

Popular Article

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Important Viral Zoonotic Diseases in India

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Abstract

The link between human and animals are very close especially in developing countries where animals provide transportation, draught power, fuel, clothing, proteins etc. There are several zoonotic diseases identified in India. These diseases can lead to serious risk to public health with severe economic consequences. Examples are avian influenza, Japanese encephalitis, rabies and the Nipah virus.

Introduction

Animals provide many benefits to people. A person comes in contact with animals in their daily lives, both at home and away from home. Animals provide food, livelihoods, travel, sport, and companionship to people across the globe. However, animals can sometimes carry harmful germs that can spread to people and cause illness – these are known as **zoonotic diseases** or zoonoses. The joint expert Committee of WHO and FAO (1959) has defined Zoonoses as are "those diseases and infections which are naturally transmitted between vertebrate animals and man". Zoonoses is the word derived from Greek word "zoo" means animals and "noses" means diseases. This term was coined and first used by Rudolf Wirchow who defined it for communicable diseases. Animals have played an important role in the evolution of humans. The hunter-gatherers started settling down and domesticating animals for food, transportation, agriculture, security, war or recreational activities, etc. This resulted in increased contact with humans and sharing of habitat thus, the transmission of diseases from animals to man and man to animals became common. Industrialization needed the



mass breeding of animals for food, sports or recreation, etc. Due to technological developments and fast communication, humans, animals, and animal products started moving from one part of world to the other, many times carrying infections in the globalization era.

1. Rabies

Rabies (Latin meaning 'rage' or 'madness') is one of the oldest known zoonoses and is considered the deadliest disease with nearly 100% mortality. It has been recognized in India since the Vedic period and is described in the ancient Indian scripture Atharvaveda.

Etiology: The disease is invariably fatal with progressive encephalitis, caused by the virus belonging to the genus Lyssavirus of Rhabdoviridae family. The viruses in this family have a bullet-shaped structure enveloped with single-stranded, negative-sense RNA genome.

Epidemiology: The disease is most commonly seen in carnivores from which it is transmitted to herbivores and other hosts. The dog has been and still is the main reservoir of rabies in India. However, wild life rabies is also a big problem and challenge to India. India has the highest incidence of human rabies in the world and reported about 20,000 human deaths every year (Sudarshan *et al.*, 2008).

Mode of transmission: The major burden of human rabies is attributed to dog-mediated transmission (around 99% cases). However, the sylvatic cycle of rabies also exists, where wild animals (e.g. bats, raccoons, foxes) serve as the maintenance host for the virus. When a rabid animal bites another warm-blooded mammal, the rabies virus is primarily transmitted through saliva. The infected animal drools excessive saliva which may also drip onto its claws. Scratches from an infected animal and contact of saliva with mucous membranes or abrasions or cuts can lead to exposure to the rabies virus.

Symptoms: There are three clinical phases of the disease:

- a) Prodromal phase
- b) Excitation phase
- c) Paralytic phase

The disease begins with prodromal symptoms

such as headache, nausea, vomiting, malaise, sore throat and slight fever lasting for 3–4 days. About 80% of patients complain of pain or tingling at the site of the bite. This stage is followed by excitatory



stage thereby, showing widespread excitation and stimulation of all parts of nervous system. The patient is intolerant to noise, bright light (photophobia) or a cold draught of air. Aerophobia is considered by some to be pathognomonic of rabies. Examinations may show increased reflexes and muscle spasms along with dilatation of pupils and increased perspiration, salivation and lacrimation. Mental changes include insomnia, convulsions, tonic-clonic spasms, fibrillary muscle twitching, fear of death, anger, irritability and depression. The symptoms are progressively aggravated and all attempts at swallowing liquid become unsuccessful. The patient may die abruptly during one of the convulsions or may pass on to the stage of paralysis and coma. In paralytic stage, there will flaccid paralysis of face and head muscle which extends to the extremities. Patient dies after 3 or 4 days as a consequence of respiratory and heart failure.

Diagnosis: The clinical diagnosis of rabies is difficult unless the rabies-specific signs of hydrophobia or aerophobia are present. In most instances, rabies is fatal, and the testing of specimens is carried out in the postmortem state. The rabies diagnosis relies on laboratory tests for the detection of viral antigens. For diagnosis, an appropriate specimen is needed (e.g. brain tissue, CSF, other specific specimens). Among diagnostic tests, the fluorescent antibody test (FAT) is considered the gold standard in postmortem rabies diagnosis. Alternative antigen detection methods such as ELISA, direct rapid immunohistochemical test (DRIT) or indirect rapid immunohistochemistry tests (IRIT) are also used. For confirmation and virus isolation, the rabies tissue culture infection test (RTCIT) or the mouse inoculation test (MIT) are used. The testing of suspected rabid dead animals or human samples does not pose a high risk for further spread of the disease; therefore, Biosafety Level 2 (BSL-2) lab. facilities are adequate. Negri bodies can be stained by Seller's stain, which appear as Magenta to dark red.

