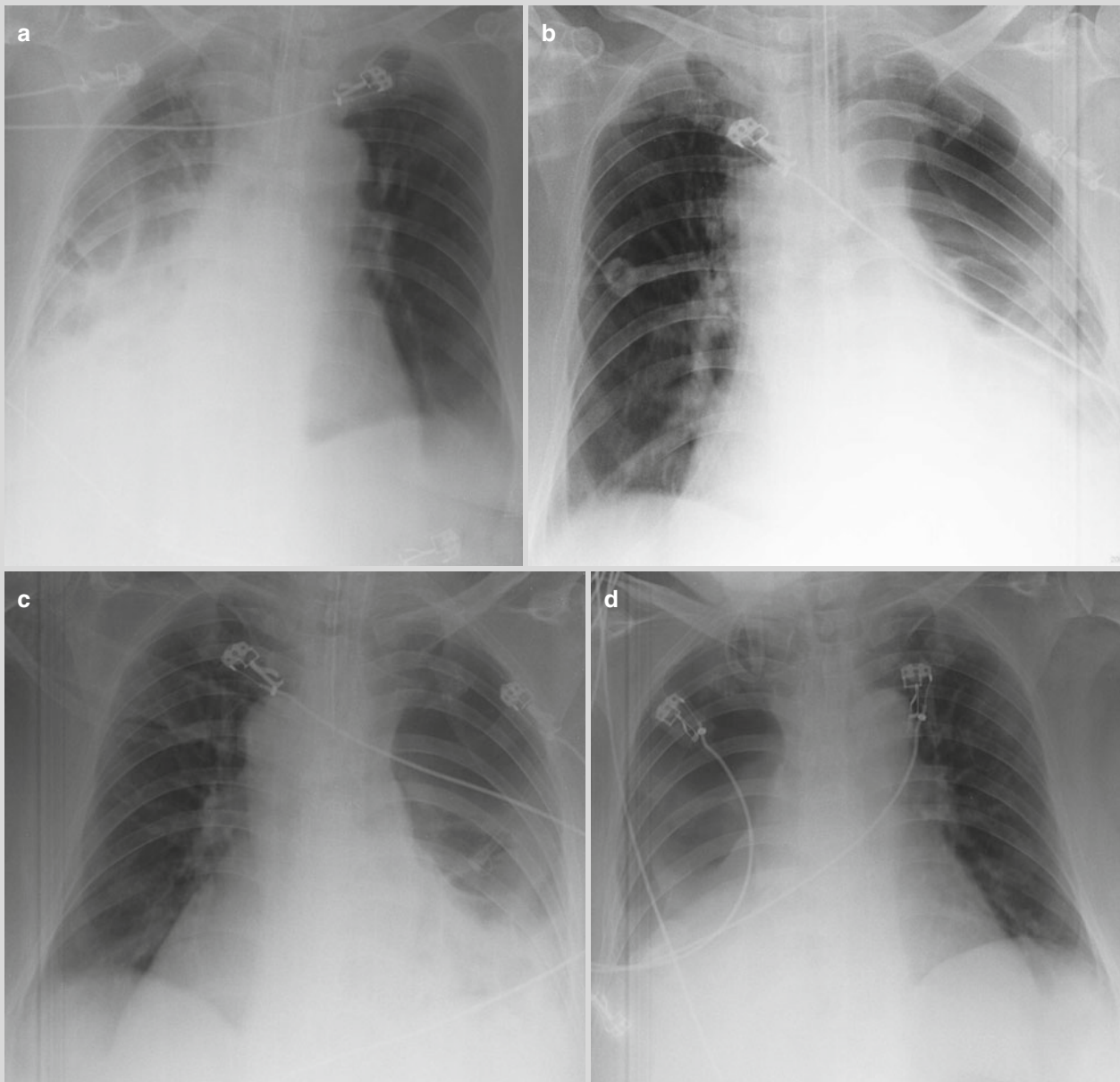


Fig. 9.23 Chest X-ray showed diffuse consolidation in the right middle and lower lung fields and in the left lower lung; poorly defined right diaphragm (a). Reexamination after treatment for 2 days, the right lung was shown with increased transparency and sporadic ground glass opacity; the left middle and lower lung fields with consolidation; poorly defined left diaphragm (b). Reexamination after treatment for 3 days, the lesions in both lower lungs progressed (c). Reexamination after treatment for 4 days, the range with consolidation in the right lung was enlarged; oval shaped area in the right upper lung with no lung markings; smaller range with consolidation in the left lung that showed decreased density (d). Reexamination after treatment for 5 days, the range with consolidations in the right lung enlarged; increased density of the con-

