Review

Diagnosis and Vaccination of Animals that are Affected by Foot and Mouth Disease
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Abstract
A virus that is both highly contagious and economically detrimental, foot and mouth disease primarily impacts animals possessing cloven hooves, including cattle, pigs, sheep, and goats. The FMD virus, responsible for causing foot and mouth disease, exists in seven distinct serotypes, complicating the challenges associated with prevention and control. The main ways that the virus spreads are through direct contact between susceptible and infected animals, contaminated food, and aerosolized viral particles. Owing to its ease of propagation, epidemics can quickly spread throughout cattle populations, resulting in significant financial losses. Fast and accurate diagnosis is crucial to halt the spread of FMD and safeguard the livestock industry. Clinical examination, serological testing, and virus isolation are examples of conventional diagnostic techniques. Several methods like Real-Time Quantitative PCR (RT-qPCR), RT-LAMP, Sandwich ELISA, Complement Fixation test (CFT) and PCR have become essential tools for FMD diagnosis in recent years. These techniques make it possible to identify the virus quickly and precisely, which facilitates the adoption of containment strategies and quick decision-making. Restrictions on migration, immunization, and the culling of diseased animals are all effective control measures. Nonetheless, the management of FMD continues to be based on prevention. Overcoming FMD in future involves vaccination, strict bio-security protocols on farms, monitoring, and outbreak readiness planning. Sustained investigation and attentiveness are essential to effectively address this persistent problem.

Keywords: Animals, foot and mouth disease, laboratory test, vaccination

Graphical Abstract
1. Introduction

Foot and Mouth Disease (FMD), is a viral infection affecting animals within the family Suidae, suborder Ruminantia, order Artiodactyla, and Camelus bactrianus, caused by the FMDV. This highly contagious disease impacts cloven-footed pigs and other animals, exhibiting symptoms such as anorexia, salivation, fever, and the development of vesicular blisters in the mouth, teats, and feet regions. Because FMD spreads across borders and is highly contagious, it is a livestock disease that needs to be reported [1]. The foot-and-mouth disease (FMD) is attributed to the foot-and-mouth disease virus, a highly contagious pathogen. This virus belongs to the genus Aphthovirus within the Picornaviridae family [2]. FMD impacts livestock characterized by cloven hooves, such as pigs, cattle, goats, sheep, and buffaloes [3].

The FMDV possesses a single-stranded positive-sense RNA genome, with an average length of 8,400 bases [3-5]. Four essential proteins, namely VP1, VP2, VP3, and VP4, encircle the RNA genome. As VP1, VP2, and VP3 are externally displayed while VP4 is situated internally within the capsid, the virus is believed to exhibit antigenic characteristics. The external exposure of VP1, VP2, and VP3 on the capsid surface facilitates interactions with the host's immune system, eliciting antibody responses and contributing to the antigenic properties of the FMD virus. The inside VP4, being shielded from immune recognition, may aid in immune evasion strategies [5]. Seven serotypes of FMDV (O, A, C, Asia 1, SAT 1, SAT 2, and SAT 3) have been identified in different regions around the world. There is not a uniform distribution of these serotypes worldwide. The typical distribution of SAT serotypes is confined to sub-Saharan Africa. The most widely distributed are types O and A, which can be found in Africa, Southern Asia, and Southern America. Type C seems to have limited to the India while Asia 1 is typically found exclusively in South Asia. Owing to the immunological variations among serotypes, an animal’s susceptibility to one infection does not no confer resistance against the remaining six types [6].

