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RESEARCH ARTICLE

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Differential diagnosis of coronavirus disease 2019 pneumonia or influenza A pneumonia by clinical characteristics and laboratory findings

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Abstract

Background: Pneumonia caused by the 2019 novel Coronavirus (COVID-2019) shares overlapping signs and symptoms, laboratory findings, imaging features with influenza A pneumonia. We aimed to identify their clinical characteristics to help early diagnosis.

Methods: We retrospectively retrieved data for laboratory-confirmed patients admitted with COVID-19–induced or influenza A–induced pneumonia from electronic medical records in Ningbo First Hospital, China. We recorded patients' epidemiological and clinical features, as well as radiologic and laboratory findings.

Results: The median age of influenza A cohort was higher and it exhibited higher temperature and higher proportion of pleural effusion. COVID-19 cohort exhibited higher proportions of fatigue, diarrhea and ground-glass opacity and higher levels of lymphocyte percentage, absolute lymphocyte count, red-cell count, hemoglobin and albumin and presented lower levels of monocytes, c-reactive protein, aspartate aminotransferase, alkaline phosphatase, serum creatinine. Multivariate logistic regression analyses showed that fatigue, ground-glass opacity, and higher level of albumin were independent risk factors for COVID-19 pneumonia, while older age, higher temperature, and higher level of monocyte count were independent risk factors for influenza A pneumonia.

Conclusions: In terms of COVID-19 pneumonia and influenza A pneumonia, fatigue, ground-glass opacity, and higher level of albumin tend to be helpful for diagnosis of COVID-19 pneumonia, while older age, higher temperature, and higher level of monocyte count tend to be helpful for the diagnosis of influenza A pneumonia.

KEYWORDS
clinical characteristics, COVID-19, differential diagnosis, influenza A, pneumonia

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1 | INTRODUCTION

Coronaviruses (CoVs) are non-segmented positive-stranded RNA viruses with a roughly 30 kb genome surrounded by a protein envelope. CoVs belong to order Nidovirales, family Coronaviridae, subfamily Coronavirinae,[1] and have been classified into four major groups: α-CoVs, β-CoVs, γ-CoVs, and δ-CoVs.[2] Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) is the seventh known coronavirus to infect humans, after 229E, NL63, OC43, HKU1, MERS-CoV, and the original SARS-CoV.[3] SARS-CoV-2 presents highly identical genome to bat coronavirus; thus, the bat is regarded pointing to bat as the natural host. Two serious coronavirus disease have outbroken in the past two decades, including severe acute respiratory syndrome (SARS) in 2003[4] and Middle East respiratory syndrome (MERS) in 2012.[5] The coronavirus disease 2019 (COVID-19) outbreak caused by SARS-CoV-2 first began in Wuhan in Hubei province, China, in December 2019[6] and marked the third introduction of a highly pathogenic and large-scale epidemic coronavirus into the human population in the 21st century. COVID-19 has spread throughout not only China but also other counties worldwide. With the number of coronavirus cases, deaths, as well as affected countries climbing, on March 11, World Health Organization (WHO) made the assessment that COVID-19 can be characterized as a "pandemic" as the virus spreads increasingly worldwide. As of September 3, WHO reported a total of 25,884,895 confirmed COVID-19 patients, including 859,130 deaths globally.

Seasonal influenza viruses cause approximately 3–5 million severe cases and 290,000–650,000 deaths each year worldwide.[7] In particular, four human pandemic influenza outbreaks occurred in 1918 (H1N1), 1957 (H2N2), 1968 (H3N2), and 2009 (H1N1). As mentioned above, COVID-19 pandemic has posed a major public health problem worldwide and shared characteristics in common with influenza pandemic: both ranking at the highest pandemic level to result in tremendous morbidity and mortality, both initiating winter respiratory disease and giving rise to pneumonia. Influenza illness is clinically characterized by non-specific signs and symptoms that also appear in other respiratory infections, such as sudden onset, fever, malaise, headache, and cough,[8] while COVID-19 patients have similar symptoms including fever, cough , fatigue, and even gastrointestinal infection symptoms.[9-11] However, treatment methods, infectiousness, and case-fatality rates in the two diseases are different. Therefore, we compared the clinical laboratory indicators of these two diseases to find specific indicators to help early diagnosis, early treatment, and early isolation.

