



**Long Island
Veterinary Specialists**

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Primary Hyperparathyroidism

Michael Larkin, DVM, DACVS-SA



Hyperparathyroidism can be classified as either primary or secondary. Primary hyperparathyroidism results from excessive production and excretion of PTH by abnormal, autonomously functioning parathyroid chief cells. Hypercalcemia develops as a result of lost negative feedback (in which high Ca^{2+} concentration normally inhibits secretion of PTH). Secondary hyperparathyroidism typically results from chronic renal failure (renal secondary hyperparathyroidism) or diet imbalances in phosphorus, vitamin D, or calcium (nutritional secondary hyperparathyroidism).

Primary hyperparathyroidism is uncommon in dogs and rare in cats. The Keeshond is the breed most at risk due to a heritable genetic mutation. Most hyperfunctional parathyroid nodules in dogs are classified as adenomas or adenomatous hyperplasia, however, up to 5% are carcinomas. Primary hyperplasia of all four parathyroid glands is rare but has been reported in dogs and cats.

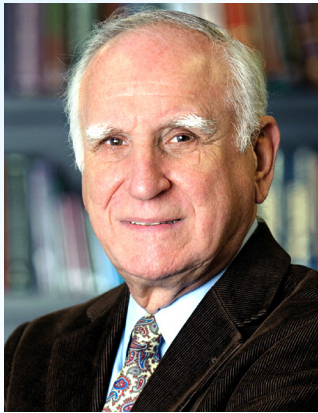
When all four glands are hyperplastic, secondary hyperparathyroidism should be suspected. Metastasis appears to be extremely rare.

With primary hyperparathyroidism, the major reason for hypercalcemia is PTH-induced resorption of Ca^{2+} from bone. At the kidney level, increased PTH initially stimulates excessive renal Ca^{2+} resorption, resulting in decreased urine excretion. As hypercalcemia worsens, renal tubular mechanisms for reabsorbing calcium become overwhelmed, and the kidney starts to excrete excessive amounts of Ca^{2+} despite the presence of PTH. Hypercalciuria increases the risk for urolithiasis and urinary tract infections. Hypercalcemia also leads to polyuria and polydipsia from an inability of the kidneys to respond normally to antidiuretic hormone, resulting in diabetes insipidus. With an increased PTH, production of vitamin D ($1,25\text{-(OH)}_2\text{-D}_3$) by the kidneys also increases, exacerbating hypercalcemia. Although vitamin D also stimulates absorption of phosphorus at the intestinal level, PTH increases excretion of phosphorus to a greater degree, with the overall effect being a decrease in phosphorus concentration.

The most common clinical signs are often attributable to PU/PD, urolithiasis or urinary tract infection as well as stranguria, pollakiuria, and hematuria, however a majority of dogs do not have any observable abnormalities on physical examination.

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A NOTE FROM THE EDITOR



We've become accustomed to the cooler weather during the last few months of the year and are adjusting to the delivery of "stuff" directly to our doors rather than the in-person shopping we all did prior to COVID. The adjustments still continue at LIVS as masks and social distancing protocols continue much as new virus variants emerge.

Construction is moving ahead as the building façade enlarges and remodeled areas open. The staff is expending considerable effort to keep all departments fully functioning servicing our community during the process. They are truly heroes!

The recent election results reflected the sentiments of the populace toward changes so earnestly described a short few months ago and clearly expressed what most are thinking regarding the politicians newly elected and some incumbents as well.

A recent report in "Infectious Diseases in Clinical Practice" stated that nearly one in four in a group of 97 Vietnam Vets studied at the Northport VA was infected with a liver fluke parasite gotten by eating undercooked freshwater fish while in "Nam." Liver flukes spend part of their life cycle in freshwater snails which release larvae that burrow into the flesh of fish and can infest the bile ducts of humans who eat the fish and can then cause bile duct cancer many years later. With the holidays and eating events approaching, careful selection of what we consume is more than prudent. Sushi lovers be prudent!

COVID has caused a severe decline in availability of mink pelts, a 49% drop; mink can transmit COVID to humans (cats can get COVID from humans, but not transmit it to their owners). A Stony Brook firm is presently testing a vaccine for use in mink. They are evaluating it in ferrets as there aren't that many uninfected mink around on whom it can be tested! A vaccine for use in household felines is "promising" in case we start seeing our pet cats get infected by us.

Events regarding pets in other parts of our world are disturbing as in some countries dogs and cats are sold as food. These dogs are tortured their entire lives before being brutally slaughtered in the most inhumane ways, often by electrocution, hanging, or beating with clubs. They are then thrown into boiling water, sometimes while still alive, to ultimately be served as a meal. This often takes place in broad daylight in full view of other living, terrified, caged dogs. Sadly, many abandoned and stolen pets end up in this industry and are subjected to the same cruelty. We might take a moment to advocate for these pets when the subject comes up in our media. We know how loyal and faithful our dogs across the world are; we all ask a lot of them - they serve us in innumerable ways: farm work, war work, police and guard duties, search and rescue, help for the disabled, guiding and guardianship, and, of course, as our loving and trusting companions.



Photos from the holiday lunches generously provided by Gail and Wendy Wallers, individuals recognized as devoted animal welfare advocates and longtime supporters of LIVS's mission.



There are those who see little difference in the way we treat our supply of meat but.... torturing (to increase hormonal release ostensibly to tenderize the meat) is not part of the cattle modus operandi industry in the US.

