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*Original Article*

# Investigation of the Clinical and Diagnostic Aspects of Peste des Petits Ruminants (PPR) in Sheep from the Southern Region of Iraq

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**Abstract**

In the southern region of Iraq, Peste des petits ruminants (PPR) has been identified and diagnosed. The study was done on (300) local sheep breeds of varying ages and sexes exhibiting PPR symptoms, while (25), healthy sheep breeds served as the control group. Additionally, the diagnosis of PPRV was confirmed by PCR. Infected sheep exhibit a variety of clinical symptoms. However, DNA sequencing was used to detect genetic links and genetic variation, and the results revealed a closed genetic relationship with the NCBI BLAST PPRV India isolate (GU014574.1) at total genetic variation (0.02-0.01%). Results indicate a large rise in PCV and ESR in conjunction with leukocytopenia and lymphocytopenia, a significant difference in clotting factor indices, and a significant increase in ALT, AST, and CK. In addition, there was a substantial variation in acute phase response. Postmortem examinations revealed various erosive lesions on the upper and lower gums, severe hemorrhagic enteritis, particularly of the small intestine, and obvious congestion of the lungs. Histopathological changes revealed an obvious flattening of the intestinal mucosa as well as an enlargement of the villi. In addition to a granuloma in the sub-mucosa, chronic inflammatory cells, primarily lymphocytes, were seen invading the mucosa. It has been determined that the sickness was circulating in the southern region of Iraq and severely afflicted sheep, which might result in significant economic losses owing to the detrimental effects of the virus that causes the disease on the various bodily parts.

**Keywords:** PPR, South part of Iraq, PCR

## 1. Introduction

Peste des petits ruminants (PPR) is a contagious illness of small ruminants caused by a virus that can result in mortality rates exceeding 50 percent (1). The illness is a significant restraint on the production of small ruminants, resulting in enormous economic losses and trade restrictions (2). Goats and sheep are both natural hosts of PPR, however goats tend to be more vulnerable and suffer from more severe clinical illness (3).

It was reported that Peste des petits ruminants was first described in Côte d'Ivoire in 1942. The disease is also known as 'goat plague,' 'Kata,''stomatitis-pneumoenteritis syndrome,' and 'ovine rinderpest' (4).

Peste des petits ruminants virus (PPRV) is the causative single-stranded negative-sense RNA virus that belongs to the subfamily morbillivirus of the family Paramyxoviridae (5). This virus was shown to encode eight distinct types of genome proteins, including the phosphor-protein (P), the nucleocapsid protein (N), the fusion protein (F), the matrix protein (M), the haemagglutinin protein (H), and the polymerase protein (L), in addition to the two non-structural proteins V and C. In addition, the causative

virus is one of the specific serotypes, but it can be divided into four distinct lines, from line I to line IV, primarily based on the protein (F) at a genetic level (6).

The illness has spread to the majority of African nations (with the exception of southern Africa), the Middle East, Eurasia (Bulgaria, Georgia, and Turkey), and Asia (4, 7, 8). It is an illness that has had an acute or perhaps chronic course. Important clinical manifestations of diseased animals include fever, ocular and nasal discharge that is predominantly purulent, stomatitis with erosive and/or necrotizing lesions, severe gastro-intestinal inflammation, diarrhea, bronchitis, and pneumonitis (2), in addition, the illness might cause abortion in pregnant animals, resulting in the death of newborns and increasing economic losses (9). The significance of the disease will depend on variables including the animal's species, the severity and virulence of the causative virus, the sexe and age of the animals, the immune status of the host, and the breed. However, previous exposure to the virus was also crucial (10). Furthermore, diseased animals (primarily sheep and goats) are the only source of the virus that causes the disease. Since saliva, ocular, and nasal discharges, as well as faeces, contained significant amounts of the viral antigen, these bodily fluids were analysed (11). Various techniques for the diagnosis of PPRV have been documented, including the detection of the antigen, viral isolation, nucleic acid amplification, indirect detection, and determination of virus-specific antibodies (7).

Little is known about PPR in the southern region of Iraq, and there is scant information available. Therefore, the purpose of this study was to conduct clinical and diagnostic research on the condition in southern Iraq.

