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Diagnosis and Control of Peste Des Petits (PPR) in Small Ruminants: A Comprehensive Review

Adnan Yousaf[1, *], Dad Muhammad Anwar[2], Mehtab Ahmed[2], Imran Qaiser[2], Rasheed Ahmed Rabbani[2], Ajeet Sing Berjang[1], Lookesh Kumar[1], Faiza Habib[2]

[1]Faculty of Animal Husbandry and Veterinary Sciences, Sindh Agriculture University, Tandojam, Pakistan

[2]Department of Livestock and Dairy Development Quetta, Balochistan, Pakistan

Email address:

dr.adnan011@gmail.com (Adnan Yousaf)

[*]Corresponding author

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Abstract: PPR (Peste des petits ruminants) is a highly infectious serious infection that affects a lot of sheep and goats. In nations like Pakistan, where small ruminant products are crucial for long-term livelihoods, the infection has a significant economic impact. Goats are known to be more sensitive to the sickness than other animals. It's a highly contagious illness that affects tiny ruminants and has a high morbidity and fatality rate. The timing of infection, the spread of the virus, and the early identification of the disease all need to be better understood. Disease has an almost universal geographic spread. The PPRV is a virus that affects both domestic and wild ruminants. In Pakistan, PPR is diagnosed mostly by postmortem examination of clinical symptoms and lesions. Conjunctivitis and rhinotracheitis, stomatitis, gastroenteritis, and pneumonia are all caused by PPRV, a lymphotropic and epitheliotropic virus. Pathognomonic symptoms include necrotizing tonsils, fibrinohemorrhagic enteritis, proliferative interstitial pneumonia, supportiv bronchointerstitial pneumonia, multinucleated giant cells [syncytia], and cytoplasmic and/or nuclear eosinophilic inclusion bodies. The acute type, which is identical to rinderpest, is most commonly found in goats. A severe respiratory condition is a common symptom of PPR. The subacute type is more common in sheep, although it can also happen in goats. Immunization, isolation, transportation restriction, and hygienic slaughter and disinfection are all part of the control and preventive programme. Moreover, in addition to the issue of resistance to antibiotics, further research into antibiotics resistance and the development of new antibiotics can aid in enhanced therapeutic intervention. Reviewing existing preventative approaches and focusing on creating new strain-based or recombinant vaccines that target specific antigens is the most effective strategy to treat the illness internationally (capsular or cellular).

Keywords: Peste Des Petit Ruminants, Morbillivirus, Epidemic Curve, Small Ruminants, Diagnosis, Control

1. Introduction

PPR is a highly infectious and acute viral illness that affects sheep and goats, with subclinical manifestations in cattle, pigs, and camels [5]. PPR, also known as ovine rinderpest, goat plague, plague of small ruminants, pneumoentritis syndrome, pneumoentritis complex, and infectious postular stomatitis [1], is an extremely infectious livestock disease that affects small ruminants. Peste des petits ruminants (PPR) is one of the most economically significant diseases that has a considerable impact on small ruminants [2], and it was formerly thought to be a developing Morbillivirus-caused viral disease of small ruminants. PPR is widely distributed over Africa, the Middle East, and Southern Asia [3]. Fever, anorexia, nasal and ocular discharges, mouth sores, pneumonia, copious diarrhea, and mortality are all symptoms of the condition [9]. Morbidity and mortality rates have been reported to range from 90 to 100 % and 50 to 100 %, respectively [19]. PPR has also been linked to a significant % age of abortion in goats infected with the virus [21]. Animals get infected by the PPRV through their oral and nasal passageways. After entering the body, the virus

replicates first in the nasopharyngeal/respiratory epithelium [4, 5], before infecting regional lymphoid organs, when a second round of replication occurs [6, 7], causing the infection to spread to distant organs. The morbilliviruses have a significant affinity for lymphoid organs, and the loss of leucocytes during infection typically results in severe immunosuppression [8]. In its acute form, the PPR virus (PPRV) generates severe clinical symptoms, which vary depending on the species, age, strain virulence, and additional infectious agents [9, 10]. Pyrexia, respiratory signs, in appetence, chronic depression, erosive stomatitis, catarrhal inflammation of the ocular and nasal mucous, profuse diarrhea that may be watery [11, 12], foetid, and blood-stained, and very often end-stage bronchopneumonia caused by bacterial complications due to immunosuppression [13]. The PPR virus can be found in a variety of secretions. Excretions of infected animals from 3 to 22 days after infection [14]. PPRV is spread mostly through contact with infected animals [15], as well as through their fresh secretions or excrement [17]. Goats are much more vulnerable than sheep and, in rare occasions, wild small ruminants [16]. The World Organization for Animal Health (OIE) and the United Nations Food and Agriculture Organization (FAO) have established a global eradication effort based on epidemiological surveillance [18], early case detection, and widespread vaccination programmes [19]. In fact, a thorough understanding of PPR illness and its clinical manifestations in target species is a necessary component of efficient surveillance [20, 21]. The pathogenesis of PPRV has been inferred from the closely related rinderpest virus and other morbillivirus infections [23]. There have been few research focusing on the pathology of PPRV, and little is understood about the processes driving the disease's development, or pathogenesis, in susceptible animals [22]. Experimental infection of two groups of goats with PPR MOR15, a lineage IV strain identified locally in 2015, was carried out in this investigation, and the pathogenesis was assessed by real-time RT-PCR [24, 50]. Furthermore, we used a quantitative time-course analysis to track illness indications and virus release, lesions, and viral load in various tissues. In comparison to two uninfected goats, we examine the pathogenesis of PPR genotype IV virus following infection of two groups of goats by viral load detection in secretions and tissues. The goal of this research is to better understand infection timelines, viral circulation, and infection early detection.

