

CASE REPORT



First detection of myxomatosis in European brown hares (*Lepus europaeus*) in West-Hungary

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ABSTRACT

The authors were the first to identify myxomatosis in brown hares (*Lepus europaeus*) in a hunting area in western Hungary. During the necropsy of brown hares, dermatitis accompanied by multiplex pustule formation and keratinisation disorders were observed primarily on the head, eyelids, ears and skin around the mouth. Subacute keratoconjunctivitis and catarrhal-purulent rhinitis were confirmed during the autopsy. In addition, multiplex, nodular pneumonia and hemorrhages in the intertubular space of the kidneys, as well as tubulonephrosis, were also observed. Hyperkeratosis and parakeratosis were seen in the epithelium, along with the appearance of myxoma cells and heterophil granulocyte infiltration. The lesions found in the hares, such as significant swelling of the eyelids and keratoconjunctivitis, significantly impaired the animals' orientation. The clinical picture observed by the authors was preceded by infection with *Leporipoxvirus myxoma*, a member of the *Leporipoxvirus* genus. This virus strain belonging to the same phylogenetic group as the classic Lausanne, Cornwall, Sussex, and Munich strains previously described in rabbits.

KEYWORDS

European brown hare, *Lepus europaeus*, myxomatosis, *Leporipoxvirus myxoma*, myxoma cells

INTRODUCTION

Myxomatosis is a disease that occurs in wild European rabbits (*Oryctolagus cuniculus*) and their domesticated counterparts, domestic rabbits. The disease was first described in 1898. The causative agent is a *Leporipoxvirus*, which causes an asymptomatic infection in Brazilian cottontail rabbits (*Sylvilagus brasiliensis*) in South America and in Californian brush rabbits (*Sylvilagus bachmani*) in the USA. In Hungary, the disease was described in domestic and wild rabbits in 1959 (Rivers, 1930; Vetési, 1990; Varga et al., 2018). Brown hares (*Lepus europaeus*) were long considered resistant to the disease (Vetési, 1990). The disease was first reported in a brown hare population in England in 2014 and then in 2018. A new recombinant strain caused an epidemic in a population of Iberian or Granada hare (*Lepus granatensis*) in Spain and the pathogen was also found in Portugal. In the same year, the

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disease was also found in Corsican hares (*Lepus corsicanus*) in Sicily (Rossini et al., 2024; Carvalho et al., 2020).

Myxomatosis in wild and domestic rabbits occurs seasonally, when the primary mechanical vectors, various mosquito species, develop. However, independently from the season, rabbit fleas (*Spilopsyllus cuniculi*) and rabbit lice (*Haematopinus lyrocephalus*) can also transmit the disease (Vetési, 1990; Varga et al., 2018). In domestic rabbits, the main peak of the epidemic tends to occur in the autumn (Varga et al., 2018). During an epidemic in the Iberian rabbit population in Spain, it was observed that deaths began in mid-summer, in July, and after peaking between August and October, they suddenly stopped at the beginning of winter in January (García-Bocanegra et al., 2018). A correlation was found between environmental factors and mortality in connection with the outbreaks, which exerted its effect through the gradation of the mosquito population (Lázaro et al., 2023). The disease was characterised by high mortality rates in Iberian rabbit populations, which was as high as 56.7% during the first outbreak in southern Spain (García-Bocanegra et al., 2019). In the Iberian Peninsula, cases of disease in brown hares were sporadic (García-Bocanegra et al., 2019; Dalton et al., 2019). The first cases of myxomatosis in brown hares were identified in England in 2014 (Barlow et al., 2014), followed by its appearance in Spain, where infection is also known to occur in coexistent populations of European and Iberian hares living in the same area. Infections caused by the classical myxomatosis virus (MYXV) and the recombinant (ha-MYXV) strain have been proven in brown hares (Cardoso et al., 2025). The disease was also detected in closed farms in Spain, where the epidemic started in August and then peaked in September (Rosell et al., 2019).

