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Retrospective analysis of clinical characteristics of 405 patients with COVID-19

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Abstract

Objective: This study was performed to investigate the clinical characteristics of patients with coronavirus disease 2019 (COVID-19), which is caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2).

Methods: We analyzed the electronic medical records of 405 hospitalized patients with laboratory-confirmed COVID-19 in the Third Hospital of Wuhan.

Results: The patients' median age was 56 years, 54.1% were female, 11.4% had a history of smoking, and 10.6% had a history of drinking. All cases of COVID-19 were community-acquired. Fever (76.8%) and cough (53.3%) were the most common clinical manifestations, and circulatory system diseases were the most common comorbidities. Gastrointestinal symptoms were present in 61.2% of the patients, and 2.9% of the patients were asymptomatic. Computed tomography showed ground-glass opacities in most patients (72.6%) and consolidation in 30.9%. Lymphopenia (72.3%) and hypoproteinemia (71.6%) were observed in most patients. About 20% of patients had abnormal liver function. Patients with severe disease had significantly more prominent laboratory abnormalities, including an abnormal lymphocyte count and abnormal C-reactive protein, procalcitonin, alanine aminotransferase, aspartate aminotransferase, D-dimer, and albumin levels.

Conclusion: SARS-CoV-2 causes a variety of severe respiratory illnesses similar to those caused by SARS-CoV-1. Older age, chronic comorbidities, and laboratory abnormalities are associated with disease severity.

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Introduction

In December 2019, a series of unexplained cases of pneumonia were reported in Wuhan, China. On 7 January 2020, the Chinese Center for Disease Control and Prevention identified a novel lineage B β-coronavirus from a throat swab sample. This virus was named severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) by the World Health Organization.[1] Patients infected with SARS-CoV-2, which causes coronavirus disease 2019 (COVID-19), develop fever, dry cough, dyspnea, and fatigue. In severe cases, COVID-19 may lead to severe acute respiratory syndrome and even death.[2,3] The disease began to spread worldwide in February 2020.

A large body of evidence has proven that the main route of SARS-CoV-2 transmission is through respiratory droplets and close contact. Zhang et al.[4] reported that viral nucleic acids can also be found in fecal samples and anal swabs of patients with COVID-19. Therefore, the possibility of fecal–oral transmission must be considered.[5–7] A retrospective study in Wuhan showed that the clinical manifestations of COVID-19 include fever, cough, fatigue, headache, and some gastrointestinal symptoms.[4,8] These gastrointestinal symptoms mainly include diarrhea, nausea, and vomiting, and a significant proportion of patients initially present with such symptoms.[1]

In the months following the disease outbreak, China gradually curbed the viral spread through active isolation measures. Unfortunately, the virus was transmitted to dozens of other countries, including the United States, Italy, Spain, and Germany. Although the clinical features of patients with COVID-19 have been described in many articles, few reports have described the characteristics of the gastrointestinal symptoms in patients with COVID-19. Therefore, we performed the present study to improve our understanding of COVID-19 by investigating (i) the clinical and laboratory characteristics of patients with COVID-19 hospitalized from January to March in the Third Hospital of Wuhan, including differences between patients with severe and non-severe disease, and (ii) the relationship between COVID-19 and gastrointestinal symptoms.

Methods

Patient inclusion and data collection

This study included all hospitalized patients who were admitted to the Third Hospital of Wuhan (one of the designated facilities for hospitalization of patients with COVID-19) from 12 January to 8 March 2020 and diagnosed with COVID-19 according to a positive result of high-throughput sequencing or real-time reverse-transcriptase polymerase chain reaction assay using nasal or pharyngeal swab specimens. Complete data were available for all patients involved in this study, and all patients lived in Wuhan during the disease outbreak. We communicated directly with the patients or their families to collect epidemiological and

symptom data not present in the electronic medical records. This study was approved by the Ethics and Science Committee of Wuhan University Tongren Hospital (KY2020-021). In view of the urgent need to collect clinical data, the requirement for written informed consent was waived.

