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

**INFEERENCE**

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.

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