dolidation opacity; no change in the area with no lung markings in the right upper lung; multiple light flakes of opacity in the left lung, with thickened lung marking (e). Reexamination after treatment for 6 days, the right lung was shown with shrinkage and increased density of the lesions; the left lower lung was shown with multiple cloud like and cotton like consolidation; poorly defined bilateral diaphragm (f). Reexamination after treatment for 6 days, CT scan showed consolidation in the right lung and obvious shrinkage of the right lung, with a small amount of normal lung issue; right pneumothorax with air-fluid level; multiple cords like opacity in the left lung and a small quantity of pleural effusion in the left thoracic cavity (g, h) (Reprint with permission from Hongjun Li and Ning Li (Eds), *Radiology of Influenza A (H1N1)*, Springer, 2013)

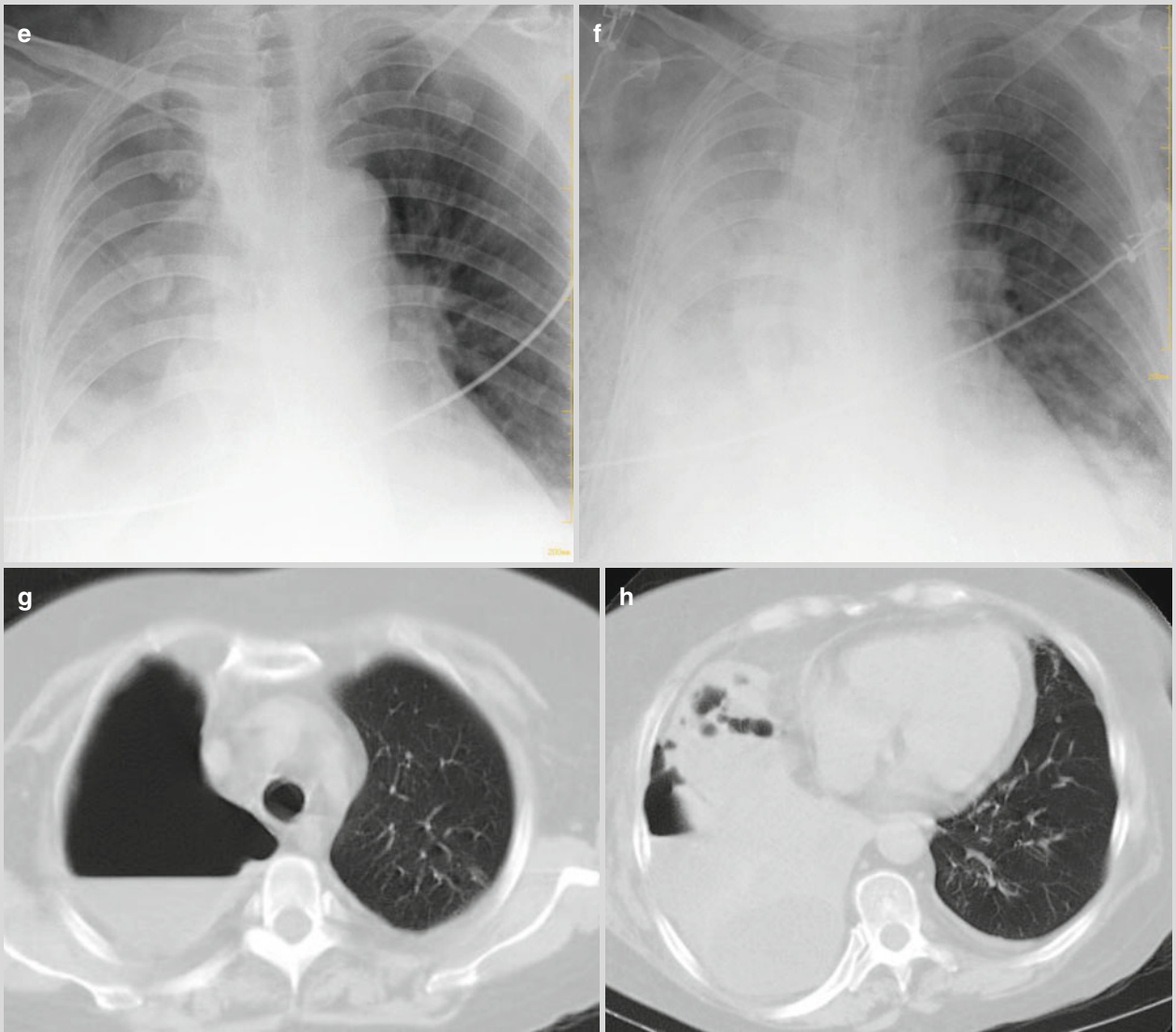


Fig. 9.23 (continued)

Case 24**[Brief Case History]**

A 45-year-old woman complained of aversion to cold and fever for 5 days with the highest body temperature of 39.5 °C. She also experienced symptoms of dry cough, expectoration of a small quantity of sputum, chest distress, choking and eye upset. In another hospital, she was diagnosed with bronchitis and was then treated by anti-inflammatory therapy, but showing symptoms of chest distress and shortness of breathing then. After the patient was transferred to our hospital, death occurred due to respiratory failure. She denied a history of contact to patient with type A influenza or patient with flu like symptoms. By physical examination, the body temperature 39 °C and the

pharynx congested. By laboratory test, the throat swab showed the M gene of type A influenza virus negative, the NP gene of swine H1N1 influenza virus positive, and the HA gene of type A H1N1 influenza virus positive. By routine blood test, WBC $3.39 \times 10^9/L$, GR% 81.1%, and LY% 14.5%. Blood gas analysis revealed pH 7.466, PCO_2 35.5 mmHg, and PO_2 76 mmHg. Liver function examination showed ALT 21.1U/L, AST 51.3U/L, UREA 3.34U/L, and CREA 41.0U/L.

[Radiological and pathological demonstrations as well as gross observation] Fig. 9.24

[Diagnosis] Critical pneumonia induced by type A H1N1 influenza virus.