It is known that in domestic rabbits, mosquitoes and possibly other blood-sucking parasites transmit the virus into the skin, where it undergoes the primary replication and then enters the regional lymph nodes, on the second or third day, where it replicates again. Around the fourth day after infection, the virus enters the lymphatic and blood circulation and after viraemia, characteristic lesions (multiplex, circumscribed, serous-proliferative inflammation) develop in predilection sites (primarily in the skin) (Kardeván, 1976; Vetési, 1990; Varga et al., 2018). After the generally observed 7-day incubation period, the classic clinical symptoms (appearance of serous vesicles and myxoma cells) associated with millet-sized myxomatous swellings and nodular, so-called dry (skin lesions without fluid) forms can be observed (Vetési, 1990). In Iberian rabbits, small lesions are formed in the skin, along with eyelid oedema and catarrhal conjunctivitis (Carvalho et al., 2020). In brown hares, the first description of the disease reports the appearance of proliferative lesions measuring 0.5–2 cm (Barlow et al., 2014).

In myxomatosis, the virus settles in the skin cells, causing them to degenerate and form small serous blisters (Vetési, 1990; Varga et al., 2018; Quesenberry et al., 2021). A characteristic feature of the disease is the formation of star-shaped cells with protruding cytoplasm, known as

myxoma cells, forming from mesenchyma cells in the subcutis and dermis, which are 16–50 µm in size. Accumulation of mucoid material is common in the corium layer (Rivers, 1930). Virus replication in the cytoplasm of myxoma cells is also associated with the formation of inclusions. Heterophil granulocyte infiltration is also evident in this process (Vetési, 1990; Kerr, 2012). In Iberian rabbits, bilateral blepharconjunctivitis, epistaxis and systemic oedema were mostly observed, while in domestic rabbits, characteristic myxomatosis foci (proliferation of histiocytes and formation of myxoma cells) (Kardeván, 1976) were not characteristic in this species (Agulló-Ros et al., 2022). In domestic and wild rabbits, it typically takes the form of well-defined inflammatory foci accompanied by cell proliferation embedded in mucoid material (Vetési and Dobos-Kovács, 2006). While this is less common in *Lepus* sp. species, such as the brown hare, in addition to the swelling of the basal epithelial cells in the epithelium, large inclusion bodies in the cytoplasm have also been reported (Barlow et al., 2014). The eyelid swelling observed in Iberian rabbits was caused by pronounced hyperkeratosis and ballooning of the epithelial cells (García-Bocanegra et al., 2019), which was also observed in the Czech epidemic on brown hares (Cukor et al., 2025).

MATERIALS AND METHODS

On October 1, 2025, two adult female hare carcasses were submitted to the Department of Exotic Animal, Wildlife, Fish and Honeybee Medicine from a hunting ground in western Hungary for pathological and diagnostic examination. Both animals were found next to a busy public road.

The hares were dissected according to professional standards and small pieces of organs showing lesions were fixed in a 10% buffered formaldehyde solution. We prepared 3–4 µm sections from the paraffin-embedded organ pieces, which we then stained with haematoxylin-eosin and PAS after mounting them on slides.

The samples were cultured from the subcutaneous connective tissue and nasal cavity of hare carcasses onto blood agar and Drigalsky culture media, which were incubated at 37 °C under aerobic conditions for 24 h.

In addition to the organs with visible lesions (such as the skin), small pieces of lungs, liver and spleen were added to 700 µL of 1x sterile PBS solution and homogenised using a TissueLyzer II (Qiagen, Hilden, Germany) instrument, then centrifuged at 3,000 rpm. Nucleic acid extraction was performed from 200 µL of supernatant using the MagCore Viral Nucleic Acid Extraction Kit 203 (RBCBioscience, Taiwan) and the MagCore II nucleic acid extraction machine (RBCBioscience, Taiwan). The sample was eluted in 60 µL RNase-free water and stored at –80 °C until PCR testing.

