

Research article

Imaging manifestations and pathological analysis of severe pneumonia caused by human infected avian influenza (H7N9)[☆]

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Received 27 November 2014; accepted 7 January 2015

Available online 2 March 2015

Abstract

Objective: To investigate the imaging and pathological findings of severe pneumonia caused by human infected avian influenza (H7N9), and therefore to further understand and improve diagnostic accuracy of severe pneumonia caused by human infected avian influenza (H7N9).

Methods: The relevant clinical and imaging data of 19 cases, including 10 males and 9 females, with pneumonia caused by human infected avian influenza (H7N9) was retrospectively analyzed. One of the cases had received percutaneous lung biopsy, with the clinical, imaging and pathological changes possible to be analyzed.

Results: The lesions were mainly located at lower lobes and dorsal of lungs, involving multiple lobes and segments. Ground-glass opacities and/or pulmonary opacities were the more often imaging manifestations of severe pneumonia caused by human infected avian influenza (H7N9) in early and evolving phases (19/19,100%). By biopsy following percutaneous lung puncture, exudation of slurry, cellulose, RBC and neutrophils, formation of hyaline membrane, squamous metaplasia and organizing exudates were observable at the alveolar space. Some of alveoli collapsed, and some responded to show compensatory emphysema.

Conclusion: The imaging features of severe pneumonia caused by human infected avian influenza (H7N9) include obvious ground-glass opacity and pulmonary consolidation, mainly at lower lobes and dorsal of lungs, with rapid changes. The cross-analysis of imaging and pathology preliminary can elucidate the pathological mechanisms of ground-glass opacities and pulmonary consolidation of severe pneumonia. Such an intensive study is beneficial to prompt clinicians to observe and evaluate the progress of the disease. In addition, it is also in favor of managing the symptoms and reducing the mortality rate.

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Keywords: Pneumonia; Severe cases; Viral; Avian influenza A (H7N9); Radiography; Lungs; Tomography; X-ray computed; Pathogenic manifestations

Human infected avian influenza (H7N9) is an acute respiratory infection caused by H7N9 subtype of avian influenza A virus [1]. The disease was firstly reported in the spring of 2013 in Yangtze River Delta, China [2]. A total of 23 cases of human infected avian influenza (H7N9) was admitted to our

hospital from December, 2013 to April, 2014, with 19 cases suffering from severe pneumonia. In this study, we retrospectively analyzed imaging and pathological manifestations of the disease to shed light on its clinical diagnosis, therapeutic efficacy evaluation and prognosis.

1. Materials and methods

1.1. Basic data

The clinical, radiological and pathological data of 19 cases with severe pneumonia caused by human infected avian influenza (H7N9) from December 18, 2013 to April 18, 2014

[☆] Foundation project: major project of the knowledge innovation program in Shenzhen. (Serial number: JCYJ20130401164750006). The Medical Research Foundation of Guangdong Province. (Serial number: A2011543).

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Peer review under responsibility of Beijing You'an Hospital affiliated to Capital Medical University.

in Shenzhen Third People's Hospital, China, were collected. All the patients were tested positive to nucleic acid of H7N9 subtype of avian influenza virus by CDC of Guangdong province and Shenzhen city, China, in line with the diagnostic criteria of severe human infected avian influenza. The 19 cases included 10 males and 9 females, aged 31–82 years with a median of 55 years. Two patients had preexisting hypertension; 3 had hypertension and diabetes; 1 had tuberculosis; and 1 had right pulmonary embolism.

1.2. Clinical manifestations

Fever was the most common symptom, found in all 19 cases (100%), and ardent fever (39 °C or above) showed up in 15 patients (78.9%). Cough was also the most common symptom, occurring in all 19 cases (100%), with expectoration in 13 cases (68.4%) and 1 case coughing up dark red bloody sputum. Anhelation occurred in 11 cases (57.9%). All patients were admitted to our hospital at d 4–14 after onset, averagely 8.4 days, and received antiretroviral and respiratory supporting therapies.