2. Etiology

The genus Morbillivirus, family Paramyxoviridae, and order Mononegavirales are responsible for PPR caused by an RNA coated virus [25]. Rinderpest virus, Measles virus, Canine distemper virus, Porcine distemper virus, Cetacean morbillivirus, the morbillivirus of marine animals, and Feline morbillivirus are among the other morbilliviruses [26]. The PPR virus is a one-stranded RNA virus with 15948 nucleotides and eight genes [3'-N-P/C/V-M-F-HN-L-5'] [27]. N-[nucleocapsid], P-[phosphoprotein], M-[matrix protein] are the six structural proteins produced by these genes. F-[fusion protein], HN-[haemagglutininaminidase protein], L-[large/polymerase], and two nonstructural proteins [protein C and protein V] have all been identified [28, 29]. There are four PPR virus lineages known. Lineage 1 began in Western Africa, lineage 2 in West African nations, including Ivory Coast, Guinea, and Burkina Faso, lineage 3 in Eastern Africa, including Sudan, Yemen, and Oman, and lineage 4 in the Arabian Peninsula, Middle East, and South Asia [9, 13-15].

3. Geographical Distribution of Disease

PPR has a vast geographical reach. The illness was initially discovered in West Africa in the 1940s [21, 22], and it has since spread to North and Central Africa, the Middle East, and portions of East Africa and Asia [24, 29, 34], as well as Europe. PPRV was initially identified in East Africa in Ethiopia in 1991 [42], while diseased goat flocks in Ethiopia's Afar area were accused of having PPR much earlier in 1977 [43, 47]. The infection was initially discovered in sheep and goats in Côte d'Ivoire, West Africa, in 1942 [1]. PPR is now widely recognized not just in West Africa, but also in the Middle - east [30]. Strain-4 PPRV was detected from Sudan in 2000, 2004, 2008, and 2009 [31]. PPRV mobility in the herd also was demonstrated by spontaneous propagation [38]. Anti-PPRV titers were shown to be greater in goat flocks than in sheep [22, 32, 33]. PPRV was discovered in Egypt between 1987 and 1990 [34]. Iran, Saudi Arabia, Iraq, Israel, Jordan, Kuwait, Libya, Oman, the United Arab Emirates, and Yemen have all isolated PPRV. Seroepidemiologic investigations have revealed that the illness is widespread in Turkey and Syria [37]. In 2000, there was no evidence of PPR seroprevalence in Saudi Arabia [35], but recent investigations reveal that incidence has risen substantially [36]. In Jordan and Lebanon, several seroprevalence tests were carried out [39, 40]. PPRV was identified using a PCR approach in Pakistan's Punjab area [41]. Due to the frequency of PPRV, Ahmad discovered that healthy goats were seropositive [42]. PPRV is extensively disseminated throughout Pakistan, according to several seroprevalence investigations [43, 44]. In the year 2000, Iraq was hit by a virus that caused considerable high disease and low fatality [45, 52, 53]. The detected pathogen had already been in existence, according to sero screening [46]. In Iran, the PPR is common and causes economic losses in sheep and goat herds [75]. An epidemic of PPR in camels was initially recorded in Iran [47], and then in India [178].

4. Sensitive Species & Transmission

Small domesticated and wild ruminants [51, 52], as well as camels [53, 54], are all affected by the PPRV. Cattle, goats, and sheep make up a substantial portion of the animals raised by Pakistani communities as sources of protein and income [48, 49], with sheep and goats providing for around 22% of meat consumed in Pakistan [226]. Pastoralists choose goats