The presence of the myxoma virus genetic material was also tested using conventional PCR to rule out the presence of the Borghi and SG33 vaccine strains. For this purpose, the Phusion High-Fidelity PCR Kit (ThermoFisher Scientific, UK) was used, with the following composition for a 20 µL reaction volume: 4 µL 5× Phusion HF buffer, 0.4 µL 10 nM

dNTP, 0.2 μ L Phusion DNA polymerase, 8.4 μ L RNase-free water and primers at a final concentration of 0.5 μ M. For virus identification, Serp2_F: 5'-GTCGACGCTCTTCGTTTCTC-3', Serp2_R: 5'-ACTCTGGATGCGACGGTTAC-3' (Albini et al., 2012), and for vaccine strains, we used the primers Vax_F: 5'-ACAAGAATATACTAAAGAATACCACG-3' and M144_R: 5'-CCGCGTCGTGTCGATGATACA-3' (Cavadini et al., 2010). The reaction heat profile was as follows: 98 °C for 30 s, 35 cycles of 98 °C for 10 s, 63 °C/60 °C for 15 s, 72 °C for 15 s, followed by 72 °C for 5 min for final elongation (Albini et al., 2012).

In the positive samples, we tested for the presence of the partial sequence of the genes encoding the M070R and M071L coat proteins of the myxoma virus, which then was sequenced. For this, we also used the Phusion High-Fidelity PCR Kit (ThermoFisher Scientific, UK) with the primer pair M071_F: 5'-ACCCGCCAAGAACCACAGTAGT-3' and M071_R: 5'-TAACGCGAGGAATATCCTGTACCA-3' (Cavadini et al., 2010). After separating the PCR products by agarose gel electrophoresis, we cut out the amplicon of the appropriate length (~474 bp) with a sterile scalpel. The gel fragments were then purified using the Qiagen Gel Extraction Kit (Qiagen, Germany) according to the manufacturer's instructions. Sequencing was performed by Sanger sequencing at a commercial service laboratory (Eurofins Biomi Kft., Hungary).

The raw electropherograms were visualised using Chromas 2.6.6 software (Technelysium Pty Ltd., Australia), the reliability of the bidirectional sequencing results was compared and any errors and discrepancies were corrected. The sequences obtained were first identified using BLASTn (NCBI) online software and then the representative M070R–M071L protein-coding sequences available in GenBank were collected. Sequence alignment was performed using the

MAFFT 7 online software with the E-INS method (Katoh and Toh, 2008). Maximum likelihood (ML) analysis and phylogenetic tree reconstruction were performed using MEGA X software (Kumar et al., 2018), with the appropriate model selected using the MODELS setting (Nei and Kumar, 2000) and bootstrap values determined based on 1,000 repetitions. The phylogenetic tree was visualised and edited using MEGA X software.

RESULTS AND DISCUSSION

The two female brown hares (Stroh-negative, i.e., older than one year) were well developed and in good nutritional condition. Their bodies were covered with fur typical of the species. In both animals, the skin around the eyes, nose, mouth and anus was swollen, with millet- and hempseed-sized swellings visible in places. Millet-sized, dense, dark gray nodules were visible on the skin of the auricle (Fig. 1A).

The skin and hair around the mouth were contaminated with saliva and a slight watery discharge was also observed. Apart from the serous exudate, no pathological abnormalities were visible on the lips. The teeth, hard and soft palate, gums and tongue were found to be free of pathological abnormalities during necropsy.

The fur and skin around the nostrils of both animals were covered with a reddish discharge and the skin was oedematous and inflamed. The eyelids were closed, partially stuck together, difficult to open during examination, the eyeballs with a watery, inflamed discharge. Pinpoint haemorrhages were visible on the inner surface of the eyelids in the bright pink, moist, shiny mucous membrane. We found a small amount of catarrhal, stringy discharge in the corner of the eye in both individuals (Fig. 1B–D). The auricle was of



Fig. 1. Skin lesions in myxomatosis in a European brown hare. A. Swelling of the nasal region and eyelids with tiny nodules observable on the auricles (arrow) in a European brown hare, B. Mucous discharge in the corner of the eye and nodules on the edge of the eyelid (arrow), C. Swollen eyelids with severe oedema, D. Petechial haemorrhages (arrow) in the mucosa of the oedematously infiltrated eyelid, E. Hyperkeratosis and oedema in the epithelium of vulva and anus

normal shape and size and the ear canal was free of pathological abnormalities, except for dark gray nodules visible on the skin.