Here at LIVS, our staff was delighted with early holiday lunches as savory dishes were provided by the Wallers, Gail and Wendy....individuals recognized as devoted animal welfare advocates and longtime supporters of LIVS's mission. They exult in helping our creatures, have been doing so for decades and we sincerely thank them.

LIVS remains open 24 hours daily as always and our clinicians have extended their hours to better accommodate your needs.

Again, we welcome your observations emailed to lmario@livs.org.

-Leonard J. Marino, MD, FAAP, LVT



Primary Hyperparathyroidism

Continued from Front Cover

Suspicion of hyperparathyroidism is based on history, physical examination and results of CBC, chemistry panel, and urinalysis.

Definitive diagnosis is usually based on results of ionized calcium, PTH, and PTH-related peptide (PTH-rp) concentrations [Figure 1]. Animals with primary hyperparathyroidism have a decreased PTH-rp concentration and an inappropriate (normal or increased) PTH in the face of an increased ionized calcium. It is important to note that animals that are hypercalcemic and have PTH concentrations within the reference range are still considered to have excessive concentrations of PTH. Patients with increased PTH-rp should undergo further diagnostics for neoplasia (e.g., lymphoma, anal sac apocrine gland carcinoma). Unlike animals with chronic renal failure, animals with primary hyperparathyroidism usually have a decreased phosphorus level and are not azotemic. In contrast to many other causes of hypercalcemia, primary hyperparathyroidism rarely requires medial management.

After a diagnosis of primary hyperparathyroidism, patient referral to a specialty center for surgical intervention is the treatment of choice. Cervical ultrasonography is often used to further support the diagnosis of a parathyroid mass (or masses) and is very helpful for preoperative determination of mass location. Ultrasonography is very accurate for identification of abnormal parathyroid glands, but negative ultrasound results do not rule out a tumor.

The parathyroid glands are small, ellipsoid discs that are anatomically intimately associated with the thyroid glands. Each thyroid gland has a pair of parathyroid glands - an external parathyroid gland (cranial pole of thyroid gland) and an internal parathyroid gland (caudal pole of thyroid gland). Adenomatous external parathyroid glands are typically firm, somewhat spherical, and enlarged (4 to 10mm in diameter) [Figure 2].