and sheep because of their toughness and capacity to tolerate the severe desert and semi-arid conditions [61]. They are generally managed in large-scale management systems that include community grazing and, in some cases, housing [54]. Goats are badly impacted among small ruminants [55, 49]. In mixed [sheep and goat] flocks, morbidity and seroprevalence rose considerably [56, 57]. Cattle, buffalo [58], pig [59], and camel [60] have all been reported to have viral seroprevalence, although no clinical indications have been found in any of these species save the camel [66]. In an experimental research, death and clinical symptoms were recorded in PPRV-infected calves [63]. In India, the PPR virus was identified from domestic buffalo [65]. Many findings suggest that camels may be affected with the PPRV virus [67]. In Ethiopian epizootic epidemics, the virus was isolated from camels [68, 69]. PCR was used to isolate PPRV from camels in Sudan [71, 72]. Iran [73] reported a clinical case of PPR in camels. Also vulnerable are Dorcas gazelle, Thomson's gazelle, Gemsbok, and Ibex [73, 76]. In Iran, the virus was isolated from wild goats and Bharals [77]. PPRV was identified from Sindh Ibex in Pakistan using PCR and immuncapture ELISA [IC ELISA] techniques [80].

4.1. Transmission

PPRV is found in tears, nasal discharges, cough secretions, and in infected animal faeces [81]. At the conclusion of the sickness, the virus is shed from the gut and identified in faeces [83]. After being exposed to the virus, swine and cattle exhibit no clinical indications [85, 86]. Following experimental inoculation, cattle exhibit no clinical indications [87]. However, there are just a few reports on the prevalence of cattle PPR in calf and immunosuppressed animals [9, 88]. Sneezing and coughing of infected animals discharge little infectious particles into the environment [90]. The Food and Agriculture Organization of the United Nations (FAO) and the World Organisation for Animal Health (OIE) have launched a global project to eradicate PPR by 2030 [62, 64]. To do this, it is necessary to comprehend the illness's particular epidemiological aspects as well as identify the socio-economic elements that must be considered in order to prevent disease transmission [70, 74].

Pathogenesis

PPRV is an epitheliotropic and lymphotropic virus that causes conjunctivitis, rhinotracheitis, stomatitis, gastroenteritis, and pneumonia [91]. After PPRV infection, antigen presentation cells (APCs) in the inter epithelial space and respiratory nasopharynx acquire the virus [92], which is subsequently transported to local lymphoid tissue for viral replication [93, 94]. Infected lymphocytes in the blood and lymphoid system transmit the virus throughout the body [95, 97]. Pneumonia develops late in the illness and after a high dosage of viral exposure [98]. Due to lymphoid tissue necrosis [spleen, payer patch, lung, lymph node] [100, 102], the blood lymphocyte count in afflicted animals is lowered. Following PPRV infection, polymorph nuclear cells [PMN] triggered apoptosis in vitro [103, 104]. There is a prolonged fever, hyperemia, muco-nasal discharge, anorexia, and diarrhea 3-5 days following the incubation period [106]. The viral dosage and the incubation time have a negative relationship [107, 111]. Following PPRV exposure in sensitive species, lung acute congestion and edoema are prevalent, and mortality is typically the result [112, 114]. Chronic infection is rare and must be distinguished from bronchopneumonia. These creatures might live for up to a month. The effects of infection differ by animal and are dependent on the immune response [113, 116]. Infestation with parasitic worms and nutritional state may have a role in illness severity, morbidity, and death [117, 118].

4.2. Immune System Response

Despite the fact that morbillivirus is an immunosuppressor, animals that have recovered from an acute infection frequently develop long-term immunity against reinfection [126, 131]. F, N, and H proteins are frequently the targets of protective immunity [132, 133]. Rinderpest Glycoprotein F and H, as well as PPRV, elicit a humoral reaction [136]. There is evidence that the cellular immune reaction is important in PPR protection [139, 140]. Despite the fact that the virus's N- protein has the highest incidence, it does not elicit a neutralizing antibody response [141, 143]. [142, 144] N-protein triggers a cellular immunological response. PPRV infection causes a characteristic inflammatory response in goats, which is marked by increased production of cytokines as IFN, IFN, IL-4, IL-1, IL-8, IL-10, IL-6, and IL-12 [146, 147].

5. Clinical Findings

PPR has clinical indications that are similar to rinderpest, however rinderpest in small ruminants is uncommon [150]. Clinical symptoms in sheep range from mild to deadly in goats [155]. Non-clinical infection has been reported in both species, particularly in flocks travelling through endemic areas [153]. Anorexia, emaciation, severe depression, fever (40–41C), diarrhea, mucopurulent nasal and ocular discharge, and erosive and necrotic stomatitis were among the prominent symptoms of PPR documented [156, 157]. There were also nodular lesions and abortion [158]. Sudden death, yellowish diarrhea [165, 166], stomatitis, ulcerative keratitis conjunctivitis, fever, respiratory distress, pneumonia, enlargement of lymph node, dermatitis, obvious dehydration [169, 170], abortion, subcutaneous edoema, submandibular enlargement, chest pain, and infrequent coughing are the most notable findings in affected goats and sheep. In addition, post-mortem examination of confirmed instances of PPR [177] revealed lung congestion and consolidation, as well as increased interalveolar wall thickness, suggesting pneumonia [179].