The fur around the perineum was intact, but the vulva was grayish brown, uneven, dry, covered with flaky skin and appeared slightly swollen in both individuals during the examination (Fig. 1E). The nipples were intact and free of lesions. A similar abnormality was observed in the area around the anus as in the perianal area. The claws, footpads and areas between the toes were intact, but the skin of the feet, especially the hind feet, showed millet-sized, dark gray, dry, thickened patches covered with dry skin.

When examining the subcutaneous connective tissue, regional lymph nodes, and skeletal muscles, no other significant abnormalities were observed except for bloody indentations on the rib cage on both sides of the chest wall in both animals. After opening the abdominal cavity, normal peritoneum was observed in both hares and the position of the abdominal organs showed no pathological abnormalities. Partially clotted blood was observed in the abdominal cavity of one female hare, where the blood clot was partially attached to the cracked surface of the liver capsule.

The spleen showed no pathological abnormalities in either animal. The stomachs of both animals were partially filled with well-chewed, slightly sour-smelling, mushy contents. No lesions were observed on the gastric mucosa and the intestinal tracts of both hares appeared normal. The liver of one individual was intact and free of lesions, while in the other individual, which had partially clotted blood in its abdominal cavity, the liver capsule, which was of normal shape and size, showed longitudinal discontinuities on the diaphragmatic surface.

The renal and urinary tracts showed no pathological abnormalities. The reproductive organs were calm and inactive, free of lesions, with one female having two grayish-black speckled bands on the right uterus and three on the left, while the other had four on each side.

After opening the chest cavity, partially clotted blood was visible in the free thoracic cavity of both female hares. The pericardium and heart of neither of the hares deviated from the normal autopsy findings. The lungs were normal shape and size but were dark red mottled on the costal surface and otherwise free of pathological abnormalities in both animals.

During the necropsy of the nasal cavity of the two hares, the mucous membrane of both animals was severely inflamed, veined, sticky, mucous and covered with debris (Fig. 3A). No abnormalities from the normal findings were observed in the cranial cavity or brain of either animal.

No pathogenic bacteria were cultured during bacteriological examination of the subcutaneous connective tissue of the hares' eyelids. Aerobic culture of samples taken from the nasal cavity revealed mixed bacterial flora.

The PCR testing of organ samples taken from the liver, spleen and kidneys of the two hares to detect calicivirus and rabbit haemorrhagic disease virus (RHDV) genetic materials yielded negative results. The presence of myxoma virus was

detected in all tissues tested using PCR. The PCR method designed for the SG33 and Borghi vaccine virus strains yielded negative results.

Macroscopic examination of the hares' intermediate coat did not reveal any signs of ectoparasites (ticks, fleas) during thorough examination of the fur.

With histopathological examination, pronounced cornification disorders were observed in the skin epithelium around the body orifices, including hyperkeratosis and parakeratosis, resulting in keratinised layers up to several tenths of a millimeter thick on the epithelium (Fig. 2A and B). In some areas of the epithelium, in the cells of the stratum basale, but also in the other layers, cytoplasmic vacuolisation and significant enlargement and rounding of the cells were observed as signs of ballooning degeneration. In these cells, the nucleus was also enlarged and pronounced perichromasia was observed (Fig. 2A and B). The dermis was thickened and loosened, with clusters of sprouting fibroblasts visible in places in the watery matrix. Some cells in the epithelial layer were unusually rounded and their nuclei contained chromatin of heterogeneous density, partly condensed to the nuclear membrane. Signs of varying degrees of vacuolisation were also visible in the cytoplasm of these cells, indicating signs of ballooning degeneration. In the dermis, PAS staining revealed large PAS-positive cells among the loosened fibers in the watery matrix, among which a significant number of heterophil granulocytes could also be observed in places. The disintegration of some of these PAS-positive cytoplasmic cells was also observed, while in the intact cells, the cell nucleus was visible in a pericytoplasmic location (Fig. 2C). In some places, these cells contain nearly spherical, bright red inclusion bodies that were larger than the cell nucleus and slightly displaced toward the periphery, surrounded by a light-coloured halo (Fig. 2C). In these cells, the perichromasia of the cell nucleus was more pronounced than in those where no cytoplasmic inclusion was visible. In the oedematous, filtered layer of the subcutis, the nuclei of the endothelial cells were significantly enlarged, bulging into the lumen of the vessel and the cytoplasm, having lost its flattened shape, also bulged into the cavity, where the basement membrane also thickened and lost its regular course (Fig. 2D). Inflammatory cell proliferation, perivascularitis, could also be observed around these vessels.