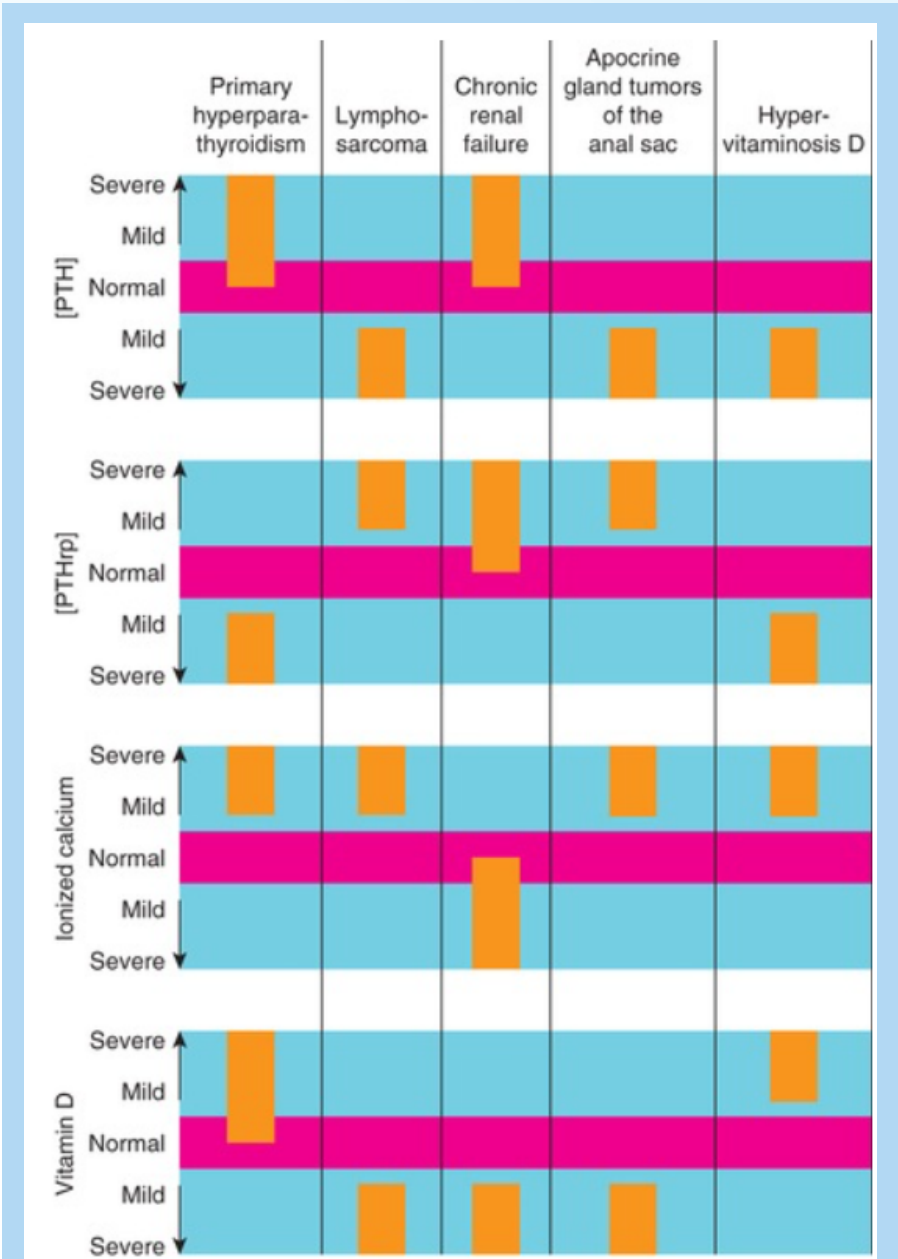


Figure 1 Results of serum parathyroid hormone (PTH), parathyroid hormone-related peptide (PTHrp), ionized calcium, and vitamin D concentrations in conditions causing hypercalcemia in dogs.

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Primary Hyperparathyroidism

Continued from Page 3

Adenomatous internal parathyroid glands undergo similar changes and are usually palpable or visible through the ventral or dorsal aspect of the thyroid parenchyma. Parathyroidectomy is a relatively delicate surgical procedure; fine instruments, bipolar cautery, hemoclips, and sterile cotton-tipped applicator swabs are often used for dissection. The affected gland is removed and submitted for histologic evaluation.

After surgery, approximately 35% to 70% of dogs develop hypocalcemia, and 25% of these dogs develop associated clinical signs. Clinical signs of hypocalcemia include facial rubbing, twitching, convulsion, and tetany. Hypocalcemia after parathyroidectomy can be seen as early as 12 hours and as late as 20 days after surgery but most commonly develops between the second and sixth day after surgery.

Prophylactic oral supplementation with both Vitamin D and calcium is often pursued, sometimes starting as early as the morning of surgery. Calcitriol is the vitamin D of choice because of its rapid onset of action and short biologic half-life. The goal of calcium and vitamin D therapy is to maintain the serum calcium concentration in the low to low-normal range. Serum calcium concentration in this range prevents clinical signs of hypocalcemia, minimizes the risk of hypercalcemia, and is low enough to stimulate recovery of function in the remaining atrophied parathyroid glands.

Calcium concentrations are checked frequently during the post-operative recovery period. After serum calcium concentration has reached a steady state, the dose of calcitriol is tapered down. It is usually withdrawn by gradually extending the time interval between each dose. The entire withdrawal process can take 3 to 6 months, although the exact amount of time necessary to prevent hypocalcemia is unknown.

Failure to achieve a cure can result from the presence of multiglandular disease, ectopic parathyroid glands, malignant disease with subsequent metastasis, or because of incorrect intraoperative decisions that resulted in incomplete excision of autonomously functioning tissue. Recurrence of hyperparathyroidism has also been reported. A second parathyroid adenoma or parathyroid hyperplasia can be detected within months to years in up to 8% of dogs after initial parathyroidectomy. When hyperparathyroidism recurs, a second surgery is usually highly successful at treating the condition again.

The prognosis for dogs with primary hyperparathyroidism is excellent with appropriate treatment and monitoring. Even with a parathyroid carcinoma, the prognosis can still be excellent. Although primary hyperparathyroidism is rare in cats, their prognosis also appears to be excellent.

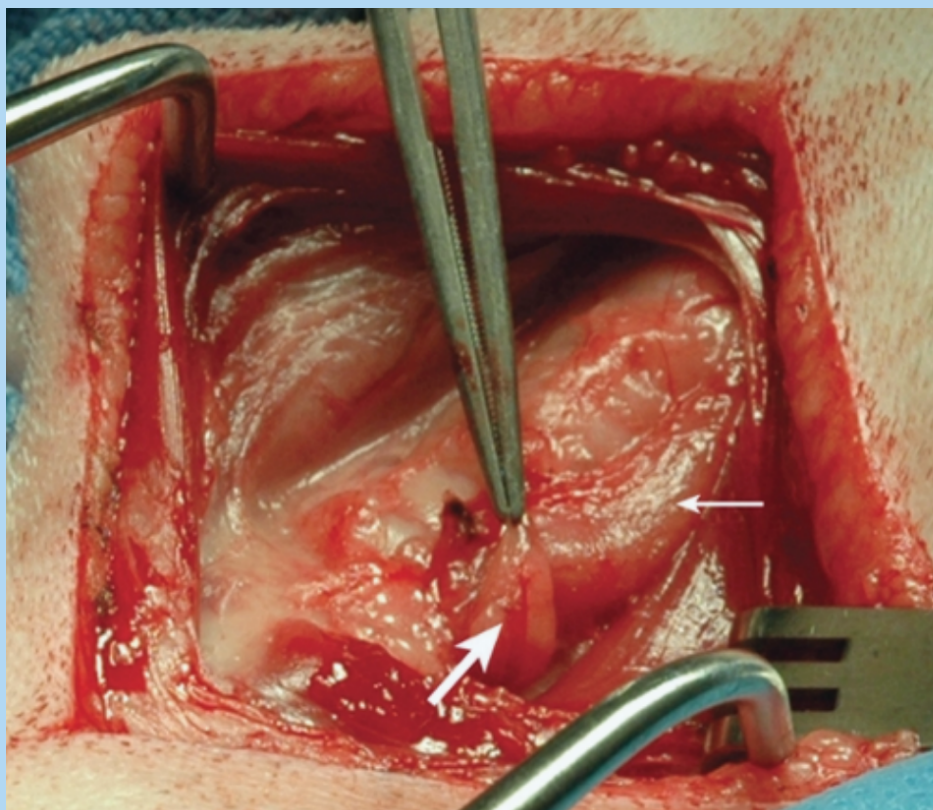


Figure 1 Intraoperative photograph of a dog with a parathyroid adenoma. The small arrow points to the right thyroid gland, and the large arrow points to the enlarged right parathyroid gland. The patient's head is toward the bottom left of the photograph.