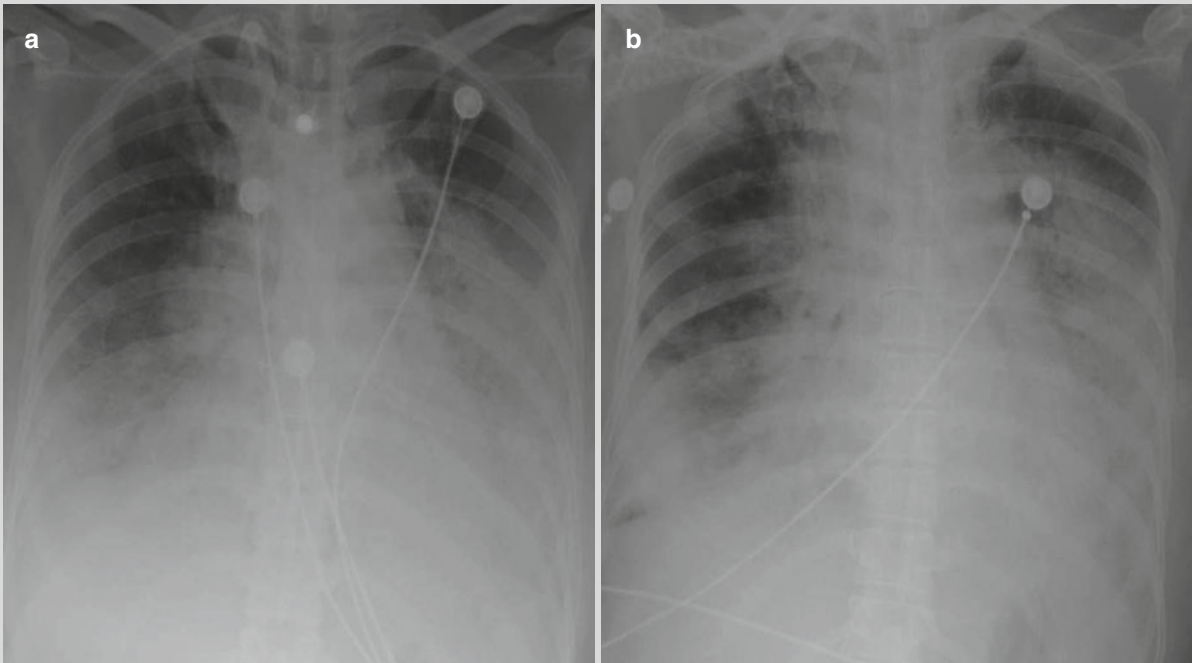


Fig. 9.24 Chest X-ray showed large flakes of poorly defined high density opacity in the middle and lower lung fields of both lungs; poorly defined bilateral hilum and diaphragm (a). Reexamination after treatment for 11 days showed enlarged range with lesions in both lungs and increased density of the lesion (b). Reexamination after treatment for 18 days showed diffuse high density opacity in both lungs (c). Reexamination after treatment for 32 days showed absorption and improvement of the lesions in both upper lungs (d). Reexamination after treatment for 33 days showed increased transparency of the left middle and upper lung fields (e). Reexamination after treatment for 34 days showed progression of the lesions in both lungs, in white-lung change (f). After treatment for 37 days, death occurred. CT scan after death for 72 h showed rightward shift of the mediastinum, bilateral pleural effusion in large quantities with air-fluid level in the left pleural cavity (g). By autopsy after

death for 72 h, the gross specimen showed lung tissue fibrosis and patches of hemorrhage (h); the microscopic observation showed a small amount of alveolar cells, fibrosis of most lung tissues, many inflammatory cells, and intra-alveolar hemorrhage (H&E staining, $\times 10$) (i); a small quantity of alveolar cells, fibrosis of most lung tissues, absence of bronchiolar epithelial cells, a large quantity of inflammatory cells that predominantly macrophages. All of these findings were in consistency with necrotic bronchitis induced diffuse alveolar damages and alveolar hemorrhage (H&E staining, $\times 40$) (j); many inflammatory cells in the interhepatocytic space ((k) H&E staining, $\times 10$; (l) H&E staining, $\times 40$); renal sinus dilation, multiple inflammatory cells in the renal parenchymal space ((m) H&E staining, $\times 10$; (n) H&E staining, $\times 40$); a small amount of inflammatory cells in the myocardial cells space ((o) H&E staining, $\times 10$; (p) H&E staining, $\times 40$)

