Chapter 4

Iodine Deficiency in Goats

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Additional information is available at the end of the chapter

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Abstract

Iodine deficiency is a common problem among humans and livestock throughout the world. Prevalence is very high in goats due to less access to soils and browsing habits of goats. It is primarily due to deficiency of iodine in soil, feed, fodder, and water or secondarily due to the presence of goitrogens in diet of animals. Clinical deficiency is characterized by cardinal signs of goiter, whereas subclinical deficiency is difficult to diagnose because clinical signs are not evident. Clinical signs are more prevalent in kids as compared to adults. Diagnosis is on the basis of clinical sign of goiter and estimation of thyroid hormones, the plasma organic iodine level. Milk and urine iodine levels are good indicators of iodine deficiency. Deficiency can be prevented by daily supplementation of iodine and avoiding diets high in goitrogens.

Keywords: iodine, goats, goiter, deficiency

1. Introduction

Iodine is an important micromineral that has a vital role in the synthesis of thyroid hormones like triiodothyronine and thyroxine. These hormones have a role in thermoregulation, increasing cellular respiration and energy generation and have widespread effects on intermediary metabolism, growth, development, reproduction, muscle function, immune defense, and circulation [1].

The synthesis of thyroid hormones takes place in the thyroid gland of all species of animals and also in goats. The size of the gland is approximating 0.20% of bodyweight [2]. It contains the highest concentration of iodine (0.2–5%) of dry weight and in the largest amount (70–80%) of total body iodine.

It is a bilobed structure situated slightly behind the larynx. Right and left lobes of thyroid gland lie lateral to the trachea and are joined by a thin isthmus that passes across the ventral aspect of the trachea [3]. The thyroid gland is a highly vascularized tissue constituted by the functional unit called thyroid follicle. Each follicle has a spherical structure composed of an

outer monolayer of follicular cells surrounding an inner core of colloid, the thyroglobulin-hormone complex, which is the storage reservoir of thyroid hormone. The colloid stored in the lumen is a clear, viscous fluid. The size of the follicles and the height of their cells vary according to the functional state of the gland. The cells may vary from an inactive squamous cell to the highly active, tall columnar cell.

Thyroid hormones (T4, T3, and rT3) immediately on entering the circulation are bound to transport proteins, mainly to thyroxine-binding globulin (TBG), in lesser amounts to thyroxine-binding prealbumin (TBPA), and to albumin. There is a wide spectrum of species variation in hormone binding by serum proteins. TBG is the major binding protein for hormone, but not all species have TBG; however, TBPA is present in all species [4].

The soil in large geographic areas of the world is deficient in iodine. About 29% of the world's population, living in approximately 130 countries, is estimated to live in areas of deficiency.

Iodine deficiency is more prevalent primarily in mountainous regions such as the Himalayas [5], the European Alps, and the Andes, where iodine has been washed away by glaciations and flooding. Iodine deficiency also occurs in lowland regions far from the oceans, such as Central Africa and Eastern Europe. Globally, 2.2 billion people are at risk for iodine deficiency disorders (IDD). Of these persons, 30–70% have goiter and 1–10% have cretinism. The clinical disorders of iodine deficiency tend to be more profound in geographic areas associated with coexisting selenium and vitamin A deficiencies and in regions where goitrogens are fed in diet [6].

Iodine deficiency in large areas of the world is associated with iodine cycle in nature. Iodine occurs in the soil and the sea as iodide. Iodide ions are oxidized by sunlight to elemental iodine which is highly volatile. The concentration of iodide in seawater and air is about 50 μg/l and 0.7 μg/m^3, respectively. Iodine in atmosphere is returned to the soil by rain and snow which has a concentration in the range of 1.8–8.5 μg/l. The return of iodine is slow and small in amount compared to the original loss, and repeated flooding further decreases iodine in the soil. High rainfall, snow, and floods increase the loss of soil iodine due to melting of glaciers in hilly area due to global warming [7].