## 2. Materials and Methods

### 2.1. Animals and Design of the Study

This study was intended to assess (325) local sheep breeds of varying ages and sexes. Three hundred indigenous sheep breeds with evidence of PPR were inspected and randomly selected from three provinces, Al –Basrah, Thiquar, and Mason province (one hundred animals (100) from each province), along with twenty-five (25) healthy animals that served as the control group. Both the sick and control groups were clinically assessed. In addition, laboratory testing was performed on their faecal samples using the normal procedure.

### 2.2. Blood Sampling and Hematological Evaluation

10 mL of blood from each animal was extracted from the jugular vein of each animal, 2.5 mL of the blood samples mixed with Ethylene diamine tetra acetic acid (EDTA) to measure the Erythrocyte count (RBC), the haemoglobin concentration (Hb), the packed cell volume (PCV), the total platelets, the mean platelet volume, the platelet distribution width, and the total leukocytes count (HEMATOLOGICAL ANALYZER, GENEX FROM USA). In addition, the differential leukocyte count was determined using the Giemsa stain technique (12). Another 2.5 mL of blood was combined with trisodium citrate (using plasma) in accordance with the examination of prothrombin time and activated partial thromboplastin time (BIOLABO, FRANCE). In addition, ESR is approximated using the Wintrobe technique, which Weiss and Wardrop (12) previously described.

### 2.3. The Estimation of the Acute Phase Response

It involves the assessment of haptoglobin (Serum Elisa Method), which was measured according to the manufacturer's instructions (BIOTECHNOLOGY CO - CHINA). Additionally, the evaluation of fibrinogen time was performed using plasma, as per the manufacturer's instructions (BIOLABO, FRANCE).

### 2.4. Histopathological Examination

Dead animals due to PPR were quickly subjected to post-mortem histopathological examinations. According to Cullen, Stalker (13) 1 $cm^3$ samples were extracted from the colon and immediately placed in a 10 percent buffered neutral formaldehyde solution. The histological specimens were then cleaned with tap water following 72 hours of fixation. After that, regular handling and processing was performed with a series of alcohols with concentrations ranging from 70 percent

to 100 percent absolute alcohol for two hours at each concentration in order to remove water from the processed tissues. In addition, xylol will be used during the clearing procedure. In addition, specimens were embedded using liquid paraffin wax at 58°C in two phases to create specimen blocks, which were sectioned at a thickness of 5 m for all tissues. Under a light microscope, all manufacturing tissues will be stained with the usual hematoxylin and eosin stain.

**2.5. Molecular Detection of PPR Viruses (PCR Technique)**

**2.5.1. Collection of Samples**

PPR viral RNA was isolated from sheep blood samples using GO TAQ® G2 Green master mix and AccuZolTM Total RNA extraction kit (BIONEER, KOREA) according to the manufacturer's instructions; finally, pedestals were cleaned and 1l of total RNA sample was pipetted for analysis.

**2.5.2. One-Step Reverse Transcription PCR (RT-PCR)**

One-step RT-PCR was performed for identification directly of ovine PPRV based on specific amplification of nucleoprotein genes. The RT-PCR was done according to the following steps:-

1- RT-PCR master mix preparation:

RT-PCR master mix was prepared by using (One-Step RT-PCR Premix Kit) and done according to the company instructions in two steps. Standard RT-PCR master mix components contain 5 µL of RNA template 5-100 ng, 1µL of N gene forward primer (10 pmol), 1 µL of Ngene reverse primer (10pmol), 8 µL of Ngene reverse primer (10 pmol) and 5 µL of DEPC water to total volume 20 µL. These RT-PCR reaction mixes were placed in sterile 0.2 ml PCR tubes and mixed and centrifuged for 3000rpm for 3 minutes by Exispin centrifuge after that transferred into PCR thermocycler.

**2.5.3. PCR Thermocycler Conditions**

PCR thermocycler conditions were set by using Optimise protocol writer and according to primer annealing temperature as in the following programmer, one cycle of Pre-Denaturation 95 °C 2 min., 35 cycles of Denaturation 95 °C 30 sec.; Annealing 59.3 °C 30 sec. and Extension 72 °C 1min. The final extension was 72 °C 5 min. for one cycle. The PCR products were analyzed by agarose gel electrophoresis, then visualized by using a UV transilluminator.