The primary method of infection in ruminants is intake of small particles, but infections can also be contracted by insemination with contaminating semen, consumption of contaminating feed, and through contaminated vaccines. While vaccination is a rare route of infection, each transmission method poses risks: ingestion may lead to systematic spread, insemination can introduce the virus into reproductive tissues, and contaminated feeds may initiate widespread outbreaks if not properly managed. Animals that are infected through the respiratory system experience viral multiplication in the pharynx, lungs, and other affected tissues prior to the onset of an acute illness. These areas develop a primary aphta, which multiplies throughout the course of three to eight days of incubation and induces viremia lasts for four to five days. Virus settles in the oral mucosa during viremia, especially in the tongue, skin, mammary gland, thyroid, interdigital areas and cause small vesicles. The virus then spread across the body, ending up in various locations including the mouth, throat, heart, foot, and oro-pharynx that are conducive to viral reproduction. These locations provide favorable conditions, such as susceptible cells and optimal environments, for the replication FMD virus. Symptoms of FMD encompass anorexia, fever, and lesions at mucous membranes, particularly in the foot, mouth and uterus. Lesions in uterus during foot and mouth disease can impact fertility and embryonic survival in livestock, posing reproductive challenges [7].

The distinctive blisters on the teats, coronary artery bands, oral and nasal mucosa, and interdigital areas are indicative of higher mortality associated with FMDV. This is linked to impaired feeding, mobility issues, and respiratory distress, intensifying the overall impact on the affected animals health and survival. Fever can still proceed to loss of appetite prior to vesicles occur. The clinical symptoms of FMD vary depending on the FMDV strain and serotype, which may include vesicular blisters on the teats or mammary glands in females, interdigital areas of the feet, and other hairless skin regions, accompanied by fever, shaking and drooling. The kind and variant of the virus, together with the species afflicted, all determine the morbidity and fatality rates of FMD. Morbidity is a serious issue that can get close to 100%. Although higher mortality rates are commonly seen in very young animals, primarily from acute
myocarditis, adult animal mortality is normally minimal (1 to 5 percent) [8]. Cattle typically exhibit fever, anorexia, shivering, and reduced milk yield for two to three days prior to the development of vesicular blisters on the cardiac band, mucous membranes, and between the fingers. The vesicles will burst in about a day, and it will take eight to fifteen days for them to heal. Cattle with high levels of saliva frequently have 80 percent lower milk production. Elevated stress levels can lead to a decrease in milk production, as stress hormones can interfere with the hormonal balance needed for lactation. Pigs that have serious blemishes are usually found on their feet, snout, udder, hock, and elbow. Pigs are more unlikely than cattle to salivate excessively, and their mouth blisters are milder than those seen in other animals. In sheep and goats, there are fewer clinical symptoms, vesicular blisters on the teats or mammary glands in females, inter digital area of the feet and other without hair areas of the skin, fever, shaking, lameness, and drooling. Although they might appear on the heel, mouth lesions are typically not visible [1].

Diagnostic symptoms, the epidemiology, pathologic injuries, and specialized identification methods such as culture separation, agar gel immune diffusion (AGID), hemagglutination tests, Immuno capture enzyme-linked immunosorbent Assay, and competitive ELISA are all important in the detection of viral diseases [9]. The reverse transcriptase polymerase chain reaction and the virus neutralization test are employed along with to the previously mentioned methods. In addition to lowering the disease's financial costs, controlling FMD is crucial for raising cattle productivity. Since FMD is a global disease that restricts the trading of farm livestock products from the nation, its control may potentially create new export opportunities [10]. This study is conducted to assess the precision and effectiveness of diverse diagnostic methods for the early detection of Foot and Mouth Disease in animals, including cattle, sheep, goats, pigs, and buffaloes. Additionally, it aims to identify shortcomings in current diagnostic approaches for potential enhancements. The study also assesses the efficiency, safety, and feasibility of various vaccination strategies in controlling and preventing Foot and Mouth Disease in livestock, such as cattle, sheep, goats, and pigs. Factors like vaccine types, delivery methods, and their influence on disease transmission and animal welfare are taken into consideration.

