

Review on Foot and Mouth Disease and the Status in Ethiopia

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Abstract: Foot-and-mouth disease (FMD) is an acute systemic infectious disease of all cloven-hoofed domestic and wild animals. It is caused by Aphthous virus which belongs to genus Aphthovirus of family Picornaviridae. There are Seven different serotypes (O, A, C, Southern African Territories (SAT) 1, SAT2, SAT3 and Asia1) and numerous variants of FMDV have been identified. The disease is characterized by a clinical signs of vesicles, which are similar to blisters, that quickly pop and cause erosions in the mouth or on the feet, resulting in excessive salivation or lameness. The disease is widespread around the world; it is endemic in Asia, Africa and South America however developed countries such as America, New Zealand, Australia and some European nations are considered free of FMD. Foot and Mouth Disease virus can easily transmit by ingestion, aerosol, direct and indirect contact as well as spread by traveling through wind. The control and prevention measures for FMD, like other trans-boundary animal diseases, include: surveillance, animal movement control, vaccination, disinfection, quarantine and slaughter mass. FMD is endemic and known for its wider distribution in Ethiopia, . It is found in many parts of Ethiopia; including: Arisi Zone, Dire Dawa region, North wollo, round Debre zeit and Addis Ababa.

Key words: Foot and Mouth Disease • FMDV • Contagious • Serotypes

INTRODUCTION

Foot-and-mouth disease (FMD) is a highly contagious extensive and economically devastating vesicular disease affecting cloven hoofed animals epizootic disease of cloven footed animals such as pigs, sheep, goat, cattles and other ruminants [1]. Foot and Mouth Disease is caused by Aphthous virus known as foot and mouth disease virus. Which is a small, non-enveloped, positive-sense, single stranded RNA (8.4 kb in length) virus belonging to the genus Aphthovirus of the family Picornaviridae [2].

A report that was first written description of FMD probably occurred in 1514, when Fracastorius refer to a similar disease of cattle in Italy. Almost 400 years later, in 1897, Loeffler and Frosch demonstrated that a filterable agent caused FMD [3]. It is recognized as a significant epidemic disease threatening the cattle industry since the sixteenth century and till date it is a major global animal health problem. The history of FMD may be traced to era of Hieronymus Fracastorius, a monk who described a disease outbreak in 1546 A.D. that occurred in cattle near Verona, Italy [4].

FMD is the most contagious Trans boundary animal disease (TAD) affecting cloven hoofed animals of domesticated and wildlife. Among species of the domesticated animals; cattle, sheep, goats, pigs and buffalo are susceptible. Foot and Mouth Disease Virus is an RNA virus with seven antigenically different serotypes such as A, O, C, Southern African Territories' (SAT) 1, SAT2, SAT3 and Asia1 as well as over 60 subtypes. Foot and mouth disease is still wide spread throughout the world, particularly in Asia, Africa and the Middle East. Even though the disease can occur in any countries; Japan, New Zealand, Australia and some other countries are FMD free countries [5].

Foot and Mouth virus is resistant to the environment; it can survive outside a host for a month or more in moist soil or in meat from the carcass of infected animals. What is particularly dangerous is that FMD travels by air within water droplets. This virus can blowout through wind some 60 kilometres over land and 250 kilometres over water [6].

Foot and Mouth Disease is characterized by fever, loss of appetite, salivation and vesicular eruptions on the feet, mouth and teats. It is a list A disease according to

OIE disease classifications [7]. FMD is highly contagious which is easily spread to animals nearly 100 %. However it comprises low mortality rates that are below 5%, but even so it is considered the most important disease of farm animals since it causes huge losses in terms of livestock productivity and trade. Although FMDV rarely causes death in adult animals, the virus can cause severe lesion in the myocardium of young animals, leading to high mortality rates in those young animal [8, 9]. This disease has been controlled successfully in many countries with the strategies such as mandatory vaccination of susceptible animals and slaughtering of infected animals. However, no country has been considered safe due to the highly contagious nature of the virus and the intensified international trade of animal or animal products [10].

The present paper discusses on FMD virus and the disease it causes, method of transmission, pathogenesis, clinical feature, diagnostic techniques, control and prevention methods and the status in Ethiopia.

