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# Pathology in Practice

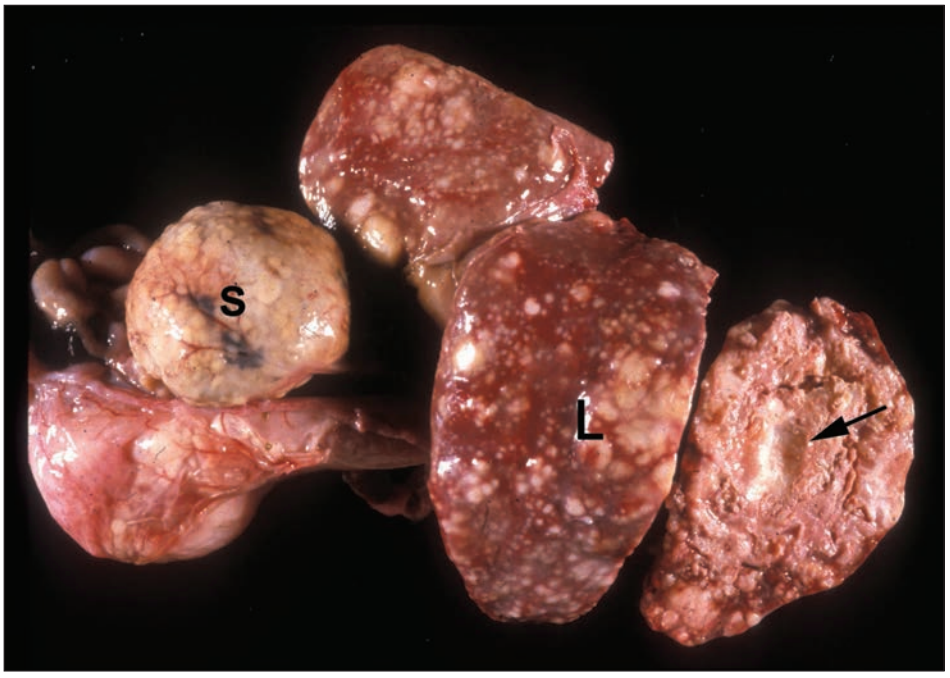


Figure 1—Photograph of the liver and spleen from an eastern screech owl (*Megascops asio*) that was found dead with no signs of trauma. The serosal surface of the liver (L) is covered with multifocal to coalescing, slightly raised, tan-white nodules that vary in size from pinpoint foci to roughly 2 cm in diameter. On cut section (far right), some nodules have necrotic centers (arrow). The spleen (S) is enlarged and largely replaced by coalescing tan-white nodules.

## History

A 95-g (0.21-lb) female eastern screech owl (*Megascops asio*) was found dead in Glynn County, Ga. The bird was either subadult or adult on the basis of feathering and time of year. It was submitted to the Southeastern Cooperative Wildlife Disease Study, College of Veterinary Medicine, University of Georgia, for testing for West Nile virus (WNV) infection and necropsy. There was no evidence of trauma, and no other dead birds were found in the immediate vicinity.

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## Clinical and Gross Findings

The bird was severely underweight with markedly decreased pectoral muscle mass; no other remarkable external lesions were identified. Visceral adipose tissue was also markedly decreased, and approximately 1 mL of free serosanguineous fluid was present in the coelomic cavity. The left side of the liver was approximately 4 times the size of that organ in a healthy eastern screech owl. Several tan nodules, ranging in size from pinpoint foci to roughly 2 cm in diameter, distorted the surface of the liver (**Figure 1**). The cut surfaces of some of these masses were firm and solid, and others contained necrotic or red fluid centers. The spleen was enlarged to approximately 10 times what would be considered normal size and composed of coalescing pale nodules. On cut surface, most of the spleen was replaced by caseous material.

**Formulate differential diagnoses from the history, clinical findings, and Figure 1—then turn the page →**

## Histopathologic Findings

Sections of liver, spleen, lungs, heart, kidneys, brain, and gastrointestinal tract were processed routinely for histologic evaluation; major pathological changes were limited to the liver and spleen. Approximately 75% of the hepatic parenchyma was replaced by variably sized, circumscribed, expansile nodules (**Figure 2**). These nodules consisted of aggregates of neoplastic myelocytic cells separated by a fine fibrous stroma. Nodules had foci of coagulative necrosis, especially near the center of the larger nodules. Although marked pleomorphism was present, the neoplastic cells were generally larger than typical myelocytes and contained brightly eosinophilic spherical granules within the cytoplasm. Nuclei were round, deeply basophilic with condensed chromatin, and eccentrically positioned. Moderate anisokaryosis was present. There were 3 to 5 mitotic figures/ten 400X fields. The splenic parenchyma was almost completely replaced by clusters of similar neoplastic myelocytic cells and necrosis.