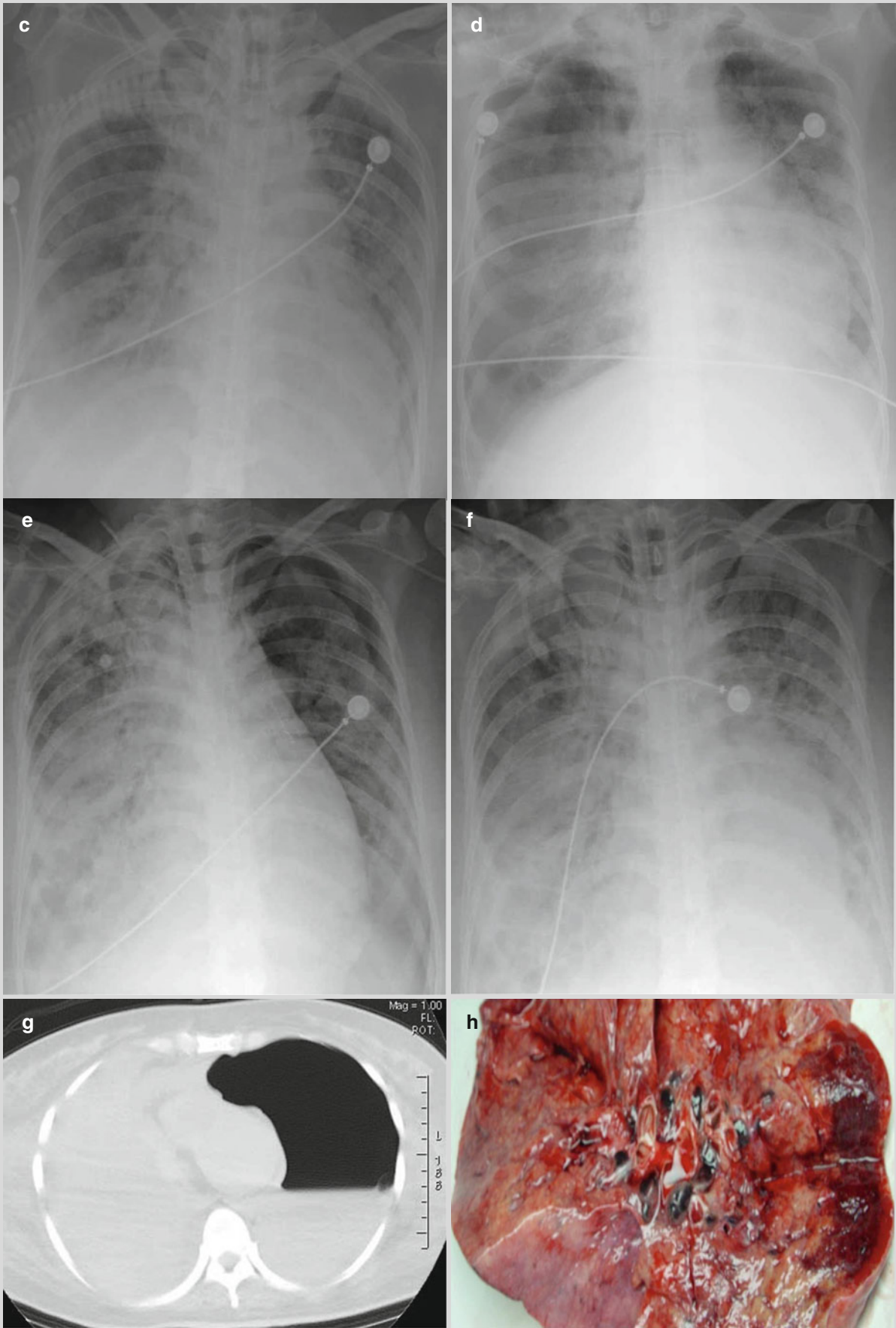


Fig. 9.24 (continued)