Etiology and Structure of the Virus: Foot and mouth disease virus (FMDV) is a member of the genus *Aphthovirus* in the family Picornaviridae. There are seven major viral serotypes: O, A, C, SAT 1, SAT 2, SAT 3 and Asia 1. Serotype O is the most common serotype worldwide. It is responsible for a pan-Asian epidemic that began in 1990 and has affected many countries throughout the world. Other serotypes also cause serious outbreaks; however, serotype C is uncommon and has not been reported since 2004 [11]. Moreover, there can be great changes in antigenicity between developing serotypes; virulence also change dramatically. There are also biotypical strains which become adapted to particular animal species and then infect other species only with difficulty [12].

By electron microscopy, the FMD virion appears to be a round particle with a smooth surface and a diameter of about 25 nm. The viral serotypes are determined by the fine structure of viral capsid using X-ray crystallographic techniques. The structural proteins, VP1 to -3, fold into an eight-stranded wedge-shaped barrel which fit together to form the majority of the capsid structure [3]. The viral particle, or virion, contains a single-stranded RNA of positive polarity, approximately 8500 nucleotides long [13]. The RNA includes three separate parts i.e. the 5' untranslated region (5' UTR), a long coding region and the 3' untranslated region (3' UTR). A small protein (24 or 25 residues long), termed VPg, which is encoded by the 3B

portion of the viral genome region, is covalently linked to the 5' end of the genome. The 5' UTR is about 1300 nt in length [14]

Host Species: FMD is extremely contagious disease which affects all cloven footed animals, including cattle, pigs, sheep, goats and buffalo. Wild animals which are cloven hoofed such as deer, antelope, wild pigs, elephant, giraffe and camelids are susceptible to FMD. Old World camels may be resistant to natural infection with some strains and South American camelids such as alpacas and llamas are mildly susceptible, but are probably of no epidemiologic significance. African buffalo are the only wildlife species to play a significant role in the epidemiology of FMD. The Strains of FMD virus that infects wild pigs and deer can also infect cattle. Capybaras and possibly hedgehogs are susceptible. Rats, mice, guinea pigs and armadillos can be infected experimentally [15]. Although Horses, dogs and cats can spread the virus by carrying it on their hair, they are not susceptible for the disease [16].

Geographical Distribution of FMD: The disease has been present in almost every part of the world where livestock are kept. More than 100 countries are still affected by FMD worldwide and distribution of the disease roughly reflects economic development [14]. Although the FMDV found every part of the world, the serotypes of FMDV are not distributed uniformly around the world. The serotype O, A and C viruses have had the widest distribution and have been responsible for outbreaks in Europe, America, Asia and Africa. However, the last reported outbreak due to serotype C FMDV was in Ethiopia during 2005 and so serotype C viruses may no longer exist outside of laboratories. The SAT1-3 viruses are normally restricted to sub-Saharan Africa. However, there have been some limited outbreaks due to SAT1 viruses in the Middle East between 1962–1965 and 1969–1970 and then in Greece in 1962 [14].

While, Foot and mouth disease is prevalent in parts of Asia, Africa, the Middle East and South America, Industrialized countries such as North and Central America, New Zealand, Australia, Greenland, Iceland and Western Europe had eradicated FMDV from their country. Western Europe was affected by some recent outbreaks (Eradication was successful), but FMD has not been reported in North America for more than 60 years. The last U.S. outbreak occurred in 1929, while Canada and Mexico have been FMD-free since 1952-1953 [11].

Methods of Transmission: The predominant route of FMD virus infection is respiratory tract, although ingestion of contaminated food or direct inoculation also both highly effective in transmitting infection [17]. FMDV can be Transmitted through Direct contact between infected and susceptible animals, by inspiration of aerosols, by Consumption (Primarily by pigs) of untreated contaminated meat products (Swill feeding), by mechanical carriage through humans or vehicles, on fomites through animal products by Artificial insemination with contaminated semen. Most of time Young calves acquire the disease from their mother through ingestion of milk. Airborne transmission can occur, especially temperate zones (Up to 60 km overland and 300 km by sea), Humans can harbour FMDV in their respiratory tract for 24–48 hours, leading to the common practice of 3-5 days of personal quarantine for personnel exposed in research facilities During an active outbreak, this may be reduced to an overnight period of time after thorough shower and shampoo, change of clothing and expectoration [18]. The Virus may be recovered from all body secretion (Tears, nasal, saliva, urine, feces, milk, vaginal, semen and the placenta of aborted foetus). The survival of virus in such excretions depends up on temperature, PH and humidity [17].