Dr. Dominic J. Marino named AVCS Founding Fellow in Joint Replacement Surgery (JRS)



Dr. Dominic J. Marino is one of the most experienced hip replacement surgeons in the country. He is recognized as an ACVS Founding Fellow in Joint Replacement Surgery (JRS). ACVS Founding Fellows are distinguished leaders as evidenced by their exemplary training, extensive experience, innovative research, and committed practice. They devote a significant portion of their professional effort in seeking to prevent, diagnose, treat, and rehabilitate patients in the specialized field of joint replacement surgery. Dr. Marino has performed over 2,000 THR procedures, starting in the early nineties. His areas of special interest include joint replacement surgery, brain surgery, and spine surgery.

Including Dr. Marino, there are only 15 veterinary surgeons recognized by the ACVS as Founding Fellows in Joint Replacement Surgery.

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CEMENTED THR



HYBRID THR



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To learn more about Total Hip Replacement procedures and other Hip Dysplasia treatment options, or to refer a case: 516-501-1700 | livs.org

Superficial Digital Flexor Tendon Luxation

Catherine Loughin, DVM, Dip. ACCT, Staff Surgeon



The superficial digital flexor tendon extends from the caudal distal aspect of the femur, down the caudal aspect of the lower limb, over the tuber calcanei (fig 1), and down to the plantar aspect of the paw and branches to each digit. It is a component of the common calcanean tendon, together with the tendon of the gastrocnemius muscle and the combined tendon of the biceps femorus, gracilis, and semitendinosus muscles. This tendon functions to flex the digits. The tuber calcanei provides a shallow groove where the SDFT runs over it, and also at this position, connective tissue bands (retinaculum) extend medial and lateral from the SDFT to the tuber calcanei to further stabilize the tendon.

Superficial digital tendon luxation occurs when the SDFT slips out of its groove on the point of tuber calcanei. It more commonly occurs laterally, but sometimes can be medial. This is typically the result of a tear through the medial or lateral retinaculum. Most commonly, the medial component tears, allowing the SDFT to slip off the tuber calcanei to the lateral side. This prevents normal flexion of the digits, and impairs normal limb use.

Tearing of the medial retinaculum is usually associated with rotational forces exerted during vigorous activity such as herding or agility work. Frequently, patients who experience a SDFT luxation also are found to have a shallow groove, potentially predisposing them to luxation. Shetland Sheepdogs and Collie breeds are most commonly affected, although this condition has been diagnosed in a wide variety of medium and large breed dogs.

Patients with SDFT luxation typically present with a history of intermittent weight-bearing hind limb lameness that occurs as the tendon pops in and out of its groove spontaneously (Fig 2). On physical exam, the tissues at the point of the hock are usually swollen or thickened. The digits may be more extended than normal, with the nails angling cranially and dorsally rather than contacting the ground. The SDFT can usually be palpated and manually luxated and reduced, but in chronic cases a large amount of scar tissue may form, keeping the tendon fixed in a luxated position. Radiographs are useful primarily to rule out other causes of hind limb lameness, but a swelling at the tuber calcanei is usually visible (Fig 3). Ultrasound and MRI can be utilized to confirm the diagnosis and determine the severity of the injury. It also enables the evaluation of the associated tendons to identify potential concurrent injuries.

The treatment for superficial digital luxation is a surgical procedure in which the torn retinaculum is repaired, thus returning the tendon to its correct location. If the groove at the point of the tuber calcanei is found to be abnormally shallow, it can be deepened to further increase stability.

Following surgery, a splint is placed on the limb to hold the hock in partial extension, preventing strain on the superficial digital flexor tendon while the retinaculum heals.

After two to three weeks, this is replaced with a soft padded bandage, which provides a decreased level of support and allows a small amount of movement. After one to two weeks, the soft padded bandage is removed, and the patient is gradually returned to full activity over the next few weeks. Even with the protection of the splint, it is essential to restrict physical activity over the first month post-surgery to prevent re-injury as well as bandage irritation and wounds. No running, jumping, or playing with other pets should be allowed during this period. Exercise should be limited to short, slow leash walks until the soft bandage is removed. At that point, leash walks should be gradually increased in length over the next two weeks before off-leash activity is permitted.

This procedure is associated with a relatively low risk of complications. Surgical site infection, wounds from the bandage, and re-luxation of the superficial digital flexor tendon may occur. Infections and wounds are managed medically with antibiotics and topical dressings. Re-luxation, while rare, may necessitate a second surgery. The prognosis following surgery is excellent, with most dogs returning to full activity within two to three months. Most dogs have no further problems with their superficial digital flexor tendon, although re-luxation can occur in rare cases.