Nutritional iodine deficiency in livestock is the leading cause of thyroid gland disorders/goiter. It generally occurs in farm animals wherever human goiter is endemic. Goat is considered as indicator species of iodine deficiency because of browsing habits and less ingestion of soil compared to other grazing animals [3].

2. Material and methods

This chapter is regarding iodine deficiency in goats. It includes the etiology, clinical findings, diagnosis, treatment, and control of iodine deficiency in goats. The authors also include figures of an outbreak of abortion and premature birth of kids with goiter in a flock of goats of Jammu and Kashmir state. Blood samples were collected for hematology, thyroid profile, and estimation of plasma iodine. Urine and water samples were also examined for iodine estimation. Histopathology of thyroid gland was also performed. Affected goats were treated with iodized oils with complete control on occurrence of such condition in future.

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3. Etiology

Iodine deficiency is of two types, that is, primary and secondary or conditional deficiency.

Primary deficiency is an environmental deficiency due to the low level of iodine in soil, water feed, and fodder of animals. A deficiency of iodine in soil and subsequently in fodder crops is the primary reason for iodine deficiency in animals. Soil deficiency may be due to leaching of iodine from surface soil and poor replenishment with airborne iodine [8]. Drinking of groundwater containing iodine less than 2 μg/l leads to iodine deficiency.

In general sandy soils are low in iodine. High clay content and high pH of soil interfere with the iodine uptake by plants growing on such soils.

Iodine content of plant varies with species, strains, climatic and seasonal conditions, and chemical fertilizer supplemented to plants. Cereals, wheat bran, and oil cakes are poor in iodine, whereas straws and green fodders contain marginally adequate content of iodine as per requirement by livestock. Stage of maturity and cutting time significantly affect the iodine content of the fodder. Iodine content of fodder decreases with fall in environment temperature and vice versa [9, 10]. The excessive use of chemical fertilizers like DAP and potash decreases the uptake of iodine from soil; conversely, supplementation of seaweeds in soil will increase the iodine content of soil.

Secondary or conditional deficiency is due to the presence of certain substances present in some plants called goitrogens. It results in iodine deficiency disorders in animals despite of normal iodine intake (1–4 ppm of dry matter), because it interferes with the utilization of dietary iodine or with its metabolism in hormone synthesis. Goitrogens in feed and fodders of animals increase the usual dietary requirement of dairy animals by four to five times.

The presence of goitrogens in diets consisting largely of cruciferous plants like cabbage, *Brassica* spp., peanut, soybean, and yellow turnip contains cyanogenetic glycosides that are goitrogenic because on hydrolysis yields of hydrocyanic acid due to structural damage of the cell wall of plants and further converted to thiocyanates by ruminal microbes. Action of thiocyanates can be overcome by increasing supplementation of dietary iodine. Goitrin (thiooxazolidone) is a thiouracil type of goitrogen present in the seeds of rape, kale, and other *Brassica* spp. It inhibits hormone synthesis, and its action cannot be overcome by extra supplementation of dietary iodine. Linseed meal contains glycoside arachidoside (linamarin) which is converted into thiocyanates in the rumen. Subabool (*Leucaena leucocephala*) contains an alkaloid mimosine (3–6%) which not only inhibits the utilization of iodine but also prevents the availability of iodine from other ingredients in the ration fed to animals. Drinking of grossly bacterial-contaminated water and ingestion of such feedstuffs reported to develop goiter in ruminants. A continued intake of the grass (*Cynodon aethiopicus*) and African pearl millet (*Pennisetum typhoides*) with low iodine and high cyanogenetic glycoside contents may cause goiter in lambs and goats, respectively.

Excess intake of calcium decreases the absorption of iodine from the gastrointestinal tract. High fluoride ingestion is also implicated as one of the important factors for development of goiter in animals. Deficiency of cobalt and thus vitamin B_{12} reported to increase thyroxine levels accompanied by marked hypertrophy and hyperplasia of thyroid gland [11, 12].

