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Case report

The Outbreak Report of Peste des Petits Ruminants (PPR) in Wild Goats (*Capra Aegagrus*) in Chaharmahal and Bakhtiari Province (Iran), A Case Report

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Peste des petits ruminant (PPR) is a highly acute disease of small ruminants with high morbidity and mortality. The disease is caused by a single-stranded negative-sense RNA virus classified within the genus Morbillivirus in the family *Paramyxoviridae* (1, 2). This disease affects mainly sheep and goats, sometimes wild small ruminants and camels (3).

Recently, PPR becomes endemic across sub-Saharan Africa, the Middle East, the Arabian Peninsula, Turkey, Iran, Iraq, Pakistan, India, Bangladesh, Tajikistan, and Kazakhstan in Central Asia (4). Iran is one of the countries with a high incidence of the disease in the Middle East, and 93 flocks of sheep and goats were reported positive for PPR between March and September 2005 (5).

In the current outbreak report, we demonstrated the outbreak of a newly emerging acute disease in 29 wild goats of Chaharmahal and Bakhtiari province in the southwest of Iran caused by a PPR virus.

Recently, an outbreak of the fatal disease in 29 wild goats from the Tang Sayad area located at 15 km of Shahrekord city of Chaharmahal and Bakhtiari province southwest of Iran was reported to the office of department of environment. The dead wild goats are shown in figure 1.



Fig. 1. The wild goats dead from PPR disease

The animals included 29 individual wild goats with clinical signs of sudden death, oral erosion, and ecthyma-like lesions (Figure 2), severe diarrhea, bronchopneumonia, enlargement of lymph nodes, obvious dehydration, and dermatitis in infected animals. The history taking indicated a total of 29 deaths in the last six days.

After clinical examination, lymph nodes, ocular and nasal swabs were collected from death animals. Samples were left in cold boxes at 4°C and then were transferred to the laboratory for more evaluations. In the laboratory, the samples were centrifuged at 4000×g for 10 min and were kept at −20 °C until usage. RNA extraction from the swabs obtained from the animal, using RNA-Plus Solution (CinnaGen® Iran). RNA extracts were stored at -70°C until use.

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Fig. 2. Oral lesions in death wild goats

The synthesis of cDNA of PPRV was performed according to the manufacturer's procedures of AccuPower® Cycle Script RT PreMix (dN6) from all samples (Bioneer Corporation, Korea). RT products were cooled on ice and stored at -20 °C until used. For RT-PCR, oligonucleotide primers corresponding to N gene sequence were used. These primers were adopted from O.I.E. proposed primers with some modification. Upstream primer sequence: (NP4–19) 5'CCTCCTCCTGGTCCTCCAG3´. Downstream primer sequence: (NP3–17) 5'G-TCTCGGAAATCGCCTC3´ (6). This primer pair amplifies 352 bp region of N gene. The PCR was performed by AMPLICON master mix (Denmark) according to the manufacturer recommendation.

The main findings were severely dehydration, erosive and ecthyma-like lesions in the lips and gums, congestion and consolidation of the lung with pneumonic lesions and foam material in trachea. Paleness of the liver, enlargement and edematous of lymph nodes, and dermatitis with or without inflammation in some areas of skin particularly around limb were other findings.

These results demonstrate appropriate clinical criteria were applied for the sample collection procedure. RT-PCR of samples also confirmed that all samples were positive for PPR.

Further, for molecular detection, RT-PCR was conducted to amplify parts of N gene and to confirm this amplification, electrophoresis of RT-PCR products was performed and the expected band was observed at 352 bp.

Clinical signs, history taking, gross lesions, and laboratory findings confirmed the etiology of the observed condition in the wild goats in the affected region to be PPRV. PPR was first reported in 1995 in small ruminants from a western province in Iran (7). The disease was later reported from most provinces over a period of 10 years (5).

Latter studies revealed that the Iranian PPRV isolates belong to lineage IV and are closely related to the Pakistan, Saudi Arabia, Turkey, India, Tajikistan, and Chinese isolates (8, 9). Iran is bordered to the east by Pakistan and to the west by Turkey. PPRV infection has been reported in the southeastern Turkey, near the Iranian border (8). Sharing border between mentioned countries is probably the most important cause of PPRV transmission and spread in Iran. The disease occurs in summer after the arrival of domestic livestock in the area and the use of shared drinking water with wildlife.

In this study, Infection and mortality of males is much higher than females and more deaths have occurred in wildlife infants between the ages of one and three years.

One of the most remarkable clinical signs that were observed in the current outbreak was ulcerative keratitis and conjunctivitis which had previously been reported in wild small ruminants (10). In addition, RT-PCR revealed amplified sequences of PPR viral genome as well identified from isolates that were recovered from the samples. This is the outbreak case report of PPR in some wild goats in Iran which could be an important alarm for spreading the disease to other hosts if animal movements, quarantine, and regular vaccinations are neglected.

In spite of being endemic in Iran, outbreaks of PPRV are regularly occurring, and limited information is available on the genetic nature of PPRV. Proper understanding of this virus circulation will help PPRV endemic countries such as Iran develop effective control strategies. Therefore, phylogenetic studies are suggested to better understanding of this subject.

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Conflict of interest

The authors declare that they have no competing interests.

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