The patients' epidemiological characteristics (including recent exposure history), clinical symptoms and signs, and laboratory findings were extracted from the electronic medical records and return visits, bedside consultations, and telephone interviews. Laboratory assessments included blood cell counts and concentrations of C-reactive protein (CRP), procalcitonin (PCT), lactate dehydrogenase (LDH), creatine kinase (CK), and D-dimers. All patients' medical laboratory data were obtained from the laboratory of the Wuhan Third Hospital. The severity of COVID-19 was defined according to the diagnostic and treatment guideline for COVID-19 issued by the Chinese National Health Committee (version 5-7). Severe COVID-19 was diagnosed when the patient fulfilled one of the following criteria: (i) respiratory distress with a respiratory rate of $\geq$30 breaths/minute, (ii) oxygen saturation of $\leq$93% at rest as measured by pulse oximetry, or (iii) an oxygenation index (arterial partial pressure of oxygen/inspired oxygen fraction) of $\leq$300 mmHg.

Statistical analysis

Categorical variables are presented as frequencies and percentages, and continuous variables are presented as median values. The frequencies of categorical variables were compared using the chi-square test or Fisher's exact test, as appropriate. All statistical analyses were carried out using GraphPad Prism version 7.00 software (GraphPad, San Diego, CA, USA). A P value of <0.05 was considered statistically significant.

Results

Demographics and clinical characteristics

The patients' demographic and clinical characteristics are presented in Table 1. Of the 405 patients with COVID-19 included in the present study, 257 and 148 patients were categorized into the non-severe and severe subgroups, respectively. The patients' median age was 56 years (range, 17–95 years). Most of the patients with severe disease (71.0%) and less than half of those with non-severe disease (44.7%) were >60 years of age. Of all 405 patients, 54.1% were female, 11.4% had a history of smoking, and 10.6% had a history of drinking. Because no patients had been directly exposed to Huanan wet markets or wildlife, we presumed that all cases were community-acquired. Among the 405 patients, 20 were hospital workers, and the family members or friends of 62 patients were also infected with SARS-CoV-2. There was no significant difference in the exposure history between the severe and non-severe groups. Of the 405 patients, 247 (61%) had at least one chronic disease, including circulatory system disease, gastrointestinal disease, endocrine disease, respiratory disease, urinary system disease, motor system disease, or nervous system disease. The most common of these was circulatory system disease (38.5%). Of the patients with non-severe disease, 130 (50.6%) had at least one chronic disease, and of the patients with severe disease, 117 (79.1%) had at least one chronic disease. This difference was statistically significant ($P < 0.01$). Fever (76.8%) and cough (53.3%) were the most common symptoms, and 61.2% of the patients had gastrointestinal symptoms. In addition, 2.9% of the patients were asymptomatic.

Because more than half of the patients had gastrointestinal symptoms, we also analyzed their demographic and clinical

Table 1. Clinical characteristics of 405 patients with COVID-19.

		Disease severity		
	All patients (n = 405)	Non-severe disease (n = 257)	Severe disease (n = 148)	*P* value
Age, years	56 (17–95)	52 (17–87)	64 (33–95)	<0.001
Age groups, years				
<30	12 (3.0)	12 (4.7)	0 (0.0)	<0.001
30–44	63 (15.6)	53 (20.6)	10 (6.7)	
45–59	109 (26.9)	76 (29.6)	33 (22.3)	
60–74	182 (44.9)	106 (41.2)	76 (51.4)	
≥75	39 (9.6)	10 (3.8)	29 (19.6)	
Sex				
Male	186 (45.9)	113 (43.9)	73 (49.3)	0.298
Female	219 (54.1)	144 (56.1)	75 (50.7)	
Exposure history				
Hospital staff	20 (4.9)	16 (6.2)	4 (2.7)	0.115
Familial/cluster infections	62 (15.8)	44 (17.1)	18 (12.1)	0.182
Smoking history				
Yes	46 (11.4)	21 (8.2)	25 (16.9)	0.008
No	359 (88.6)	236 (91.8)	123 (83.1)	
Drinking history				
Yes	43 (10.6)	25 (9.7)	18 (12.2)	0.444
No	362 (89.4)	232 (90.3)	130 (87.8)	
Chronic medical disease	247 (60.1)	130 (50.6)	117 (79.1)	<0.001
Circulatory system disease	156 (38.5)	79 (30.7)	77 (52.0)	<0.001
Gastrointestinal disease	102 (25.2)	62 (24.1)	40 (27.0)	0.517
Blood endocrine disease	88 (21.7)	42 (16.3)	46 (31.1)	0.001
Respiratory disease	31 (7.7)	7 (2.7)	24 (16.2)	<0.001
Urinary system disease	28 (6.9)	12 (4.7)	16 (10.8)	0.019
Motor system disease	18 (4.4)	5 (1.9)	13 (8.8)	0.001
Nervous system disease	5 (1.2)	1 (0.4)	4 (2.7)	0.061
Other disease	9 (2.2)	8 (3.1)	1 (0.7)	0.21
Signs and symptoms				
Fever	311 (76.8)	201 (78.2)	110 (74.3)	0.372
Cough	216 (53.3)	135 (52.5)	81 (54.7)	0.669
Chest tightness	124 (30.6)	68 (26.5)	56 (37.8)	0.017
Fatigue	155 (38.3)	89 (34.6)	66 (44.6)	0.047
Insomnia	103 (25.4)	54 (21.0)	49 (33.1)	0.007
Sore throat	29 (7.2)	22 (8.6)	7 (4.7)	0.15
Chest pain	23 (5.7)	17 (6.6)	6 (4.1)	0.284
Gasping/dyspnea	9 (2.2)	4 (1.6)	5 (3.4)	0.397
Headache/dizziness	5 (1.2)	3 (1.2)	2 (1.4)	0.602
Gastrointestinal symptoms	248 (61.2)	153 (59.5)	95 (64.2)	0.354
Asymptomatic	12 (3.0)	12 (4.7)	0 (0.0)	0.018