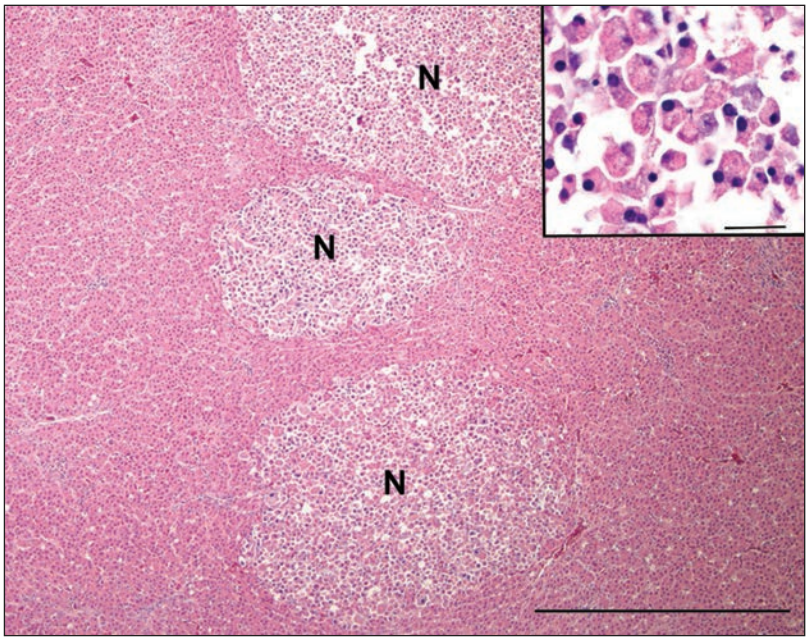


Figure 2—Photomicrographs of a section of the liver from the screech owl in Figure 1. Notice that well-circumscribed, unencapsulated, expansile masses of variable sizes have replaced much of the hepatic parenchyma (N). These masses are composed of myelocytes that vary in degree of maturity. H&E stain; bar = 500 µm. Inset—Higher-magnification image of neoplastic myelocytes with abundant cytoplasm filled with eosinophilic granules. H&E stain; bar = 20 µm.

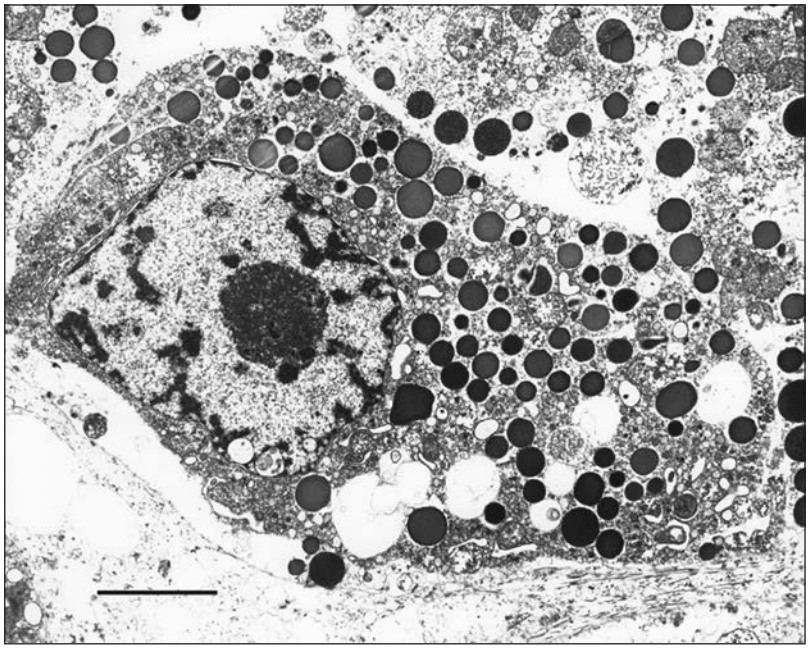

Figure 3—Transmission electron micrograph of a nodule-bearing section of liver from the screech owl in Figure 1. In this image, a neoplastic myelocyte with heterogeneous granules of variable size (maximum dimension, 0.25 to 1.9 µm) is visible. The granules are spherical with moderate to high electron density. The nucleus has a prominent, electron-dense, granular nucleolus and clumped chromatin. No viral particles are evident. Lead citrate–uranyl acetate stain; bar = 5 µm.

Small pieces of formalin-fixed liver were transferred into 2% (para)formaldehyde with 2% glutaraldehyde in 0.1M phosphate buffer and processed for transmission electron microscopy. The neoplastic cells had multiple, round, variably sized (maximum dimension, 0.25 to 1.9 µm) membrane-bound, electron-dense granules in the cytoplasm, typical of myelocytes (**Figure 3**).[1] Viral particles were not observed. Results of virus isolation and PCR assay on brain and heart tissues and immunohistochemical analysis on brain, heart, lung, kidney, spleen, and intestinal tissues for WNV were negative.