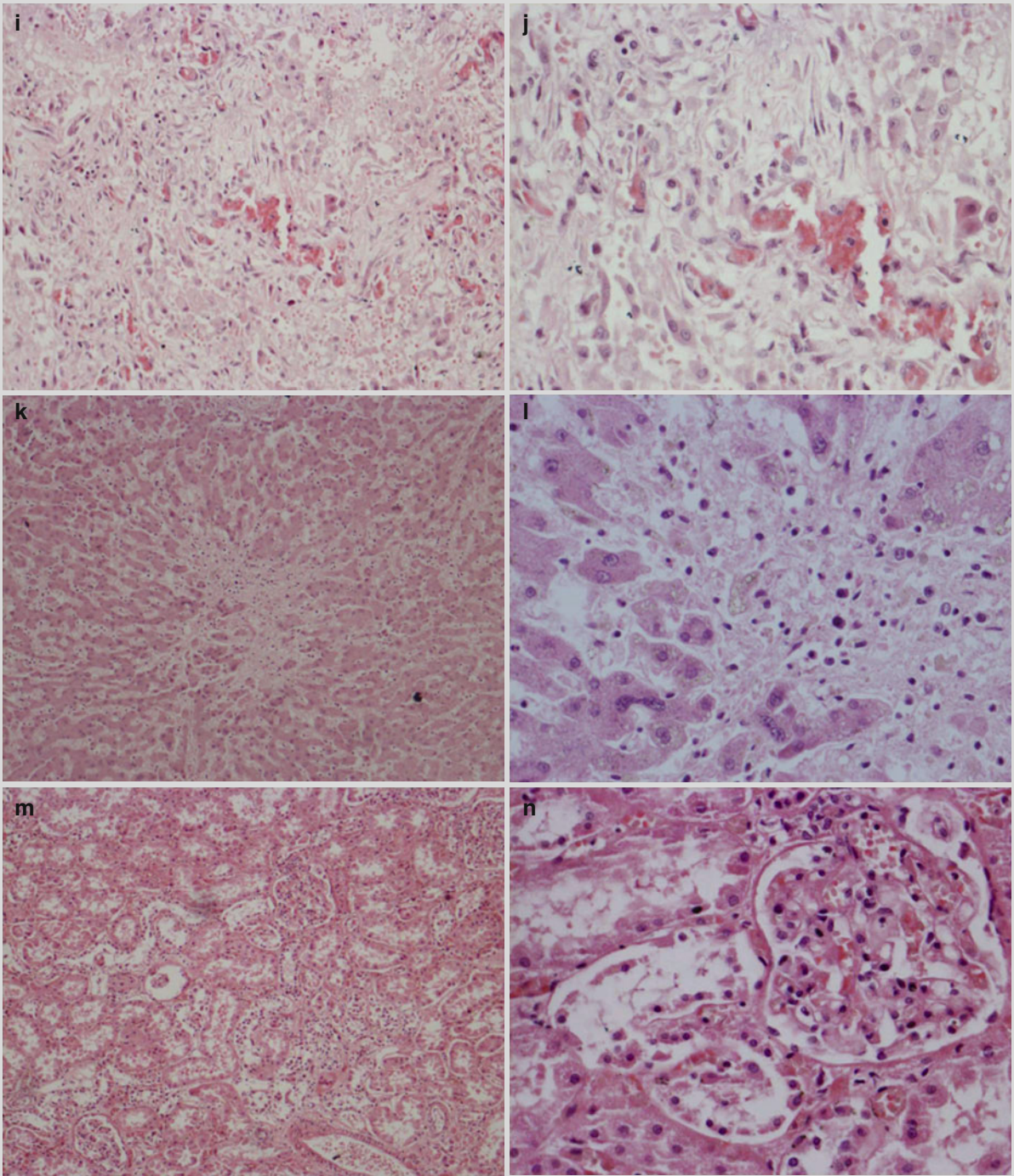


Fig. 9.24 (continued)

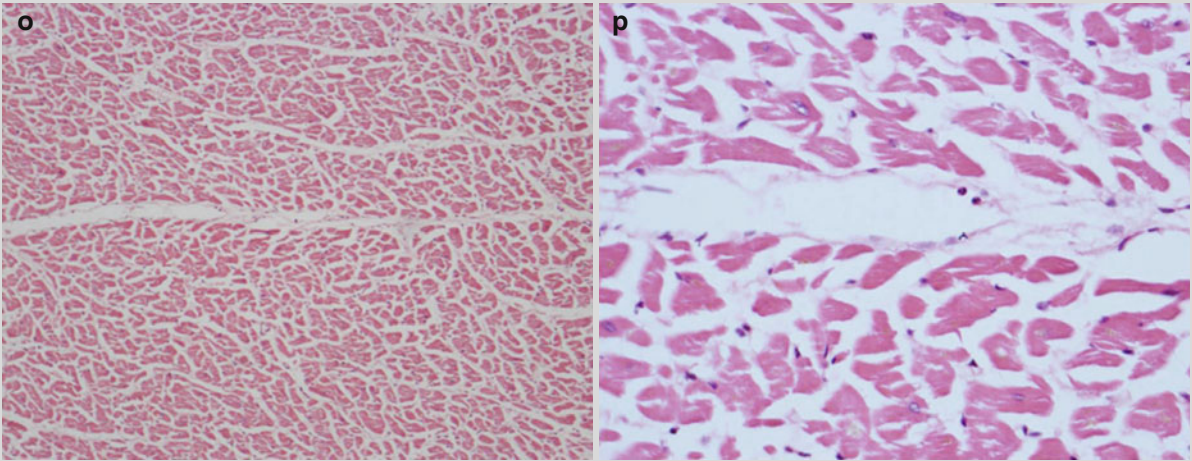


Fig. 9.24 (continued)

Case 25

[Brief Case History]

A 48-year-old man complained of fever for 5 days with the highest body temperature of 39 °C. He also experienced expectoration of yellowish thick sputum, shortness of breathing and chest distress. He had medical histories of diabetes, chronic renal insufficiency and chronic bronchitis. He denied a history of contact to patient with type A influenza. By physical examination, the pharynx congested and tonsil swollen in degree I. By laboratory test,

the throat swab in laboratory of CDC showed the M gene of type A influenza virus positive, the NP gene of swine H1N1 influenza virus positive, and the HA gene of type A H1N1 influenza virus positive. Routine blood test showed WBC $8.1 \times 10^9/L$, RBC $2.49 \times 10^{12}/L$. Blood gas analysis revealed pH 7.13, PCO_2 41 mmHg, PaO_2 60 mmHg. Blood chemistry revealed AST 76.3U/L, ALT 13.4U/L. The clinical diagnosis was made as critical influenza A (H1N1). Autopsy was performed after occurrence of death.