2 | METHODS

2.1 | Data sources

This was a retrospective cross-sectional observational study. All of the COVID-19 pneumonia and influenza A pneumonia subjects were confirmed by laboratory tests and were admitted to Ningbo First Hospital. COVID-19 pneumonia patients were hospitalized between January 1 and March 31, 2020, while influenza A pneumonia patients were hospitalized at the same period: All of the patients met the criteria to diagnose pneumonia as an acute respiratory disorder characterized by the presence of cough and at least one of the new-onset focal chest signs, fever for more than 4 days or dyspnea/tachypnea.[12] Following fulfillment of these criteria, all of the patients with COVID-19–induced or influenza A–induced pneumonia were included in this study. The study has been reviewed and approved by the Medical Ethical Committee of Ningbo First Hospital (2020-R037). The requirement for written informed consent was waived for the urgent need to collect clinical data according to the policy for public-health-outbreak investigation of emerging infectious diseases issued by the National Health Commission of the People's Republic of China, and no potential harm could be done to patients in light of retrospective study.

2.2 | Inclusion criteria

Only hospitalized patients with laboratory-confirmed pneumonia cases were included the analysis. A laboratory-confirmed case with COVID-19 or influenza A is defined as a positive result of the respiratory tract specimens by real-time RT-PCR assay.

2.3 | Exclusion criteria

Outpatients, nonlaboratory-confirmed cases, not manifesting as pneumonia patients, or ones with pneumonia caused by mycoplasma, chlamydia, bacteria, and other pathogens were excluded the analysis.

2.4 | Data collection

The recent exposure histories, clinical symptoms or signs, and laboratory findings on admission were extracted from electronic medical records. Radiologic assessments included chest radiography or computed tomography (CT). We determined the presence of a radiologic abnormality on the basis of the documentation or description in medical reports. Laboratory assessments consisted of blood routine, infection-related biomarkers, and blood chemical analysis. All data were checked by at least two experienced specialists.

2.5 | Statistical analysis

Data analysis was performed using SPSS 17.0 (IBM Corp., Armonk, NY) software. Categorical variables were summarized using frequencies and percentages, and continuous data are presented as the medians (interquartile ranges [IQRs]). Chi-square test was used for categorical variables, and nonparametric rank sum test of independent samples was used for continuous variables. Variables with a p

value <0.05 in chi-square test and rank sum test were entered into multivariate logistic regression analysis to identify independent risk factors associated with COVID-19 or influenza A. All of the *p* values less than 0.05 were considered to be statistically significant.

3 | RESULTS

3.1 | Comparison of the clinical characteristics between COVID-19 pneumonia and influenza A pneumonia

The median age of influenza A pneumonia cohort was 62 years old, which was significantly higher than that of COVID-19 pneumonia cohort (54 years old, $p = 0.037$). There was no significant difference in gender ratio and underlying diseases (including hypertension, type 2 diabetes, cardiovascular and cerebrovascular diseases, tumor, hepatitis, and autoimmune diseases) between the two cohorts. The median value of highest temperature of influenza A pneumonia cohort was 38.8°C, significantly higher than that of COVID-19 pneumonia cohort (37.9°C, $p < 0.001$), but there was no significant difference in admission temperature between the two cohorts. The proportion of pleural effusion in influenza A pneumonia cohort was significantly higher than that of COVID-19 pneumonia cohort (18.75% vs. 2.94%, $p = 0.031$) (Table 1). Both of COVID-19 and influenza A pneumonia cohorts presented high proportions of cough, but there was no significant difference between two groups. However, the proportions of fatigue (67.65%), diarrhea (32.35%) in pneumonia patients with COVID-19 were higher than those of influenza A cohort (9.09%, $p < 0.001$; 3.64%, $p < 0.001$, respectively) (Table 1).