## Morphologic Diagnosis and Case Summary

Morphologic diagnosis and case summary: hepatic and splenic myelocytomatosis in an eastern screech owl.

## Comments

Avian myelocytomatosis with massive involvement of liver and spleen was the most likely cause of death for the owl of the present report. Data regarding myeloid neoplasms in owls are limited. However, in chickens, avian leukosis virus (ALV) subgroup J is a well-documented cause of myeloid neoplasms (myelocytoma and myelomonocytic leukemia). Infection with avian myelocytomatosis virus MC29 (an acutely transforming strain of ALV) can cause myelocytomatosis and myelocytomas in chickens and can be pathogenic in guinea fowl, quail, turkeys, pheasants, and partridges.[2] Other ALV strains (including BAI A, R, and ES4)[2,3] could have potentially caused similar disease in the bird of the present report; however, the bird was not tested (ie, virus isolation or PCR assay) for those viruses. Moreover, viral particles were not identified via electron microscopy, so potential viral induction was unclear. Myelocytic tumors in wild birds have been described sporadically, and there have been documented cases of retroviral-associated neoplasia in free-ranging wild turkeys (*Meleagris gallopavo*), Attwater's prairie chickens (*Tympanuchus cupido attwateri*), and greater prairie chickens (*Tympanuchus cupido*) that were reared

in captivity. In general, however, the pathogenesis of myelocytic tumors in wild birds remains undetermined.[4]

Regardless of the cause, a myelocytoma is characterized by proliferation of neoplastic myelocytes, which are immature cells of granulocyte lineage that includes heterophils, eosinophils, and basophils. As these cells mature, the characteristic heterophilic and eosinophilic cytoplasmic granules become more prominent. In birds, the gross postmortem findings of myelocytoma associated with ALV strains include hepatomegaly, splenomegaly, a mottled liver, and yellowish-white nodules of variable sizes composed of neoplastic myelocytes. These nodules can be found in the liver, spleen, kidneys, lungs, and bones.[2] The neoplastic myelocytes of these nodules are well-differentiated with moderate amounts of cytoplasm containing intensely eosinophilic granules but have anisocytosis, anisokaryosis, and a high mitotic rate.[2] Infection with either the ALV subgroup J or MC29 strain can result in concurrent myelocytic leukemia, but circulating neoplastic cells indicative of leukemia were not noted in the case described in this report.

Other causes of hepatic or splenic nodules in birds considered as differential diagnoses for the bird of this report included granulomatous lesions (as a result of chronic systemic infections with *Staphylococcus* spp, *Streptococcus* spp, *Escherichia coli*, or *Salmonella* spp; mycobacteriosis; and chronic fungal infections),[5] ascaridiasis,[6] trichomoniasis,[7] and various neoplasms (eg, carcinomas, melanomas, and sarcomas).[5] Staphylococcal infections are most often caused by *Staphylococcus aureus* and typically result in an altered gait, joint swelling, reluctance to move, gradual emaciation, and often death.[2] Hepatic granulomas in poultry with more chronic infections have been reported.[8] Chronic avian streptococcosis can be the result of infection with either *Streptococcus faecalis* or *Streptococcus zooepidemicus*. Necrotic foci are often detected in the liver at necropsy in conjunction with enteritis and a fibrinous exudate in the coelom. Hepatomegaly and splenomegaly can also be present,[2] with focal granulomas in various tissues secondary to septic emboli.[9] Avian colibacillosis can also cause necrotic foci in the liver as well as granulomas in the liver, ceca, duodenum, and mesentery; there are no reports of splenic lesions, however. The colibacillosis-related granulomas are known as coligranulomas (Hjarre's disease) and have been identified in chickens and turkeys. Coagulative necrosis often accompanies the coligranulomas and can involve as much as half of the liver parenchyma. *Salmonella enterica*, the causative agent of fowl typhoid, also has the ability to induce granulomatous lesions in many avian species. Gray to white foci or nodules can be found in the liver, spleen, heart, pancreas, and gizzard of affected birds. Often, chronic disease results in hepatomegaly, splenomegaly, and renomegaly, with foci of necrosis in those organs.[10]

Avian mycobacteriosis, generally caused by *Mycobacterium avium* or *Mycobacterium genavense*,[11] was an important differential diagnosis in the case described in the present report because of the zoonotic potential in immunocompromised individuals. Owls, like other birds of prey, have the potential to be infected via consumption of infected prey.[12] Postmortem findings expected with mycobacteriosis can vary among species but often include loss of body weight (owing to the chronicity of the disease), hepatosplenomegaly, and disseminated granulomatous inflammation commonly involving the liver, spleen, intestines, lungs, air sacs, and thoracic and abdominal cavities.[13] More discrete granulomas can range from pinpoint to several centimeters in diameter.[14]