The liver basically retained its normal tissue structure in the hares examined, but multiplex haemorrhages could be observed around the smaller bile ducts, along with cell proliferation indicating mild inflammation. In some cases, pronounced vacuolisation of the cytoplasm was also observed in individual liver cells, where intense perichromasia was also evident in the nuclei of the affected cells (Fig. 3D). In both hares, the glomeruli showed a basically normal structure, but in some podocytes, kariomegaly was observed with homogeneous, even chromatin distribution in the kidneys. In the tubules, however, the degeneration of the tubular epithelium and its detachment from the basement membrane was characteristic in the sections. In some places, haemorrhages could be observed in the intertubular space

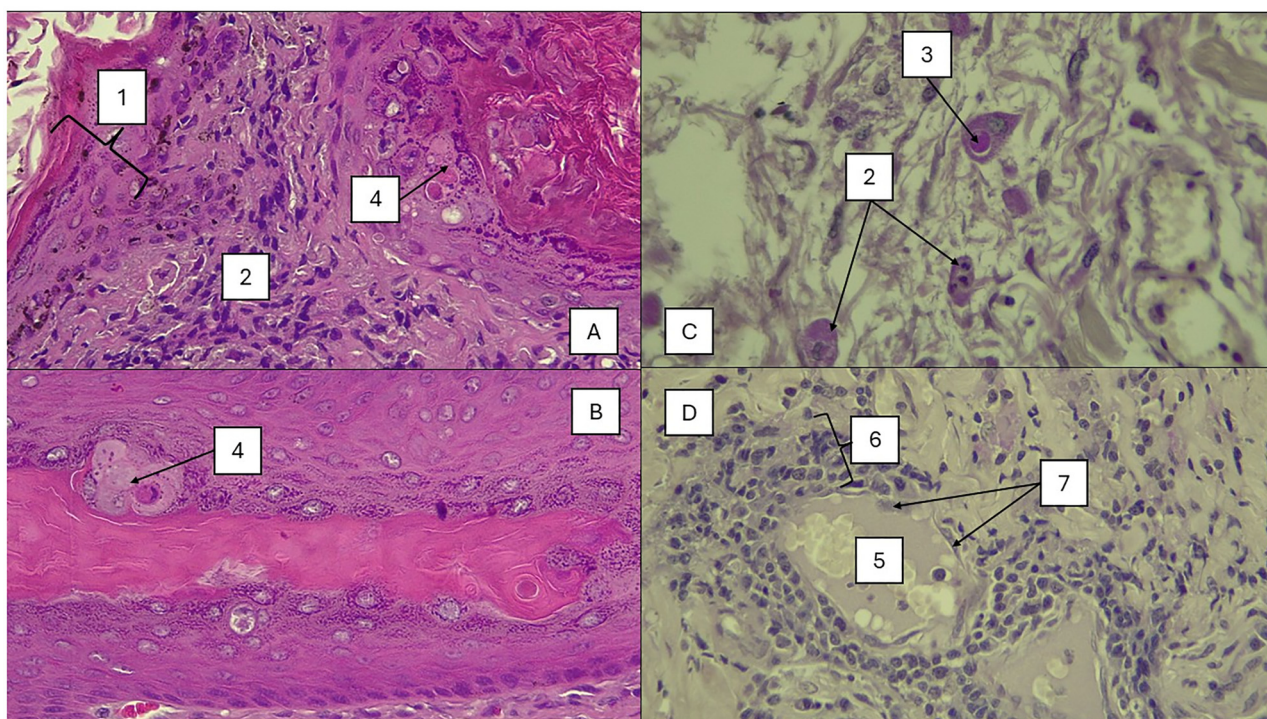


Fig. 2. Microscopic skin lesions in myxomatosis in a European brown hare. A. Alongside ballooning degeneration and keratinisation disorders (hyper- and parakeratosis) of certain epithelial cells, myxoma cells appear in the subcutaneous connective tissue (Staining: hematoxylin-eosin, magnification: 200 $\times$), B. Ballooning degeneration in the epithelial layer (Staining: hematoxylin-eosin, magnification: 200 $\times$), C. Intracytoplasmic inclusion body in the myxoma cell (Staining: PAS, magnification: 400 $\times$), D. Perivascularitis and degeneration of endothelial cells (Staining: PAS, magnification: 200 $\times$), 1. Hyper- and parakeratosis, 2. Myxoma cells, 3. Intracytoplasmic inclusion body, 4. Ballooning degeneration, 5. Lumen of blood vessel, 6. Perivascularitis, 7. Endothelial cell degeneration

(Fig. 3C). Although there were no macroscopic signs of inflammation in the lungs of the hares, the sections showed signs of multiplex, focal interstitial pneumonia, with inflammatory cells filling the space between the alveoli and compressing the air sacs in the affected area. The alveolar spaces in the unaffected, adjacent regions were intact. The pulmonary blood vessels, especially in the inflamed areas, were dilated and filled with red blood cells (Fig. 3B).

