Aspartate Aminotransferase (AST)

Interpretive Summary

**Description:** Aspartate Aminotransferase (AST) is a cellular enzyme that primarily increases due to liver and muscle damage.

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**Decreased AST**

**Common Causes**

- Not clinically significant

**Increased AST**

**Common Causes**

- Inflammatory
  - Infectious causes
    - Bacterial cholangiohepatitis
    - Leptospirosis
    - Feline infectious peritonitis (FIP)
    - Histoplasmosis
    - Infectious canine hepatitis
  - Non-infectious
    - Chronic hepatitis
    - Cirrhosis
    - Pancreatitis
- Toxic
  - NSAIDS
  - Phenobarbital
  - Corticosteroids
- Liver hypoxia or hypoperfusion
  - Anemia
  - Congestive heart failure
  - Shock
- Metabolic disorders
  - Hyperthyroidism (cats)
  - Hepatic lipidosis (cats)
  - Cushing’s disease
  - Diabetes mellitus/diabetic ketoacidosis
  - Equine hyperlipidemia
- Neoplasia
- Trauma

**Uncommon Causes**

- Skeletal myopathy
  - Myositis
  - Exertional disorders
  - Nutritional myopathies
  - Toxicity
  - Neoplasia
- Toxic
  - Sago palm
- Aflatoxicosis
- Ragwort (horses)
- Tetracycline
- Idiosyncratic drug reactions

- Inherited
  - Copper storage disorders (certain dog breeds)
  - Lysosomal storage disorders

- Nutritional hepatopathies

**Related Findings**

- **Inflammatory**
  - Increased ALT, ALP, GGT, total bilirubin
  - Decreased albumin, cholesterol, glucose, BUN in severe cases
  - Increased bile acids and ammonia
  - Positive titers or PCR for leptospirosis, feline corona virus (FIP), histoplasmosis
  - Positive bacterial or fungal culture of liver/bile
  - Histopathology/cytology findings consistent with inflammatory hepatic diseases
  - Increased Spec cPL® or Spec fPL® with pancreatitis

- **Toxic**
  - Phenobarbital
    - Therapeutic drug monitoring of phenobarbital may predict toxic levels

- **Metabolic**
  - Hyperthyroidism
    - Increased T4, free T4, free T4 by equilibrium dialysis
  - Hepatic Lipidosis
    - Increased ALP, ALT
    - GGT usually normal unless concurrent inflammatory disease is present
    - Enlarged liver on radiographs, hyperechoic liver on ultrasound
    - Cytology/histopathology consistent with hepatic lipidosis
  - Cushing’s Disease
    - Increased ALP
    - Decreased urine specific gravity
    - Stress leukogram: increased neutrophils and monocytes, decreased lymphocytes and/or eosinophils
    - Adrenal function tests consistent with Cushing’s disease
  - Diabetes Mellitus
    - Increased serum glucose and glucosuria
    - Increased fructosamine
    - Ketonuria (in severe cases)

- **Neoplasia**
  - Enlarged/irregular liver on radiographs and/or ultrasound
  - Cytology/histopathology findings consistent with neoplasia

**Additional Information**

**Physiology**

- AST is present in the cytosol and mitochondria of many cell types but increased activity primarily reflects liver and muscle disease, with less specificity than ALT (liver) or CK (striated muscle), respectively.
- AST is also known as serum glutamic-oxaloacetic transaminase (SGOT).
- Significant damage to hepatocytes must occur before AST activity increases so it is less sensitive for liver disease than is ALT.
- The plasma half life of AST is short (dogs 5 hours, cats 1.5 hours).
• In large animals, AST is used to detect liver disease despite its lack of specificity. Clinical signs and concurrent CK activity help discriminate between hepatic and other diseases.
• Plasma half life of AST is longer than that of CK; concurrent measurement of AST and CK may help assess the course of muscle disorders. In horses, AST activity peaks 12-24 hours after liver or muscle injury and remains increased for 5-6 days.
• Erythrocytes are an important source of AST, so hemolyzed samples have mild to moderate increases in AST activity; lipemia may also interfere.

References


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