2. FMDV etiology and taxonomy

Other names for foot and mouth disease include aphthous fever, epizootic aphthae, infectious aphthous stomatitis, Aftosa in Italian and Spanish, fever aphthe use in French, and Mauland Klavenseuch in German. These are alternative names for FMD in different languages. It was initially discovered in South Africa in 1780 after an outbreak close to Verona, Italy, in 1546. Though it was not well recognized till the late 1800s, the disease presented a serious danger to the cattle business in earlier decades due to the culling of infected animals and trade restrictions. The twentieth century saw the discovery of much knowledge regarding FMDV, notably its genetic makeup and physical makeup, which, when examined by crystallography using X-rays, seems to be three-dimensional [7].

Loffler and Frosch, two scientists, discovered FMDV as the configurable viral cause of animal sickness in 1897. This discovery laid the foundation for understanding and controlling this highly contagious viral disease in livestock [11]. The International Committee on Virus Taxonomy then made the initial identification of it in 1963. It belongs to the Picornaviridae family and the Aphthovirus genus. The term Picornaviridae originates from the Latin words "Pico" signifying little, and "rna" denoting RNA, elucidating the nature and size of the virus's genome. 'Aphthovirus' is the genus name for the vesicular lesions that show in the mouths and feet of organisms [12]. The FMD virus component, also known as the virion, consists of a non-enveloped icosahedral protein coat (capsid) and the genetic material [13]. The virus's outer layer, or capsid, is made up of 60 capsomers, each of which has four basic polypeptides. The molecular weight of FMDV's single-stranded positive-sense RNA genome varies from 7.2 to 8.4 kb, and the sedimentation coefficient of the viral particles, as a whole, is 146S [14].
3. Incidence of the disorder
The seven distinct serotypes of the virus A, O, C, SAT1, SAT2, SAT3, and Asia1 are present globally, with a higher prevalence observed in Asia, Africa, and the Middle East. Nonetheless, there is no FMD in Australia, New Zealand, Japan, or certain countries. Out of the seven FMDV serotypes, serotype O is widely disseminated worldwide, whereas serotype C has fewer cases, with the most recent one being in Kenya in 2005 [15]. Around the world, FMD still affects over 100 countries, and it's estimated that the illness still exists in almost two thirds of the world's cattle population [11].

4. The host species
FMD is extremely transmissible illness. Animals possessing cloven hooves, including pigs, cattle, goats, sheep, and buffalo, are vulnerable to infection. Wild animals prone to foot-and-mouth disease (FMD) include antelope, wild pigs, elephants, camels, and deer. With certain strains, old world camels may exhibit resistant to the innate infection. Camelids from South America, such as alpacas and llamas, are rather vulnerable. Cattle can contract the same strain of FMD that infects deer and wild pigs. The inter-species transmission potential is significant, and various strains of the virus can infect multiple species. However, susceptibility may depend on the specific strain involved, as different strains may have varying effects on different animal species. In controlled laboratory studies, experiments have shown that armadillos, rats, mice, and guinea pigs can all become infected. Despite the fact that horses, dogs, and cats may have the virus in their hair; they are not prone to FMD. These species are generally not susceptible to the disease due to variations in their cellular receptors or immune responses [16].

5. Pathogenesis and the transmission mechanisms
The primary site of disease is the respiratory tract, where the virus initially replicates in the pharyngeal mucous membrane. Afterward, it travels to second replication sites that include the mouth and its surrounding tissues, the mammary glands, and the feet, with the aid of blood and lymphatic flow. This sequence of events is typical for the course of FMD in infected animals. In cattle, the virus can be identified up to two years after infection, while in sheep, detection is possible up to six months post-infection. The detection period of the virus varies among different species and can be influenced by various
The infected animals transmitted the virus through all of their bodily fluids and excretions, comprising blood, urine, feces, milk, saliva, nasal and lachrymal fluid, and air, before the infections showed any clinical signs (Figure 2). The likelihood of transmission of virus both within and between farms is increased when the virus is present prior to the beginning of the clinical symptoms of the disease due to a phenomenon known as subclinical shedding.