Among different breeds of goats, Boer goat of South Africa seems to be more susceptible to develop iodine deficiency due to rapid growth. Indigenous breeds of Himalayan region are more resistant to iodine deficiency than Barbari and Alpine goats. The Angora goat is also reported to be very susceptible to iodine deficiency [3].

4. Pathogenesis

Inadequate iodine in the thyroid gland results in the synthesis of uniodinated inactive pre-hormone instead of the thyroxine, which stimulates the pituitary gland to secrete thyroid-stimulating hormone (TSH). This commonly results in hyperplasia of the thyroid tissue and considerable enlargement of the gland, that is, goiter (**Figures 1** and **2**).

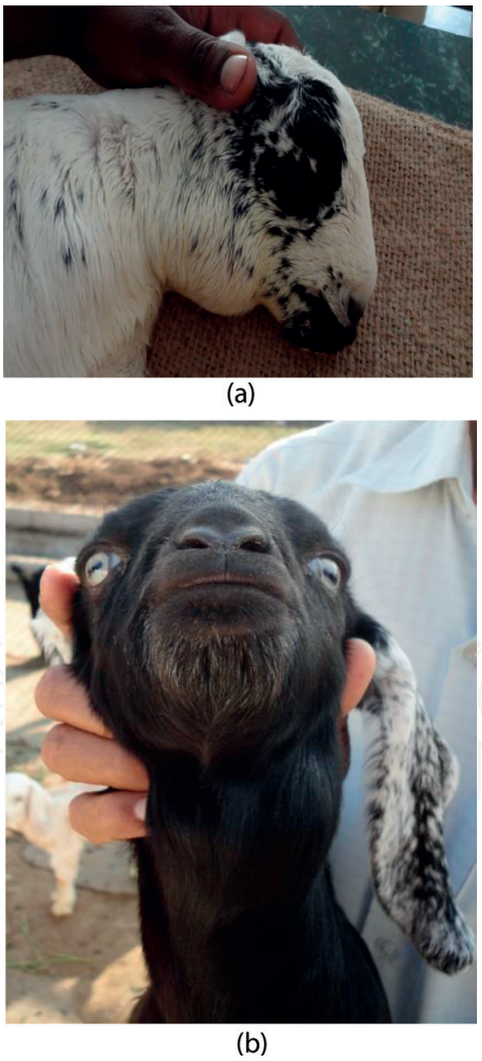

Figure 1. Enlarged thyroid gland/congenital goiter in a kid presented to the University clinics (Courtesy of Dr. R.K. Bhardwaj).

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Figure 2. Kid borne with goiter to an apparently normal goat (Courtesy of Dr. R.K. Bhardwaj).

5. Clinical findings

Iodine deficiency occurs in many species of animals and develops different signs in different animals. Goiter is a cardinal sign of iodine deficiency manifested mostly in young ones, that is, kids. The normal thyroid gland is 0.20% of body weight, but it is markedly enlarged up to the size of an orange in iodine-deficient goats [3]. The thyroid gland of goats/kids may be graded as palpable and plum size (+), easily palpable and lemon size (++), and duck egg size, hanging and visible from the distance (+++) (**Figure 3c**). Kids who survive the initial danger period after birth may recover except for partial persistence of goiter (**Figure 1**). The gland may pulsate with the normal arterial pulse and may extend down a greater part of the neck and cause some local edema. Auscultation and palpation of the jugular furrow may reveal the presence of a murmur and thrill (the thyroid thrill) due to the increased arterial blood supply of the glands [12, 13].