3.2 | Comparison of radiologic and laboratory findings between COVID-19 pneumonia and influenza A pneumonia

In terms of radiologic characteristics, ground-glass opacity was a characteristic representation of COVID-19 pneumonia (73.53%) (Table 2) (Figure 1A,B), while few influenza A pneumonia patients presented ground-glass opacity (6.25%, $p < 0.001$ Figure 1D). In addition, two cohorts were prone to harbor bilateral patchy shadowing, but there was no significant difference (82.35% vs. 77.08%, $p > 0.05$).

As far as complete blood count (CBC) was concerned, lymphocyte percentage, absolute lymphocyte count, red-cell count, and hemoglobin levels in COVID-19 pneumonia cohort were all significantly higher than those in influenza A pneumonia cohort (24.8% vs 14.0%, $p = 0.011$; 1.21 vs 0.80 × 10^9/L, $p = 0.035$; 4.44 vs 4.24 × 10^{12}/L, $p = 0.012$; 133.0 vs 125.0 g/L, $p = 0.003$, respectively). Moreover, monocyte count in COVID-19 pneumonia cohort was significantly lower than that in influenza A pneumonia cohort (0.38? vs 0.59?, $p = 0.005$). In terms of infection-related biomarkers, c-reactive protein level in COVID-19 pneumonia cohort was significantly lower than that in influenza A pneumonia cohort (11.13 mg/L vs 30.58 mg/L, $p = 0.005$). In blood chemistry tests, albumin level in COVID-19 pneumonia cohort was significantly higher than that in influenza A pneumonia cohort (41.3 vs 35.3 g/L, $p < 0.001$), while aspartate aminotransferase (AST), alkaline phosphatase (ALP), and serum creatinine levels in COVID-19 pneumonia cohort were all significantly lower than those in influenza A pneumonia cohort (21.5 vs 28.0 U/L, $p = 0.005$; 67.5 vs 87.0 U/L, $p = 0.001$; 58.7 vs 68.0 μmol/L, $p = 0.017$, respectively) (Table 2).

TABLE 1 Comparison results of clinical characteristics of the study pneumonia patients with influenza A or COVID-19

Characteristics	Total (*n* = 89)	Influenza A (*n* = 55)	COVID-19 (*n* = 34)	*p*
Age (years)	58.0 (42.5, 71.0)	62.0 (46.0, 76.0)	54.0 (34.0, 63.3)	0.037
Female	50 (56.18)	27 (49.09)	23 (67.65)	0.086
Underlying diseases	40 (44.94)	28 (50.91)	12 (35.29)	0.150
Hypertension	26 (29.21)	18 (32.73)	8 (23.53)	0.354
Highest temperature (IQR) (°C)	38.5 (37.7, 39.0)	38.8 (38.3, 39.1)	37.9 (37.4, 38.4)	<0.001
Admission temperature (IQR) (°C)	37.1 (36.7, 37.5)	37.2 (36.8, 37.5)	37.0 (36.7, 37.3)	0.226
Pleural effusion	10 (12.20)	9 (18.75)	1 (2.94)	0.031
Symptoms				
Fever on admission	72 (80.90)	48 (87.27)	24 (70.59)	0.052
Fatigue	28 (31.46)	5 (9.09)	23 (67.65)	<0.001
Cough	79 (88.76)	50 (90.91)	29 (85.29)	0.693
Diarrhea	13 (14.61)	2 (3.64)	11 (32.35)	0.001
Myalgia	6 (6.74)	2 (3.64)	4 (11.76)	0.293

Note: Underlying diseases include hypertension, diabetes, cardiovascular and cerebrovascular diseases, tumor, hepatitis, autoimmune diseases. IQR denotes interquartile range.

