A Case of Avian Influenza a (H7N9) with Lactose Intolerance

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Abstract

The avian influenza A (H7N9) has infected more than 500 people in China since the first human infection was confirmed in Mar 2013. Diarrhea was present in some H7N9 infected patients, but this is the first report on the clinical characteristics of an H7N9-infected patient with lactose intolerance. Geographical location, environment, lifestyle, and host factors including genetic variations could have played a role in the susceptibility to influenza A (H7N9) infection.

Keywords

Avian Influenza a (H7N9), Lactose Intolerance, Diarrhea, Zoonotic Infection, China

1. Introduction

THE zoonotic infection of avian influenza A (H7N9) was demonstrated to originate from the mixing between three earlier avian influenza viruses, H7N3, H7N9 and H9N2 [1, 2]. As of July 18, 2015, laboratory-confirmed human-infections of H7N9 have reached 500, with a fatality rate of over 30% since the first case was confirmed in China in May 2013 [3,4]. Among the clinical manifestations of avian influenza A (H7N9), fever (100%) and cough (90.1%) were the most frequent, followed by shortness of breath (55.9%), sputum production (55.9%), fatigue (36.0%), hemoptysis (24.3%), and diarrhea or vomiting (13.5%) [4]. Diarrhea was also present in some cases of H5N1 and 2009 H1N1 infections [5, 6]. The Yangtze River delta is one of the geographical origins of avian influenza A (H7N9) [7]. Gene-culture co-evolution led to high frequency of lactose intolerance in China [8, 9]. To our knowledge, avian influenza A (H7N9) infection with lactose intolerance has not been reported.

2. Case Presentation

The patient was a 54-year-old female, who worked as a grocer selling cigarettes in a farm produce market that involved live poultry transactions in the countryside of Jiaxing [Figure 1]. She had fever and cough on Apr 13, 2013 but did not receive any treatment, and no improvement was noted. She was admitted to the hospital after developing conjunctivitis and myalgia on Apr 17, 2013. The initial laboratory tests showed leucopenia, neutropenia and lymphocytopenia in peripheral blood, elevated erythrocyte sedimentation rate, increased levels of lactate dehydrogenase and hypersensitive C-reactive protein. Antibodies to respiratory tract viruses in the serum was negative, creatine kinase and procalcitonin concentrations were normal [Table 1]. Chest CT scan on the day of admission showed infection in the left lung [Figure 2]. Intravenous piperacillin/sulbactam sodium and oseltamivir was started. The real-time reverse transcriptase polymerase chain reaction (RT-PCR) for avian
influenza A (H7N9) in a throat swab specimen was positive on Apr 17, 2013 for which the patient received anti-infective treatment with oseltamivir. Invasive mechanical ventilation, thymalfasin and methylprednisolone were not provided. Her fever and myalgia subsided, although respiratory symptoms were not resolved until Apr 19, 2013. On Apr 20, 2013, after ingestion of 20ml milk for 20 minutes, the patient developed acute diarrhea without abdominal cramps, tenesmus and vomiting. There was no blood and mucus in her lungs, and her cardiac, lung and abdominal evaluations were within normal limits. Her symptoms were suddenly relieved within five minutes. Diagnosis after admission included lactose intolerance, so she was recommended to avoid milk and administered smectite powders. On Apr 21, 2013, the patient experienced the same reaction within 20 minutes of oral milk intake, which almost completely resolved in a moment. Since then, no evidence of diarrhea recurrence after milk ingestion was found. On Apr 26, 2013, CT showed that the focus of infection had begun to resolve [Figure 2]. The patient was discharged after a throat swab sample tested negative for avian influenza A (H7N9) by RT-PCR. She was asymptomatic and CT showed resolution of consolidation changes on May 14, 2013 [Figure 2].

Figure 1. The neighborhood of the confirmed H7N9 patient.

Figure 2. Chest CT of the confirmed H7N9 patient. (A) The CT showed infiltrates and partial consolidation in the left lung on Apr 17, 2013; (B) The CT showed decreased infiltrates and partial consolidation in the left lung on Apr 26, 2013; (C) The CT showed reticular changes and distortion with consolidation resolution in the previously-involved areas of parenchyma on May 14, 2013.
3. Discussion

With fresh cases reported in Shanghai, the number of humans infected with avian influenza A (H7N9) grew in three years after the first three human cases [1, 3]. The viral factors, geographical locations, temperatures and epidemiological control measures responsible for the increased incidence of human infections in southern China have been reported [10]. Herein, we reported an H7N9 infected patient with lactose intolerance. Rice, fish and poultry, especially ducks, are the main diet in the lower Yangtze River delta. Gene-culture co-evolution induced single-nucleotide polymorphisms leading to gradual loss of intestinal enzyme lactase in China, which led adults, particularly elderly, to experience gastrointestinal symptoms following ingestion of milk and dairy products [9]. This diagnosis was usually based on clinical history, but not lab tests [11]. The geographical distribution and prevalence of H7N9 flu epidemic in Shanghai, Zhejiang, Jiangsu and Guangdong provinces correlated with a high frequency of lactose intolerance. This might provide insights on the risk of environmental, host genetic, and immunological factors.

The patient works as a grocer about 40 meters away from a live poultry market stall. She was in contact with numerous customers, which may increase the chances to develop H7N9 [12]. Due to limited person-to-person H7N9 transmission, her family members, close contacts and others exposed to the poultry market without any personal protective equipment did not develop any influenza-like symptoms [13]. Seven family clusters were identified, and exposure of only two non-blood relatives resulted in infection. Possible genetic polymorphisms among blood relatives could contribute to susceptibility to influenza infection [3, 14]. A study identified subclinical H7N9 infection in 396 poultry workers who produced antibodies against avian influenza A (H7N9), suggesting that host factors including genetic variations may play a role in the disease severity [15]. The interferon-induced transmembrane protein 3 (IFITM3) rs12252-C allele is highly frequent in Han Chinese but rare in Northern Europeans [16], and may contribute to a higher risk of developing severe influenza infection among Chinese [17]. Two studies identified the host genetic variations that contribute to the outcome and disease severity of H7N9 infection [18, 19]. The co-evolutionary dynamics of antagonistic interactions between avian influenza A (H7N9) and the host could facilitate rapid selection or evolution of host antiviral restriction factors [20-22]. Gene–culture interactions may have generated selection that affected genetic variation to protect against zoonotic infection during the switch from a nomadic hunter-gatherer lifestyle to an agricultural lifestyle. Outside of this environment, this genetic variation might be harmful [23, 24]. Future studies should identify whether the host genetic variations that influence susceptibility, severity and clinical manifestation of influenza virus infection in humans are ancestral or derived.

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References