Acute form: The acute type, which is identical to rinderpest, is most commonly found in goats. A severe respiratory condition is a common symptom of PPR. [184, 185] The incubation period is 3-6 days. Young animals are particularly vulnerable to PPR, which has a poor prognosis and often results in mortality [186, 189]. Sudden fever (40-

41.3°C) [190, 191], sneezing, tears, and nasal secretions [192] are all symptoms of the acute type. Necrotic lesions in the nasal cavity and inside the oral cavity appear 1-2 days later [193]. Diphtheria plaque is formed, and nasal secretions give off a foul odour [198]. The muzzles of affected animals are dry, and mucopurulent discharges are present [200, 201]. At the end of the disease, dyspnea and cough were observed [202]. There have been reports of vaginal and prepuce erosions, as well as abortion [203, 204]. Death happens seven to ten days after the commencement of the fever [206].

Subacute form: The subacute type is more common in sheep, although it can also be found in goats [209]. The majority of the animals infected by necrotizing ulcers recover [210]. [207] Death is an uncommon occurrence. The illness frequently lasts longer than two weeks [213].

5.1. Abortions

It has been observed that blood samples from aborted dams tested positive for PPR antibodies, suggesting that PPR illness has a probable link to mortality and prevalence in goats [4]. Furthermore, abortions can occur at any stage of pregnancy if the animal is infected with the PPR virus [10].

5.2. Morbidity and Mortality

The morbidity rate is 100 %, and death is 100 % in severe epidemics [11, 12]. The rates of morbidity and death vary, although they can exceed 100% [13]. In endemic locations, these rates are frequently lower (mortality of 20% or less), and sero-surveillance is sometimes the sole way to detect illness [14]. It has been observed that mortality in acute cases ranges from 70 to 80 %, with death occurring between 10 and 12 days [15]. Furthermore, sucklers had greater rates of illness and death than adult animals [16].

6. Seasonal Occurrence

The incidence of PPR is influenced by climatic conditions. In the wet season, outbreaks are reduced due to reduced animal movement, increased fodder supply, and improved nutritional and health condition [16]. Because illness spreads and cases peak in April [17], the dry and dusty season combines with poor nutrition in December-February. In Pakistan, significant PPR seroprevalence was recorded in December to February, as well as September and October [32]. Disease frequency was observed to be higher in January to April, with 33% of cases reported in March [35]. As a result, we may conclude that the sickness occurs throughout the year, with varying degrees of severity depending on the weather [116].

6.1. Risk Factors of PPR for Sheep and Goats Population

6.1.1. Age

Young animals are particularly susceptible to the illness, with greater rates of morbidity and death [132]. Sero-prevalence is often higher in animals over the age of two. Furthermore, older animals are more likely to be PPR seropositive than younger animals [116]. Specie although the illness affects both sheep and goats, the incidence and severity of the condition differs [133]. In numerous studies across Pakistan, the prevalence of PPR has been found to be greater in goats than in sheep [142]. PPR can also impact wild ruminants, and a large epidemic [11] was observed in which the Sindh Ibex was seriously harmed [142].

6.1.2. Sex

Sheep ewes had greater seropositivity than rams, according to one study [142]. Furthermore, this occurrence has been reinforced in Pakistan's village-based production system [185], where male sheep and goats are routinely slain at a young age while female sheep and goats are kept.

6.1.3. Season

Despite the fact that the disease is considered prevalent in Pakistan, there have been few instances of seasonal incidence. Due to the abundance of fodder available during the rainy season, PPR incidence dropped, resulting in enhanced disease resistance [202]. PPR illness is caused by large flock sizes, animals that attend the livestock market, and insufficient veterinary care [185].

6.2. Spatial Distribution of Ppr in Various Locations of Pakistan

Although both goats and sheep are vulnerable to infection and can develop illness, they are not always afflicted at the same time. For example, in Africa, PPR is more typically observed in goats, but sheep are the most visible victims in western and southern Asia [214]. However, PPR affects both goats and sheep in Pakistan, as it does everywhere, although in many places, only goats are afflicted [217], and data [215] back this up. PPR seroprevalence in sheep is 49.5% in different parts of Sindh province, compared to 56.3% in goats [216]. The majority of PPR cases appear at the start of the summer season, peaking in the months of April to July before declining again [219]. PPR virus antibody prevalence in small ruminants in Punjab was 51.3% [132]. In the months of December, January, and February, antibodies against PPR virus were observed at 67.7%, 71.1%, and 60.2%, respectively, and 50.7% and 53.0% in September and October [219]. The availability of local fodder and the nutritional state of the animals may play a crucial influence in disease spread [221]. PPR virus distribution in several districts of Pakistan, which is 48.30 % [222]. The illness was shown to be more widespread in young sheep and goats than in adults, and the lymph nodes remained the PPR virus's preferred location [224].