Based on macroscopic and histopathological changes in the two female brown hares submitted for diagnostic examination, as well as PCR testing of organ samples, we diagnosed the acute form of myxomatosis caused by the myxoma virus (sp. *Leporipoxvirus myxoma*). This disease, previously known only in wild rabbits and their domesticated variants (Vetési, 1990; Varga et al., 2018; Rosell et al., 2019; Agulló-Ros et al., 2022), has been detected in different species of the genus *Lepus* throughout Europe in recent years (England (Barlow et al., 2014), Spain (García-Bocanegra et al., 2018; Lázaro et al., 2023; Rosell et al., 2019), Portugal (Carvalho et al., 2020), the Czech Republic (Cukor et al., 2025) and Italy (Rossini et al., 2024)). The disease has spread rapidly across the continent since it was first detected in Spain (Dalton et al., 2019; Carvalho et al., 2020) and our diagnostic tests were the first to confirm its occurrence in Hungary.

After feeding on blood, the mosquitoes infect their hosts and a few days later, lesions develop on the skin, primarily on the head (eyelids, around the nostrils and on the skin of the ears), but also around the anus and on the skin of the genitals. Unlike the lesions characteristic of the classic form in domestic rabbits (Vetési, 1990; Varga et al., 2018; Agulló-Ros et al., 2022), the formation of blisters is not as common in brown hares. Instead, the carcasses showed oedema and swelling of the affected skin areas (Fig. 1). The brown hares examined showed hyperkeratosis and parakeratosis, the appearance of myxoma cells in the skin and the formation of cytoplasmic inclusions in these cells (Figs 2 and 3). Other histological changes observed by us, such as inflammatory cellular infiltration in the skin, perivascularitis around small blood vessels in the skin and endothelial cell degeneration, had also been observed in domestic rabbits and other species of the genus *Lepus* (Vetési, 1990; Varga et al., 2018; García-Bocanegra et al., 2018; Lázaro et al., 2023; Rosell et al., 2019; Agulló-Ros et al., 2022).

In our case, the death of the hares examined may have been caused by a mechanical trauma (presumably a collision with a motor vehicle - coagulated blood was observed in the chest cavities of both animals), which could have been a result of the substantial, oedematous infiltration of the eyelids potentially impairing the animals' orientation.