Affected kids are born with enlarged thyroid gland or goiter, and enlargement of pituitary gland is also reported [14]. There may be birth of premature kids which are very weak and die within few hours of birth due to severe dyspnea caused by compression of the trachea by enlarged thyroid glands. Majority of new born kids may be hairless/bald (**Figure 3a**) or covered with very fine hair due hypoplasia of hair follicles [15]. Kids may appear dumb or unwilling to suckle the dam [3]. Growth rate of kids is stunted. Fertility of does is affected. Iodine supplementation in goats is found to increase conception, succeed in the first insemination, and reduce abortion rate and dystocia due to goiter [16, 17].

Figure 3. (a) Premature kids aborted with goiter and baldness. (b) Dead kid with goiter. (c) Exposed enlarged thyroid gland (Courtesy of Dr. R.K. Bhardwaj).

6. Diagnosis

Diagnosis of iodine deficiency is based on the history of supplementation of mineral mixture and iodine in the diet of animal and clinical signs like stillbirth, abortion, birth of weak kids, or dead kids with congenital goiter. It is easy to diagnose clinical iodine deficiency. The subclinical deficiency is of more importance as it is difficult to diagnose and goes unnoticed. Subclinical iodine-deficient animals show few or no clinical signs, but production, growth rate, and fertility are affected.

Thyroid hormone (triiodothyronine and thyroxine) assay is used to confirm iodine deficiency or hypothyroidism in goats. Normal range of thyroxine is 3.3–7.0 μg/dl and is decreased in iodine deficiency [3]. Triiodothyronine is an active form of thyroid hormone at the cellular level which needs to be converted from thyroxine to triiodothyronine by selenium-containing enzyme deiodinase. Deficiency of selenium may result in increase in the level of thyroxine and decrease in triiodothyronine and elevated plasma cholesterol.

Estimation of protein-bound (organic) iodine in blood is highly sensitive for diagnosis of iodine deficiency. Low protein-bound (organic) iodine below 8.1 μg/dl is suggestive of iodine deficiency [3].

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The rate of excretion of iodine in milk and urine can provide useful diagnostic criteria in simple iodine deficiency since iodine intake is positively correlated with urinary iodine excretion. The milk iodine level of sheep is 80 μg/l and in the deficiency level is below 8 μg/l. Similarly, lower normal limit of iodine in urine is 50 μg/l.

Normal thyroid gland contains acini lined by low cuboidal epithelium filled with colloid, whereas in goiter, it is replaced by tall columnar epithelium, papillary in foldings, and reduced colloid. A hyperplasic goiter is characterized by enlarged gland with much colloid also known as colloid goiter when dietary supplementation of iodine is made to goats. Iodine content of the thyroid gland is reduced (**Figure 4a–c**). Subclinical iodine deficiency can be diagnosed histopathologically by hyperplasia of the thyroid epithelium with grossly normal size of the thyroid gland [3].