Pathogenesis: The predominant entrance of virus is most commonly through the upper respiratory tract by inspiration of infected aerosols, but infection may also occur through a skin injury. The virus begins to replicate in the upper respiratory tract (Pharynx), mucosa or skin [1]. The epithelial cells of animal are the cells which are preferred by the FMD virus, causing ballooning degeneration and vesicle formation. Thereafter, the FMD virus enters the bloodstream, muscles, lymph glands, bone marrow and organs. In young animals the virus invades the myocardial cells of the developing heart, causing necrosis and death of the affected cells and consequent heart failure, which can be seen as white scars post mortem (Tiger heart). In the carrier animal, the virus can be found in the basal layer of the stratified squamous epithelium of the dorsal soft palate. It is not clear why it is non-lytic in these animals. Viraemia lasts 3 to 4 days, detectable 48 h before the onset of clinical signs [19].

The presence of virus in blood results in widespread distribution of FMDV to various tissues and organs, including epithelia, visceral organs and across the blood–brain barrier. The virus is excreted during Viremia,

all excretions and secretions may contain virus during the viremia and clinical phases of disease. The presence of virus in blood is characterized by the onset of the FMD marks of vesiculation and erosion of various epithelial sites including the, feet, mouth, prepuce, teats and pillars of the rumen the animal recovers after the development of specific antibodies in the blood (IgM and IgG) and secretions (IgA), detectable from 10 days after infection. [1, 19]

Clinical Signs: The period between the infection of an animal by the virus and showing the clinical signs takes between 2 and 14 days, which depends on the route of infection, the dose and the strain of virus and the susceptibility of the host. Following an initial pyrexia in the region of 40°C (104°F), lasting one or two days, a variable number of vesicles develop on the tongue, hard palate, dental pad, lips, muzzle, coronary band and interdigital space. Vesicles may also be seen on the teats, particularly of lactating cows [20]. An animal that is acutely affected by the disease may show a signs such as stamp their feet, drool their saliva profusely and prefer to lie down. Ruptured oral vesicles can coalesce and form erosions but heal rapidly, roughly 11 days after vesicle formation. A vesicle that is found on the Feet takes longer to heal and are susceptible to secondary bacterial infection leading to chronic lameness. The involvement of bacteria on the vesicles of udder result Secondary bacterial mastitis this is followed by the animal to be resistant to milking. After vesicular disease develops, cattle quickly lose condition and milk yield, which can persist chronically [21]. Young calves may die before the development of vesicles because of a predilection by the virus to invade and destroy the cells of the developing heart muscle [20].

Post Mortem Lesions: The formation of fluid-filled vesicles or bullae is one of the characteristics of FMD, in which the vesicles can be either in single or multiple varying in size from 2-10 mm in diameter [22]. Lesions may be seen in any stage of development from a small white area to a fluid filled blister, sometimes joining with adjacent lesions. The vesicles rupture, leaving a red eroded area, which is then covered with a gray fibrinous coating. This coating becomes yellow, brown, or green then is replaced by new epithelium with a line of demarcation that gradually fades. Occasionally the fluid may escape through the epidermis instead of forming a vesicle. These “Dry” lesions appear necrotic instead of

vesicular. “Dry” lesions are more common in the pig oral cavity. Lesions at the coronary band progress similarly: the skin and hoof separate and, as healing occurs, a line showing evidence of coronitis appears on the hoof [23]. Involvement of heart in the form of cardiac degeneration and necrosis which mostly appear as gray or yellow streaks in the myocardium (“Tiger heart” lesions) are observed in young calves [22].

Mortality and Morbidity: The FMD virus is highly contagious disease which is easily spread to animals and nearly 100% of exposed animals become infected. On the other hand mortality rate in adults is typically 1–5%. Younger animals are more likely to die from FMD, often due to inflammation of the heart. The virus can spread from infected animals, contaminated animal feed or water, contaminated shoes or clothing and contaminated vehicles or farm equipment.