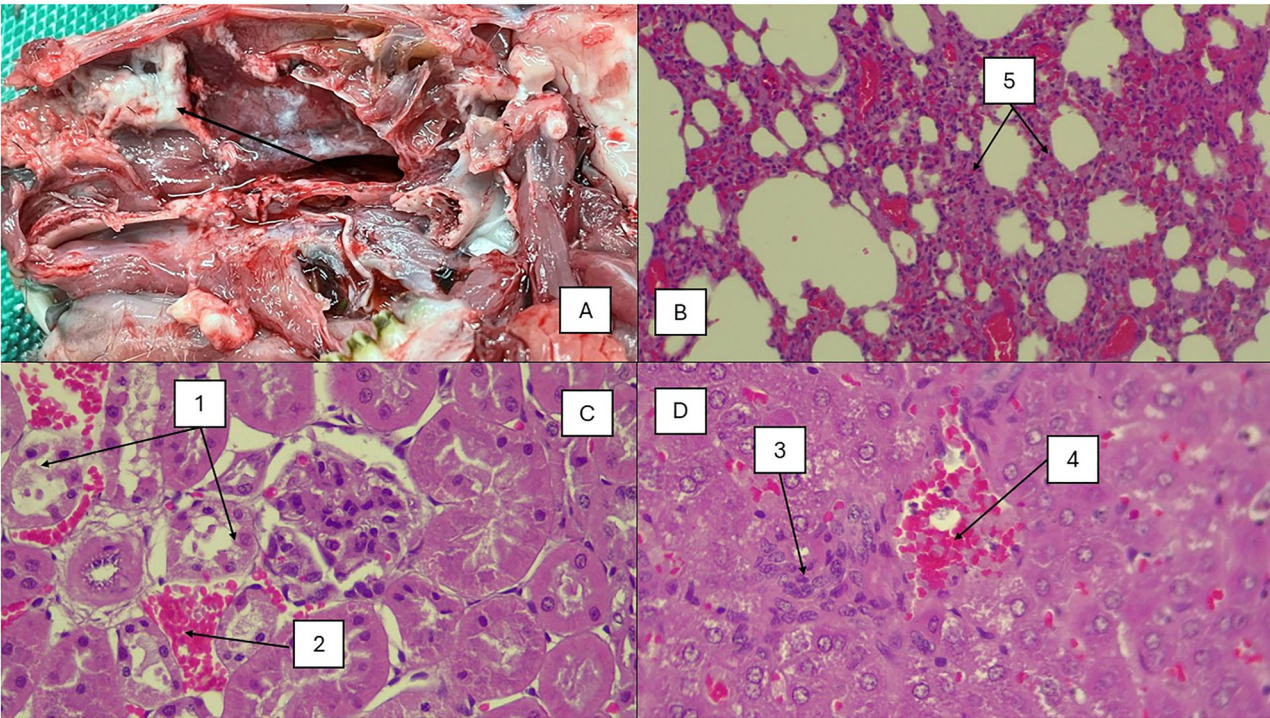


Fig. 3. Lesions in other organs. A. Acute catarrhal-purulent like rhinitis in the nasal cavity, B. Subacute interstitial pneumonia (Staining: hematoxylin-eosin, magnification: 100×), C. Tubulonephrosis and multiple haemorrhages in the intertubular space (Staining: hematoxylin-eosin, magnification: 400×), D. Subacute inflammation around the bile vessels and hemorrhage in the liver (Staining: hematoxylin-eosin, magnification: 400x), 1. Tubular epithel cell degeneration, 2. Bleeding in the intertubular space, 3. Perivascularitis, 4. Pericapillar bleeding, 5. Thickened interstitium

