A Case Report on Histomorphological Features in Death Due to H1N1 Influenza

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DOI: 10.36348/sjpm.2022.v07i07.009 | Received: 25.06.2022 | Accepted: 18.07.2022 | Published: 22.07.2022

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Abstract

Swine influenza strain can be transmitted from human to human and causes normal symptoms of influenza. Transmission mainly occurs between pigs and pigs and humans. Our present study is of a 72yrs old male who died of H1N1 infection. Postmortem findings showed congested and edematous lungs with patchy areas of consolidation. Histopathological findings in lung revealed edema, pneumonic changes, hyaline membrane formation and focal cytophagocytosis.

Keywords: Influenza, congested, edematous, atheromatous, pandemic, pneumonia

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INTRODUCTION

Swine flu is a respiratory infection caused by type A influenza virus which first emerged in pigs. It has person to person transmission and can occur as outbreaks [1]. In 1918, a deadly influenza pandemic caused by H1N1 influenza virus, also known as the Spanish flu, infected approximately 500 million people around the world and resulted in the deaths of 50 to 100 million people (3% to 5% of the world population) worldwide, distinguishing it as one of the most deadly pandemics in human history. Influenza outbreak occurs in two distinct patterns: Endemic (seasonal) and Pandemic. H1N1, H2N2 and H2N3 are the influenza virus subtypes associated with pandemics. Infection is due to exposure to infected droplets expelled by coughing or sneezing via inhalation or contaminated hands or surfaces. Symptoms include fever, cough, malaise, headache, rhinitis, sore throat, muscle and joint pain and sometimes diarrhoea or vomiting. But most of the patients have only mild illness and have full recovery even without treatment. The cyclical process of endemic and pandemic pattern of influenza A virus is due to the ability of the virus to modify its surface proteins namely Hemaglutinin and Neuraminidase. Both of these surface proteins play an important role in the pathogenesis. American media named the initial outbreak as “Swine flu” or “H1N1 Influenza” while U.S Centres for Disease Control and Prevention referred it as “2009 H1N1 FLU” or “novel Influenza A(H1N1)”. Cases of H1N1 virus infection was first diagnosed in Mexico and US IN April 2009 which promptly reached pandemic proportions. The potential retention of influenza virus strains in swine after these strains have disappeared in the human population, essentially make pigs a reservoir where swine influenza viruses could persist, and later emerge to reinfect humans once their immunity to these strains has waned [2]. The present study was carried out to assess various histopathological findings in a case of death due to H1N1 at Kottayam district in Kerala state in India.

CASE HISTORY

Our case is a 72yrs old male, moderately nourished, who was a known case of systemic hypertension, dyslipidemia and peripheral vascular occlusive disease who died and the post-mortem examination was conducted at forensic department, Government Medical College, Kottayam. He had complaints of fever, cough and dyspnoea four days prior to hospitalisation. After admission with SpO2-72%, he was on Bipap mechanical ventilator support. Chest X-ray revealed alveolar shadows on both lungs. Crepitations were present on examination.

In the Forensic department, samples of blood, urine and viscera were preserved and sent for chemical analysis. Tissue bits were sent for histopathological examination. Throat swabs, samples of blood, urine and
CSF were sent for virology examination. Test result for virological examination was done at National Institute of Virology, Alapuzha and was found to be positive for pandemic H1N1. Antibody test for Dengue and Leptospira was done at Department of Microbiology, Government medical college, Kottayam and were found to be negative.

**POSTMORTEM FINDINGS**

Brain was congested and edematous. Right and left chest cavities contained straw coloured fluid. Air passages were congested. Lungs were congested, edematous and showed patchy areas of consolidation. Heart was flabby and chambers and valves were normal. Anterior descending branch of left coronary artery showed atheromatous thickening of walls with 70% narrowing of lumen. Inner aspect of aorta showed ulcerated atheromatous plaques. Postmortem findings were consistent with death due to Pneumonia.

**HISTOPATHOLOGICAL FINDINGS**

Lung tissue showed edema, pneumonic changes, hyaline membrane and focal cytophagocytosis. Spleen also showed evidence of focal cytophagocytosis along with congestion and edema. Myocardium, brain and liver showed congestion. There was mild portal congestion. Pericardium showed mild lymphocytic infiltration. Aorta showed atheromatous plaques. Thus the cause of death was ascertained as H1N1 pneumonia, severe atherosclerotic changes and changes of hypertension and chronic ischaemic heart disease were also present.

**DISCUSSION**

The 2009 swine flu pandemics had resulted in more than 17000 deaths all over the world. When WHO declared a pandemic in June 2009, total of about 74 countries and territories had already reported confirmed cases of H1N1 (lab confirmed). Most of the deaths occurred in young children, pregnant women and those with underlying chronic lung or other medical conditions. Although it had originated in pigs, it was able to spread from human to human. When the flu spreads from human-to-human, instead of from animals to humans, there can be further mutations, making it harder to treat because people have no natural immunity [3].

India also had outbreaks of swine flu in 2009. The first case reported in India was a man travelling from US to India who was found positive for H1N1 at Hyderabad airport on 13 May 2009. Subsequently more cases were found and reported and the first death due to H1N1 in India was reported in Pune.

There are numerous previous studies on histopathological findings in death due to H1N1.

Appropriate precautions have to be undertaken to prevent contact with body fluids and aerosols released in the air while coughing. Hand washing should be enforced and only a limited number of healthcare personnel should be allowed to come into contact with the infected person. Only through open communication among members of the inter professional team can the morbidity and mortality of swine flu be reduced [4].
In a retrospective study done at University of Michigan Health systems on 8 patients with fatal novel H1N1 infection, diffuse alveolar damage was seen in all cases. Bronchopneumonia was seen in 6 patients. Peripheral pulmonary vascular thrombosis was seen in 5 out of 8 patients with influenza. Cytophagocytosis was evident in all cases.

In a study of 21 patients by Mauad et al, diffuse alveolar damage was seen in 20 cases, 6 had necrotising bronchiolitis and 5 had extensive haemorrhage.

In a study conducted by Prasad et al, on 15 patients of H1N1 pneumonia in Pune, India, the histopathological findings were typically localised to lungs which included diffuse alveolar damage, intra alveolar haemorrhage, pulmonary edema, thickened alveolar septa, congested pulmonary vessels and hyaline membrane formation.

Bal et al, studied 9 autopsies of confirmed H1N1 pneumonia and found that along with lung involvement, most of them had centrilobular haemorrhagic necrosis in the liver and some had acute tubular necrosis in kidney. Haemophagocytosis in the reticuloendothelial system was also seen.

In a study of 46 patients by Shelke et al, diffuse alveolar damage was seen along with findings of spotty or focal hepatic necrosis and acute tubular necrosis.

In our patient also, lung tissue showed edema, pneumonic changes, hyaline membrane formation and focal cytophagocytosis. Spleen showed congestion, edema and cytophagocytosis. Liver showed features of mild portal inflammation and edema.

**INFERENCE**

With respect to the present study and the previous studies conducted, it is observed that H1N1 infection can lead to wide range of histopathological findings in various organs with characteristic involvement in the lungs. Lung involvement mostly includes diffuse alveolar damage, hyaline membrane formation, intra alveolar haemorrhage, necrotising features and pneumonia. The histopathological features when correlated with a clinical history of flu like symptoms should raise suspicion of H1N1 infection. These studies on the post-mortem histomorphological characteristics also help clinicians for proper intervention at the earliest.

**REFERENCES**