Barry B and Richard [16] and USDA APHIS Veterinary Services [24] in a flock of sheep, when they are kept under intensive condition indoors may result high morbidity, whereas sheep that are kept under low intensive condition outside may have a much lower morbidity. Morbidity in susceptible wildlife is quite variable from high to very low, depending on the foot and mouth disease virus subtype and the species involved [17].

The occurrence of variation in the morbidity rate may depend on species, age, sex as well as the status of the immunity. Self-recovery in the animals is the result of immunity against the infecting serotype of the virus. Frequently, foot and mouth disease occurs due to one type of virus and development of immunity also remains confined against specific serotype, thus no immunity develops to other serotypes, a reason behind occurrence of the disease in the endemic areas [25]. Mortality in suckling pigs and lambs ranges from 20-75% in most extreme cases and it is highly age dependent. In animals infected under 4 weeks of age, the mortality is high and decrease rapidly as animals get older (>4weeks). During outbreaks in endemic and developed countries, most deaths are due to a slaughter policy that usually involves all susceptible animals and herds in contact with or within a certain radius of infected herds [26]

Control and Prevention: Among animal infectious diseases, FMD is one of the most difficult infectious disease which is tough to control. Control of foot and mouth disease is difficult due to its highly contagious

nature, viral stability, multiple hosts, multiple antigenic types and sub types, short term immunity and Because of the disease occurs in many parts of the world, there is always a chance of its accidental introduction into an unaffected country. The type of control strategies applied in a country depends on the goal of the control programme. The control strategies varies from country to country based on their epidemiological condition, importance of livestock sector in the national economy and economic capability of the country to invest in control strategies [12, 27]. Countries in different regions of world adopt FMD control policies depending on the epidemiology of disease. Slaughtering infected animals is one of the best ways to control FMD in developed countries. In FMD free countries, slaughter of all infected and susceptible in contact animals, quarantine of infected animals, strict animal and animal product import regulation and animal movement restrictions are practiced. FMD endemic countries do not follow stamping out policy and use only vaccination as a measure of control. Vaccination for FMD is possible, but because the virus mutates quickly, vaccination isn't always effective [28, 29]. Protection of free zones by border animal movement control and surveillance, Quarantine measures Slaughter of infected, recovered and FMD-susceptible contact animals, Cleaning and disinfection of premises and all infected material, such as implements, cars and Clothes as well as Proper disposal of carcasses, bedding and contaminated animal products in the infected area [15].

Mass-Slaughter or ‘De-Population’ of Infected/ Susceptible Animals: The killing practice of the animals which are affected and those suspected of being affected by FMD in the herd and, those in other herds which have been exposed to infection by direct animal to animal contact, or by indirect contact of a kind likely to cause the transmission of the causal pathogen. All susceptible animals, vaccinated or unvaccinated, on an infected premises should be killed and their carcasses destroyed by burning or burial, or by any other method which will eliminate the spread of infection through the carcasses or products of the animals killed [30]. Here, animals on infected premises and susceptible animals identified as ‘dangerous contacts’ (e.g. contiguous premises, or, animals within a certain radius), are culled humanely and the carcasses incinerated, rendered, or disposed of within licensed commercial landfill sites. In the 2001 UK outbreak, for example, some 6, 134, 078 animals (Cattle, sheep, pigs, goats, deer etc.) were slaughtered. These

measures should be accompanied by livestock movement bans, restricting public access, comprehensive cleansing and disinfection and heightened biosecurity measures at the national level [31].

Vaccination: Foot-and-mouth disease can be controlled by using vaccination, which is practiced in many parts of the world. Yet it is a constant drain on resources, since effective vaccination requires a high proportion of animals to be vaccinated two or more times per year. The highly infectious nature of FMD means that gaps in coverage emerge as disease outbreaks [32].