7. Diagnosis

In Pakistan, PPR was diagnosed primarily through postmortem observation of clinical signs and lesions, followed by the use of a monoclonal antibody-based competitive enzyme-linked immunosorbent assay (cELISA) for the detection of PPRV antibodies to determine whether

the patient had previously or currently been infected [225]. In addition, several of the investigations used confirmatory molecular approaches to detect the PPRV genome [227]. Because the clinical indications of PPR and rinderpest are so similar, it's difficult to tell the two apart [96]. PPRV and rinderpest antibodies against nucleocapsis protein and haemagglutinin protein aided illness identification by serologic assays [99]. Swabs of conjunctival, nasal, and oral discharges and ulcers, whole blood, and serum samples for serology were taken for testing [101]. For the detection of viral RNA, portions of the intestines, lungs, and lymph nodes were also taken and homogenised [105]. PPRV genome was detected in buffy coat, homogenised tissue samples, and nasopharyngeal and ocular swabs of probable patients using real-time reverse transcription polymerase chain reaction (rRT-PCR) targeting the PPRV nucleoprotein (N) gene [108, 109]. Virus isolation, antigen and antibody detection, and molecular approaches are all used in diagnosis. For PPR diagnosis, viral isolation is the gold standard approach. In vitro, the virus has been isolated in sheep and cattle primary cells [227], Vero cells [18], and marmoset B-lymphoblastoid-B95a cells. In compared to RT-PCR, this approach takes longer, needs special equipment, and has lower sensitivity [228]. Indirect immunofluorescence test [IFAT] and Immunoperoxidase [108]; CIE [111] AGID, AGPT [112]; Haemagglutination Tests [229]; MAb based immunocapture ELISA [52, 232]; sandwich ELISA (s-ELISA) [234]; and dot-ELISA [114, 233] are some of the virus antigen diagnostic methods. Although the AGPT is the most popular way of diagnosing PPR and rinderpest, it is ineffective in differentiating PPR and rinderpest [236]. The hemagglutinin of PPRV as a morbillivirus is suitable [85, 117-119]. In compared to AGPT, HA is more sensitive to viral antigen [14]. For the differential diagnosis of PPR from rinderpest, HA is the preferred approach [237]. The CIE as AGPT, on the other hand, is not capable of distinguishing PPR from rinderpest, but it is more sensitive and easier to use [111]. PPR and rinderpest antigen isolation may be done using CIE [239]. The monoclonal antibody-based immunocapture ELISA is a legitimate and sophisticated approach extensively employed in reference laboratories [240, 241, 242], with a sensitivity and specificity of 89% and 93%, respectively, when compared to IC ELISA [194]. Another study looked at the effectiveness of a sandwich ELISA for detecting PPR. The sensitivity and specificity of this study's findings were found to be satisfactory [78]. Dot ELISA is a monoclonal antibody-based agonist protein M or N that is frequently utilized when the number of referred samples is higher than normal [243]. This approach has a sensitivity and specificity of 82.5% and 91%, respectively [114]. VNT [244], C-ELISA, and b-ELISA [54, 122, 123] are examples of antibody-based detection techniques. VTN is the preferred approach for distinguishing PPRV from rinderpest virus [245]. For PPR screening, ELISA is a quick, simple, and sensitive serologic approach. C-ELISA and b-ELISA are sensitivity and specificity-adequate monoclonal antibody-based techniques [antiprotein H and antiproton N antibody] [247]. In comparison to VNT, Singh et al. found that c-ELISA was 92.2% sensitive and 98.4% specific [246]. For PPRV diagnosis, molecular procedures such as PCR, RT-PCR, and cDNA hybridization are extremely exact, although they are time consuming and need specialized equipment [248]. Genes P, F, M, and N are the target genes in PPRV PCR techniques [249, 250]. The RT-PCR approach relies on the detection of PPRV specific genes such as M and N [251, 252].

7.1. Necropsy Finding

Necrotic ulcers can be detected in the gastrointestinal tract and respiratory system, but they can also appear in other organs [253]. The animals that are afflicted are generally malnourished and have dry secretions on their faces. Necrotic lesions in the lower lip and surrounding gum, hard and soft palate, and ventral side of the tongue that reached to the esophagus were discovered. Stomatitis is characterized by erosions and nodules in the oral cavity, which are often visible near the end of the infection [133]. Serous and mucopurulent discharges are seen in the nasal cavity, which is hyperemic. Bronchopneumonia is characterized by coughing and necrotic foci in the cranial and cardiac lobes of the lung in the end stage of the disease. Lymphadenopathy can be detected when the gastrointestinal and lung lymph nodes are implicated. The abomasum has regulatory delineated erosions and hemorrhages, while the initial section of the small intestine has erosions less often. The large intestine is more seriously damaged by diffused bleeding along the fold of mucous in the large intestine, resulting in a streak of congestion and the appearance of a zebra line [133]. A rotten odor emanates from the cadaver [133]. Antigen testing requires samples from the lungs, small and large intestines, oral mucosa, and mesenteric lymph nodes [254].