**2.6. DNA Sequencing Method**

RT-PCR products of positive nucleoprotein (N) gene for PPRV samples were sent to Macrogen Company in Korea for performed the DNA sequencing by (AB DNA sequencing system). The analysis of DNA sequencing was performed by using the phylogenetic tree UPGMA method (MEGA 6.0 version), Multiple alignment analysis based on ClustalW alignment analysis, and NCBI-BLAST for homology sequence identity. The positive PCR products (5 samples) of PPRV were sequenced (BIONEER, KOREA).

**2.7. Statistical Analysis**

The scientific data in the current study were statistically calculated and the statistical difference between infected sheep and the controls was estimated using a student t-test (14).

## 3. Results

Clinical signs of infection in the infected animals included sadness and loss of appetite in 88.6 %, erosive mouth lesions with foamy salivation in 77 %, diarrhoea and/or dysentery in 74.6 %, and foul-smelling mouth lesions. Additionally, 67.3 % of the sick sheep exhibit indicators of dehydration and weight loss. In addition, some infected sheep exhibit indicators of pneumonia, including coughing, increased belly respiration, the presence of severe and/or mucous nasal discharge, and evidence of conjunctivitis with ocular discharge in 57.6 % of affected animals. However, during a chest auscultation, an aberrant lung sound was heard (Table 1).

Additionally, during a clinical assessment, sick animals exhibit significantly higher values for their body temperature, respiration rate, and heart rate compared to the control group ($P$<0.05) (Table 2).

Moreover, the PCR technique is used to validate the diagnosis of PPRV (Figures 1-3). Given that all of the blood samples utilized in the current investigation tested positive.

**Table 1.** The clinical manifestations of sheep infected with PPR

| Clinical manifestations | Diseased sheep n=300 | % |
|---|---|---|
| Loss of appetite with depression | 266 | 88.6 |
| Erosive mouth lesions with frothy salivation and foul smelling | 231 | 77 |
| Diarrhea and / or dysentery | 224 | 74.6 |
| Dehydration and loss of body weight | 202 | 67.3 |
| Conjunctivitis with ocular discharge | 173 | 57.6 |
| Sings of pneumonia with abnormal lung sound | 132 | 44 |

* ($P$<0.05)

**Table 2.** Body temperature, respiratory and heart rate of diseased sheep with PPR and controls

| Clinical parameters | Controls n=25 | Diseased sheep n=300 |
|---|---|---|
| The body temperature C° | 38.45±0.32 | 41.8±1.43* |
| The respiratory rate/mint | 23.56±4.21 | 61.5±4.3* |
| The heart rate/mint | 83.23±6.87 | 112.8±16.3* |

* ($P$<0.05)

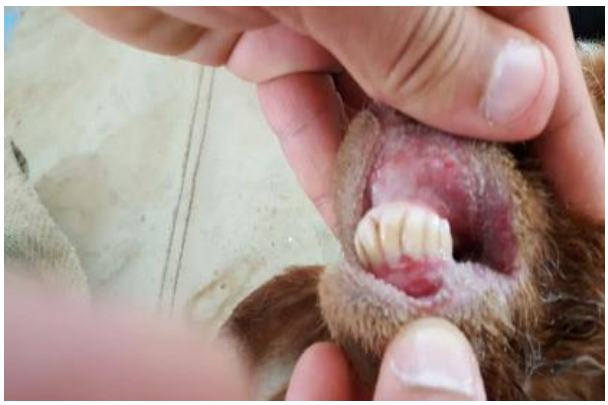

**Figure 1.** Erosive mouth lesions with frothy salivation

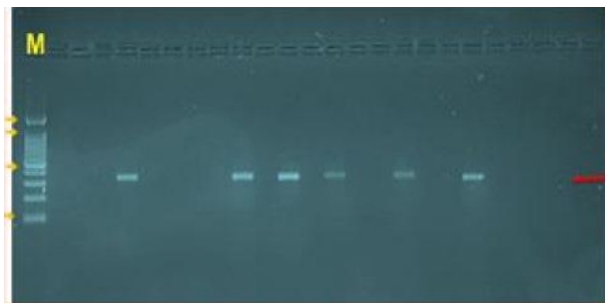


**Figure 2.** Agarose gel electrophoresis image showed Nested PCR product analysis of nucleoprotein (N) gene in Peste des petits ruminants virus from sheep blood samples. Where M: marker (1500-100bp). The positive PCR amplification samples were showed at (333bp) nested PCR product

