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The Essentials of EIAV: Essential Facts on Diagnosis and Control

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Abstract: Equine infectious anaemia (EIA) is a disease that affects horses caused by the equine infectious anaemia virus (EIAV), which first appeared in 1840. This article provides an in-depth examination of Equine Infectious Anemia Virus (EIAV), a devastating viral disease affecting horses, donkeys, and mules. EIAV belongs to the lentivirus family, sharing similarities with retroviruses in terms of long-term infections and immune system targeting. The control of EIAV presents challenges due to its persistence and potential for spreading. The article explores the virus's biology, transmission routes, clinical manifestations, and diagnostic methods. It delves into various control strategies, including regulations, testing programs, biosecurity practices, and advancements in vaccine development and antiviral therapies. By fostering an understanding of EIAV's nature and its association with lentiviruses, this article aims to enhance knowledge and promote effective disease management practices.

Keywords: Structure, Clinical pathology, Transmission and Pathogenesis, ELISA, AGID and PCR, Vaccine development, Immune reaction.

Introduction

Equine Infectious Anemia (EIA), also known as swamp fever, is a devastating viral disease that affects horses, donkeys, and mules [1]. It is caused by Equine Infectious Anemia Virus (EIAV), a member of the lentivirus family. Lentiviruses are a group of retroviruses known for their ability to establish long-term infections and weaken the immune system, leading to chronic and progressive diseases [3]. However, controlling and managing EIA poses significant challenges for horse owners, veterinarians, and regulatory authorities due to the virus's persistence and potential for widespread transmission [2]. This article aims to delve into the intricate details of Equine Infectious Anemia Virus, encompassing its characteristics, transmission routes, clinical manifestations, and diagnostic methods. Furthermore, it explores the diverse strategies employed to control this virus, with the ultimate goal of minimizing its impact on equine populations and curtailing its further spread [9]. Gaining a comprehensive understanding of EIAV and its association with lentiviruses is crucial for implementing effective control measures. By comprehending the virus's biology and transmission dynamics, veterinarians and horse owners can make informed decisions to safeguard the health and well-being of equine populations [19]. Additionally, a thorough knowledge of diagnostic techniques and surveillance methods is essential for early detection of infected animals and prevention of disease transmission. Control measures for EIA encompass strict regulations, testing programs, and the implementation of robust biosecurity practices. These measures are designed to identify infected animals, isolate them from the general population, and minimize the potential for transmission [20]. Furthermore, ongoing advancements in vaccine development and research into antiviral therapies offer promising avenues for future control strategies [30]. By emphasizing the significance of lentiviruses, specifically Equine Infectious Anemia Virus, and the imperative to implement effective control measures, this article seeks to contribute to the broader understanding of the virus's impact on equine health and the adoption of efficient disease management practices. Through sustained research efforts, collaborative initiatives, and increased awareness, we can work towards reducing the prevalence of EIA and safeguarding the welfare of our cherished equine companions.

Etiology

Based on the ultrastructure, genomic organization, opposing transcriptase activity, and serologic cross-reactivity, EIAV, the etiologic agent of EIA, is recognized as a member of the Retroviridae family and the Lentivirinae subfamily [4,3].

It has been linked to a variety of lentiviruses, including caprine arthritis-encephalitis virus, sheep Visna/maedi virus, and human and feline immunodeficiency viruses [3]. EIAV is an enveloped RNA virus with a thick, conically shaped centre and surface "knobs" to protect it (core) [4,5]. The gp90 and gp45 glycoproteins are viral-specific glycoproteins that are likely required for virus entry of host cells and can also act as strong immunostimulants [2]. The virus contains viral RNA in a dense main core. Three short pieces of RNA are thought to code for viral regulatory proteins of a lesser size. Surface glycoproteins are more sensitive to antigenic strain than non-glycosylated structural core proteins [3]. The central proteins, p26, elicit a humoral immune response in the vast majority of infected horses and are utilized as serologic diagnostic tests for the virus [1].

Clinical Signs

The clinical signs (Figure 1) and duration of infection with EIAV are variable and depends upon virulence of the viral strain, viral dose, and susceptibility of the horse [1,5]. Incubation period of EIAV is 15-45 days. It may be longer in naturally acquired cases of infection [6]. After incubation period, classic cases of the disease have been identified as progressing through three clinical phases [6]. The first and acute phase persists 1-3 days and is characterized by depression, fever and thrombocytopenia [7,9]. As these signs can be mild, they are overlooked and misdiagnosed.

