Disseminated Idiopathic Myositis in Young Marshall Farms Ferrets

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An apparently new, severe, and previously unknown illness has recently been seen in various parts of the United States, affecting young ferrets from Marshall Farms, usually between the ages of 5 and 10 months. The illness can occur in isolated ferrets that have lived alone for months, or in multiple ferret households, wherein the other ferrets in the house seem (so far) to be unaffected. The disease strikes rapidly, and most cases reported have been fatal. This article discusses the general appearance of the illness and possible causes and treatments.

DEFINITION

Disseminated Idiopathic Myositis is a descriptive term for what the disease does: "disseminated" means widespread; "idiopathic" means "of unknown origin or cause"; and "myositis" means muscle inflammation. This illness causes widespread muscle inflammation and damage, and no known cause has been found. Other tissues can also be affected, but the inflammation seems to focus especially on muscles. In our practice, we have seen four cases of this disease in the past three months. Signs typically are lethargy, fever (often over 104° F), weakness and reluctance to move, sometimes pain when handled (especially over the back or hips). Appetite may be depressed. Some patients have external lymph node swelling, sometimes in a single node, such as in a leg or in the neck area.

The blood test results in these ferrets are unusual, and provide the strongest initial evidence of this disease. The CBC is particularly abnormal, with extremely elevated white cell counts, far above what most ferrets would produce even with severe infections. The white cell counts in our patients have varied from around 14,000 to nearly 60,000, with most cells being neutrophils, not lymphocytes. Some patients show a mild to moderate anemia, which initially may be nonregenerative but can become strongly regenerative (many red blood cells being produced). The combination of high WBC and regenerative anemia can superficially resemble autoimmune anemia. However, there is usually no elevated bilirubin in the blood (which would be expected if the immune system was destroying red cells). Another form of immune-mediated anemia can occur if the immune system damages the bone marrow, but the anemia in those cases is nonregenerative (no new red cells being made).

The serum chemistry tests are usually unremarkable, though one of our four patients had elevated ALT suggesting liver damage (hepatitis). Surprisingly, although this disease causes extensive muscle inflammation, CK values (which detect muscle damage) are usually fairly normal in these patients.

When an external lymph node is enlarged, biopsy of the node shows suppurative to granulomatous inflammation, with no infectious organisms seen microscopically. Bacterial cultures yield no growth. Complete necropsy and microscopic exam of deceased patients shows widespread suppurative inflammation with the muscles of the body being the most heavily targeted. The esophagus and skeletal muscles seem particularly hard-hit by this disease, but even the heart and gastrointestinal muscles are affected. Some non-muscular organs such as liver can be affected, as well. Again, no infectious organisms such as bacteria, rickettsia, etc., have been seen in any of the affected tissues, even with special stains applied. Electron microscopy has also failed to find a visible infectious agent, including viruses.

"Suppurative" inflammation (involving neutrophils as the primary white blood cell in the inflamed tissues) is typical of bacterial disease rather than viral. The high neutrophil counts in the blood would also fit severe bacterial disease. However, one would expect to see such organisms with microscopic exam of the tissues in most cases. Also, these patients routinely fail to improve (other than briefly) on any antibiotic combinations we have tried, including penicillins, cephalosporins, tetracyclines, quinolones (such as Baytril®), metronidazole, or chloramphenicol. Brief improvement on antibiotics might be due to treating a secondary bacterial infection, which took advantage of the already-weakened patient.

CAUSE OF THE DISEASE

We are forced to conclude that a likely cause of the disease is viral. An immune mediated disease attacking the muscles isn't out of the question; however, none of our cases have improved when given high dosages of cortisone. An immune deficiency (inherited?) which shows up in young adults could be hypothesized, but if a ferret were overwhelmed by infection due to immune deficiency, then infectious (bacterial) organisms should be

A new illness seems to be targeting young Marshall Farms ferrets; encourage your veterinarian to become familiar with the symptoms.

PATTY ASHEUER

easily found in the tissues, which is not the case here. One piece of evidence favors the viral theory: one of our patients survived and initially made a good recovery after using Interferon, an antiviral drug. But this drug can affect immune function and other disease processes, too, so it isn't certain why the drug seemed to produce improvement. (More recently, this patient relapsed after weeks of normal behavior, and the white cell count is again above 40,000.) Other possibilities include a myelocytic leukemia (cancer of the neutrophils), or a genetic defect involving the white cell line in the bone marrow (similar diseases have been reported in dogs).

DIAGNOSIS AND TREATMENT

A presumptive diagnosis is made based on physical signs combined with an extremely high white cell (neutrophil) count; definitive diagnosis can be made via biopsy of external skeletal muscle (such as on a leg) or via complete necropsy after death. Treatment should include supportive care (hand feeding and IV fluids if the patient is very ill); broad-spectrum antibiotics, such as Baytril® and amoxicillin, may minimize secondary infections. Orally administered Interferon-alpha (600 I.U. daily) may be started and maintained for several weeks at least. The duration of the illness isn't known. If the patient responds to treatment, it may be advisable to use Interferon for at least two months.

CONCERNS

We have some concerns. Is this disease is due to an infectious organism? If so, is it contagious to ferrets other than young Marshall Farms animals? If the animals were infected while at the breeder facility, why does it not show up earlier? Why have other ferrets that have been exposed to these pets not become affected?

It is possible that this illness is initially slow to develop, taking months before symptoms appear. This could explain why affected animals are young adults rather than kits; it could also mean that other exposed ferrets may develop symptoms months after exposure to a sick ferret. Only time will tell; within the next 6 to 12 months it will become obvious if this illness has spread to other exposed ferrets in multiple-pet households. We hope not, as this disease is very serious and usually fatal. Our apparent success with Interferon in one patient has yet to be duplicated in other ferrets. Perhaps our patient's initial recovery was spontaneous and not due to the medication.

We do not have a complete understanding of this illness, so a definitive paper cannot be written at this time. With increased recognition, it will be easier to track how many cases of this illness are being seen around the U.S.

We produced this article to enable ferret owners and their veterinarians to become aware of this new illness, possibly leading to the discovery of more cases and ultimately to an increased success rate in treatment and cure.

Veterinarians are encouraged to contact Dr. Burgess or Dr. Ramsell at burgess@swanimalhospital.com or 503-643-2137 if they see a similar case.