<u>Treatment</u>: Rabies is 100% fatal once the disease is developed. There is no specific treatment for the disease except management. The patient should be kept in a calm, quiet and isolated room. Analysics and muscle relaxants are prescribed to alleviate pain and muscular spasms. Intensive care may allow an occasional patient to survive.

Vaccines and immunization: Introduction of cell culture vaccines like human diploid cell vaccine, purified chick embryo cell vaccines and purified Vero cell vaccine have been a boon in rabies treatment and prophylaxis. There are two recommended intramuscular regimens for post exposure vaccination recognized by WHO. These include the Essen regimen given on days 0, 3, 7, 14 and 28 in the deltoid muscle (a booster dose on 90th day is optional) and the Zagreb regimen where one

dose of vaccine is administered at two sites on day 0 and at one site on day 7 and 21.

2. Kyasanur forest disease

A Flaviviral disease, transmitted by the ticks (*Haemaphysalis spinigera*) between monkeys, primates, cattle and man. Man is dead end host.

Etiology : KFD virus belongs to Russian Spring Summer Encephalitis group, a member of family Flaviviridae

Epidemiology: It was firstly identified in Kyasanur forest of Shimoga district in Karnataka. Epidemics of KFD coincide with nymphal activity during Jan-May. The outbreak which occurred in Wayanad district of Kerala is the largest outbreak ever reported from Kerala. Earlier KFD was restricted to a few districts of Karnataka but recent evidences point to its spread to Kerala, Goa and Maharashtra.

Symptoms - Incubation period: 3-8 days.

- 1. Acute phase Sudden onset with fever (6-11 days), bradycardia, hypotension, abdominal pain, cough, and hemorrhage in gums, nose, stomach and intestine, and finally prostration.
- Second phase Afebrile with mild meningo-encephalitis (9-21 days), severe headache, stiffness of neck, mental confusion, tremors, giddiness and abnormal reflexes. CFR - 5-10%.
 Serious with dehydration in low socio-economic group because of poor state of nutrition and chronic diseases.
- 3. Convalescence Prolonged 4 weeks or more. Physical effort is difficult and tremors due to muscle weakness. Leucopenia in first week followed by leucocytosis in third and fifth week, thrombocytopenia and decrease haematocrit is seen.

Prevention and control: Control of ticks – r BHC/lindane as spray -1 kg/hectare around the spot of monkey's death and forest tracks frequented by man, Application of repellants- dimethyl phthalate, restriction of cattle movement in tick infected area, killed KFD vaccine – population at risk, protective clothing etc.

3. Avian influenza

Swine and avian influenza are acute respiratory infection characterized by fever, cough and dyspnoea.

Etiology:- Influenza A viruses are negative sense, single-stranded, segmented RNA viruses under the family-Orthomyxoviridae. Several subtypes of influenza viruses exist in nature depending on the hemaglutinin and neuraminidase antigens. So far, 17 H and 10 N antigens were identified. Some

subtypes such as H5N1, H7N3, H7N7, H7N9, and H9N2 are very pathogenic to human beings.

Epidemiology: Initially, in India, the disease was being reported mainly in backyard poultry in vicinity of migratory birds/water bodies particularly in North-Eastern States and West Bengal. The ducks used to be reservoir of the virus, harbouring the infection. However, 4 four occurrences were reported from the poultry farms of central government such as DADF, ICAR and State Governments later on. Disease also shifted from Northern East to East and southern parts of India such as Odisha, Karnataka and Bihar.

Symptoms: sudden death,

- o respiratory distress,
- swelling of the head,
- o purple discolouration of comb and wattles,
- o coughing, sneezing,
- o rasping breathing,
- o rapid decrease in feed and water intake,
- decreased egg production,
- ruffled feathers,
- depression,
- closed eyes,
- o diarrhoea, and
- o occasionally, nervous signs.

Prevention and control:

- i. All countries must maintain the public and private components of Veterinary services, which comply with OIE standards on quality, including appropriate legislation, early detection and response capacities in face of biological events in animals, compensation mechanism establishment and management, efficient veterinary laboratories, use of vaccination in relevant epidemiological situations when appropriate.
- ii. During outbreaks, HPAI viruses are normally eradicated by depopulation of infected flocks, combined with other measures such as movement controls, quarantines and emergency vaccination. Insect and rodent control, disposal of contaminated material and thorough cleaning and disinfection are also important to prevent the further spread of virus.



4. Chandipura virus encephalitis

Etiology: Chandipura virus (CHPV) is a vesiculo-virus which belongs to the family Rhabdoviridae.

Epidemiology: Vesiculo viruses were isolated in 1965 in the Chandipura (Nagpur) region of India from two adult patients with febrile illness during an outbreak caused by chikungunya and dengue viruses. In the recent past, the noticeable association of CHPV with pediatric sporadic encephalitis cases as well as a number of outbreaks in Andhra Pradesh (2004, 2005, 2007 and 2008), Gujarat in (2005, 2009-12) and Vidarbha region of Maharashtra (2007, 2009-12) have been documented.