Data are presented as median (range) or n (%).
COVID-19, coronavirus disease 2019.

Table 2. Clinical characteristics of 248 patients with COVID-19 and gastrointestinal symptoms.

		Disease severity		
	All patients (n = 248)	Non-severe disease (n = 153)	Severe disease (n = 95)	*P* value
Age, years	60 (17–95)	55 (17–85)	66 (33–95)	<0.001
Age groups, years				
<30	11 (4.4)	11 (7.2)	0 (0.0)	<0.001
30–44	44 (17.7)	37 (24.2)	7 (7.4)	
45–59	63 (25.4)	44 (28.8)	19 (20.0)	
60–74	107 (43.1)	57 (37.2)	50 (52.6)	
≥75	23 (9.4)	4 (2.6)	19 (20.0)	
Sex				
Male	111 (44.8)	62 (40.5)	49 (51.6)	<0.001
Female	137 (55.2)	91 (59.5)	46 (48.4)	
Smoking history				
Yes	30 (12.1)	11 (7.2)	19 (20.0)	0.004
No	218 (87.9)	142 (92.8)	76 (80.0)	
Drinking history				
Yes	28 (11.3)	13 (8.5)	15 (15.8)	0.043
No	220 (88.7)	140 (91.5)	80 (84.2)	
GI disease	85 (34.3)	52 (34.0)	33 (34.7)	
Chronic gastritis	29 (11.7)	16 (10.5)	13 (13.7)	0.537
Peptic ulcer	20 (8.1)	10 (6.5)	10 (10.5)	0.225
Cholelithiasis/cholecystitis	19 (7.7)	12 (7.8)	7 (7.4)	0.799
Chronic liver disease	16 (6.5)	11 (7.2)	5 (5.3)	0.291
Gastroesophageal reflux disease	4 (1.6)	4 (2.6)	0 (0.0)	0.131
GI tumor	3 (1.2)	1 (0.7)	2 (2.1)	0.345
Hepatic cyst	3 (1.2)	1 (0.7)	2 (2.1)	0.636
Enteritis	2 (0.8)	2 (1.3)	0 (0.0)	0.364
Gastrointestinal polyps	1 (0.4)	1 (0.7)	0 (0.0)	0.604
GI symptoms as the first symptoms	185 (75.6)	107 (69.9)	78 (82.1)	0.032
Signs and symptoms				
Poor appetite	170 (68.5)	94 (61.4)	76 (80.0)	<0.001
Diarrhea	112 (45.2)	74 (48.4)	38 (40.0)	0.67
Nausea/vomiting	76 (30.6)	43 (28.1)	33 (34.7)	0.407
Upper abdominal discomfort	41 (16.5)	26 (17.0)	15 (15.8)	0.906
Sour regurgitation/belching	37 (14.9)	28 (18.3)	9 (9.5)	0.602
Constipation	15 (6.0)	11 (7.2)	4 (4.2)	0.418
Poststernal discomfort/heartburn	26 (10.5)	20 (13.1)	6 (6.3)	0.072
Improved symptoms after 1 week of treatment	201 (81.0)	134 (87.6)	67 (70.5)	0.001