Most of time FMD is occurred in winter season and for this reason, vaccination is advised in this season. It is given to all breeders and stock over 12 weeks of age and then again every 4 months thereafter. Serotype O FMD virus is endemic in HK and vaccination is carried out routinely with a killed serotype O FMD virus. Protection is short-lived, lasting only about 4-6 months and vaccination breakdowns often occur if the disease virus challenge is very high or if disease virus strain is dissimilar to the strain used in the vaccine. There are many different strains within serotype O and careful selection of the correct strain for incorporation into the vaccine is essential to ensure the effectiveness of the vaccine [16]. Vaccination against one serotype of FMDV does not cross-protect against the other serotypes. Within a serotype, vaccination against one strain may not cross-protect against other strains, depending on the antigenic similarity of the strains [24].

Disinfection: Chemicals which are used commonly house hold bleach is effective disinfectant of FMD virus at concentration of 3 %. It can be used as on infected properties, but not a good choice for disinfection of equipment and foot paths. Vinegar at 4-5 % dilution also kills the virus. Lye can be used at 2 % dilution, but this is highly caustic. New disinfectants like Virkos S (Per oxygen molecule/ organic acid/ surfactant combination) appear to have wider spectrum of activity against many germs including FMD virus. Another compound based on per oxy acetic acid (Oxy-sept333), now Environmental Protection Agency (EPA) approved for FMD virus [28].

Diagnosis

Clinical Diagnosis: Identifications of FMD by clinical signs by means of close physical examination of susceptible animals are a best way of diagnosis in areas where in which laboratory equipment not available.

Clinical outcome of the disease is depends on the Host species, environment and the virus strain. Within 24–48 hours after infection, the affected animals refuse their feed, the oral mucosa showed redness, fever and Viremia start in cattle and pigs and leads to viral spread in the body resulting in the production of vesicles preferentially in the mouth and feet should make foot-and-mouth disease a differential consideration. Gross lesions resembles those seen before death and include vesicular, erosive and ulcerative lesions of mouth, feet and teat ends; Foot and Mouth disease also cause lesion of mammary gland and ruminal epithelium. Most of the affected animals eventually recovered, However, those animals which are recovered from the disease may become carrier of FMDV in which virus can be recovered after 28 days of infection. Duration of carrier status may vary from few weeks to several years [14, 33, 34].

Laboratory Diagnosis: A sample for laboratory diagnosis is collected from epithelium or vesicular fluid from an animal that is suspected with foot and mouth disease. Samples of choice in the cattle are lesions from tongue tissue, buccal mucosa, wounds from feet and hoofs. In pigs fluid filled vesicles wound from the tongue, snout, coronary band hoof shall be collected [17].

Virus Isolation: The presence of relatively high levels of FMDV antigen in vesicular material can be detected by ELISA. However, when the virus concentration is too low to be detected by ELISA, then it has to be propagated in susceptible cell cultures. [35] In the determination of foot and mouth disease, isolation of the virus is still considered the international gold standard. Laboratory diagnosis of FMD is like any other viral disease by demonstration of antigen using serological and nucleic acid based methods and demonstration of antibody against structural proteins, or alternatively against non-structural proteins (NSP) for differentiation of infected from vaccinated animals (DIVA). Primary cell cultures like bovine thyroid and fetal lamb kidney are highly susceptible but difficult to maintain. Culture has to be examined for 48 hours for the presence of cytopathic effect. Virus isolation is slow and requires 4-5 days for giving definite diagnosis. Recently, transfection of RNA isolated from clinical samples yielded more than 70% revival/isolation rate [34].

There are some drawbacks of virus isolation techniques of diagnosis. Some FMDVs fail to grow in a specific cell type. Thus the absence of apparent growth

does not guarantee absence of the virus and therefore samples collected from a suspected case of FMD should be subjected to further investigations, e.g. using another testing system. Additional disadvantages include the problems associated with obtaining and maintaining a regular supply of cells; possible contamination of cell cultures and the necessity to confirm any apparent virus growth by ELISA. These issues may delay the initiation of control measures to contain outbreaks [14].

Enzyme Linked ImmunoSorbent Assay ELISA: The ELISA appears to be the most suitable of the antibody-detection tests and can be a complement, rather than an alternative, to test based on cellular immunity [36]. Although methods based on virus isolation or the demonstration of FMD viral antigen or nucleic acid in samples of tissue or fluid or culture products is sufficient for a positive diagnosis, the ELISA using type-specific serological reagents is the preferred procedure for the detection of FMD viral antigen and identification of viral serotype in the early stages of research. Owing to it is more specific, sensitive and efficient and it is not impacted by pro- or anti-complement factors the ELISA has access to better development and even replaced complement fixation (CF) in most laboratories in the early research phase of FMD. Contrast to CF and virus isolation, almost the equivalent, even the higher of sensitivity was achieved in ELISA [37].