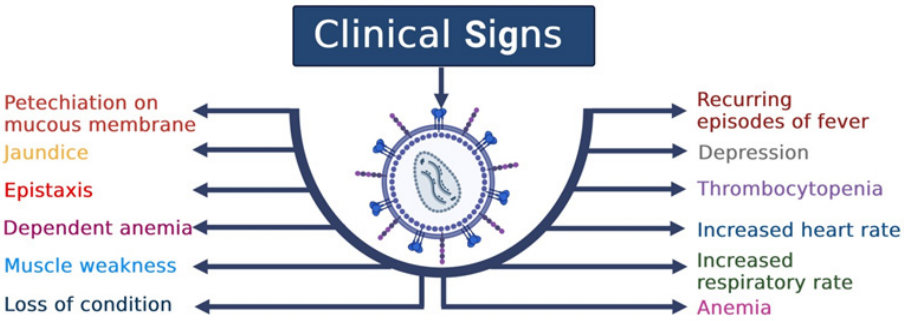

Figure 1: General clinical signs of EIA exhibited by the horses, donkeys, and mules

The duration between the episodes can range from days to weeks or months [12,9]. In most of the cases the infection finishes within a year and infected horses become non-carrier and non-reservoir of EIAV. Many of these horses remain normal but chronically infected horses may experience these episodes varying from fever and thrombocytopenia to the clinical signs have been described [7]. Such episodes are linked with intercurrent infections or other sources of stress. After the acute phase, some EIA outbreaks are linked with peracute infection which appear as uncontrolled primary viral infection and can characterized by very high fever, severely reduced platelet counts, and acute depression and epistaxis leading to death [9]. It is impossible to diagnose EIA based on clinical signs due to wide variation in natural cases of infection.

Lesions

Mostly the gross lesions seen in acute cases of EIA infection. These may include enlargement of spleen, liver, and abdominal lymph nodes, dependent edema, and mucosal haemorrhages [10,9]. Chronic cases of infection are characterized by emaciation, pale mucous membranes, petechial haemorrhages on internal organs especially spleen and kidney. Thrombosis of blood vessels has also been identified. Histopathologically, nonsuppurative hepatitis and in some cases a glomerulonephritis, periventricular leukoencephalitis, meningitis or encephalitis can be seen. In chronic cases, ocular lesions can be seen [9]. Proliferation of reticuloendothelial cells is seen in many organs like liver along with accumulation of hemosiderin in Kupffer cells [7,10]. Lymphocyte accumulation in perivascular areas can also be found in various organs.

Pathogenesis and Transmission

The disease condition induced by EIAV is the result of macrophage infection that meddles with host-cell gene expression and generates certain pro-inflammatory mediators or cytokines, i.e., interleukin 1 (IL-1), interleukin 6(IL-6), transforming growth factor, and tumor necrosis factor alpha (TNFα) [38,35]. These pro-inflammatory mediators activate the arachidonic pathway to increase the production of prostaglandin E2 (PGE2), which elicits the febrile responses. Moreover, the cytokine-mediated suppression of growth of equine megakaryocyte colony growth can also induce thrombocytopenia [38]. The retardation of the process of erythropoiesis by TNF-alpha plays role in anemia [14]. Adaptive immune responses are also significantly involved in the pathology of this infection. Bound IgM or IgG is present in substantial amounts in the platelets of infected horses. This leads to the immune-mediated destruction, which ultimately contributes to the enlargement of spleen and liver [8]. Figure 2 explains the pathogenesis of EIA.