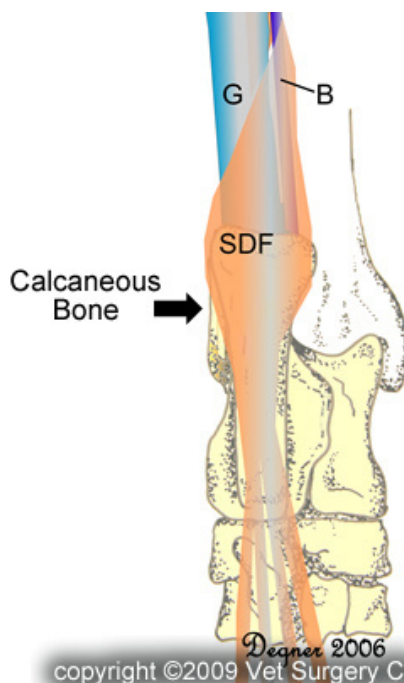


Figure 1 The superficial digital flexor tendon (SDF) extends over the tuber calcanei and is part of the calcanean tendon complex with the gastrocnemius (G), the biceps femorus (B), gracilis, and semitendinous tendons.

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Superficial Digital Flexor Tendon Luxation

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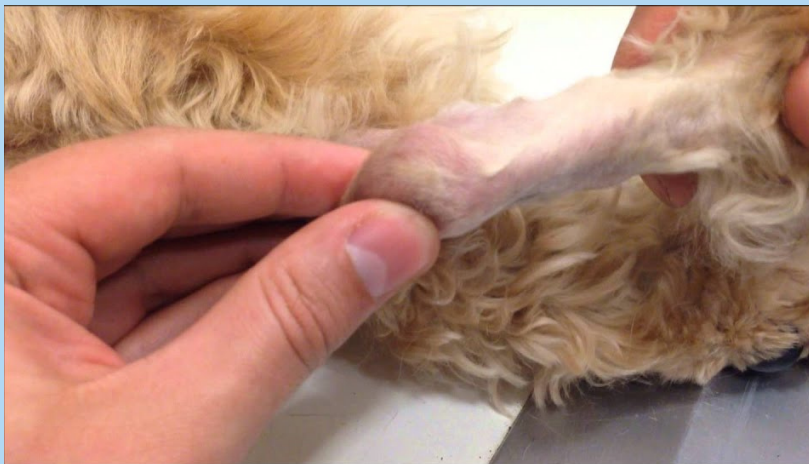


Figure 2 Palpation of the SDFT at the tuber calcanei resulting in luxation of the tendon.



Figure 3 Radiograph of the lateral hock with arrows pointing to soft tissue swelling associated with calcaneal tendon injury.

Radioiodine Therapy

Radioiodine therapy at Long Island Veterinary Specialists is the preferred choice! Radioiodine therapy (I-131) is the safest treatment for hyperthyroidism in cats and has been proven to be 96-98% effective, employing a single treatment. At LIVS, our specially designed radioiodine facility allows us to accommodate many patients and permits quick access to this life-saving therapy.

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Diagnostic Approach to Elevated Liver Enzymes in the Dog

Joshua W. Tumulty, DVM, DACVIM (SAIM)



The presentation of elevated liver enzyme activities may present a diagnostic challenge because they are common, often nonspecific biochemical findings. A systematic approach to interpretation of increased liver enzyme activities should be utilized to confirm the significance and determination for advanced diagnostics (Image 1). The first step is to develop an integrated clinical picture based on the patient's signalment, history, clinical signs, and concurrent laboratory abnormalities. Careful evaluation of this picture aids in ranking diagnostic differentials.

Increases in liver enzyme activities are categorized as cholestatic, hepatocellular leakage, or mixed. A cholestatic or inducible pattern is characterized by predominant increases in alkaline phosphatase (ALP) and γ -glutamyl transpeptidase (GGT) activities.

A hepatocellular leakage pattern has predominant increases in the activities of alanine aminotransferase (ALT) and aspartate aminotransferase (AST) secondary to hepatocyte injury or necrosis. A mixed pattern of liver enzyme activity increase suggests concurrent hepatocellular injury and cholestasis and may be caused by concurrent disease processes or progressive disorders.

The magnitude and duration of increase in liver enzyme activities should be considered in addition to the pattern. Mildly increased activity of a single liver enzyme in a clinically asymptomatic dog may be spurious and should be rechecked. Lipemia and hemolysis may falsely increase activities of serum liver enzymes and should be considered when interpreting laboratory data. Because increases in hepatocellular leakage enzyme activity are proportional to the severity of damage, increases greater than twice the reference interval warrant further investigation. Increased activity of multiple liver enzymes, particularly if a hepatocellular or mixed pattern of injury is present, also warrants close attention because of an increased likelihood of severe disease.

Once spurious or self-limiting increases of liver enzyme activities have been ruled out, the patient should be evaluated for breed associations (Image 2).