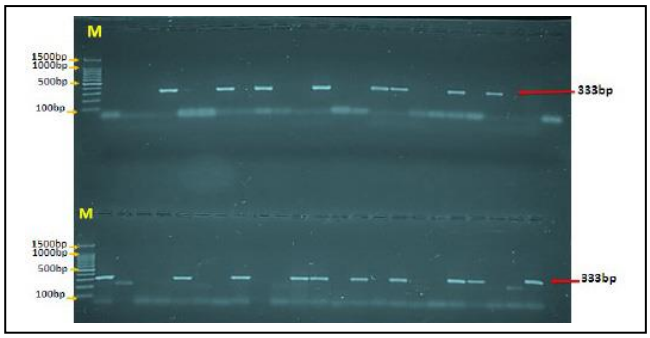


**Figure 3.** Agarose gel electrophoresis image showed Nested PCR product analysis of nucleoprotein (N) gene in Peste des petits ruminants virus from sheep blood samples. Where M: marker (1500-100bp). The positive PCR amplification samples were showed at (333bp) nested PCR product

### 3.1. DNA Sequence Results

The NCBI-Blast linked countries Peste-des-petits-ruminants (PPR) virus isolates and local Peste-des-petits-ruminants (PPR) virus isolates were used to uncover genetic relationships and genetic variation (substitution mutations) study in the viral nucleocapsid (N) gene.

The local PPRV isolates (IQN.No.1 - IQN.No.6) exhibited close genetic relationships to the NCBI BLAST PPRV of isolate from India (GU014574.1) at total genetic change (0.02-0.01 percent), according to the phylogenetic tree genetic relationship analysis (Figure 4).

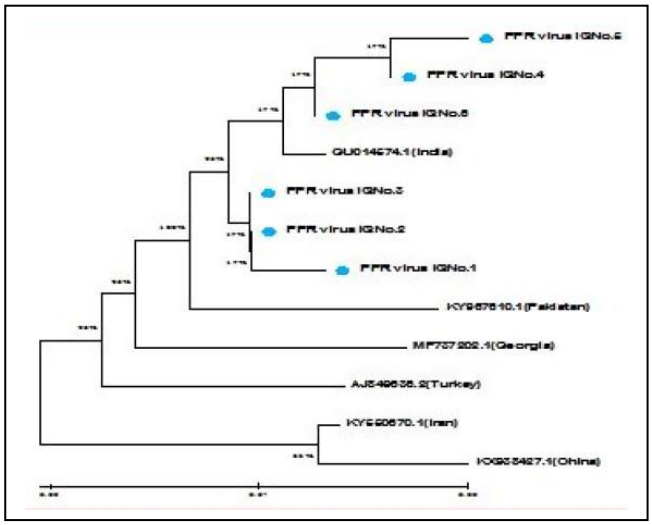


**Figure 4.** The phylogenetic tree genetic relationship analysis

Figure 4 shows a phylogenetic tree based on the partial sequence of the viral nucleocapsid (N) gene from local PPRV isolates that was used for genetic connection analysis. The Unweighted Pair Group Method with Arithmetic Mean (UPGMA tree) was used to create the phylogenetic tree (MEGA 6.0 version). The local PPRV isolates (IQN.No.1 and IQN.No.6) were identified as being genetically identical to the NCBI-BLAST PPRV India isolate (0.02-0.01%).

The NCBI BLAST related Peste-des-petits-ruminants (PPR) virus India isolates and local PPRV isolates (IQN.No.1 - IQN.No.6) showed genetic homology sequence identity varied from (98.94-99.65%) (Table 3).

One to three substitution mutations were detected in the nucleocapsid (N) gene of the NCBI-Blast related PPRV India isolate and local PPRV isolates (IQN.No.1 to IQN.No.6) in the genetic variation (substitution Mutations) investigation (0.35-1.06 %) (Table 4).