Infected animals may discharge the virus for years afterwards re-infection, and those with the infection can spread the virus for a few days until symptoms show up. A susceptible animal can become infected with just a few of infectious particles. The primary method of ruminant infestation is by inhaling of the airborne virus, which based on the direction that and the velocity of the wind, can travel great distances. Because of its extremely basic form, the virus may spread easily through the air. Various potential infection mechanisms involve the consumption of contaminated food, direct inoculation of susceptible animals, and infection through skin lesions [11].

The most common method of foot-and-mouth disease (FMD) transmission is direct contact between infected and susceptible animals. This can happen through the mechanical transmission of the virus from infected to susceptible animals. Virus enters via cuts, abrasions, or mucosal membranes. Infection spread by means of the accumulation of tiny particles or droplet-nuclei (aerosols) in the respiratory system of its recipient animals. It is swiftly spread from one animal to another. All animals in a hamlet or farm become infected when an animal arrives. Instantly, neighboring countries that are experiencing an epidemic of FMD take precautions. Trade limitations are imposed by other countries as preventive measures. There have been notable advancements in the knowledge of FMD epidemiology during the past 15 years.

The mobility of animals affects spread. Keeping sick animals alongside healthy ones leads to infection. The most frequent means of viral transmission, in addition to animal migration, is infected animal production. Animals can contract the virus from one another through their saliva, wool, skin, and hair. Additionally, shoes worn by individuals entering barns, tyres from moving cars, infected grasses, fodder, and seeds can all spread the virus. FMD is an extremely contagious disease that can propagate through various routes. Sick animals excrete substantial quantities of the virus. Products of animal include meat, milk, and other tissues that have been contaminated and live animals that are diseased are among the most typical means of transmission. People, vehicles, equipment, hay or bedding infected with infected animals' faeces or urine, among other things, can all be indirectly carriers of the disease. Large-scale animal migrations of any kind caused by intense animal husbandry techniques are particularly hazardous [17].

6. Clinical symptoms

A preliminary diagnosis of a disease is made using the clinical symptoms of that condition (Figure 2). To distinguish between illnesses with comparable clinical features, it is crucial to study the differential diagnosis. There is variation among species and a range of clinical symptoms for FMD [18]. Under natural settings, the length of the time of incubation differs according to the type of virus, the host's sensitivity, the amount of being exposed, and the point of entrance. In the majority of instances, it can linger everywhere from two to fourteen days. Since the bovine species is an indicator host, signs are most commonly observed in cattle. The main symptoms of FMDV include vesicular blisters on the teats or mammary glands in females, the interdigital area of the feet, and other hairless skin regions, accompanied by fever, drooling and shaking [11]. Following infection, the virus persists in the esophageal-pharyngeal tract for 28 days or more, even in animals that do not display clinical symptoms [19].

7. Rates of morbidity and mortality

In animals with FMD infection, the morbidity rate is 100%. However, 2% of adult animals (have age two years or older) and 20% of young animals (have age below 2 year) die (Figure 2). The morbidity rate is influenced by immune level, species, and sex. Immunity to the virus's serotype leads to self-recovery. The primary cause of FMD in epidemic locations is usually a single virus form, to which immunity is restricted towards a particular serotype and does not develop across other serotypes.
The mortality rate of 20-75% in lambs and suckling pigs is typically observed under conditions of inadequate nutrition, poor management practices, and insufficient veterinary care. Factors such as maternal health, disease prevention, and environmental conditions can significantly impact the survival rates of these young animals. Animal mortality is higher at younger ages, such as less than four weeks, but it rapidly declines as an animal gets older (more than four weeks). In both endemic and developed countries, the majority of animal deaths are caused by slaughter regulations that include all susceptible animals throughout a pandemic [21].