Data are presented as median (range) or n (%).
COVID-19, coronavirus disease 2019; GI, gastrointestinal.

characteristics (Table 2). Of all 405 patients, 248 had gastrointestinal symptoms (153 and 95 in the non-severe and severe subgroups, respectively). Among these 248 patients, 44.8% were male, 12.1% had a history of smoking, 11.3% had a history of drinking, and 34.3% had a history of gastrointestinal disease. Chronic gastritis (11.7%) was the

most common gastrointestinal disease, followed by peptic ulcers (7.7%) and cholelithiasis/cholecystitis (7.7%). The statistical analysis showed that the severe subgroup had a significantly higher proportion of patients with a history of smoking and drinking than did the non-severe subgroup ($P < 0.05$). Of the 248 patients with gastrointestinal symptoms, gastrointestinal symptoms were the initial symptom in 185 patients. Among them, 107 had non-severe disease and 78 had severe disease. The statistical analysis showed that compared with the non-severe subgroup, the severe subgroup had a significantly higher proportion of patients with gastrointestinal symptoms as the first symptom ($P < 0.05$). The gastrointestinal symptoms mainly included a poor appetite, diarrhea, nausea, vomiting, epigastric discomfort, acid regurgitation, belching, poststernal discomfort, heartburn, and constipation. Among all gastrointestinal symptoms, a poor appetite (68.5%) and diarrhea (45.2%) were the most common. The statistical analysis showed that compared with the non-severe subgroup, patients in the severe subgroup were significantly more likely to have a poor appetite ($P < 0.01$). The gastrointestinal symptoms of most patients (80%) improved significantly after using proton pump inhibitors, probiotics, and/or liver protection drugs for 1 week. The vast majority of patients recovered from their gastrointestinal symptoms when they were discharged, including those with abnormal liver function. The statistical analysis showed that compared with the severe subgroup, the gastrointestinal symptoms of the patients in the non-severe subgroup were significantly more easily relieved ($P < 0.01$). However, during the follow-up process, we found that a few patients had elevated transaminase concentrations after discharge. Unfortunately, further analysis of this abnormality could not be performed because of the limited number of patients.

Radiological and laboratory findings

Table 3 shows the chest computed tomography (CT) scan and laboratory assay results on admission. Abnormal CT scan results were obtained for 378 patients (93.3%), among whom 349 patients (86.2%) had changes in the bilateral lungs. Most patients (n = 294, 72.6%) showed typical ground-glass opacities, and 125 patients (30.9%) showed consolidation. In addition, 5.4% of the patients had pleural effusion. The laboratory findings showed that on admission, 72.3% and 71.6% of patients had lymphopenia and hypoproteinemia, respectively. Leukopenia was observed in 17.8% of the patients. Most patients had elevated CRP and D-dimer levels, but elevated levels of PCT, LDH, and CK were less common. About 20% of patients had abnormal liver function. Patients with severe disease had significantly more prominent laboratory abnormalities, such as an abnormal lymphocyte count and CRP, PCT, alanine aminotransferase, aspartate aminotransferase, D-dimer, and albumin levels ($P < 0.05$).

The patients' second CT scans and routine blood tests (about 7 days after hospitalization) (Table 4) showed that 375 patients (93.3%) had abnormal CT scan results, among whom 87.9% had changes in the bilateral lungs. Still, 72.6% of patients showed ground-glass opacities in the lungs, 30.9% showed pulmonary consolidation, and 5.2% had pleural effusion. The lymphocyte count was low in 48.3% of patients, and there was a significant difference between patients in the non-severe and severe subgroups ($P < 0.01$).

Discussion

In this study, we evaluated 405 patients with community-acquired COVID-19. Most patients were middle-aged or elderly; their median age was 56 years, which is

Table 3. CT and laboratory findings of 405 patients with COVID-19.

