Metabolic diseases

Metabolic diseases may be inherited or acquired, the latter being more common and significant. Metabolic diseases are clinically important because they affect energy production or damage tissues critical for survival.

Parturient paresis

- an acute to peracute, afebrile, flaccid paralysis of mature dairy cows that occurs most commonly at or soon after parturition. It is manifest by changes in mentation, generalized paresis, and circulatory collapse.

- onset of lactation results in the sudden loss of calcium into milk. Serum calcium levels decline from a normal of 10-12 mg/dL to 2-7 mg/dL.

- The disease may be seen in cows of any age but is most common in high-producing dairy cows >5 yr old

Clinical Findings and Diagnosis:

- Parturient paresis usually occurs within 72 hr of parturition. The disease can contribute to dystocia, uterine prolapse, retained fetal membranes, metritis, abomasal displacement, and mastitis.

- During stage 1, animals show signs of hypersensitivity and excitability. Cows may be mildly ataxic, have fine tremors over the flanks and triceps, and display ear twitching and head bobbing. Cows may appear restless, shuffling their rear feet and bellowing. If calcium therapy is not instituted, cows will likely progress to the second, more severe stage.

- Cows in stage 2 are unable to stand but can maintain sternal recumbency. Cows are obtunded, anorectic, and have a dry muzzle, subnormal body temperature, and cold extremities. Auscultation reveals tachycardia and decreased intensity of heart sounds.

- Peripheral pulses are weak. Smooth muscle paralysis leads to GI stasis, which can be manifest as bloat, failure to defecate, and loss of anal sphincter tone. An inability to urinate may be manifest as a distended bladder on rectal examination. Cows often tuck their heads into their flanks, or if the head is extended, an S-shaped curve to the neck may be noted.

- In stage 3, cows lose consciousness progressively to the point of coma. They are unable to maintain sternal recumbency, have compete muscle flaccidity, are unresponsive to stimuli, and can suffer severe bloat. As cardiac output worsens, heart rate can approach 120 bpm, and peripheral pulses may be undetectable. If untreated, cows in stage 3 may survive only a few hours.

Differential diagnoses

- include toxic mastitis, toxic metritis, other systemic toxic conditions, traumatic injury (eg, stifle injury, coxofemoral luxation, fractured pelvis, spinal compression), calving paralysis syndrome (damage to the L6 lumbar roots of sciatic and obturator nerves), or compartment syndrome.

Treatment:

- Treatment is directed toward restoring normal serum calcium levels as soon as possible to avoid muscular and nervous damage and recumbency. Recommended treatment is IV injection of a calcium gluconate salt, although SC and IP routes are also used.
- 1 g calcium/45 kg (100 lb) body wt

Neonatal Hypoglycemia:

- Hypoglycemia develops as a result of decreased caloric intake and/or increased catabolism. Decreased intake can be associated with inability to nurse due to neonatal factors such as weakness, prematurity, peripartum asphyxia, or competition between siblings, or it may be due to maternal factors such as agalactia, mastitis, or maternal rejection.

- Neonatal hypoglycemia is most common in piglets <1 wk old. It is a contributing factor that leads to death in many diseases and accounts for 15-35% of total piglet mortality.

- Piglets are predisposed to hypoglycemia if the sow has any disease that decreases or inhibits milk production or letdown. Large litter size with an inadequate number of teats precludes proper nursing. In addition, if the lower rail of the farrowing crate impairs access to the udder, inadequate milk intake and hypoglycemia can result.

- One or more piglets in a litter may be involved. Initially, behavior changes from vigorous sucking or play alternating with sleep to solitary lassitude. Affected piglets wander aimlessly with a faltering gait and cry weakly. The piglets are gaunt with poor muscle tone and pale, cold, clammy skin. They are hypothermic and unresponsive to external stimuli.

- As incoordination increases, piglets may stand with legs splayed, followed by sternal or lateral recumbency. Terminally, they exhibit convulsions with jaw champing, salivation, opisthotonos, nystagmus, forelimb and hindlimb contraction, coma, and death. Many affected piglets are crushed by the sow.

- Blood glucose levels fall from a normal of 90-130 mg/dL to as low as 5-15 mg/dL; piglets usually manifest clinical signs when levels are <50 mg/dL. Any condition that impairs food intake by neonatal pigs can complicate the diagnosis.

- Piglets should be treated with 15 mL of 5% glucose given IP and placed in a warm environment. A heat lamp can be used.
- Active piglets learn quickly to drink from a dish. Foster-suckling of piglets is possible in batch farrowing units; most sows accept piglets during the milk letdown period if introduced quietly within the first 24 hr after farrowing.

Rickets

- Rickets is a disease of young, growing animals. The most common causes are dietary insufficiencies of phosphorus or vitamin D.

Clinical Findings and Lesions:

- The characteristic lesions of rickets is mineralization in the area of provisional calcification of the physis. This pathology is most obvious in the metaphyses of the long bones. There may be a wide variety of clinical signs, including bone pain, stiff gait, swelling in the area of the metaphyses, difficulty in rising, bowed limbs, and pathologic fractures. Animals fed all-meat diets are commonly affected.

Treatment:

- Correction of the diet is the primary treatment. The prognosis is good in the absence of pathologic fractures or irreversible damage to the physes. If the animals are housed, exposure to sunlight (ultraviolet radiation) will also increase the production of vitamin D_3 precursors.