Avian Goiter (Thyroid Hyperplasia or Dysplasia)

Thyroid hyperplasia is an enlargement of the thyroid glands due to abnormal proliferation of the epithelial cells lining the follicles. The glands are located in the neck region. Thyroid hyperplasia has been observed in many species of birds, including pigeons, canaries, budgerigars, and wild birds. It is the most common disease of the thyroid gland in the budgerigar.

Several conditions associated with damage to the thyroid gland result in goiter. The most common is a dietary iodine deficiency. Substances called goitrogenic agents, which interfere with the normal production of thyroxine by the thyroid glands, may also result in the disease. Feeds containing goitrogenic agents include soybean, flax, rapeseed, kale, cabbage, broccoli, and turnips. Septicemic diseases with an acute onset may affect the thyroid glands causing inflammation and hyperplasia. Exposure to toxic levels of organophosphates or chlorinated biphenols may also result in enlargement and hyperplasia of the glands.

An inadequate level of iodine in the diet leads to a lack of available iodine in the thyroid glands. Iodine is needed for the production of thyroxine. When low levels of thyroxine are in the bloodstream, the brain sends signals to the thyroid glands causing a proliferation of follicular epithelial cells. These cells are responsible for thyroxine production. Hyperplasia of the thyroid gland is a response to the body's need to produce more thyroxine. With sustained iodine deficiency, the signals continue to stimulate epithelial cell proliferation and produce thyroid enlargement.

Goitrogenic substances act by blocking the production of thyroxine, even though adequate levels of iodine are present. Clinical signs and lesions similar to those described for the iodine deficiency occur.

The clinical signs of thyroid hyperplasia are due to abnormal function of the thyroid glands and marked increase in gland size.

The signs associated with abnormal function of the glands are variable and include:

1. Immune deficiency - the birds easily develop infections
2. Depression and lethargy - the metabolic rate of the birds is decreased
3. Abnormalities of the skin and feathers
4. Reproductive problems - birds experience increased embryo mortality and decreased hatchability.

Clinical signs associated with enlargement of the glands result from mechanical pressure on neighboring body organs. These include

1. Convulsions and sudden death - the glands put pressure on the heart and major vessels
2. Vomiting, weight loss, and difficulty swallowing-blockage of the crop and esophagus so food cannot pass normally
3. Respiratory abnormalities and loss of voice - obstruction of the trachea preventing normal passage of air.

Recent findings suggest that clinical signs may *not* result from direct mechanical pressures. It is now suspected that excessive fluid secretions in the crop and lower intestine and accumulations in the lungs are responsible.

Gross lesions associated with thyroid hyperplasia include markedly enlarged glands which are brownish in color and possibly contain yellow granules. Large fluid-containing cysts may also be seen. In severe cases, birds will have grossly visible swelling in the neck.

The diagnosis of thyroid hyperplasia involves examining the history, clinical signs, and diet of the birds. On physical examination, palpation often reveals enlarged masses in the neck region. Blood thyroxine levels can be measured to determine if they are within the normal range. Necropsy of the bird allows direct visualization of the thyroid glands. The glands can be examined microscopically if doubt still exists.

In areas where iodine deficiency is known to exist or birds are fed goitrogenic substances, iodine supplementation is recommended. Providing one drop of dilute Lugol's solution in 1 oz. of fresh drinking water once a week is the suggested preventative.

Other diseases of the thyroid glands which need to be differentiated from hyperplasia and dysplasia include thyroid tumors and cysts.

*Source*: edis.ifas.ufl.edu/VM029

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**Footnotes**

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