TABLE 2 Radiographic and laboratory findings at admission in pneumonia patients with influenza A or COVID-19

Variable	Total (*n* = 89)	Influenza A (*n* = 55)	COVID-19 (*n* = 34)	*p*
Radiologic findings				
Unilateral patchy shadowing (%)	17 (20.73)	11 (22.92)	6 (17.65)	0.562
Bilateral patchy shadowing (%)	65 (79.27)	37 (77.08)	28 (82.35)	
Ground-glass opacity (%)	28 (34.15)	3 (6.25)	25 (73.53)	<0.001
Laboratory findings				
Blood routine				
White-cell count (×10^9/L; 3.50–9.50)*	5.60 (4.19, 8.51)	6.74 (4.13, 10.01)	5.20 (4.33, 6.83)	0.133
Neutrophils (%; 40.0–75.0)	70.7 (59.5, 81.9)	74.9 (59.3, 85.2)	66.7 (59.4, 76.3)	0.136
Lymphocyte (%; 20.0–50.0)	18.5 (10.0, 30.3)	14.0 (8.0, 26.9)	24.8 (15.8, 30.9)	0.011
Monocytes (%; 3.0–10.0)	7.7 (5.6, 10.9)	8.0 (5.8, 12.4)	7.1 (5.1, 9.4)	0.201
Neutrophils count (×10^9/L; 1.80–6.30)	3.80 (2.52, 6.21)	4.50 (2.50, 8.20)	3.52 (2.52, 4.64)	0.137
Lymphocyte count (×10^9/L; 1.10–3.20)	1.10 (0.60, 1.37)	0.80 (0.60, 1.30)	1.21 (0.87, 1.45)	0.035
Monocyte count (×10^9/L; 0.10–0.60)	0.43 (0.30, 0.70)	0.59 (0.31, 0.88)	0.38 (0.29, 0.49)	0.005
Red-cell count (×10^{12}/L; 3.80–5.10)	4.33 (4.05, 4.59)	4.24 (3.96, 4.52)	4.44 (4.22, 4.88)	0.012
Hemoglobin (g/L; 130.0–175.0)	129.0 (119.5, 136.5)	125.0 (116.0, 135.0)	133.0 (127.0, 141.0)	0.003
Platelet count (×10^9/L; 125–350)	196.0 (146.0, 247.0)	196.0 (128.0, 250.0)	202.0 (147.5, 244.5)	0.688
Platelet crit (%; 0.19–0.36)	0.20 (0.15, 0.25)	0.19 (0.15, 0.25)	0.21 (0.16, 0.24)	0.801
Infection-related biomarkers				
C-reactive protein (mg/L; 0.00–5.00)	18.94 (6.61, 64.46)	30.58 (9.38, 100.49)	11.13 (2.66, 38.52)	0.005
Blood chemistry				
Alanine aminotransferase (U/L; 9.0–50.0)	19.0 (12.3, 25.8)	17.5 (10.8, 30.3)	20.0 (14.0, 24.3)	0.480
Aspartate aminotransferase (U/L; 15.0–40.0)	24.0 (18.0, 34.8)	28.0 (19.0, 43.8)	21.5 (16.0, 26.3)	0.005
Gamma glutamyltransferase (U/L; 10.0–60.0)	22.0 (15.3, 38.0)	21.0 (14.8, 46.5)	22.5 (15.8, 33.0)	0.870
Alkaline phosphatase (U/L; 45.0–125.0)	76.5 (58.3, 105.3)	87.0 (61.8, 114.5)	67.5 (49.8, 86.5)	0.001
Albumin (g/L; 40.0–55.0)	38.0 (33.9, 41.4)	35.3 (31.0, 38.3)	41.3 (39.0, 45.4)	<0.001
Total bilirubin (μmol/L; 3.40–20.5)	8.22 (5.95, 13.11)	7.95 (4.75, 11.01)	10.45 (6.78, 13.98)	0.116
Direct bilirubin (μmol/L; 0.00–6.80)	3.30 (1.93, 5.38)	3.28 (1.78, 6.16)	3.30 (2.28, 4.15)	0.873
Lactate dehydrogenase (U/L; 120.0–250.0)	224.0 (188.3, 287.5)	224.0 (175.5, 306.0)	224.0 (192.8, 276.5)	0.963
Urea (mmol/L; 3.10–8.00)	4.47 (3.23, 6.72)	4.65 (2.84, 8.68)	4.40 (3.75, 5.24)	0.630
Serum creatinine (μmol/L; 57.0–97.0)	62.0 (54.9, 80.0)	68.0 (56.0, 98.5)	58.7 (54.4, 68.0)	0.017

*The bracketed content represents the unit and normal ranges of the index.