**Table 3.** The NCBI-BLAST Homology Sequence identity percentage between local Peste-des-petits-ruminants (PPR) virus isolates and NCBI-BLAST India closed related PPRV isolate

| PPRV isolate | Accession number | Homology sequence identity (%) | | |
|---|---|---|---|---|
| | | Country related NCBI | Accession number | Identity (%) |
| IQN.No.1 | | India | GU014574.1 | 99.30% |
| IQN.No.2 | | India | GU014574.1 | 99.65% |
| IQN.No.3 | | India | GU014574.1 | 99.65% |
| IQN.No.4 | | India | GU014574.1 | 99.30% |
| IQN.No.5 | | India | GU014574.1 | 98.94% |
| IQN.No.6 | | India | GU014574.1 | 99.65% |

**Table 4.** The NCBI-BLAST genetic variation analysis between local PPRV isolates and NCBI-BLAST India closed related PPRV isolate

| PPRV isolate | Accession number | Homology sequence identity (%) | | |
|---|---|---|---|---|
| | | Number Mutations | Type of Mutation | Mutation % |
| IQN.No.1 | | 2 | A/T, T/C | 0.70% |
| IQN.No.2 | | 1 | A/T | 0.35% |
| IQN.No.3 | | 1 | A/T | 0.35% |
| IQN.No.4 | | 2 | T/C , T/A | 0.70% |
| IQN.No.5 | | 3 | T/C, A/T, T/A | 1.06% |
| IQN.No.6 | | 1 | A/T | 0.35% |

The local PPRV isolates (IQN.No. 1 - IQN. 6) were subsequently submitted to the NCBI Gen-bank and assigned the accession codes Banklt2565840 Seq1 ON087834, Banklt2565840 Seq2 ON087835, Banklt2565840 Seq3 ON087836, Banklt2565840 Seq4 ON087837, and Banklt2565840 Seq5 ON087838.

On the other hand, results regarding the haematological changes in PPR-affected sheep showed a significant rise ($P \leq 0.05$) in PCV and ESR levels in affected sheep compared to controls. Additionally, when comparing infected lambs with PPR to controls, a significant ($P \leq 0.05$) lymphocytopenia and leukocytopenia were found. (Table 5).

PPR-infected animals have a considerable change in their clotting factor indices. As the total volume of the diseased sheep's platelets was significantly lower than that of the control group ($P \leq 0.05$), additionally, when comparing diseased sheep to controls, a significant increase ($P \leq 0.05$) was observed in the values of the platelet distribution width, mean platelet volume, clotting time, prothrombin time, and activated partial thromboplastin time (Table 6).

When compared to control healthy sheep, diseased sheep with PPR have a substantial increase ($P$0.05) in ALT, AST, and CK values (Table 7). In addition, the acute phase response values of ill sheep with PPR differ significantly ($P \leq 0.05$), Results show that the amount of haptoglobin and the fibrinogen time values are significantly higher ($P \leq 0.05$) in infected sheep than in controls (Table 8).

**Table 5.** Hematological changes of sheep infected with PPR and controls

| Parameters | Controls n=25 | Diseased sheep n=300 |
|---|---|---|
| RBC×$10^6$ | 7.65±1.45 | 7.58±1.33 |
| Hb g/dl | 11.67±1.35 | 11.96±2.32 |
| PCV% | 30.4±1.56 | 37.56±2.12* |
| ESR mm/24hr | 4.45±1.3 | 21.34±7.89 * |
| TLC×$10^3$ | 12.76±1.23 | 9.65±3.87* |
| Nutrophiles/absolute | 6876±734.55 | 6191.21±365.13 |
| Lymphocytes/absolute | 5320±121.11 | 3211.43±135.45* |
| Monocytes/absolute | 171±46.11 | 173±22.51 |
| Esinophiles/absolute | 203±22.45 | 201.12±51.21 |
| Basophiles/absolute | 32±3.21 | 33.11±12.33 |

* ($P$<0.05)

**Table 6.** Clotting factor indices changes of sheep infected with PPR and the control group

| Parameters | Controls n=25 | Diseased sheep n=300 |
|---|---|---|
| Platelet count/g/ L | 467.54±12.43 | 365.34±55.34* |
| The platelet distribution width % | 16.43±3.32 | 21.87±6.34* |
| The mean platelet volume/fL | 9.56±1.44 | 14.56±6.43* |
| Clotting time/mint. | 3.32±1.45 | 4.44±1.87* |
| Prothrombin time/Sec. | 14.87±1.54 | 18.12±3.65* |
| The activated partial thromboplastin time/sec. | 51.69±3.33 | 65.41±5.78* |