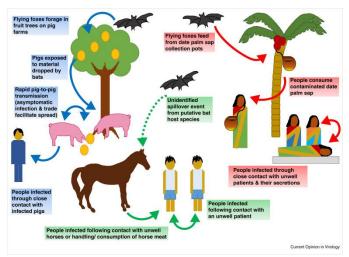
Clinical signs: Fever and other symptoms similar to those of flu, and acute encephalitis.

5. Nipah virus infection

Nipah is an emerging bat-borne zoonosis that causes sporadic outbreaks of fatal encephalitis in regions of South Asia.

Etiology: The Nipah virus is an enveloped, non-segmented, negative-sense, single-stranded RNA virus belonging to the family Paramyxoviridae.

Epidemiology: In India, the first Nipah virus outbreak was reported in Siliguri town in 2001, followed by a second outbreak in Nadia district in 2007, both in West Bengal. Another outbreak was reported in Kerala during 2018. The index case contracted the virus from fruit bats and later, the transmission of the disease was observed from person to person and to healthcare workers.



Signs and symptoms: Symptoms typically appear in 4-14 days. The illness initially presents as 3-14 days of fever, headache, and includes signs of respiratory illness, such as cough, sore throat, and difficult breathing. A phase of encephalitis may follow with symptoms like drowsiness, disorientation, and mental confusion, which can rapidly progress to coma within 24-48 hours.

Prevention: Practice handwashing regularly with soap and water.



- Avoid contact with sick bats or pigs.
- Avoid areas where bats are known to roost.
- Avoid eating or drinking products that could be contaminated by bats, such as raw date, palm sap, raw fruit, or fruit that is found on the ground.
- o Avoid contact with blood or body fluids of any person known to be infected with NiV.

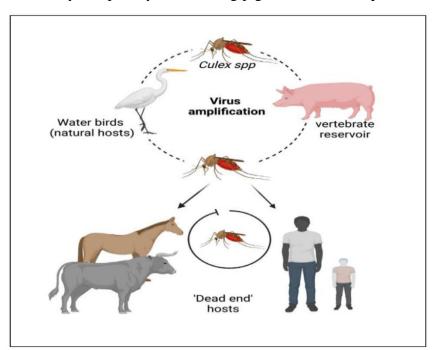
6. Japanese encephalitis

Japanese encephalitis (JE) is a mosquito-borne viral disease that affects the central nervous system, causing encephalitis in humans as well as in horses and abortion in pigs.

Etiology : Japanese encephalitis virus (JEV) belongs to the genus Flavivirus of family Flaviviriade and is a small, enveloped virus with a single stranded, positive sense RNA.

Epidemiology : There are two subtypes of JE virus namely, Nakayama and JoGAr – 01 (Beijing), however GP78 (Nakayama strain) is thought to be the most common Indian strain. It was clinically diagnosed for the first time in India in 1955 at Vellore, erstwhile North Arcot district of Tamil Nadu. Since then, the disease has been reported from 24 states/Union Territories so far. JEV is endemic in Gorakhpur and Basti divisions of eastern Uttar Pradesh.

<u>Transmission</u>: Pigs acts as amplifying host of JE virus and ardeid birds acts as its reservoirs. Virus is maintained in nature by complex cycle involving pigs, birds and mosquito as vectors.



Clinical signs and symptoms: Most JEV infections are mild (fever and headache) or without apparent symptoms, but approximately 1 in 250 infections results in severe clinical illness.

Incubation period is 4-14 days. Severe disease is characterized by rapid onset of high fever, headache, neck stiffness, disorientation, coma, seizures, spastic paralysis, and ultimately death.

Prevention and control: Safe and effective JE vaccines are available. Currently, 4 main types of vaccines are in use: inactivated mouse brain-derived, inactivated Vero cell-derived, live attenuated and live recombinant (chimeric) vaccines. WHO recommends having strong JE prevention and control activities, including immunization in all regions where the disease is recognized, public health priority, strengthening surveillance and reporting mechanisms. Vaccination of humans should be prioritized over vaccination of pigs.

Conclusion

The wide range of animals (domestic, pets, companion, synanthropic) acts as a reservoirs and carriers of many zoonotic disease organisms that are distributed in the natural ecosystem such as soil, water, food, and aerosol. The link between human and animals with their surrounding are very close especially in developing countries where, animals provide transportation, draught power, fuel, clothing, proteins etc. This can lead to serious risk to public health with severe economic consequences. As the population continue to increase and new area are opened up for food production, both for humans and their livestock, which are more frequently exposed to disease agents as a result of encounters with wild animals thereby, increasing human exposure to rare zoonotic infections. Increased urbanization allows faster spreading of any new disease between populations and also within an area.

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