3.3 | Multivariate analysis

Variables with a *p* value <0.05 in chi-square test and rank sum test were entered into multivariate logistic regression analysis. Compared with parameters in influenza A pneumonia cohort, COVID-19 pneumonia cohort had a greater disposition to exhibit symptoms of fatigue (OR 13,886.443, 95% CI [29.734–648,538,7.882], $p = 0.002$), present ground-glass opacities in chest CTs (OR 3308.148, 95% CI [9.040–1,210,575.505], $p = 0.007$), and harbor higher level of albumin (OR 1.204, 95% CI [1.048–1.383], $p = 0.009$). Furthermore, compared with additional parameters in COVID-19 pneumonia cohort, influenza A pneumonia cohort was more older (OR 0.850, 95% CI [0.750–0.965], $p = 0.012$), had higher temperature (OR 0.008, 95% CI [0.001–0.238], $p = 0.005$), and higher monocyte count (OR 0.009, 95% CI [<0.001–0.264], $p = 0.006$) (Table 3).

4 | DISCUSSIONS

The outbreak of COVID-19 initiated in December 2019, which was consisted with winter respiratory virus season, including influenza. Huang et al[13] indicated that it was difficult to differentiate COVID-19 from influenza via only clinical manifestations prior to viral identification. However, viral pneumonia is rarely investigated separately. Therefore, we make great efforts to analyze the clinical characteristics of patients with COVID-19 pneumonia or influenza A pneumonia. To avoid the interference of seasonal changes, corresponding hospitalized patients with influenza A were selected from the same period. In addition, two cohorts were both admitted in the same hospital to ensure the consistency of medical records to minimize bias. Tang et al[14] explored the different clinical presentations between COVID-19 and influenza