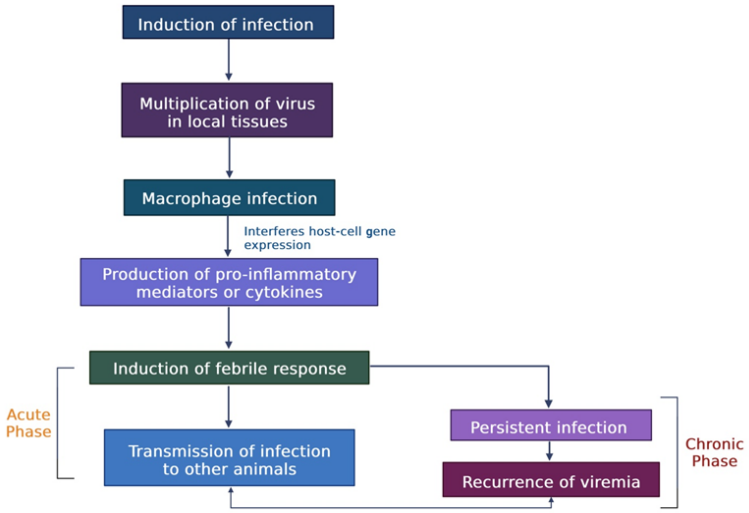

Figure 2: Pathogenesis of EIA

EIAV is spread by the exchange of blood or blood products between sick and healthy horses [11]. When large insects (horseflies and deerflies) of the Tabanidae family feed on blood, transmission occurs. Iatrogenically, infected blood transfusions, infected or nonsterile hypodermic needles and infected surgical tools (overall enamel tools) can spread the infection [13]. There has also been evidence of transplacental and colostral transfer. The plasma virus titers of acutely infected febrile horses are higher than those of afebrile horses by a factor of 1000 to 100,000. As a result, a feverish horse may have a higher risk of viral transmission than a horse without a scientific symptom. As a result, horses exhibiting clinical signs are the primary source of disease transmission and propagation [15]. Virus transmission through the venereal route has also been described. The virus is transmitted by subcutaneous injection of experimental horses using raw or fresh semen from an infected stallion [16]. One mare grazed with EIAV-uninfected animals, after being mated with a chronically ill stallion, developed a slight vulva hemorrhage. Stallions infected with the virus may have lower fertility. Semen from chronically infected stallions showed decreased sperm motility, a lower sperm count, and aberrant sperm shape. Transplacental switch, feeding infected colostrum or milk, and transmission by vectors are all possible pathways for viral transmission between mares and foals [17]. When a mare has acute febrile with high titer of virus in the blood during pregnancy chances of EAIV transmission through the transplacental route are increased [32]. This might potentially lead to abortion. When horse fetuses were contaminated before 203 days of pregnancy, it resulted in abortion. Between 21 and 64 days following infection, abortion occurred. Foals would be infected and die within 60 days if fetuses were infected in late pregnancy (Issel and Foil 2015). In one research, not more than 10% of foals born to mares (with no pronounced clinical signs of EIA during pregnancy) tested positive for virus and antibodies. Anti-EIAV uninfected foals who consume colostrum from an infected mother acquire antibodies from colostrum [17]. Antibodies present in colostrum are usually undetectable until the age of six months. There are a few examples of viral transmission in colostrum or milk.

Diagnosis

Diagnosis is based on serologic tests. The clinical signs appear in acute phase are nonspecific. Diagnosis can be confirmed by the presence of antibodies against the virus in blood. Competitive enzyme-linked immunosorbent assay and Coggins test (infection AGID) are the two currently authorized laboratory techniques for determining EIAV (C-ELISA) [19]. States may have different policies about whether tests are approved. Both assays look for antibodies to the EIAV p26 core protein. In the early 1970s, the AGID or Coggins test methods were developed. It's a lot more delicate and particular [20]. This test is still the most extensively used laboratory test for diagnosing EIAV infection since it is at least 95% accurate. Within 45 days of infection, equines seroconvert to AGID. The Coggins test is done using a Petri dish with agar. The Centre well of the agar gel plate contains the soluble antigens of EIAV. The test sera, as well as negative or positive control sera, are placed in the remaining wells of the agar plate [21] Soluble reagent radially dissolves from the wells into the agar. Upon acquiring the required amounts of soluble anti-EIAV antibody and antigen, a precipitate line appears (opaque line) [22] Between the wells containing Dissolvable antigen and negative serum, no line of precipitation develops. Despite the high sensitivity and specificity of the AGID test, it has been documented to provide misleading findings [23]. Many stallions ultimately tested positive for EIAV infection, which were showing consistently negative or inconclusive results before. The C-ELISA for EIAV detection was only recently approved. There were 100 agreements between the AGID test and C-ELISA findings in a research work that compared sera of 420 horses (122 positives, 297 negatives, and 1 margin positive sample) [26].

Virus detection assays such as reverse transcription PCR re not used to diagnose EIA as they may not detect virus in carrier horses with very low viral loads. Animal inoculation test is highly sensitive for EIAV detection, but it is no longer in use due to logistic and economic reasons.