8. Diagnosis

Clinical indicators are the primary basis for most FMD diagnoses established in the clinic; however, testing in the laboratory is also crucial, particularly to differentiate FMDV from other vesicular illnesses that share similar clinical characteristics. Laboratory diagnosis can be performed using a variety of materials, including blood (used to detect the presence of pathogens or antibodies, providing information on systemic infections), sperm (for studying reproductive diseases), serum (useful for detecting antibodies), vesicular fluids (allows direct examination of the fluid for pathogens), epithelial samples (to identify infections affecting the skin or mucous membranes), oro-pharyngeal fluid and throat swabs (useful for diagnosing respiratory or oral infections, aiding in the identification of pathogens in these areas). Epithelial tissue samples are the most desired kind of specimen [18]. Various methods can be utilized to detect the virus, including reverse transcription-polymerase chain reaction (RT-PCR), enzyme-linked immunosorbent assay (ELISA), multiplex polymerase chain reaction (mPCR), complement fixation test (CFT), and virus isolation [23].

8.1. Virus separation

ELISA can be employed to detect FMDV antigen in vesicular material. If the virus quantity is insufficient for ELISA detection, it is necessary to cultivate the virus on a suitable cell culture. FMD viral samples are introduced into primary pig kidney cell cultures, incubated at 37°C, and observed for cytopathic effect within 24 to 48 hours after infection. A limitation of virus isolation techniques is their in capacity to cultivate viruses on specific cell types. Therefore, the absence of virus development does not necessarily indicate the absence of the virus in the collected cell sample. Additional disadvantages of this approach include contaminating of cell cultures, ELISA's confirmation of virus growth, and the need to regularly replenish cell supplies [4].

8.2. Sandwich ELISA

This method is quick and easy to use. It is the main test used to diagnose FMD. To identify FMDV structural proteins, the assay uses poly clonal antibodies specific to a certain serotype that are raised in guinea pigs and rabbits. In terms of FMDV identification, the test yielded 80% sensitivity and 100% specificity [24] (Table 1).

8.3. Complement fixation test

The CFT is a technique that dates back to the early days of clinical virology. The antigen-antibody complex is attacked by the complement. The complement binds when the Ag-Ab complex is present. Red blood cells (RBCs) from sensitized sheep were employed as an indicator. Hemolysis-free outcomes are linked to positive outcomes. The CFT is labor-intensive and insensitive, despite its convenience and inexpensive material cost [25] (Table 1).

8.4. RT-PCR

By replicating FMDV genomic sequence in diagnostic samples using generic or serotype-specific primers, reverse transcription PCR could be employed for identification [7]. The serotype-specific forward primers are derived from the hyper-variable regions of the capsid coding gene (VP1/1D), while the universal reverse primer (BES-VP1R) used to amplify and identify all FMDV serotypes from specimens is obtained from the conserved 2B region alignment of VP1 genomic sequences of serotypes accessed from the GenBank nucleotide database. Because the RT-PCR procedure is automated, more diagnostic options are available. Real-time RT-PCR in comparison to conventional RT-PCR offers a number of benefits. It can be carried out in a closed one-tube system, is faster and more delicate, and eliminates the possibility of the cross-contamination throughout the preparation of samples for post-
PCR analysis. Real-time detection of PCR product formation can be accomplished by using dual-labeled hydrolysis (Taq Man) probes, which are made up of complementary sequences inside the target gene [26].

8.5. RT-LAMP

Out of 50 samples, 38 tested positive using RT-LAMP, with identified serotypes being A (15/50), O (15/50), and Asia-1 (8/50). An exceptionally sensitive molecular analysis for the rapid and straightforward detection of FMDV, isothermal nucleic acid amplification technique operates at a constant temperature and doesn't require a cycler like PCR [27]. The RT-LAMP successfully amplified the 3D polymerase gene target sequences of serotypes A, O, and Asia-1 at 60°C over 15–60 minutes, resulting in lengths of 199, 209, and 187 base pairs, respectively. Reverse transcriptase loop-mediated isothermal amplification (RT-LAMP) was developed by incorporating both generic and serotype-specific genes in a single tube. This test is simple to run and takes about 60 minutes to detect FMDV at the serotype level. Furthermore, it is comparable to real-time PCR and reverse transcriptase PCR in terms of sensitivity and specificity [28].