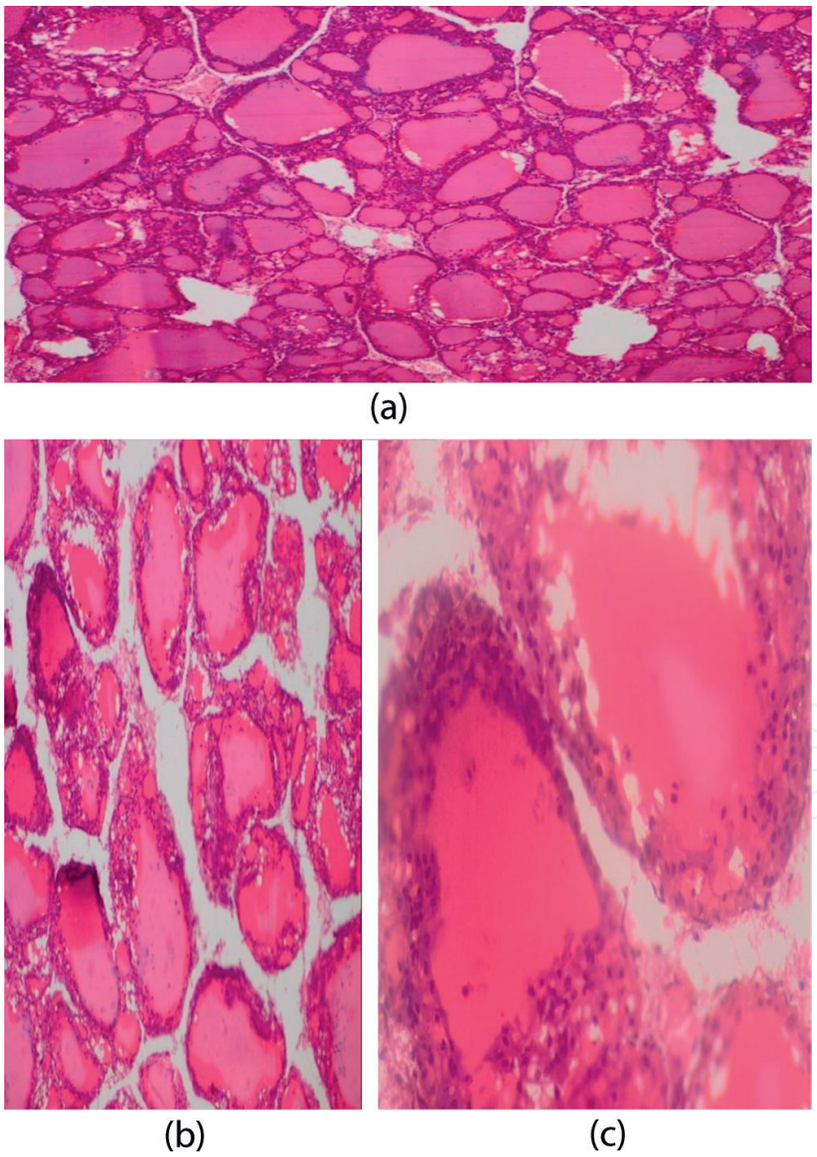

Figure 4. (a) Histopathology of enlarged thyroid gland at 10×, (b) 40×, and (c) 100× showing various sizes of thyroid follicle with colloid and hyperplasia of the epithelium of thyroid follicles (Courtesy of Dr. R.K. Bhardwaj).

7. Treatment and control

The actual dietary iodine requirement is 0.8 mg/kg dry matter for lactating goats and 0.5 mg/kg dry matter for the rest of the flock. When goats are fed cruciferous plants, iodine requirement is approximately 2 mg/kg dry matter to prevent iodine deficiency [19].

Iodine deficiency goiter is treated or prevented by supplementation of iodine to the goats especially to pregnant does in the form of iodized salts. The recommended iodine content of salt is 0.0190%; it should be supplemented to livestock as 2% in concentrates or 0.5% of the total dry matter intake. The daily iodized salt requirement for goats is 4.5 g for adults and 2–2.5 g for kids [18].

Oral daily supplementation of 130 mg potassium iodide or application of 1 ml of tincture of iodine weekly on the back during pregnancy successfully prevented goiter in goats.

The author has treated outbreak of stillbirth in goats with 28 g of potassium iodide in 1 l of distilled water and 10 ml drench to each doe checked abortion in flock.

Prophylactic injection of 375 mg of iodized oil before servicing of goats prevented relapse of stillbirth and congenital goiter in goats.

In general goiter can be prevented by avoiding goitrogenic diet and forages especially during gestation period and regular supplementation of iodine in the diet of goats.

8. Conclusions

Iodine deficiency is a most common problem in goats all over the world due to browsing habits of goats and ingestion of plants containing goitrogens. Deficiency of iodine and development of goiter in goats are suggestive of iodine deficiency in the area. It can be treated by supplementation of iodine in oral or injectable before servicing and avoiding goitrogens containing feed during gestation period.

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