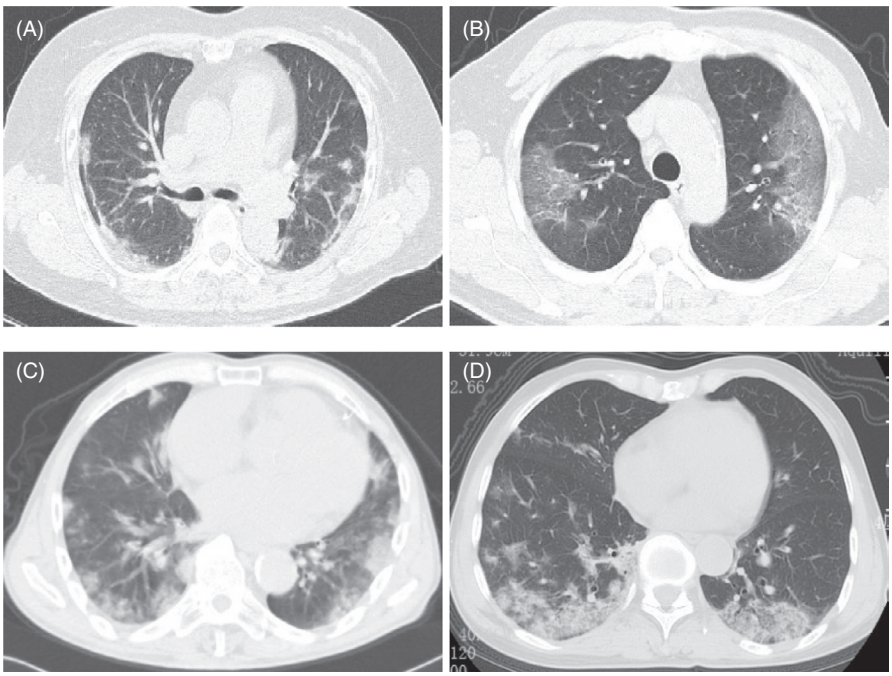

FIGURE 1 Imaging characteristics of chest computed tomographies from COVID-19 pneumonia patients and influenza A pneumonia patients. (A) 63-year-old female patient with COVID-19 pneumonia exhibited multiple ground-glass opacities and stripe shadows in both lungs. (B) A 67-year-old male patient with COVID-19 pneumonia exhibited diffuse ground-glass opacities and paving stone shadows in both lungs. (C) A 67-year-old male patient with influenza A pneumonia exhibited exudation and consolidation distributed with bronchus in multiple lobes and segments. (D) A 59-year-old female patient with influenza A pneumonia exhibited multiple patchy ground-glass opacities with grid shadow of map-like change in both lungs

TABLE 3 Multivariate logistic regression analysis of independent risk factors for differentiating COVID-19 pneumonia from influenza A pneumonia

						95% CI	
Index	***B***	***SE***	**Wald**	***p***	**OR**	**Lower limit**	**Upper limit**
Age	−0.162	0.064	6.359	0.012	0.850	0.750	0.965
Highest temperature	−4.773	1.703	7.858	0.005	0.008	<0.001	0.238
Pleural effusion	3.870	2.360	2.688	0.101	47.920	0.469	4891.541
Fatigue	9.539	3.136	9.252	0.002	13,886.443	29.734	6,485,387.882
Diarrhea	0.708	5.538	0.016	0.898	2.030	<0.001	105,015.819
Ground-glass opacity	8.104	3.012	7.242	0.007	3308.148	9.040	1,210,575.505
Lymphocyte %	−0.062	0.040	2.314	0.128	0.940	0.869	1.018
Lymphocyte count	0.303	0.855	0.126	0.723	1.354	0.253	7.233
Monocyte count	−4.666	1.700	7.531	0.006	0.009	<0.001	0.264
Red-cell count	−0.610	1.333	0.210	0.647	0.543	0.040	7.403
Hemoglobin	0.079	0.050	2.543	0.111	1.083	0.982	1.194
C-reactive protein	−0.004	0.009	0.176	0.674	0.996	0.978	1.015
Serum creatinine	−0.035	0.020	3.221	0.073	0.965	0.929	1.003
Aspartate aminotransferase	−0.048	0.030	2.570	0.109	0.953	0.899	1.011
Albumin	0.186	0.071	6.895	0.009	1.204	1.048	1.383
Alkaline phosphatase	−0.008	0.007	1.164	0.281	0.992	0.979	1.006

A (H1N1) pneumonia in patients with acute respiratory distress syndrome (ARDS). In this study, the clinical characteristics and laboratory findings of pneumonia patients caused by COVID-19 or influenza A were analyzed and compared; different subtypes of influenza A were not subdivided.

Guan et al[10] reported that fever (43.8% on admission, 88.7 during hospitalization) and cough (67.8%) were the dominant symptoms in COVID-19 patients also in Shi's report.[15] Wang et al[16] reported that fever, fatigue, and dry cough were the most common symptoms of COVID-19 patients, which is consistent with our study.

Furthermore, the highest temperature and fatigue were independent risk factors to differentiate two cohorts. COVID-19 pneumonia cohort had a greater disposition to present symptom of fatigue, which is consistent with Tang's research,[14] while influenza A pneumonia patients were more inclined to have a higher temperature. Although diarrhea (32.35%) was uncommon in COVID-19

pneumonia cohort, there was a significant difference between pneumonia patients infected with COVID-19 versus influenza A.