* ($P$<0.05)

**Table 7.** Some serum biochemical changes of infected sheep with PPR and control group

| Parameters | Controls n=25 | Diseased sheep n=300 |
|---|---|---|
| ALT (U/L) | 37.78±5.22 | 97.87±12.88* |
| AST (U/L) | 98.43±6.87 | 132.43±33.78* |
| CK (U/L) | 122.67±4.78 | 181.56±23.6* |

* ($P$<0.05)

**Table 8.** The acute phase response of diseased sheep and control group

| Parameters | Controls n=25 | Diseased sheep n=300 |
|---|---|---|
| haptoglobin g/dl | 0.022± 0.09 | 0.033.±0.003* |
| fibrinogen time/Sec. | 22.86±5.34 | 28.46±8.12* |

* ($P$<0.05)

The results of post-mortem exams show various erosive lesions on the upper and lower gums, severe hemorrhagic enteritis, particularly of the small intestine, as well as clear lung congestion. Additionally, in addition to the villi enlargement, histopathological alterations showed a clear flattening of the intestinal mucosa. Along with a granuloma in the sub-mucosa, chronic inflammatory cells, primarily lymphocytes, were also seen to be flooding the mucosa (Figures 5-7). However, sub-mucosal vascular congestion was also clearly visible.

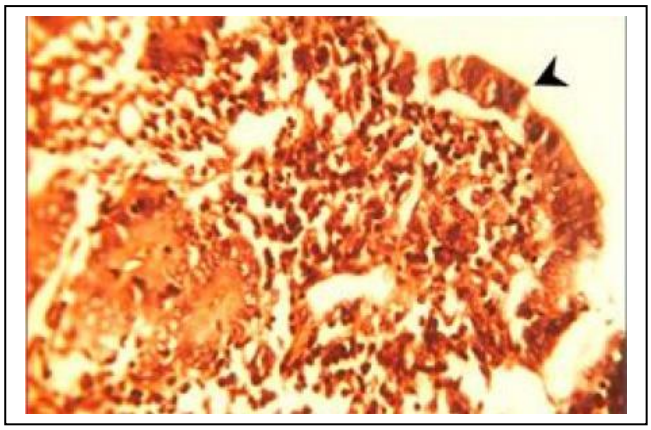

**Figure 5.** Section of sheep intestine with PPR shows flattening of the mucosa (black arrow) H&E 125X

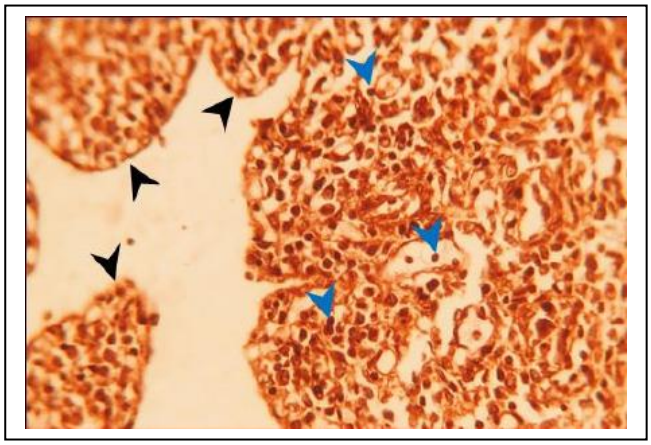

**Figure 6.** Section of sheep intestine with PPR shows swelling of the villi (black arrow), flooding of the mucosa by chronic inflammatory cells, predominantly lymphocytes (blue arrow) H&E 500X

