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Ocular Manifestations of Rabies

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Introduction

Rabies is a zoonotic fatal and progressive neurological disease that is caused by the rabies virus (RABV) and its replication in the central nervous system (CNS). RABV is a negative-strand RNA virus that is part of the rhabdovirus family and causes an infection that can affect all warm-blooded animals [1]. This disease is still endemic to many countries in the world. It is most efficiently transmitted through a bite of an infected animal whose saliva contains the RABV and is mainly associated with bats in the Americas and with dogs in Europe, Asia, and Africa. This infection has a highly variable incubation period that ranges from 2 weeks to 6 years and often presents with severe neurological signs. An early diagnosis and treatment are critical at preventing disease progression [2,3]. Rabies virus can induce an ocular disease in humans through invasion of the optic nerve [4].

Epidemiology

Even though the rate of rabies infection has steadily decreased for decades, the disease is still prevalent around the world and endemic to certain regions with over 60,000 yearly casualties caused by the disease, and around 15 million individuals receiving rabies post-exposure prophylaxis. The majority of rabies cases occur in developing nations in Asia and Africa, most notably in India, Bangladesh, and Nepal. The urbanization of dog populations and dog slaughterhouses are considered the main contributor for the high transmission of rabies in endemic areas. Human cases have seen a severe decrease in Latin America despite the presence of certain endemic regions. Even though rabies still exists in North America and Europe, human cases have mostly disappeared. These advances in decreasing human cases are most likely due to the enforcement of animal vaccination policies, especially in dogs [3,5].

Disease Pathogenesis

The RABV causes a slowly progressive disease that turns fatal once clinical symptoms are noted. The incubation time of the virus depends on the inoculation site and concentration of inoculated virus. Post-exposure the virus attaches to G-proteins in myocytes and local neurons where it amplifies and remains for up to 18 days after which it is transported by retrograde microtubule-dependent axonal transport to reach the CNS and infects the nerve cells [6]. Once the virus reaches the CNS, it rapidly multiplies in neuronal bodies and is then transported to the synapse where it spreads to another neuron. This trans-synaptic spread continues until the infection is disseminated in the CNS and the disease has caused encephalitis. The spread of the RABV to diverse end organs such as the salivary glands, muscle fibers, and the eye occurs in the late stages of infection via anterograde transport, which highlights a difference in the mechanism of propagation of the virus in late infection [7].

Ocular Presentations and Pathology

Open globe injuries with rabid animals can cause virus transmission. Injury to the eye that is caused by a scratch wound or bite can pass along the RABV. The virus has a higher infection rate and shorter incubation period in those bitten in the face. Corneal transplantation with infected tissues is also a possible route of RABV infection and CNS dissemination [8]. Notably, the RABV can directly enter nerves without previous replication in the muscle [9]. Ophthalmologists need to be aware of this mode of transmission and quickly administer

post-exposure rabies prophylaxis to prevent disease spread in such patients [10]. Once the RABV reaches the CNS, it can invade the optic nerve and infect the retinal ganglion cells and the trigeminal ganglia resulting in infection of the cornea. Apoptosis of uninfected photoreceptors and morphological alterations of dorsal root ganglia occurs in RABV ocular infection [4]. Additionally, following brain infection, the RABV can infect the retina where it induces inflammation by promoting infiltration of neutrophils and T-cells into the eye [11]. This infection can cause endothelial damage and perivascular inflammation in the retinal veins and the spilling of exudate into the outer plexiform and subretinal layers of the retina. Chronic inflammatory infiltration of the choroid and ciliary body with destruction of the retinal ganglion cells is noted [4]. The visual symptoms of rabies infection are non-specific and may include transient visual limitations, photophobia, and blurring of disk margins [12,13].

Diagnosis

Clinical diagnosis of rabies can only be made with confidence in cases of furious rabies where patients present with autonomic dysfunction, phobic spasms, and fluctuating consciousness. Infection can also present as paralytic rabies where there is flaccid muscle weakness starting at the site of infection, and then progressing to gradual paralysis. This form of infection cannot be clinically diagnosed as it often mistaken for Guillain-Barré Syndrome. Multiple tests are required to diagnose a flaccid RABV infection ante-mortem in humans: reverse transcription followed by polymerase chain reaction of the patient's saliva, testing for antibodies in serum or cerebrospinal fluid, and/or skin biopsy specimens that are examined for rabies antigen in cutaneous nerves [14]. RABV infection can be identified using a corneal impression test due to induced infiltration of neutrophils into the eye. Ophthalmologists should consider performing corneal smears when rabies is a possible diagnosis [15].

Management

There is currently no proven treatment for rabies. For potential rabies exposure, the immediate recommendation is to thoroughly clean bite wounds and scratches with running water, soap, and povidone-iodine or other antiseptics for fifteen minutes [16]. Following wound disinfection, combination therapy of rabies vaccination, ribavirin, ketamine, and interferon-alpha is advised [17]. Rabies vaccination has two components: pre-exposure and post-exposure prophylaxis. Pre-exposure prophylaxis is recommended for individuals with an increased risk of RABV due to residence, occupation, or travel to endemic areas. They should be offered to individuals with continuing risk every one to three years. Postexposure prophylaxis should be given on the same day following RABV, one-week post-exposure, and two-three weeks post-exposure [18]. Bilateral optic neuritis, retinal artery occlusion, and multiple evanescent white dot syndrome have been reported following post-exposure prophylaxis vaccine administration given for RABV [19-21].

Summary

Rabies is a zoonotic disease most transmitted through the bite of an infected animal but can also be transmitted via corneal transplant with infected tissues. The virus can replicate in the CNS and cause encephalitis. It can also reach the eye and cause a wide variety of pathological processes but does not present with any specific ophthalmic findings. Clinicians should consider post-exposure prophylaxis for potential rabies infections.

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