Sandwich ELISA is a much faster approach to detect viral antigens, but it has low sensitivity, so its primary indication is to confirm and type the FMDV after isolation in cell culture. As a consequence, several researchers have been developing alternative assay systems that allow more rapid confirmation of clinical diagnosis, which do not require a laboratory setting and may be performed 'Pen side.' [38]

Reverse Transcription Polymerase Chain Reaction. (RT-PCR): Foot-and-mouth disease virus (FMDV) can be diagnosed by using a reverse transcription-PCR (RT-PCR) which is highly sensitive and specific for the detection of the virus. The test allowed the detection of viral RNA from a variety of animal samples and from a wide range of FMDV isolates of different origins and serotypes [39]. RT-PCR can be used to amplify genome fragments of FMDV in diagnostic materials including epithelium, milk, serum and OP samples. RT combined with real-time PCR

has sensitivity comparable to that of virus isolation and automated procedures enhance sample throughput. Serotyping primers have also been developed. Simplified RT-PCR systems for potential field-use are under development. The RT-PCR assay consists of the three successive procedures of (i) extraction of template RNA from the test or control sample followed by (ii) RT of the extracted RNA, (iii) PCR amplification of the RT product and (iv) Detection of the PCR products by agarose gel electrophoresis [40].

Complement Fixation Test: The CFT has been of great value in the past in many FMD laboratories. The CFT is serotype dependant and requires a good practical knowledge of anti-complement reactions. Near Europe it is still used nowadays in some Transcaucasian countries and e.g. in regional laboratories in Iran. CFT could be replaced by the more sensitive antigen-ELISA but the availability of an ELISA reader, ELISA plates and reagents are essential factors. The necessity of using a more sensitive test depends on the phase of the FMD control campaign [41, 42].

Differential Diagnosis: Typically, Foot-and-Mouth Disease is difficult to distinguish by clinical signs from other vesicular diseases of the viral origin such as Swine vesicular disease, Vesicular exanthema, Vesicular stomatitis, Infectious bovine rhinotracheitis, Rinderpest, Bluetongue, Bovine papular stomatitis, Peste des petits [43].

Economic Impact of FMD: Foot and mouth disease is the most contagious Trans boundary animal disease affecting cloven footed animals. The highly contagious character of the virus results a great potential for causing severe economic loss by its high morbidity and the export trade restrictions imposed on affected countries. Many studies highlighted the severe impact on national economies with introduction of FMD could have in a FMD free country [28].

In general as the Fig. 1 shows that, the economic impact of FMD can be separated into two components direct and indirect impacts, the direct impacts includes reduced milk production, reduced livestock growth, mortality in young stock, problems with fertility and the indirect economic impacts of FMD includes Additional costs such as movement control and vaccination costs [44, 45].

