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Conceptual Paper

Conceptual Note on Rabies Virus

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Abstract

In this article we have discussed about the Rabies Virus, introduction, transmission, signs and symptoms of rabies and prevention protocol.

Keywords: Rabies Virus; lyssavirus; Nucleoprotein

Introduction

Rabies Virus is of lyssavirus genus under rhabdoviridae family which is ss RNA virus of "-"ve sense. It is having a bullet shaped structure. Rabies has been recorded in various parts of the world although Australia, New Zealand, Hong Kong, Singapore, Britain, Hawaii free from rabies.

The antigenic property of rabies Virus is due to both glycoprotein G and Nucleoprotein. Glycoprotein G is pathogenic as it binds to the Ach receptor in neural tissues first step in pathogenesis causing neuroinvasiveness, hemagglutinin activity, antibodies produced because of this antigen are generally protective in nature so we can used the purified form of this antigen for vaccination. The nuecleoprotein non pathogenic. The antibodies produced by this antigen can't be used for the production of vaccine as they are not protective in nature.

Animal susceptibility

- Very highly susceptible -fox, jackals, wolves, cotton rats
- Highly susceptible - Rabbit, cattle, cat, hamster, racoons, bats
- Moderately susceptible - dogs, goats, sheep, horses
- Low susceptible - opossum
- Vampire bats acts as a vector

Strains of Rabies Virus

It was recognized long ago that the strain of virus known as the 'street virus' differed in same way from 'fixed virus' which had been cultivated for vaccine production (grown in cell culture or passaged through serial generations of laboratory animals specially rabbit).

Street Virus Fixed virus Freshly isolated Isolated after serial intercerebral passage in rabbits/cell line.

- Produce Negri Do not produce.
- Affect salivary gland Do not produce.
- Pathogenic non-pathogenic
- Incubation period long and Incubation period is fixed.
- Variable and short

Incubation period

Depends on the site of the bite. The nearer the bite to the head, the shorter the Incubation period. It may vary from 2 weeks to 9 months.

Pathogenesis

If animal carrying the rabies virus bite/lick on mucosa local multiplication of rabies virus happens in the skeletal muscle also in epithelial cells where the neuromuscular junction also present

in neuromuscular junction it attaches to the nicotine Ach receptor and then travels to dorsal root ganglia of spinal cord (centripetal spread) via motor nerves then goes all the way to C.N.S mainly hippocampus and cerebellum then via centrifugal spreading goes to various tissue mainly salivary gland, also skin cornea, liver, pancreas, heart etc goes to salivary glands via facial, glossopharyngeal nerve. In C.N.S it parasitizes the ganglia cell, damage the nerve cell and vascular endothelial, causing irritation of nerve cell increases excitability (furious form). Neuronal degeneration.

- Paralysis of the muscle of deglutination - unable to swallow/ dribbling of saliva.
- Paralysis of jaw muscle- Propped jaw.
- Paralysis of respiratory muscle- Asphyxia and death.

Virus is shed even in infected human's saliva but human to human transmission is not confirmed.

Brain

- Mononuclear cell infiltration.
- Perivascular cutting of lymphocytes.
- Babes' nodules consisting of glial cells.
- Negri bodies present (Intracytoplasmic eosinophilic in brain neurons consisting of viral protein and RNA.) The Negri bodies help in detection of but only after a patient is dead (Postmortem diagnosis).
- Perkinjee cells of cerebellum and pyramidal cells of hippocampus contain this Negri bodies.

Classification of exposure: It is classified as Class 1,2 and 3, depending upon nature of exposure.

- Class 1: Slight or negligible exposure, all cases of licks except on fresh cuts and scratches.
- Class 2: Moderate exposure: Two types---- a) all bites except those on the head, neck, face, palm, and fingers and less than minor wounds. B) Licks on fresh cuts and scratches drawing blood.
- Cla**s**s 3: Severe exposure: All bites on the neck or above palm and fingers. Lacerated wounds anywhere in the body multiple bites (more than 5 bites).