Undoubtedly, the most common patterns on chest CT of COVID-19 pneumonia patients were ground-glass opacity and bilateral patchy shadowing and this proportion in this study was higher than that in a research by Guan WJ.[10] The radiological findings of 81 patients with COVID-19 pneumonia showed that diffused bilateral ground-glass opacities were the most predominant pattern of abnormalities in chest CTs within 1–3 weeks after disease onset.[17] Compared with features in influenza A pneumonia cohort, ground-glass opacities in chest CTs were characteristics of COVID-19 pneumonia cohort and were independent risk factors to differentiate COVID-19 pneumonia from influenza A pneumonia. In contrast, only 3 patients with influenza A pneumonia presented ground-glass opacity (Figure 1D). Therefore, radiologic assessments have been applied to the diagnosis of COVID-19 pneumonia in Chinese guidelines.[18]

Guan WJ et al[10] reported that lymphocytopenia was present in 83.2% of the patients, thrombocytopenia in 36.2%, and leukopenia in 33.7% on admission. Lymphocytopenia was also common in other reports.[13,14,19] But lymphocytopenia was not common in our study, which is due to different subjects: COVID-19 pneumonia patients in our study, while COVID-19 patients including severe ones in other studies. For the prevention and control policy as well as enough capacity of medical treatment, many COVID-19 pneumonia patients in our study were admitted before confirmation of SARS-CoV-2 for epidemiological history, suspected clinical signs and symptoms or suspected chest CT images.[20] Therefore, we speculate that blood indexes of COVID-19 pneumonia patients at the early stage may not immediately respond to SARS-CoV-2. Interestingly, we also found in our other study that severe COVID-19 pneumonia patients presented lymphocytopenia and thrombocytopenia while mild ones did not (unpublished data). It perhaps explains the differences of blood indexes between this study and other ones. Furthermore, monocyte count is an independent risk factor for differentiating COVID-19 pneumonia from influenza A pneumonia: compared with the influenza A pneumonia infection, COVID-19 pneumonia cohort was more inclined to have lower level of monocytes.

Blood biochemistry of patients with COVID-19 pneumonia was short of characteristics and the consistency according to previous reports.[10,14] In the study by Guan et al,[10] the levels of alanine aminotransferase, aspartate aminotransferase, and creatine kinase were elevated in some COVID-19 cases. Tang[14] reported that the levels of aspartate transaminase (AST) and lactate dehydrogenase (LDH) in COVID-19 patients were all significantly lower than those in influenza A patients. In our study, albumin level in COVID-19 pneumonia cohort was significantly higher than that in influenza A pneumonia cohort, while the levels of AST, ALP, and creatinine in COVID-19 pneumonia cohort were all significantly lower than those in influenza A pneumonia cohort. Moreover, albumin is an independent risk factor for differentiating COVID-19 pneumonia from influenza A pneumonia. In addition, Chen et al[21] reported that neutrophil count, hypersensitivity C-reactive protein, creatine kinase, and blood urea nitrogen are the risk factors of COVID-19 severe patients with poor outcomes on admission.

There were some limitations of our present study. On the one hand, in our research, only patients with influenza A pneumonia admitted to hospital were included. Therefore, these findings might not be representative of mild to moderate influenza A. Among COVID-19 patients in our cohort, most cases were mild and only a few cases were severe. On the other hand, the prognosis of two cohorts were not compared for the treatment of COVID-19 pneumonia have being changed depending on more and more clinical evidences. Lack of sufficient evidence in the early stage, oxygen therapy, lopinavir /ritonavir (100 mg bid po), and arbidol (200 mg tid po) were administrated on COVID-19 pneumonia patients, depending on Chinese guidelines.[19] At present, with accumulation of clinical evidence, and the recent UK RECOVERY trial[22] has shown that dexamethasone might reduce death by a third in patients who are on mechanical ventilation as a result of severe respiratory complications from COVID-19, while oxygen therapy and oseltamivir are standard treatment to treat influenza A pneumonia.[23]

In summary, clinical characteristics and laboratory findings contribute to differential diagnose COVID-19 pneumonia and influenza A pneumonia.

CONFLICT OF INTEREST

All authors: No reported conflicts.

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DATA AVAILABILITY STATEMENT

The data used to support the findings of this study are available from the corresponding author upon request.

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