		Disease severity		
	All patients (n = 405)	Non-severe disease (n = 257)	Severe disease (n = 148)	*P* value
Chest CT images				
Abnormal	378 (93.3)	238 (92.6)	140 (94.6)	0.44
Single lung	29 (7.1)	19 (7.4)	10 (6.8)	0.71
Bilateral lungs	349 (86.2)	219 (85.2)	130 (87.8)	–
Ground-glass opacity	294 (72.6)	181 (70.4)	113 (76.4)	0.198
Lung consolidation	125 (30.9)	77 (30)	48 (32.4)	0.604
Pleural effusion	22 (5.4)	8 (3.1)	14 (9.5)	0.007
Laboratory findings				
Leukocytes				
Increased	31 (7.7)	14 (5.4)	17 (11.5)	0.089
Decreased	72 (17.8)	47 (18.3)	25 (16.9)	
Lymphocytes				
Decreased	293 (72.3)	166 (64.6)	127 (86.8)	<0.01
C-reactive protein				
Increased	205 (50.6)	111 (43.2)	94 (63.5)	<0.01
Procalcitonin				
Increased	107 (26.4)	42 (16.3)	65 (43.9)	<0.01
Alanine aminotransferase				
Increased	84 (21.7)	40 (15.6)	44 (29.7)	0.001
Aspartate aminotransferase				
Increased	82 (20.2)	50 (19.5)	32 (21.6)	0.601
Lactate dehydrogenase				
Increased	140 (34.6)	92 (35.8)	48 (32.4)	0.493
Creatine kinase				
Increased	37 (9.1)	30 (11.7)	7 (4.7)	0.02
D-dimers				
Increased	257 (63.5)	136 (52.9)	121 (81.8)	<0.01
Albumin				
Increased	290 (71.6)	169 (65.7)	121 (81.8)	0.001

Data are presented as n (%).
CT, computed tomography; COVID-19, coronavirus disease 2019.

consistent with that reported by Wang et al.[9] and close to those reported by Zhang et al.[4] (57 years) and Chen et al.[2] (55 years). Patients with severe disease were much older than those with non-severe disease. The proportion of patients with chronic comorbidities was 79.1% and 50.6% in the severe and non-severe subgroup, respectively. Circulatory diseases (38.5%), gastrointestinal diseases (25.2%), and endocrine diseases (21.7%) were the most common chronic comorbidities, which is consistent with other recent reports.[8] We can infer that elderly patients with chronic diseases are more likely to develop severe COVID-19. Smoking and drinking were not associated with disease severity.

Consistent with previous reports,[8,10,11] fever (76.8%) and cough (53.3%) were the

Table 4. Secondary CT and lymphocyte counts findings of 405 patients with COVID-19.

		Disease severity		
	All patients (n = 405)	Non-severe disease (n = 257)	Severe disease (n = 148)	*P* value
Chest CT images				
Abnormal	375 (92.6)	235 (91.4)	140 (94.6)	0.243
Single lung	19 (4.7)	11 (4.3)	8 (5.1)	0.137
Bilateral lungs	356 (87.9)	222 (86.4)	134 (84.8)	–
Ground-glass opacity	294 (72.6)	185 (72)	109 (69)	0.718
Lung consolidation	112 (27.6)	64 (24.9)	48 (30.4)	0.103
Pleural effusion	21 (5.2)	8 (3.1)	13 (8.2)	0.013
Laboratory findings				
Lymphocytes				
Decreased	196 (48.3)	87 (33.9)	109 (73.6)	<0.01

Data are presented as n (%).
CT, computed tomography; COVID-19, coronavirus disease 2019.

most common symptoms in patients with COVID-19, and the proportion of patients with fever and cough was significantly higher among patients with severe than non-severe disease. However, the proportion of patients with fever and cough was lower in the present study than previously reported. Notably, 2.7% of patients in our study were asymptomatic. These patients had a history of close contact with other patients who had COVID-19, and they were therefore examined. When they were found to be positive as well, they were hospitalized; however, they did not develop COVID-19 symptoms. In addition, we found that more than half of the patients had digestive symptoms, the most common of which were a poor appetite, diarrhea, nausea, and vomiting. In previous reports, however, digestive symptoms were uncommon.[2,4,10,11] In the present study, 248 patients (61.2%) had digestive symptoms (153 and 95 in the non-severe and severe subgroups, respectively). The gastrointestinal symptoms were mainly a poor appetite, diarrhea, nausea, vomiting, epigastric discomfort, acid regurgitation, belching, poststernal discomfort, heartburn, and constipation; 34.3% of the patients had a history of gastrointestinal diseases, with chronic gastritis (11.7%) being the most common, followed by peptic ulcers (8.1%). Based on these data, we can infer that SARS-CoV-2 infection may cause acute gastritis or enteritis.