EIAV Control

The advancement of detecting technology is complementing EIAV Precautions and Control. Clinical complete diagnosis (CCD) was commonly employed in the early stages of EIAV because isolation was the only known control tool [24]. During 1974 and 1978, two serological approaches based on antibody Presence were developed to fight EIAV. Complement-fixation (CF) test was the first, which identified fixed complement when antibodies interacted with the specific antigen [25]. As an indicator system hemolysis was utilized to evaluate residual complement. Agar gel immunodiffusion (AGID) was another serological test. EIAV-positive horses were either euthanized or quarantined life long, according to the local rules [27]. To control EIAV infection, these two tests were used in China. Not only are CF, KCCD and AGID testing difficult to interpret and time-consuming, but some sick horses have been misdiagnosed since some horses test positive for only one of the two serological tests [28]. In 1979 enzyme-linked immunosorbent test (ELISA), being simple, fast, sensitive, and specific, resolved all these drawbacks. EIAV is difficult to eradicate alone with isolation measures. Because the concentrations of antibodies in infected horses aren't consistent, all the subclinical cases are impossible to detect [29]. A comprehensive control program was implemented, which included vaccination and isolation. This not only prevented the spread of EIAV by excluding the majority of afflicted animals but also allowed the majority of healthy equines to build immunity. These efforts hastened China's management and elimination of EIAV.

EIAV Live Weakened Vaccine Development

Live Weakened Pathogens vaccinations, which have been widely utilized to suppress pandemics of multiple contagious diseases such as measles, smallpox and polio for more than a century, have been the most effective vaccines [30]. EIAV has cost the Chinese economy a lot of money since the 1960s. Chinese scientists created an efficient Weakened-EIAV vaccination after almost two decades of hard labor. Equine vaccines were given not only against homologous EIAV strains, but also against a variety of other viral types [34]. The Chinese Weakened vaccine is the world's first large-scale lentiviral vaccine and serves as a paradigm for other lentivirus vaccine development. Chinese researchers isolated EIAVLN, a highly pathogenic strain identified from equines in Liaoning and attempted to infect the tissues or cell lines of other animals [31]. Except for Donkey, none of these attempts were successful. In Donkey, the virus survived 117 passages (EIAVDV117), with increased virulence rather than attenuation. There found an increasing reduction of virulence after 121 monthly passages of EIAVDV117 strain on jackass leukocyte (EIAVDLV121). EIAVDLV121 is a potent vaccination capable of eliciting defensive immunity [32]. For horses, this vaccination provided 85 protections while for jackass, it provided 100. Despite the fact that separating jackass leukocytes was expensive and time-consuming, scientists were able to effectively develop EIAVDLV121 in fetal jackass dermal cells after 13 passes. EIAVFDDV13 was shown to be equally effective as EIAVDLV12.

Immune Reaction Triggered through EIAV Vaccine

Horses who have been vaccinated can have a massive humoral immunological response. Agar Gel Immunodiffusion (AGID) antibodies Complement-fixation (CF) and may be found two weeks following inoculation [33]. The AGID and CF antibody seroconversion ratios were 100 percent three weeks after inoculation and have remained at 45 percent to this day [35]. The rate of AGID antibody and CF positivity began to diminish after 45 days. At 360 days, this rate had dropped to 22%. Neutralizing antibodies (Nab) were discovered after the AGID antibodies and CF had been found. 60 days after vaccination, Nab is discovered [36]. At day 180, Nab ranges peaked, and the Nab seroconversion rate jumped to 100% and stayed there for a long time with minimal change. This suggested that Nab may market vaccine-induced immunity as well. The immunological response to EIAV-precise cells was also studied [37]. After vaccination, CD8 + T cells proliferated and generated CTL response specific to equine infectious anemia virus. The vaccination elicited a strong cytokine response, including EIAV-specific cytokines. In addition, INF beta production and TLR3 activation induced by virus, were superior to molecular clone strains. Through a virulent horse infectious anaemia virus (EIAV), infection of equine monocyte-derived macrophages with a weakened EIAV generates a significant level of disease resistance [38].

Conclusion

Equine Infectious Anemia Virus (EIAV) poses significant challenges to the equine industry, affecting horses, donkeys, and mules. This article has provided an exploration of EIAV's characteristics as a lentivirus and discussed strategies for its control. EIAV, known as swamp fever, shares traits with retroviruses by establishing long-term infections and targeting the immune system.

These factors contribute to the difficulties in controlling its persistence and spread. By understanding EIAV's biology, transmission routes, clinical manifestations, and diagnostic methods, stakeholders such as veterinarians, horse owners, and regulatory authorities can make informed decisions to prevent its dissemination and safeguard equine populations. Effective control measures are crucial in managing EIAV. Strict regulations encompassing testing programs and biosecurity practices play a vital role in identifying infected animals, segregating them, and reducing transmission risks. Ongoing research on vaccine development and antiviral therapies offers promising avenues for more efficient control strategies in the future.By disseminating knowledge and raising awareness, this article aims to contribute to a broader understanding of EIAV and its control. Through the implementation of appropriate measures, we can strive to minimize the impact of this devastating virus on equine health and welfare.

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