7.2. Differential Diagnosis

Other viral infections such as goat pox, blue tongue, contagious pustular stomatitis, foot and mouth disease (FMD), and contagious caprine pleuropneumonia (CCPP) should be distinguished from PPR [255].

7.3. Vaccine and Vaccination

In Pakistan, the PPR is considered an endemic illness of sheep and goats. The live attenuated Virus of Lineage I [230] was used in a variety of immunization regimens. PPR outbreaks are common, despite stringent vaccination regimens and other preventative and therapeutic interventions. Furthermore, a variety of PPR vaccines, including conventional, thermostable, recombinant, and edible vaccines, have been produced and are widely employed in the control and eradication of the illness [84]. Vaccination is now advised in several parts of the country. The Nig75/1 vaccine is based on lineage II, whereas field isolates from Pakistan are classified as lineage IV [10, 11]. The genetic characterization of field strains will lay the groundwork for developing vaccines from local strains, as India has just done [21]. A summary of research based on PPR vaccination is

shown below. It was investigating the responsiveness of sheep and goats in Pakistan to a locally manufactured live attenuated PPR cell culture vaccination [258]. The vaccine generated a high serological titer within 21 days of vaccination and was shown to be safe, with the vaccine titer remaining high for a year after vaccination. Furthermore, PPR vaccination demonstrated a strong reaction to control the issue in the face of an epidemic, according to [56]. The data can be used to plan effective disease management in small ruminant subsistence farming in the KPK [257]. In field trials, a homogeneous vaccination has been created and tested. To minimize misunderstanding with Rinderpest during serological surveys, this PPR vaccination is strongly recommended [173]. It is now available for purchase. In addition, a live attenuated cell culture vaccine was developed and tested for protection against PPR illness in small ruminants (sheep/goats), which are the species most vulnerable to the virus [22]. Following immunization, no unpleasant effects were reported. Antibodies were produced in large quantities in all vaccinated animals (PI > 50) [89]. As a result, this live attenuated PPR cell culture vaccine may be used to safely immunize small ruminants against PPR illness, reducing the devastating economic losses. While, utilising the Vero cell line, a tissue culture-based live freeze-dried PPR virus (PPR 75–1) vaccine was generated [1] and tested for validity, safety, sterility, and effectiveness [11]. They came to the conclusion that this PPR vaccination would be a useful strategy for limiting PPR illness in goats and reducing economic losses caused by the disease in Pakistan [172]. The humoral immune response evaluated by haemagglutination inhibition (HI) and agar gel immunodiffusion (AGID) assays in sheep and goats was used to assess the efficacy of PPRV vaccinations available in Pakistan. At the 63rd day after vaccination in sheep, the geometric mean titer (GMT) of antibodies against locally made PPRV vaccine was greater (207.9) than Pestivec (73.3), a vaccine imported from Jordan; the comparable values in goats were 147.0 and 48.5, respectively. Both diagnostic tests revealed that all of the animals in the control group were antibody-free. Furthermore, it was shown that the efficiency of PPR virus vaccines is dependent on storage temperature, buffer pH, and immune response in sheep is superior to goats [51].

7.4. Challenges for the Control of PPR

PPR has spread over much of Pakistan despite several vaccination programmers [110]. Low knowledge among small ruminant farmers, merchants, and transporters was cited as a key restriction in the management of PPR [120], as were uncontrolled livestock movements [115], a lack of diagnostic instruments [119], insufficient surveillance and reporting, and a lack of capacity to implement rules. PPRV's persistence in Pakistan is due to a combination of reasons, including unregulated livestock movement [121], inadequate zoo-sanitary practices among farmers, and a lack of appropriate local and national management measures [123].