Chronic active hepatitis can be characterized by a fibrotic, pale, nodular liver in birds. Multiple infectious agents and toxic insults have been implicated as causes. There are no reports of chronic active hepatitis in owls, to our knowledge, but other birds, especially psittacines, have been affected.[5]

Very few neoplastic processes in owls (particularly screech owls) have been documented. For great horned owls (*Bubo virginianus*), there have been reports of a cutaneous mast cell tumor,[15] a presumptive histiocytic sarcoma,[16] pulmonary carcinoma,[17] a lymphoproliferative disorder,[18] and chronic myelogenous leukemia.[19] In that case of chronic myelogenous leukemia, a presumptive diagnosis was based on disease chronicity, severe leukocytosis of unknown cause, and lack of response to traditional treatments.[19] Postmortem examination findings confirmed the diagnosis; however, no nodular lesions were present.[19]

To our knowledge, there have been no prior reports of myelocytomatosis in owls of any species. A survey of eastern screech owls would be necessary to estimate the prevalence of myeloid neoplasms in that population. If viral induction was the definitive cause, heavy population losses could be incurred depending on virulence and host susceptibility.

## References


1. Young KM, Meadows RL. Chapter 43: eosinophils and their disorders. In: Weiss DJ, Wardrop KJ, eds. *Schalm's veterinary hematology*. 6th ed. Ames, Iowa: Wiley-Blackwell, 2010;282, 975.
2. Saif YM. Chapter 15: neoplastic diseases. In: *Diseases of poultry*. 11th ed. Ames, Iowa: Iowa State Press, 2003;465–489.
3. Bolognesi DP, Langlois AJ, Sverak L, et al. In vitro chick embryo cell response to strain MC29 avian leukosis virus. *J Virol* 1968;2:576–586.
4. Thomas NJ, Hunter DB, Atkinson CT. Chapter 11: retroviral infections. In: *Infectious diseases of wild birds*. Ames, Iowa: Iowa State Press, 2007;216.
5. Schmidt RE, Reavill DR, Phalen DN. Chapter 4: liver. In: *Pathology of pet and aviary birds*. Ames, Iowa: Iowa State Press, 2003;74–86.
6. Saif YM. Chapter 28: internal parasites. In: *Diseases of poultry*. 11th ed. Ames, Iowa: Iowa State Press, 2003;941–944.
7. Saif YM. Chapter 29: protozoal infections. In: *Diseases of poultry*. 11th ed. Ames, Iowa: Iowa State Press, 2003;1006–1008.
8. Merck Veterinary Manual. *Streptococcus*: introduction. Available at: www.merckvetmanual.com/mvm/index.jsp?cfile=htm/bc/204300.htm. Accessed Jan 10, 2011.
9. Merck Veterinary Manual. *Staphylococcus*: introduction. Available at: www.merckvetmanual.com/mvm/index.jsp?cfile=htm/bc/204200.htm. Accessed Jan 10, 2011.
10. Center for Food Security and Public Health. Fowl typhoid and pullorum disease. Available at: www.cfsph.iastate.edu/Factsheets/pdfs/fowl_typhoid.pdf. Accessed Jan 10, 2011.
11. Tell LA, Woods L, Cromie RL. Mycobacteriosis in birds. *Rev Sci Tech* 2001;20:180–203.
12. Bunn DS, Warburton AB, Wilson RDS. *The barn owl*. Vermillion, SD: Harrell Books, 1982;169.
13. Shivaprasad HL, Palmieri C. Pathology of mycobacteriosis in birds. *Vet Clin North Am Exot Anim Pract* 2012;15:41–55.
14. Heidenreich M. Chapter 9: infectious diseases. In: *Birds of prey: medicine and management*. Oxford, England: Blackwell Science, 1997;108–118.
15. Swayne DE, Weisbrode SE. Cutaneous mast cell tumor in a great horned owl. *Vet Pathol* 1990;27:124–126.
16. Sacré BJ, Oppenheim YC, Steinberg H, et al. Presumptive histiocytic sarcoma in a great horned owl (*Bubo virginianus*). *J Zoo Wildl Med* 1992;23:113–121.
17. Rettenmund C, Sladky KK, Rodriguez D, et al. Pulmonary carcinoma in a great horned owl (*Bubo virgnianus*). *J Zoo Wildl Med* 2010;41:77–82.
18. Kelly TR, Vennen KM, Duncan R, et al. Lymphoproliferative disorder in a great horned owl (*Bubo virginianus*). *J Avian Med Surg* 2004;18:263–268.
19. Wiley JL, Whittington JK, Wilmes CM, et al. Chronic myelogenous leukemia in a great horned owl (*Bubo virginianus*). *J Avian Med Surg* 2009;23:36–43.