Breeds Associated With Increased Activities of Liver Enzymes^a

Hepatocellular leakage pattern

- ▶ Idiopathic hepatitis
 - American and English cocker spaniels
 - Labrador retriever
 - Standard poodle
- ▶ Copper-associated hepatitis
 - Bedlington terrier
 - Dalmatian
 - Doberman pinscher
 - Labrador retriever
 - Skye terrier
 - West Highland white terrier
- ▶ Amyloidosis
 - Chinese shar-pei

Cholestatic pattern

- ▶ Benign hyperphosphatasemia
 - Scottish terrier
 - Siberian husky
- ▶ Hyperlipidemia
 - Miniature schnauzer
- ▶ Gallbladder disease
 - Shetland sheepdog

Image 2 Breed-associated disease associated with elevated liver enzyme activities

A careful dietary history may identify risk factors for infection (e.g., raw or undercooked meat) or intoxication (e.g., poor food storage practices, recalled diets). Depending on the geographic location, access to *Amanita* mushrooms and cycad palm plants should be determined. The owner should be carefully questioned about medications to which the patient may have access, including supplements, nutraceuticals, and topical glucocorticoids. Owners may not notice a pet's consumption of prescription medications or know that common items for human use, such as those containing xylitol, can be hepatotoxic.

Abnormal liver enzymes

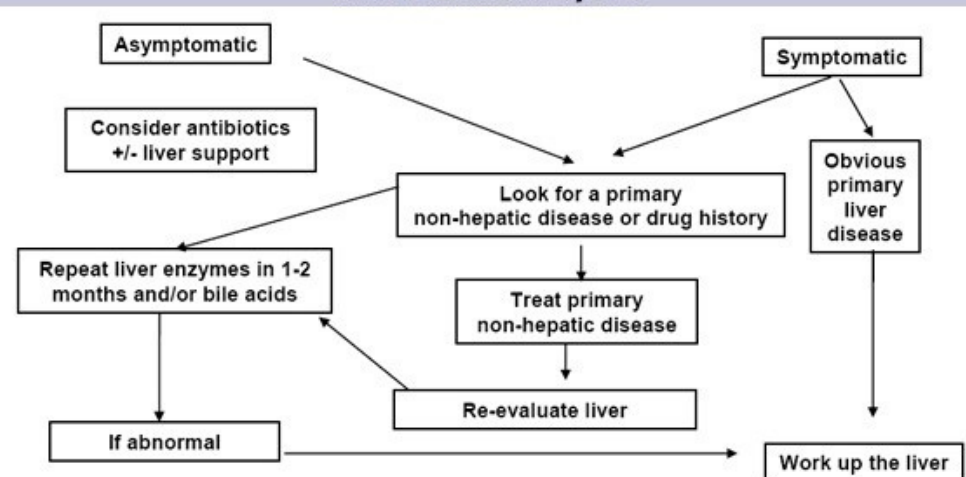


Image 1 Diagnostic flow chart

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Diagnosis of drug-related hepatotoxicity can be challenging. The time between initial administration of a medication and detection of hepatic injury can vary greatly, which makes it difficult to establish exposure preceding the onset of injury. Liver injury may worsen for days or weeks following withdrawal of the medication before improvement is seen. Repeat challenge with a suspected hepatotoxin may greatly exacerbate hepatotoxicosis. Alternatively, a patient may develop tolerance for a hepatotoxin and have negative challenge results. If potential hepatotoxins are identified, they should be discontinued and liver enzyme activities reassessed in 2 to 3 weeks.

The patient's vaccination history and potential exposure to infectious agents should be determined. Hepatopathies have been documented with *Leptospira interrogans* serovars *Icterohaemorrhagiae* and *Pomona* and *Leptospira kirschneri* serovar *Grippotyphosa*; additional associations may be identified with increased molecular testing. Canine adenovirus-1 infection, heartworm disease, ehrlichiosis, leishmaniasis, neosporosis, toxoplasmosis, and systemic mycoses should be considered as potential causes of hepatocellular disease. In dogs, bacterial infections arise primarily from hematogenous routes and breaks in host defenses, such as periodontal disease.

Lethargy, inappetence, vomiting, and diarrhea are common clinical signs. Icterus, ascites, acholic feces, or hepatic encephalopathy may occur with acute liver failure or progression of chronic disease. Physical examination may be unremarkable or may reveal abnormalities consistent with an extrahepatic disorder. Poor body condition, icterus, abdominal pain, hepatomegaly or microhepatia, and abdominal effusion may suggest hepatic dysfunction. The presence of clinical signs, physical examination abnormalities, or evidence of decreased liver function should prompt an expeditious workup to maximize chances of obtaining a diagnosis and successful therapeutic intervention.

A minimum database of a complete blood count (CBC), biochemical profile, and urinalysis should be obtained for dogs with verified increased activities of liver enzymes. Hepatobiliary disease can be associated with a variety of CBC abnormalities. Although gastrointestinal bleeding or coagulopathy secondary to hepatobiliary dysfunction may stimulate a regenerative anemia, the chronicity of many hepatic diseases often leads to anemia of chronic disease, which is nonregenerative. Microcytosis is common with acquired or congenital portosystemic shunts (Image 3). Thrombocytosis occurs with hyperadrenocorticism, chronic blood loss, and inflammatory disorders, and thrombocytopenia suggests ongoing vasculitis or consumptive processes. Biochemical changes may aid in the differentiation of extrahepatic and hepatobiliary disease. Concentrations of bilirubin, cholesterol, blood urea nitrogen (BUN), albumin, and glucose provide information about liver function despite low sensitivity and specificity. In the absence of hemolysis or sepsis, hyperbilirubinemia indicates cholestasis due to hepatobiliary disease or posthepatic obstruction. Cholesterol concentrations increase with cholestatic disease and decrease with end-stage liver disease. BUN concentration may be increased secondary to gastrointestinal bleeding or decreased by shunting of portosystemic blood or decreased hepatic production. Creatinine concentration typically remains within the reference interval. With the loss of approximately 70% of functional hepatic parenchyma, hypoalbuminemia may occur. Other causes of hypoalbuminemia should be ruled out, including gastrointestinal disease, protein-losing nephropathy, hemorrhage, vasculitis, inflammation, and prolonged anorexia. Hypoglycemia may occur after loss of 75% of hepatic parenchymal function and is a poor prognostic indicator. Other causes of hypoglycemia should be excluded, including congenital portosystemic vascular anomalies, insulinoma, and sepsis.