7.5. Control and Prevention

Quarantine, movement restriction, hygienic slaughter, and disinfection are all conventional disease control procedures. In endemic locations, a modified live vaccination is employed [106]. In the past, an attenuated heterologous vaccine (Tissue culture rinderpest vaccine) containing live rinderpest was employed [135, 136], however it is no longer an appropriate strategy since it interferes with the global eradication schedule [54]. PPRV cytotoxicity in sheep hepatocytes and adaptability to PPRV in cell culture [137]. There is an attenuated homologous PPR vaccine made in Vero cell culture [114, 138]. For more than a year, this vaccination provides protection against natural infection. Vaccinated animals make neutralizing antibodies against proteins F, H, and N in the same way as diseased animals do [95, 140]. Nigeria 75/1, a lineage 1 vaccine, was the first homologous vaccination against PPRV. The virus was isolated from a PPRV-infected deceased goat [67]. The virus was passaged 63 times in Vero cell culture to reduce the severity. The humoral immune response begins seven days after immunization and lasts three years [141]. Sungari/96 vaccine is a lineage 4 vaccine. The virus was discovered in a goat in India in 1994 [142]. After 10 passages, the isolated virus was adapted in the Marmoset lymphoblastoid 1395a cell line. The virus was passaged 54 times in the Vero cell line to reduce severity [141]. Arasure 87 and Coimbator 97 [143] are two additional vaccines available. It is recommended that the Nigeria 75/1 vaccination not be used in Asia because it may promote mutation and enhanced virulence [144]. In Asia, the vaccination Sungri/96 is suggested, whereas in Africa, the vaccine Nigeria 75/1 is advised [144]. Sungri 96 is safe to use in pregnant animals and has no immunosuppressive effects [12, 141]. South India uses Arasure 87 and Coimbator 97. Saravanan et al., 2010 found that all three vaccinations, Sungri/96, Arasure 87, and Coimbator 97, provide adequate protection against PPR in both sheep and goats [143, 145]. Although attenuated vaccinations give enough protection, the natural reaction is not recognized, hence recombinant vaccines are recommended [256]. To begin, a recombinant rinderpest vaccine was created by expressing protein H and F; however, this vaccine was not created to protect goats against PPRV, but the immune response occurs after PPRV infection [97, 146]. In goats, a recombinant Capri pox virus vaccine expressing F [14] and H protein [197] can provide PPRV protection [148]. In sheep and goats, a recombinant Capri pox virus vaccine expressing both F and H proteins is employed [149]. After two doses of the vaccine, goats were protected against the disease by the vaccine virus vector [Modified vaccine Ankara] expressing Peste des petits ruminant's virus F and H proteins [150]. Fowl pox virus [151] and recombinant adenovirus vector [151-154] are two more recombinant vaccines that have been developed.

8. Discussion

The limited number of acceptable publications retrieved for this study indicates that there is a scarcity of literature on the condition of PPR in Pakistan. The entry of PPR into Pakistan in