Clinical signs

The clinical course that usually last for 3-7 days or exceptionally for 10 to 12 days can be divided into three phases prodromal, fusions and paralytic or dumb stage. Towards the end of Incubation period and 2 to 3 days before the onset of typical clinical signs a number of-or prodromal symptoms are seen. Change in temperature is observed. Some become more affectionate than usual and other more irritable.

Slight fever, photophobia and decreased corneal reflex.

By the third day after onset of illness, the dog enters the furious stage that lasts for 3 to 7 days. It becomes irritable, restless and nervous and tends to hide under dark. During the furious phase, there is characteristic changes in its bark due to paralysis of laryngeal muscles. Profuse salivation and frothing lead to difficulty in swallowing and drinking.

In cases when the second or furious phase is extremely short or absent, the animal rapidly enters into the paralytic or dumb stage. Here lower jaw paralysis is seen when the tongue protrudes and the dog is unable to eat and drink. Finally there is exhaustion and complete in coordination of movement. Animal gradually becomes comatose and dies from the second to fourth day after the onset of the paralytic stage.

In cats

More furious form is observed than dog. Cat generally bites man and other animals on their face.

- **In cattle:** Cattle usually bellow in a characteristic low pitched voice due to vocal cord paralysis. In field cases, bellowing and excessive salivation are the most common signs.
- **In sheep and goat:** Animals show restlessness, twitching of lips, salivation, aggressiveness and death. Increased sexual libido is observed.
- **In humans:** Mild fever, nausea, headache are common early complications. Hypersensitivity, anxiety, masclar spasm, salivation are evident. In the later stage, there is development of severe spasm of the muscles of deglutination and respiration. Paralysis develops, convulsions follow and death which invariably ensures is usually from respiratory paralysis. "Babes nodules ".

Diagnosis

- Microscopical demonstration of Negri bodies.
- Fluorescent antibody teat.
- Complement fixation test, ELISA test.
- PCR test.
- Immuno Peroxidase Test (IPT)
- Complement Fixation Test (CFT)

Treatment

There is no specific treatment for rabies, Dog usually die after showing clinical signs. The site of bite should be washed with water or soap. Alkali prevents multiplication of virus. NaHCo3 may be used. Tincture iodine, 70% alcohol may be used. Wound my be cauterized with carbolic acid or HNO3. A solution of zephiran may prevent the establishment of the infection.

Antirabies serum may be infiltrated around the world. Treatment with antirabies vaccine is indicated. Dose for unprotected dog and cat (less than 15kg) _2ml s/c daily for 14 days and more than 15kg dog -5ml s/c daily for 14 days and more than 15kg dog _5ml s/c daily for 14 days. For protected dog and cat same dose for 7 days.

For all other animals unprotected animals less than 15kg _2ml s/c daily for 14 days (for protected _7days) body wt_ 15-50kg 5ml for 14 days (unimmunized) for 7 days (immunized). Body wt more than 50kg but less than 100kg 15ml for 14 days (unimmunized) for 7 days (immunized).

Pre-exposure vaccine

- **LEP (low egg passage):** Flurry strain 40-60 egg passage prepared from attenuated strain of rabies virus grown in developing chick embryo. Used in dog. 3ml i/m protection for 3 years
- **HEP (High egg passage):** Flurry strain, above 180egg passage. Used in cattle. 5ml i/m protection for 6-12months
- **Duck embryo:** attenuated duck embryo. Used for human. 2doses at interval of 6 weeks
- **Tissue culture:** Tissue culture from pig kidney used for cattle, horse and dog. Route s/c. Dog -5ml revaccination after 6months then at yearly intervals.
- Sample- prepared as 20% suspension of brain of sheep infected with a modified rabies virus. Inactivated by betapropiolactone, used in dog and cattle s/c route. Dose: Dog_ less than 15kg- 2ml, more than 15kg -5ml. × 7 injection. Cattle 30ml × 14 Injection. Repeated every year.

Conclusion

Rabies is very dangerous disease. If the disease starts showing its symptoms, it is next to impossible to recover the patient. There is only one event of rabies recovery so far in these entire worlds, that too by bat biting. So prophylaxis is the only way to prevent the disease. There are so many ways discussed in this article for immunization, we must have to follow those steps and if we want to eradicate the disease we have to start from grass root level of our society by eradicating the disease from dogs