A variety of abnormalities may be identified on urinalysis. Hyposthenuria and isosthenuria occur frequently with extrahepatic and hepatobiliary diseases.

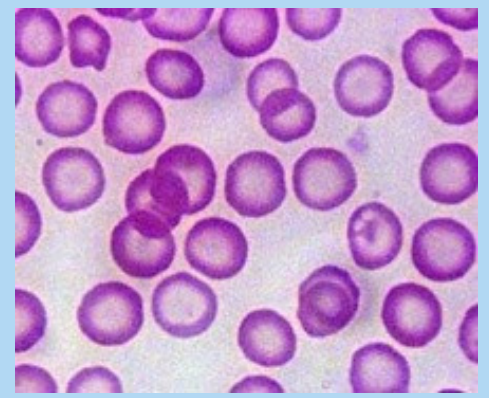


Image 3 Microcytosis

Bilirubinuria with or without hyperbilirubinemia may be identified. A small amount of bilirubinuria can be normal, particularly in male dogs.

Portosystemic shunting and end-stage liver disease may result in ammonium biurate crystal formation. Glucosuria and aminoaciduria, consistent with Fanconi syndrome, have been noted in dogs with copper-storage hepatopathy.

If no likely explanation for the laboratory abnormalities can be found there are two courses of action that one can take; either begin a diagnostic evaluation of the patient starting with bile acid determinations, or re-evaluate the patient's liver enzymes at a later date. A rational wait period for re-evaluation is 4-6 weeks giving consideration to the half-life of liver enzymes and the time needed for recovery from an acute occult hepatic injury. It is best not to delay retesting beyond 6-8 weeks in the event that an active disease process may progress.

Thorough evaluation of liver enzyme activity increases may require additional testing for extrahepatic and hepatobiliary diseases after integration of the minimum database with the clinical presentation. Hyperadrenocorticism and hypothyroidism should be considered in dogs with appropriate clinical findings and cholestatic enzyme activity increases. Thyroid hormone testing should be interpreted carefully because sick euthyroid syndrome is common in dogs with chronic disease.

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Dogs with gastrointestinal signs should be evaluated for pancreatitis and primary gastrointestinal disease. Depending on risk assessment and vaccination history, infectious disease screening may be indicated.

Liver function can be evaluated in a variety of ways. Increased bile acids are extremely sensitive for detecting decreased functional hepatic mass or portosystemic shunting in nonicteric patients.

Increased ammonia concentration occurs with portosystemic shunting, hepatic failure, and urea cycle enzyme deficiencies and is a useful biomarker for hepatic encephalopathy. Protein C is a circulating anticoagulant protein that is synthesized by the liver. Decreased protein C activity may be a useful biomarker for decreased hepatic function or hepatportal perfusion. Protein C activity may also be useful for therapeutic monitoring and differentiation between microvascular dysplasia and congenital portosystemic shunts. The liver synthesizes most clotting factors and is responsible for activation of vitamin K-dependent clotting factors. In dogs with chronic hepatitis, prolonged prothrombin time (PT) and partial thromboplastin time (PTT) have been associated with decreased survival times.

Buccal mucosal bleeding time is used to assess risk of abnormal platelet function. Because of the fragile balance between procoagulant and anticoagulant factors, it is recommended that PT, PTT, and buccal mucosal bleeding time tests be conducted no more than 24-hours before liver biopsy.

Survey radiography allows assessment of the size, shape, position, opacity, and margins of the liver (Image 4). Mineralization of hepatic parenchyma or choleliths may be detected on survey films. Abdominal ultrasonography allows visualization of focal, multifocal, or diffuse lesions within the hepatic parenchyma, although it cannot distinguish pathologic from benign lesions. Ultrasonography also allows assessment of the biliary tract and provides visualization for percutaneous cholecystocentesis, a minimally invasive method of bile collection (Image 5). Normal findings on abdominal imaging do not rule out primary hepatobiliary disease.

The optimal time for liver biopsies is early in the disease course so that they yield useful information rather than demonstrate nonspecific end-stage changes.



Image 4 Lateral radiograph, demonstrating severe microhepatica, renal calcification, and multiple bladder stones.

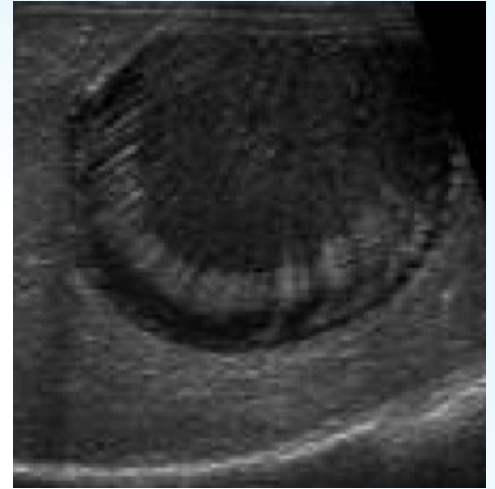


Image 5 Ultrasound image, demonstrating biliary mucocele formation.

