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A case of pneumonia following human infection with avian influenza a (H5N1)

Case Report

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Abstract: The H5N1 infection was diagnosed in 12 patients in Turkey and confirmed by the WHO. Of these 12 patients so far, 8 have been published. In this case, we are presenting a case of pneumonia that developed following avian influenza infection in Eskisehir. Our case is one of the 4 patients who were not reported previously.

Keywords: *Avian Influenza • H5N1 • Pneumonia*

1. Introduction

The first documented case of human infection with the avian influenza A (H5N1) virus occurred in Hong Kong in 1997 [1]. The cumulative number of confirmed human cases of avian influenza AI (H5N1) reported to the WHO by 10 September 2008 is 387 [2]. The main clinical features are fever, cough, sore throat, rhinorrhoea, myalgia, conjunctivitis, diarrhea and severe unexplained respiratory illness. The clinical spectrum of influenza A (H5N1) in humans is based on descriptions of hospitalized patients. The frequencies of milder illnesses, subclinical infections have not been determined.

2. Case Report

A 27-year-old male patient was admitted to our hospital after a three-day illness. He had complaints of chills, dry cough, headache, myalgia, nausea, vomiting and stomach ache. The patient lived with his wife and his two sons. One week earlier one of his sons had brought home a pigeon found in the which was street sick and incapable of flying. The pigeon died the next day. All the family members had direct contact with the pigeon. One day after the pigeon died, his two sons and his wife had symptoms of fever, chills and myalgia. Six days after the contact with the pigeon, the patient was admitted to our hospital with the same complaints as his family.

His blood pressure was 100/70 mm Hg, heart rate 72 per minute, respirations 18 per minute, and temperature 37.1°C orally. The only evidence was the nasopharyngeal hyperaemia. Laboratory investigations revealed white blood cell count at 7.2 x 10^3/mm^3 (neutrophils 65.9%, lymphocytes 22.5%, monocytes 9.9%, eosinophils 1.1%, basophils 0.6%), hemoglobin at 12.7 g/dl and platelet count at 195 x 10^3/mm^3. Erythrocyte sedimentation rate was 11 mm during the first hour; C-reactive protein was 2.47 mg/dl. Serum creatinine, alanine aminotransferase, sodium and potassium, serum protein electrophoresis, serum uric acid, cholesterol and triglyceride levels were normal. In his urinalysis, specific gravity was 1.033 (normal, 1.005-1.030), and 15 red cells per millimeter were found. The rest of urinalysis findings and the chest radiography of the patient were normal.

The patient was hospitalized for suspicion of avian influenza as a result of cases with avian influenza in our country and avian influenza in poultry in our city, Eskisehir. Also, his wife and children were hospitalized due to fever, chills, cough, headache and myalgia. After taking a nasopharyngeal swab sample from the patient,

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oseltamivir 2x75 mg was administered. The patient's fever was decreased in two days.

Since the result of the sample was weakly positive, a new sample was sent on the second day of treatment, and it was negative. Oseltamivir treatment was stopped on the fifth day. Results of nasopharyngeal swab samples from his wife and his sons were negative. The patient, whose complaint of cough continued on the sixth day of his hospitalization, had also complaint of expectorating a small amount of sputum with green color and purulent characteristic. He also had pleuritic chest pain. In chest radiography of the patient, a slight density was determined in the right paracardiac region. In the sputum examination, a great amount of polymorphonuclear leukocytes and Gram-positive diplococcus showing a similar morphology with *Streptococcus pneumoniae* were seen. No organism was isolated from the sputum and blood culture. The patient was prescribed cefuroxime axetil 4x500 mg/day orally, and then he was discharged. On the tenth day of treatment, the patient had no complaint of cough and sputum; however, his pleuritic chest pain continued for five more days. Meanwhile, no complication was observed in his wife and his sons.