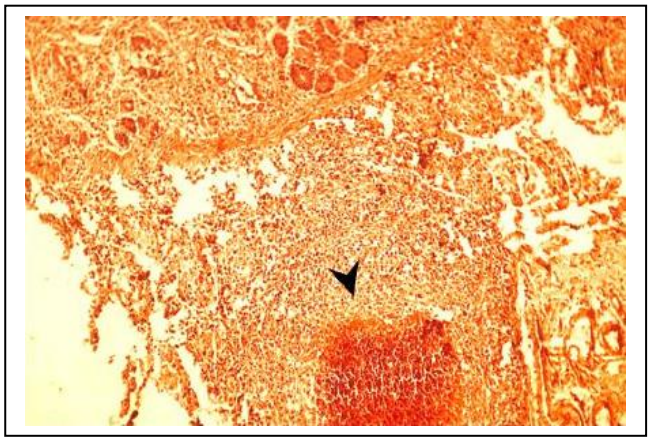

**Figure 7.** Section of sheep intestine with PPR shows granuloma in the sub-mucosa (black arrow) H&E 125X

## 4. Discussion

An acute viral illness called Peste des Petits Ruminants (PPR) is brought on by a Morbillivirus. The disease primarily affects sheep and goats, but goats are more frequently and severely affected than sheep (15). The illness is extremely contagious and affects susceptible groups with varied degrees of morbidity and mortality (1).

In the major southern provinces of Iraq (AL-Basrah, Thi-qar, and Mesan), the current study provided the first clinical report on the PPR virus in small ruminants based on molecular identification and histological diagnosis.

Animals with disease exhibit a wide range of clinical symptoms that have been observed and documented previously (1, 16). However, because of the likelihood for genetic change in the viral strains themselves, the disease's clinical indications might occasionally vary (17). Depending on the severity of the disease, scientists document the per-acute, acute, and mild types of the illness (18). Since, the severity of PPR in small ruminants may vary on a number of variables, including species and animal breed, virulence of the causative agent, immunological status of infected animals, and animal age (19).

Erosive stomatitis has been identified as one of the most prevalent clinical manifestations of disease in sheep, leading appetite loss and sadness in affected animals. Since, after viremia, the infected virus will target the mucus membranes of the mouth and after localization cause severe inflammation manifested by erosions (erosive stomatitis) with frothiness of the mouth and bad odor could also be detected, which leads to difficulty eating and chewing the food by the infected animal, which in turn could negatively impact the general health of the animal, concluded with general weakness and may also lead to a weakened immune system in general (20). In advanced cases of the PPR, the necrotic lesions may grow into a caseous deposit on the tongue, causing the animal to refuse to eat because of the pain caused by the active inflammation and the excessive production of frothiness (1). When the disease progresses, the intestinal mucosa is one of the important tissue centers that is targeted by the causative virus, causing severe intestinal inflammation and clear damage to the epithelial screcies. Furthermore, a powerful and virulent virus will cause more severe inflammation and tissue damage, which might extend to all layers of the intestine, leading to harmful effects and damage to the deep blood vessels, reflecting a hemorrhagic type of enteritis along the intestinal lyres (21). Moreover, dehydration and weight loss are undesirable outcomes of the diarrhea process in infected animals (10). In addition, the detection of a conjunctival affection in the affected sheep suggests acute conjunctivitis with ocular discharge, as well as auscultation of pulmonary rales, which indicates pneumonia with respiratory embarrassment in diseased animals. These signs are consistent with what was found by Constable, Hinchcliff (1).

Infected animals exhibited a considerable increase in body temperature, respiration rate, and heart rate compared to the control group, confirming the disease's acute phase. It has been shown that an increase in body temperature in infected animals may signify the liberation of endogenous pyrogens that cause pyrexia due to the lysis of body cells that excite the thermal regulating centres of the thalamus in brain tissue. In addition, a haemoconcentration suggested by an increase in PCV due mostly to severe diarrhea may reflect the considerable rise in respiratory and heart rate of infected sheep revealed by the present investigation (22).

It was demonstrated that the disease is quite similar to the virus that causes bovine rinderpest, is more infectious, and has severe negative effects on small ruminants (particularly goats and sheep), accompanied by high morbidities and mortalities (23). Transmission of the causative virus to clinically normal animals could occur through direct contact with infected animals and their contaminated materials, primarily

through the ocular, nasal, and oral discharges. However, the faeces of diseased animals carry a significant amount of the causative virus, and animal movement will play a significant role in increasing disease transmission (11).

It has been stated that a number of approaches have been developed for the detection of the causal virus, with the PCR technique, which identifies both the F and N gene primers, being the most widely used method for diagnostic and molecular epidemiological research to date (24). As well, the isolation of the virus and the application of the agar gel immunodiffusion technique could fail to identify the causal virus even if the samples were badly maintained. In addition, it was demonstrated that RT-PCR can indicate the likelihood of studying the relationship between strains of the causal virus for more precise molecular epidemiological research (25).