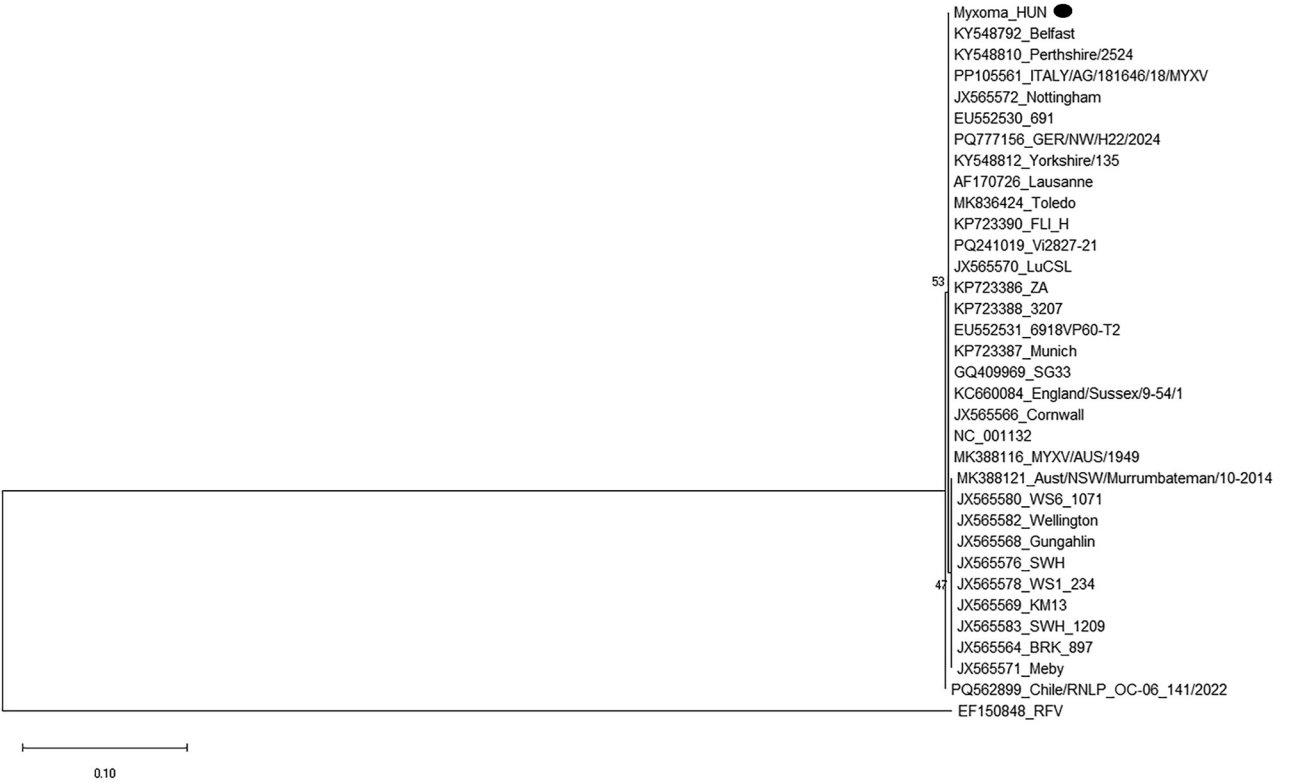


Fig. 4. Phylogenetic tree based on the comparison of reference and the Hungarian M070R–M071L coding sequences of myxoma virus downloaded from GenBank. The envelope sequence of rabbit fibroma virus (RFV) was used as an outer group. The comparison was conducted using the Maximum Likelihood method, and the resulting bootstrap values are displayed above the branches as percentages. The scale indicates 0.1 expected changes at specific locations and branches. The coding of strains appearing on the phylogenetic tree is as follows: GenBank accession number PX475730 name of the strain.

Based on the analysis of the genetic material of the virus isolated from hares, we showed that the pathogen responsible for myxomatosis is the *L. myxoma* virus (Fig. 4). Based on partial sequence determination of the capsid protein, it belongs to a phylogenetic group with the classic Lausanne, Cornwall, Sussex and Munich strains described in European wild rabbits. In the future, we plan to determine the complete genome of the identified strain, which will provide a more complete picture of the virus genome through phylogenetic analysis.

There is little data available on the impact of the disease on populations, the regeneration of the stock following an epidemic and the immune status of hares that survived the disease and reproduced in the following season. For this reason, we have also started to study the population dynamics effects of the disease.

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