Next-generation sequencing technology and molecular modeling have revealed that SARS-CoV-2 shares about 79% sequence identity with SARS-CoV-1 (another lineage B β-coronavirus), indicating that these two viruses are highly homologous.[12] Angiotensin-converting enzyme II (ACE2) is an entry receptor for SARS-CoV-1. Lu et al.[12] reported that the receptor-binding domains of SARS-CoV-1 and SARS-CoV-2 are structurally similar as indicated by molecular modeling. Therefore, SARS-CoV-2 might also use ACE2 as an entry receptor despite the presence of amino acid mutations in the SARS-CoV-2 ACE2 receptor-binding domain.[12] Related studies have shown that ACE2 is abundant in human lung and intestinal epithelium, in agreement with the notion that the pulmonary and gastrointestinal tracts are possible routes of SARS-CoV-2

infection.[8] In addition, Hashimoto et al.[13] showed that ACE2 is mainly expressed on the luminal surface of differentiated small intestinal epithelial cells and that mutations in ACE2 can reduce the expression of antimicrobial peptides and alter the gut microbial composition. Therefore, we speculate that acute gastroenteritis caused by SARS-CoV-2 infection may be related to intestinal flora imbalance. Previous studies have shown that most patients infected with SARS-CoV-2 have respiratory symptoms but that few have gastrointestinal symptoms. However, recent studies have revealed that the proportion of patients with gastrointestinal symptoms is increasing.[2,4,14] In addition, the proportion of patients with gastrointestinal symptoms in the present study exceeded 50%, while the proportions of patients with fever and cough were significantly lower than previously reported. We speculate that these differences are caused by decreased virulence with increased infectivity and altered organ susceptibility due to viral mutations. However, the mechanisms by which SARS-CoV-2 causes gastrointestinal symptoms remain unexplored.

Radiologically, the most common sign in CT scans is ground-glass opacities in both lungs,[4,10,11] and our results are consistent with this. In our study, 93.3% of the patients had abnormal CT scan results, and 86.2% had lesions involving both lungs, mainly with ground-glass opacities. However, 30.9% of patients had pulmonary consolidation, and 5.4% of patients had pleural effusion. Consistent with two recent reports,[3,8] lymphopenia and hypoproteinemia are the most common laboratory abnormalities, followed by elevated D-dimer levels. In some patients, the leukocyte count and CRP, PCT, D-dimer, and CK levels were increased, indicating that COVID-19 infection may lead to inflammation and coagulation disorders. In addition, 20% of patients had abnormal liver function, but whether this was caused by the virus or medication is unknown. The patients were reexamined about 7 days after hospitalization. The results showed that 92.6% of the patients had abnormal CT scan results, and of these patients, 87.9% had changes in the bilateral lungs; 72.6% had ground-glass opacities in the lungs, 30.9% showed pulmonary consolidation, and 5.2% had pleural effusion. The lymphocyte count was decreased in 48.3% of patients, suggesting that the duration of COVID-19 exceeds 1 week.

In summary, we have reported the clinical and laboratory characteristics of 405 patients with community-acquired COVID-19, many of whom had gastrointestinal symptoms. Because of the increasing numbers of patients with COVID-19 who develop gastrointestinal symptoms as the first symptoms or who develop gastrointestinal symptoms at some point during their clinical course, global authorities should pay more attention to these atypical patients. We hope that with joint efforts and strong support among global communities, the COVID-19 epidemic can be controlled in the near future.

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Author contributions

Ting Zhan, Yalin Tang, Xueting Cheng, and Junsheng Deng collected the data. Ting Zhan, Yalin Tang, Meng Liu, Xia Tian, and Xiaodong Huang analyzed the data and prepared the manuscript. Zheng Han contributed to the collection and interpretation of the radiological materials. Xiaoli Chen, Xia Tian, and Meng Liu were involved in the patient management and organization work. Ting Zhan and

Xiaodong Huang designed the study and reviewed the manuscript.

Declaration of conflicting interest

The authors declare that there is no conflict of interest.

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