It was discovered that Real-time RT-PCR has numerous advantages over conventional RT-PCR; where it is more delicate and swift. However, it can be used in a closed, one-tube system that prevents cross-contamination during post-PCR sample preparation (26).

Three distinct PPRV genes, notably the fusion, nucleocapsid, and matrix protein genes, have been utilised as RT-PCR amplification targets (19). As with the test conducted by Couacy-Hymann and Bodjo (19), this assay attempted to amplify a segment of the N gene, as this is the most frequently abundantly produced mRNA in infections caused by the morbillivirus family.

Hemagglutinin (H) and Fusion (F) proteins, which are both exterior glycoproteins that protect the causal virus in infected animals, are regarded as attractive subunit vaccine candidates (27). The genetic relationships between PPRVs were investigated by comparing the nucleotide sequence of PCR products amplified from the F gene to identical sequences registered in the National Center for Biotechnology Information (NCBI) GenBank database (28). The proportion of positive samples detected by RT-PCR in this study was similar to that reported by Kerur, Jhala (28). A comparison of the genomes revealed a high degree of homology between the detected viruses, indicating that the N-type gene of these infectious agents has not undergone rapid mutations. Liu, Yang (29) (30) also mentioned similar finding. RT-PCR was used to amplify the fusion, nucleocapsid, and matrix protein genes, which are three distinct viral genes. Today, it has been reported that a number of trials targeting the N gene for PCR-based detection of causal virus yielded positive findings (31).

The results of the current investigation demonstrated a considerable increase in the Packed cell volume of sick sheep. This was consistent with Aikhuomobhogbe and Orheruata (32). As, this alteration may refer to the excessive loss of body fluids in diarrheic animals, as shown by the haemoconcentration state brought on by dehydration (1). In addition, ESR readings were considerably elevated in diseased animals compared to healthy sheep. As the highly elevated levels of ESR found in the current study of infected sheep may reflect the heightened inflammatory condition caused by PPR, particularly in the bowel of ill animals, Consequently, if an inflammatory reaction begins, Hyperfibrinogenemia will occur in the bloodstream, causing erythrocytes to clump and sediment rapidly (33).

The results also demonstrated an apparent Leukocytopenia caused by a considerable drop in the absolute number of lymphocytes (Lymphocytopenia) in infected sheep, as indicated by a fall in absolute lymphocyte count. This outcome may be assured due to the lymphoid tissue lesions, damage, and reduction of lymphocytic cells often found in the acute stages of the disease. However, prior studies have demonstrated that the PPR virus is found in Peyer's patches, enterocytes, spleen, thymus, lymph nodes, tonsils, and the liver (32).

The current investigation revealed a considerable increase in fibrinogen levels. Important plasma protein fibrinogen is assumed to be an acute-phase protein in all species (34). Consequently, the computation of this plasma protein may be especially valuable for identifying inflammatory disorders. Since it may be a

sign of multiple inflammatory disorders, such as endocarditis, enteritis, peritonitis, pneumonia, and nephritis. In addition, fibrinogen and haptoglobin have historically been regarded as reliable indicators of host inflammatory response.

Localization of the causative virus in the epithelial cells of the gastrointestinal tract has been shown to cause blunting and stunting of intestinal villi with congestion and desquamation, goblet cell hyperplasia, infiltration of inflammatory mononuclear cells in the lamina propria, and submucosal edoema. In addition, Squamous epithelial syncytia may be present in the epithelium of the digestive system following PPR infection.

## Authors' Contribution

Study concept and design: G. F. A. and K. M. A. S.
Acquisition of data: G. F. A. and K. M. A. S.
Analysis and interpretation of data: G. F. A. and K. M. A. S.
Drafting of the manuscript: G. F. A. and K. M. A. S.
Critical revision of the manuscript for important intellectual content: G. F. A. and K. M. A. S.
Statistical analysis: G. F. A. and K. M. A. S.
Administrative, technical, and material support: G. F. A. and K. M. A. S.

## Ethics

We hereby declare all ethical standards have been respected in preparation of the submitted article and accepted by the ethics committee of the University of Basrah, Basrah, Iraq.

## Conflict of Interest

The authors declare that they have no conflict of interest.

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