8.6. Multiplex polymerase chain reaction

The method is faster and more accurate than traditional viral isolation (Table 1). Assays were designed to target the conserved 3D region and 5'UTR region of the FMD virus. Following that, VP1 region-directed multiplex PCR (mPCR) was created and used to distinguish between the serotypes of FMDV [10]. Two sets of primers were employed in this assay; the initial set targeted the 1D region, and the subsequent set went straight to the 2B region. The approach successfully identified the FMD serotype, detecting unique-sized products 249, 376, and 537 bp corresponding to serotypes O, A, and Asia1. The minimal detection limit for PCR has been established at $1 \times 10^3$ TCID50/mL for serotypes O, A, and Asia1 [29].

9. Immunization:

9.1. Vaccines that are not active

Most of the currently employed commercial vaccines for FMD are inactivated vaccines produced by treating noninfectious particles with binary ethyleneimine. There are three types of vaccines: monovalent, bivalent, and multivalent. These are aluminum-based, oil-emulsion, or water-based inactivated vaccines. This form of vaccination can be stored in liquid nitrogen for an extended duration after concentrating the FMDV antigen. The inactivated vaccine can undergo additional concentration to reach six times the 50% protective dose, resulting in a higher-potency effect, or it can be conventionally concentrated to three times the 50% protective dose. The primary factors of antigen concentration and amount are the type of antigen used, the intended usage, and the supplier.

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<tr>
<th>Table 1</th>
<th>Diagnosis test for FMD in animals [30, 31, 32].</th>
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<tr>
<td>Diagnosis test</td>
<td>Sensitivity</td>
</tr>
<tr>
<td>Enzyme linked Immunosorbant Assay</td>
<td>Eighty percent</td>
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<tr>
<td>Multiplex polymerase chain reaction</td>
<td>TCID 50 mL-1 minimal limit of detection of 1x 10-1</td>
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<tr>
<td>Virus isolation</td>
<td>NA</td>
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<tr>
<td>TaqMan Real time Polymerase Chain reaction</td>
<td>TCID 50 mL-1 minimal limit of detection of 1x 10-1</td>
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According to most regulations and recommendations, an animal should have two main injections separated by one month, then booster shots each year or every 4 to 6 months for specimens who are 2 years or older. The primary limitations of the presently employed inactivated vaccines include the requirement for a bio-safety level III facility, a meticulously regulated laboratory, and the inclusion of multiple serotypes, potentially imposing strain on the animal's immune system. Additionally, because FMDV is sensitive to heat, the vaccines must be stored at a low temperature. Unluckily, the majority of these vaccinations only guards against generalization (the efficacy of the vaccine in preventing the spread of the disease within an infected animal) and fail to prevent initial infection; it is likely that over half of vaccinated animals will develop into carriers. The only tests that can differentiate between vaccinated and sick animals are DIVA assays [33]. Using multiple adjuvants, a BEI-inactivated a virulent FMDV serves as the current marker-inactivated vaccine. It has inherent DIVA NS markers seen in the 3AB and Lpro proteins [34]. Mice receiving the inactivated foot and mouth disease vaccines exhibit protective effects, and the humoral and cellular immune responses are heightened when an adjuvant injection of the chemokine CCL20 plasmid is administered before immunization [35].