1.3. Epidemiology

Six patients assured of a history of contact to live poultry, by another 5 patients denied a history of contact to live poultry. The epidemiologic data of the other 8 patients was not available.

1.4. Laboratory tests

WBC count was detected to have a decrease in 7 cases, being from $2.20 \times 10^9/L$ to $7.11 \times 10^9/L$, with a mean of $4.69 \times 10^9/L$. Neutrophil percentage increased in 13 cases, but remained normal level in 6 cases, being from 63.0% to 92.9%, with a mean of 76.8%.

1.5. Diagnostic criteria of severe pneumonia

According to the diagnostic and treatment protocol for human infected avian influenza A (H7N9) established by National Council on Health and Family Planning Commission, P. R. China (edition, 2014), the cases with any one of the following criteria can be diagnosed as severe.

- (1) Chest X-ray demonstrates lesions with multiple lobes involved or lesions progress more than 50% within 48 h;
- (2) Difficulty breathing, with more than 24 breaths per minute;
- (3) Severe hypoxemia, with oxygen flow at 3–5 L per minute and $SpO_2 \leq 92\%$;
- (4) Shock, ARDS or MODS (multiple organ dysfunction syndrome).

1.6. Pathological examination

Biopsy following percutaneous lung tissue puncture was performed in one case to observe the pathological changes. The pathogenic bacteria was observed after PAS and Masson

staining. Acid-fast bacteria staining was performed to detect possible infection of *Mycobacterium tuberculosis*.

1.7. Radiological modality

Conventional chest X-ray was performed using Philips DiDi TH/VR, with the tube voltage 102 kV and automatic tube current. Bedside chest X-ray was performed using Hitachi Sirisu130HP mobile DR, with a tube voltage 100 kV and automatic tube current. CT scanning was performed using Toshiba TSX-101A 64-slice spiral CT, with a tube voltage 135 kV, automatic tube current, a pitch of 0.9, a matrix of 512×512 , an FOV of $320 \text{ mm} \times 320 \text{ mm}$, a thickness of 5.0–6.0 mm, and an interval of 1 mm. CT scanning was from the apex to the bottom of lungs continuously. Lung window was reconstructed using conventional 1 mm and high resolution of 5 mm after the scanning.

1.8. Image analysis

All the images were independent analyzed by two radiologists with a title above associate chief-physician, and consensus was reached after consultation and discussion. The images were analyzed in terms of distribution and range of lesions, morphology of the lesions as well as changes of mediastinum and pleura.

2. Results

2.1. CT scans

- (1) CT manifestations at the early phase (d 1–4 after onset)

The lesions more often onset from a lower lung lobe (17/19), only 2 cases had their lesions onset from an upper lung lobe. Poorly-defined patches of shadows and fragmental ground-glass opacities were demonstrated by CT scanning. Changes of pulmonary interstitium were observed, including interlobular septal thickening, acinar nodules, and other changes. Chest CT scanning demonstrated rapid progress of the lesions within a short period of 1–2 days, with rapid expansion, fusion, formation of large patchy opacities, and the lesions were demonstrated to involve multiple lobes (Fig. 1).

- (2) CT manifestations at the evolving phase (d 5–10 after onset)

The lesions of 18 cases involved both lungs (18/19, 94.7%), and only 1 case showed lesions with unilateral lung involved (1/19, 5.3%). The lesions of 19 cases involved median lobe (lingual lobe) or lower lobe (19/19, 100%). The lesions of 18 cases involved 4 to 6 lung segments (18/19, 94.7%).

Ground-glass opacity was demonstrated by CT scanning in all 19 cases (100%), which were poorly defined in fragments and large patches. Pulmonary consolidation was also demonstrated in all 19 cases (19/19, 100%), which was more commonly found at the lower lung lobes to cross segments or

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