2008 may be directly connected to the establishment and spread of PPR, according to peer-reviewed research [124]. The fact that the initial occurrence of PPR in Pakistan was an epidemic in Punjab (130, 134), and the PPRV strain identified belonged to lineage II [124], is indicative of this. Lineage IV's origins may be difficult to trace because it is found all over the world, including East Africa [125]. Indeed, the prevalence of an informal cross-border cattle trade [127, 128] poses a constant danger of PPR entrance, persistence, and spread across these and other countries [129, 135, 137]. PPR is prevalent across Pakistan, according to the studies examined, and it has wreaked havoc on the country's small ruminant population and pastoralists' livelihoods in recent years [138]. This is due to PPR's high transmissibility and morbidity [145], which has caused in the disease's fast expansion in small ruminant populations throughout wide swaths of Africa and Asia over the last 20 years [148]. Several studies have shown evidence of PPR transfer between species (1, 231). Although there is no reasonable evidence of self-sustaining PPRV infection in wild ruminant populations, the possible role of animals in disease epidemiology must be overlooked [235]. As a result of PPR's endemicity, it poses a threat not only to pastoralists and their livelihoods, but also to wildlife conservation and endangered wild small ruminant species [149, 151]. Major risk factors for PPR in Pakistan have been identified as events/activities that bring together flocks/herds from various farms/locations or expose ill animals to healthy ones [152, 154]. Communal grazing and lodging, the mixing of sick and healthy animals at livestock markets, and the introduction of freshly purchased or rustled animals to a herd are all examples of these behaviors. Other investigations in Djibouti [160], Chad [161], India [162], and Pakistan [163, 164] found similar risk variables for PPR. In Pakistan, a lack of access to veterinary care has been highlighted as a risk factor for PPR [175]. In rural Pakistan, the heartland of small ruminant production, veterinarians and community animal health professionals are in short supply [195, 196]. As a result, PPR management in rural Pakistan is low on the priority list [174]. PPR causes substantial annual economic losses over the world [167]. Mortalities linked with the illness, lower market value owing to poor body condition, culling, the expense of medicine, immunization, veterinary and labor services, and the cost of an embargo on livestock markets enforced by authorities are among the losses documented in this review [168]. These findings are consistent with those seen in other PPR-endemic countries, such as Ethiopia [152, 205], Kenya [154], India [180], and internationally [199]. The projected overall national loss of revenue due to PPR (92 million Euros per year) is a significant burden on the Pakistani economy, emphasizing the need of eradicating the infection [142]. Culling, confinement of diseased animals, biosecurity measures to decrease infectious fomites, denial of imports of sheep and goats from outbreak-prone areas, and mass vaccination can all help to control PPR [208]. In addition to mass/blanket vaccination, targeted vaccination and sero-surveillance measures along the borders with other PPR endemic areas/countries are necessary to develop immune belts and avoid epidemic importation [253]. Despite vaccination programmes [79], after the probable entry of PPR into Pakistan in 2008, the illness has spread throughout the country and is currently prevalent in most regions [255]. In Pakistan, a substantial rise in antibody was seen between pre- and post-vaccination goat and sheep, suggesting that the vaccine might be efficacious in an outbreak [240]. Other variables, such as inadequate vaccination coverage, lack of control of livestock movement [212], and high fecundity owing to the dynamic character of small ruminant populations [218, 220], are believed to be to blame for the disease's failure to be successfully contained by vaccination programmes [82]. Previous vaccination operations in Pakistan may have fallen short of this estimate [181], and herd immunity levels necessary for effective control of PPR transmission are in the range of 70–90 % [171, 176]. To avoid transmission, high levels of immunity must be maintained throughout time, especially in small ruminants with a short generation period and a large turnover of new/nave animals [211]. Vaccination failure can also be caused by interference with maternal immunity in young animals, poor vaccine quality, and a lack of cold chain maintenance [228]. As a result, the causes for vaccination failure and disease transmission persistence in Pakistan must be investigated. Investigations on the obstacles to vaccine use, as well as aspects that may impact vaccine effectiveness and uptake, such as cold-chain storage and proper delivery, should be promoted [152]. Farmers must be aware of the advantages of vaccination, and they and their veterinary extension consultants must understand that vaccination frequency is linked to herd dynamics [187]. For traceability, optimal vaccination coverage, and reliable sero-monitoring, good animal identification is also required [199]. If laboratory access and expenses can be regulated, establishing herd status by clinical history and serological testing would be beneficial. As proven in countries with strong PPR control strategies, such as Morocco [182], adequate surveillance of PPR is critical for control and informing vaccination efforts [231]. Indeed, the epidemiological research used in this study only covered a few districts/areas of Pakistan, leaving vast swaths of the country lacking data on PPR [183]. As a result of the techniques utilized to gather data for this review, the study sites may have been biased, and data from specific areas may have been excluded [251]. The inadequacy of most antibody tests to discriminate between infected and vaccinated animals is a serious impediment to effective monitoring [188]. This might be solved by using vaccinations with DIVA (Differentiating Infected from Vaccinated Animals) capabilities and associated diagnostic tests that allow infected and vaccinated animals to be distinguished [159]. This is critical for appropriate control programme development, implementation, and assessment [223]. Furthermore, the utilization of low-cost, simple-to-use point-of-care diagnostic tools and non-invasive sample types may improve surveillance [228]. If the goal of eliminating PPR globally by 2030 [238] is to be met, international cooperation with institutions such as the OIE and FAO should be pursued [231], along with local initiatives to alleviate the problem. This analysis highlights PPR's endemicity in Pakistan, which has significant socio-economic consequences for Pakistan's pastoralists and agro-pastoralists, as well as the local economy [247]. The disease's spread and survival have been assisted by

uncontrolled animal movement, low vaccination coverage, mixing of herds/flocks from various farms/locations, and ill animals with healthy animals [249, 250]. Farmers, the Pakistani government, international agencies (such as FAO and OIE), researchers, and multinational veterinary pharmaceutical corporations must all work together to control and eradicate PPR in Pakistan. To arrest the spread of the virus and stop disease incursion into neighboring countries, an effective widespread/national vaccination campaign must be planned and implemented, along with policies aimed at improving disease awareness, improving diagnostics, surveillance, disease reporting, and controlling livestock movement; and to achieve the global goal of eradicating PPR by 2030, policies aimed at improving awareness of the disease, improving diagnostics, surveillance, disease reporting, and controlling livestock movement must be implemented.

9. Future Perspectives

Despite the fact that PPR vaccine manufacturing capability exists in two locations across the country, no structured PPR immunization campaign is currently underway. With a present population of over 90 million small ruminants and a PPR endemic scenario, there is a constant concern of PPR posing a threat to food security. PPR has been identified as the next target disease for management and possibly eradication by international animal health authorities (OIE and FAO). As a result, a nationwide PPR control programme in the country is urgently required. Following the successful eradication of Rinderpest, the OIE has designated PPR as the next candidate disease to be eliminated. As a result, substantial efforts have been made to better understand the disease and viable solutions for its elimination. Following the successful eradication of Rinderpest, the OIE has designated PPR as the next candidate disease to be eliminated. As a result, substantial efforts have been made to better understand the disease and viable solutions for its elimination.

10. Conclusion

Despite the fact that the FAO has begun a programme in Pakistan to gradually manage PPR, a comprehensive national programme to address this threat is required. Only the joint efforts of local and national authorities, as well as political will, as well as ongoing assistance and strengthening from international organizations, could achieve this.

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