A nasopharyngeal swab sample was sent to the Refik Saydam Hygiene Institute Laboratory in Ankara to be tested by means of a rapid influenza test (Quickvue Influenza A+B test, Quidel) and an enzyme-linked immunosorbent assay (ELISA) for influenza A. The result of the rapid influenza test was weakly positive. A real-time polymerase chain reaction (PCR) assay was performed with the RoboGene Bird Flu H5N1 qualitative kit (AJ Roboscreen, Leipzig, Germany) and the WHO Influenza Reference Laboratory in London confirmed that the result was positive.

3. Discussion

The cumulative number of confirmed human cases of avian influenza AI (H5N1) reported to the WHO by 10 September 2008 is 387. Of these, 245 had died [2]. Twelve of these 387 cases were reported in our country and four died. Of these 12 cases, eight cases have been published to date and all these cases were children [3]. In all the cases reported so far, the patients have usually been children or young adults with high mortality rates. However, our patient was an adult and did not have any mortality risk. An article reported 169 patients confirmed by the WHO and they were commonly children [4]. The mortality rate in our country was lowest compared to other countries. According to the data announced by the WHO, the highest mortality rates were found in Cambodia (7/7, 100%), Indonesia (112/137, 82%) and Thailand (17/25, 68%) [2].

To our knowledge, no secondary bacterial pneumonia case following AI was reported. In an article published in Indonesia concerning all the AI cases in 2005, all patients with fatal disease presented with bilateral pneumonia and respiratory distress [4,5]. No sign of pneumonia was found in our patient during admission. He had complaints of sputum and pleuralgia in the improvement period, and then he was diagnosed with pneumonia. No change was observed in his vital findings except for fever during his monitoring by means of both AI and pneumonia. Moreover, he never needed respiratory support.

The most common laboratory findings for AI are lymphopenia, thrombocytopenia and increased aminotransferase levels [6,7]. However, none of these was seen in our patient. Instead, hematuria was observed as a striking laboratory finding. In a previous paper, it was also reported that hematuria developed in one AI case [8].

The estimated time between the exposure to poultry and the onset of illness suggests an incubation period of two to four days [6]. In our case this period was three days after exposure to the infected pigeon. The incubation period was two days for the patient's wife and his two sons. All the family members had a history of direct contact with the pigeon. In our patient, we thought that the cause of AI was direct contact with the pigeon. The result of the rapid influenza test from the nasopharyngeal swab sample was weakly positive for our patient, while those of his two sons and his wife were negative. ELISA and rapid influenza tests have limited value in diagnosing H5N1 infections and real-time PCR should be performed. In a 10-case experience observed in Vietnam, rapid influenza tests were less sensitive than real-time PCR for the diagnosis of AI (H5N1) [9]. Only rapid tests were performed for our patient's wife and his two sons, and the results were negative. As real-time PCR was not carried out, we were not able to confirm whether they had AI or not.

Although there are no controlled clinical studies, use of antiviral drugs and corticosteroids are recommended in the treatment of influenza A (H5N1) virus infections [6,7]. Due to the fact that clinical presentation of our patient was slight, we administered him only oseltamivir.

Viral influenza affects the respiratory epithelium and decreases local pulmonary host defence mechanisms. Decrease in functions of respiratory epithelium predisposes the patient to a subsequent bacterial invasion. Hence, secondary bacterial pneumonia could develop. Secondary bacterial pneumonia is a common cause of death in patients with seasonal influenza,

which has been found in 24% of all influenza-related deaths [10]. *S. pneumonia* is the most common cause of community-acquired pneumonia and bacterial co-infection with influenza A [11]. Viral influenza may be seen as three clinical presentations: viral influenza alone, viral influenza followed by early pneumonia in 1 to 3 days, and viral influenza followed by late pneumonia in 1 to 3 weeks. Usually early pneumonia may be caused by *Staphylococcus aureus* whereas late pneumonia may be caused by *S. pneumoniae* or *H. İnfluenzae* [12]. In our case, early pneumonia developed following a mild AI presentation. The agent that caused the pneumonia was probably *S. pneumoniae.* The pneumonia that developed in our patient was considered to be community-acquired pneumonia and was easily treated with oral cefuroxime axetil.

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