When selecting a biopsy technique, clinicians should consider the ability to obtain adequate samples for histopathology, aerobic and anaerobic cultures, and copper and iron quantitation. Contraindications to biopsy include coagulopathy and liver failure. Ultrasound-guided fine-needle aspiration of the liver is easy to perform and minimally invasive. Unfortunately, cytology has been demonstrated to have poor agreement (29% to 66%) with primary histopathologic diagnoses, including the diagnosis of neoplasia. In data from previous studies, 3.6% and 3.9% of cases were inappropriately diagnosed with neoplasia on cytology; 14% and 50% of cases with histologically confirmed neoplasia were identified cytologically. The poor agreement between liver cytology and histopathology and the potential ramifications of misdiagnoses should be kept in mind when considering fine-needle aspiration findings.

The least invasive method of liver biopsy is percutaneous ultrasound-guided needle biopsy. One disadvantage of this technique is the inability to directly monitor the liver for hemostasis. Needle biopsy findings concurred with findings from wedge biopsies only 48% of the time in one veterinary study. Accuracy may be improved by use of the largest appropriate biopsy instrument and collection of multiple samples.

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Laparoscopy offers excellent visualization and sampling of the liver, gallbladder, and other abdominal organs; provides better hemostatic access than percutaneous sampling; and is less invasive than laparotomy. Widespread use of laparoscopy is limited by requirements for specialized training and equipment. Laparotomy is recommended when surgical cure may be possible and in cases for which full exploration of the abdominal cavity is desired.

Histopathologic evaluation of liver biopsy samples remains the gold standard for the diagnosis of hepatobiliary disease, but it can fail to provide a definitive diagnosis. Clinicians often must incorporate information regarding the extent and type (inflammatory, neoplastic, vascular, vacuolar) of pathology into the clinical picture to make a diagnosis.

Quantification of iron and copper concentrations may aid in diagnosis, prognostication, and therapeutic tailoring. Abnormally high hepatic copper levels cause oxidative stress and are associated with low hepatic glutathione levels, which contribute to hepatocellular damage. Both inborn errors of metabolism leading to decreased copper excretion and impaired copper excretion due to chronic hepatitis and cholestatic disease may occur in dogs. Abnormal accumulation of iron may be due to increased intestinal absorption or abnormalities of hemoglobin metabolism and delivery of iron to the liver. Accumulation of iron in Kupffer cells has been correlated with inflammation and increased copper levels.

Aerobic and anaerobic cultures of hepatic tissue and bile complete a comprehensive hepatobiliary workup. In findings from one study, biliary cultures were more commonly positive (30%) than hepatic cultures (7%) in dogs.

It is unclear whether the biliary system is more susceptible to infection or whether it is a more sensitive sampling site. Cytologic evaluation of bile may aid in identification of infectious organisms.

Successful interpretation of liver enzyme abnormalities requires integrated evaluation of the patient's clinical picture and diagnostic testing. A thorough diagnostic workup for hepatobiliary disease includes a CBC, biochemical profile, and urinalysis; liver function tests, including coagulation parameters; abdominal radiography and ultrasonography; bile cytology and culture; and hepatic biopsy for histopathology, culture, and metal analysis. Employing a systematic approach facilitates diagnosis of hepatobiliary disease in a timely fashion to improve patient outcome.

Integrative Medicine at LIVS

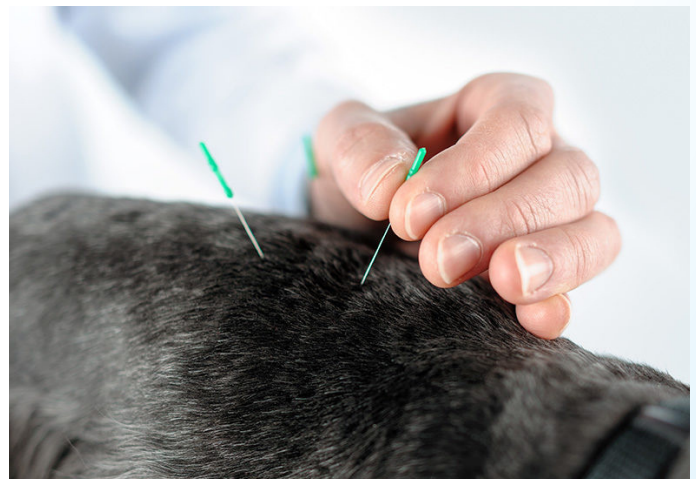


**Michel Selmer, DVM
MS, CTCVMP**

The Integrative Medicine Team takes a holistic and gentle approach to treating animal disorders. While combining techniques of both Eastern and Western medicine, our Integrative Medicine Team puts an emphasis on the patient's emotional and mental well-being. Dr. Michel Selmer is one of only a handful of Traditional Chinese Veterinary Medicine Practitioners that holds a Master's Degree in the United States.

Services offered include:

- Acupuncture
- Chinese Herbology
- Class IV Cold Laser Therapy
- Food Therapy
- Herbal Medicine
- Nutritional Consults
- Tui-na Massage



**To refer your clients to Dr. Selmer, call
516-501-1700 or visit livs.org**



Long Island Veterinary Specialists

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