9.2. Attenuated live vaccination:
FMDV can be attenuated in two ways: conventionally, by passing through cultivated cells; or, in a novel way, by optimizing or deleting certain genes using molecular virology techniques. Cattle have been immunized with mouse-attenuated live FMD vaccines made using BHK-21 cells. After that, in 1969, certain alterations and cloning in BHK-21 cells were accomplished. With one notable exception—one immunized animal had a fever—it was shown in one investigation that the live attenuated FMD vaccination shielded recipients from acquiring lesions. It is thought that the recently developed attenuated FMD vaccinations are more stable than earlier strains. Compared to traditional ones, they also bear a lower chance of reverting to virulence. Better live attenuated vaccines will only be possible with thorough research into virulence genes. The viral leader protease is one of these virulence factors; it prevents the host animal's innate immunity and hinders the induction of beta interferon mRNA. It's been demonstrated that the virus becomes incurable with deletion of the protease gene, a virulent in cattle and pigs. In cattle, this gene likewise attenuates due to an in-frame change. Following aerosol inhalation, both the leaderless and in-frame vaccines do not induce viremia or clinical symptoms; however, the leaderless variation became less widespread than the in-frame form. It has also been noted that leaderless mutants partially revert to virulence [36].

9.3. Vaccinations using live viral vectors
Viral vectors can be used to deliver immunogenic viral structural proteins that, when expressed in vector-infected cells, elicit a humoral and cell-mediated immune response. Viral vectors carrying the sequence of interest include the vaccinia virus, fowlpox virus, pseudorabies virus, alphaviruses, replication-defective human adenovirus vectors, and Semliki Forest virus. Mice that were vaccinated exhibited a robust level of specific humoral and cellular immunity against a recombinant Sendai virus containing the FMDV P1 gene [37]. The bamboo mosaic virus is a recombinant virus expressing FMDV epitopes and has been utilized as a viral vector to confer protective immunity in swine. Calves that were vaccinated against virulent infectious bovine rhinotracheitis virus (IBRV) were able to withstand challenges from the virus by developing protective levels of anti-foot and mouth disease antibodies in the humoral response. While a bovine enterovirus expressing an FMDV epitope was engineered, it has not undergone testing in a challenge experiment. In contrast, a recombinant bovine herpesvirus-1 carrying the FMDV VP1 gene induced a notable level of neutralizing antibodies in a rabbit model [38].

10. Preventative measures and controlling
To stop animal diseases from spreading further, disease control and preventative techniques are applied [18]. In veterinary science, those procedures should always make sense. Depending on the severity of the disease and the financial and
technological resources of each nation, different controlling measures may be used [39]. Limiting animal movement or immunizing against viruses is two ways to control the spread of viruses in animals. This involves lowering the animal's susceptibility to the virus and its chance of infection. Considering socioeconomic factors, cost-effective management is a crucial consideration in veterinary science [1,40].

11. Conclusion:
In Conclusion, FMD in animals is a complex and highly contagious viral infection that significantly affects both global trade and animal health. Important details of FMD, such as its mechanisms of transmission and diagnostic techniques, have been clarified by this review. First off, knowing the different methods that FMD can spread, including direct touch, fomites, and aerosols, emphasizes how crucial it is to have strict biosecurity protocols in place as well as vaccination campaigns in order to stop and manage epidemics. To stop the disease from spreading internationally, rigorous trade rules and international cooperation are necessary. Second, improvements in diagnostic procedures, such as molecular approaches and serological testing, have significantly enhanced our capacity to distinguish between distinct strains of the FMD virus. For the purpose of managing and containing outbreaks, diagnosis accuracy and timeliness are critical. The impact of FMD on animal populations and the economies that depend on them can be reduced by improving our understanding of transmission patterns and developing diagnostic tools. In the end, effective FMD control and eradication require a multidisciplinary strategy combining veterinary science, epidemiology, and international cooperation. The future perspective for FMD in animals involves implementing strict biosecurity measures, vaccination, and prompt detection and control.

Data Availability statement
The data supported this study are available on request from the corresponding author.

Conflicts of Interest
Authors declare that, they have no conflict of interest.

Author Contributions
AA, SM, KA, DM and HK: Investigation, Methodology, Writing - Original Draft; IQ: Conceptualization, Supervision; Review and Editing.

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