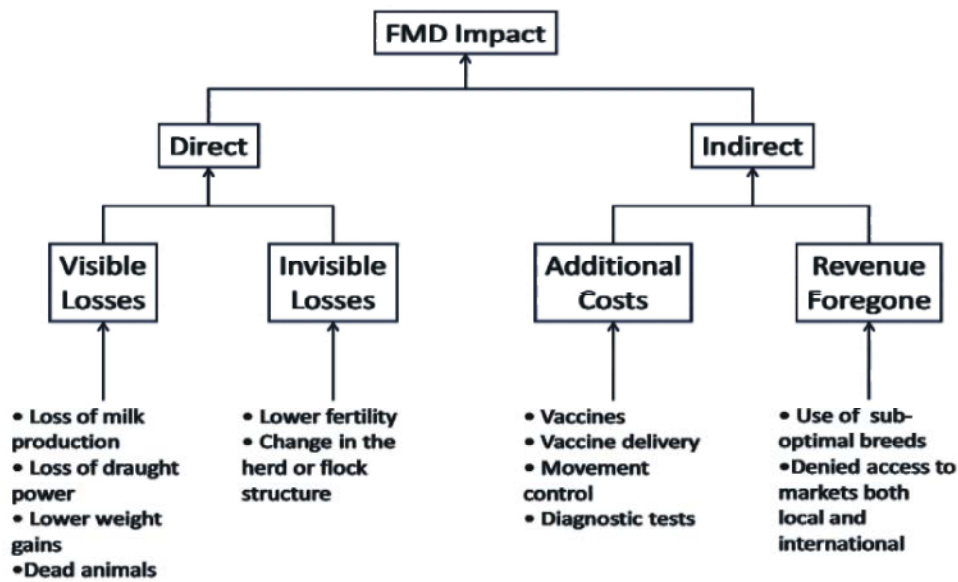


Fig. 1: The economic impacts of foot mouth disease [46].
FMD Status in Ethiopia

Foot and mouth disease is enzootic in most parts of Africa and only a few countries on the continent have managed to control the disease to allow access to lucrative export markets for live animals and animal products [47, 48]. FMD is endemic and known for its wider distribution in Ethiopia. It was first recorded in Ethiopia in 1957 when serotypes O and C were found. [49] This disease is found in many parts of Ethiopia; including: Arisi Zone, Dire Dawa region, North wollo, round Debre zeit and Addis Ababa. Extensive movement of live stock and the high rate of contact among animals at commercial markets, in communal grazing areas and watering points, have been forwarded as cause for the increasing incidence in recent years [29, 50, 51]. Factors such as the presence of high numbers of susceptible animals, wild and domestic animals sharing common grazing pastures and watering points in areas where wild life occur contribute to the frequent occurrence of FMD outbreaks and to the difficulty in controlling the disease [52]

FMD virus serotypes that are identified in Ethiopia are Serotype O, A, Namibia, Botswana and the Republic of south Africa C, SAT1 and SAT2. These serotypes of FMD were identified and characterized by the National Animal, health research centre at Sebeta and the world reference laboratory for FMD at UK in the years 1969-1994 on samples submitted by Sholla disease investigation laboratory. [53] [29] In recent researches the seroprevalence of FMD among Borana pastoral cattle in 2008 was reported to be 24.6% [54].

In Ethiopia, many of the known infectious diseases of animals occur commonly and are poorly controlled. FMD is one of infectious disease that is prevalent in Ethiopia and it is poorly controlled. Foot and mouth disease (FMD) has a great impact on economic development of the country [55, 56]. Ethiopia has large numbers of susceptible domestic and wild ruminants exist, with limited vaccination and disease reporting and investigation, serological surveys are very useful for understanding the epidemiology of the disease [57].

CONCLUSIONS

Foot and Mouth Disease is the most contagious disease of all cloven hoofed animals with a serious effect on animal production and significant impact on the economic development of the country. Its aetiological agent, the foot-and-mouth disease virus (FMDV; family Picornaviridae, genus: Aphthovirus), causes an extremely contagious disease of domesticated and wild cloven footed animals. Seven immunologically distinct serotypes [O, A, C, Asia 1, Southern African Territories (SAT) 1, SAT2 and SAT3] have been identified so far. The identification and control measures of the disease has become challenging due to the contagious behaviour of the virus and presence of seven serotypes with their multiple subtypes and strains. FMD is endemic and known for its wider distribution in Ethiopia. Foot and Mouth

disease is common during winter season. In Ethiopia, serotype O, A, Namibia, Botswana and the Republic of south Africa C, SAT1 and SAT2 serotypes were identified in different parts of the country. Uncontrolled movement of live stock and the high rate of contact among animals at commercial markets, in communal grazing areas and watering points, have been forwarded as cause for the increasing incidence of the disease.

Based on the above conclusions the following recommendations are forwarded:

- The disease serotypes should be quickly identified and quick responses should be prepared in order to control the disease effectively.
- Government should regularly monitor the occurrence of outbreak and it should widen the availability and accessibility of effective diagnostic techniques as much as possible.
- Control and restriction of the cross border animal movement and setting up quarantine station around the border area should be applied.
- Control over in the airport, during importation of live animal from other FMD endemic countries.
- Regular and Periodic mass vaccination of animals (At least two times per year)

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