[Pathological demonstration] Fig. 9.25

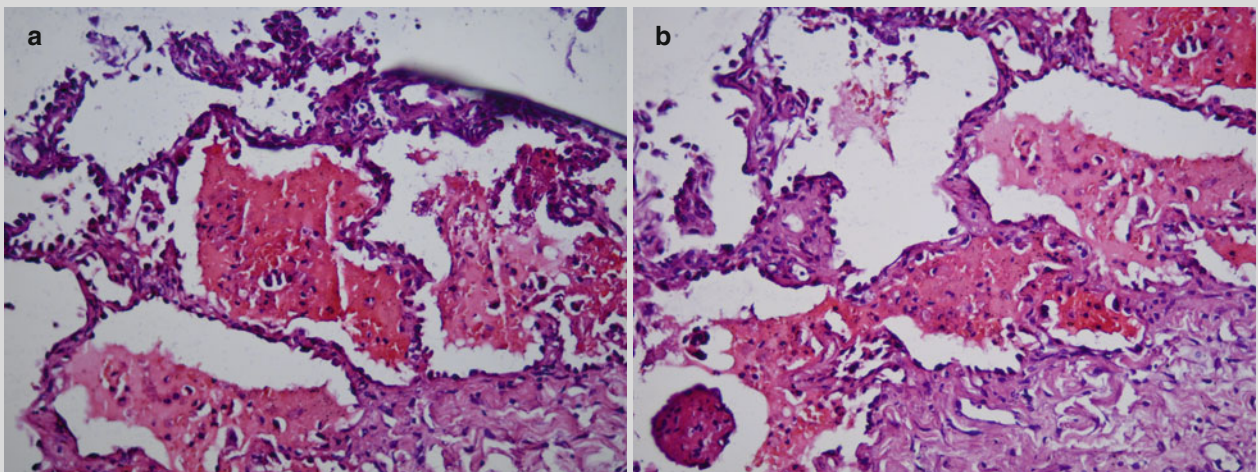


Fig. 9.25 (a, b) Capillary edema and congestion; infiltration of inflammatory cells ((a) H&E staining, $\times 40$; (b) H&E staining, $\times 20$); (c, d) infiltration of a large quantity of inflammatory cells in myocardial tissue ((c) H&E staining, $\times 40$; (d) H&E staining, $\times 20$)

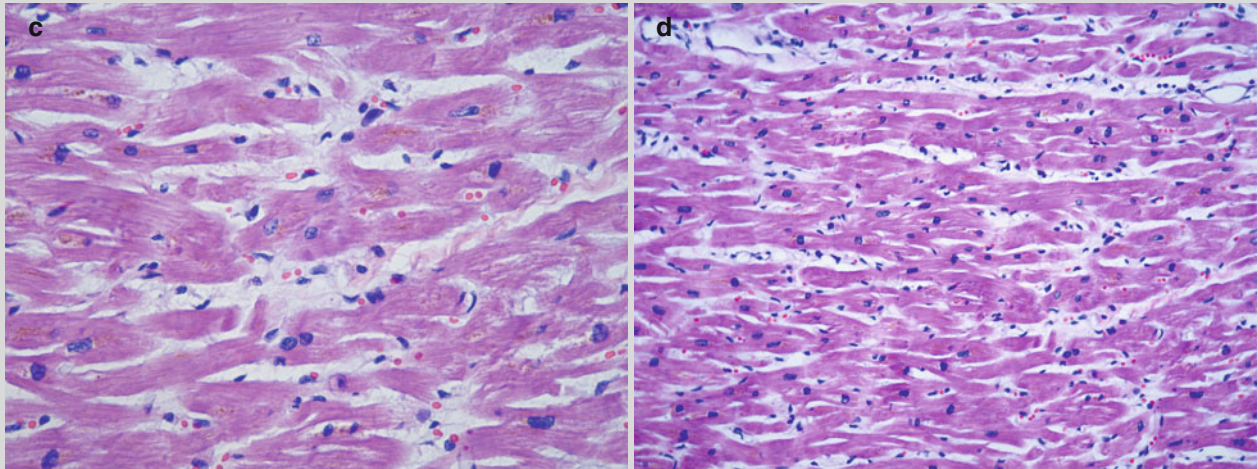


Fig. 9.25 (continued)

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