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Highly pathogenic avian influenza virus (H5N1) causes severe symptoms due to insufficient induction of humoral immune responses

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It has been reported that fatal outcome of the patients infected with highly pathogenic avian influenza (HPAI) virus (H5N1) is associated with high viral load. However, the reason why patients cannot eliminate the viruses and succumb to them is not well known. To clarify the immune responses against H5N1 HPAI virus, we investigated temporal changes of the humoral immune response in animal models infected with low pathogenic pandemic H1N1 or H5N1 HPAI viruses. BALB/c mice were infected intranasally with A/Tokyo/2619/2009 (H1N1) or A/Whooper swan/Hokkaido/1/08 (H5N1). Cynomolgus monkeys were infected with H1N1 or H5N1 viruses via oral, nasal, and tracheal routes. Mice infected with H5N1 virus exhibited significant weight loss and nearly 100% mortality. In contrast, mice infected with H1N1 survived without weight loss. The titers of neutralizing and binding antibody against H5N1 virus-infected mice were significantly lower than those of H1N1 virus-infected mice. The H5N1 virus infection induced thinner outer layers of B-cell follicles. Similarly, cynomolgus monkeys could not induce antibodies against H5N1 virus, resulted in marked weight loss and manifestation of diffuse severe pneumonia. The similar alteration in formation of B-cell follicles was also observed in the monkey model. On the other hand, H1N1 virus-infected monkeys could induce successfully virus binding and neutralizing antibodies and exhibited only partial inflammatory foci. These results imply that the severe symptoms in H5N1 virus infection were associated with insufficient activation of B-cells to induce efficient neutralizing antibodies.

Biography

Ai Ikejiri has completed her PhD from Keio University School of Medicine (Tokyo, Japan) and joined National Institute of Infectious Diseases. Since 2014 she is a researcher of Tokyo Metropolitan Institute of Medical Science. She is interested in the implication of the host immune responses in influenza pathogenicity.

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