Vitamin A In Veterinary Medicine

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VETERINARIANS now have available vitamin preparations which are intended for use as specific drugs in the prophylaxis and therapy of disease processes. Use of these products requires knowledge of indications for their use in rational therapy. This article is intended to aid in presenting this information for vitamin A.

Three forms of vitamin A have thus far been determined and they are at times collectively called the anti-xerophthalmic vitamin. Animal metabolism is the only natural source of vitamin A, but natural synthesis by animals requires carotenoids which are formed only by plants. The vitamins A are alcohols, containing a beta ionone ring, which are fat soluble and almost colorless. They are to a marked degree oxygen labile, light labile, and their activity is destroyed by hydrogenation. The esters are, in general, more stable.

Provitamins A

There are ten known forms of provitamins A which are also termed carotenoids. Alpha, beta, and gamma carotene are found in roughages and cryptoxanthine is found in yellow corn. Carotenoids have no vitamin A activity of themselves and appear almost entirely in plant tissues. They are deep red in color, but occur mainly in association with chlorophyl which masks this tint, and also gives a fairly reliable indication of provitamin A present in the feed.

Important naturally occurring sources of carotenoids or provitamins A are: Green pastures, certain root crops such as carrots, sweet potatoes, properly cured hays, green vegetables, pumpkins, and fruits. Quantitatively carotene in fresh vegetables is less than .01 per cent by weight; in carrots it is approximately .01 per cent. Palm oil contains .15 to .20 per cent carotene. Carotenoids may be found in plant tissues in the free form or combined with proteins as are many other vitamins. They occur in non-plant tissues in fat depots of the animal body, milk and butterfat, egg yolk, corpus luteum of the cow, and the adrenal glands of most animals. Carotenoids are not found to any extent in seeds. Amounts present in seeds, except yellow corn and palm kernels, are not adequate for nutrition of farm animals.

Ration Deficiency

Thus vitamin A deficiency can be suspected when the ration has consisted mainly of grains, except yellow corn. Carotene is readily oxidized in air and light and thus it is expected that hays which endured a long field curing process will be low in provitamin A and feeding them will lead to deficiency symptoms. Storage for periods of over one year materially reduces cryptoxanthine content of yellow corn. Periods of drouth, excessively rainy haying seasons, and stable-feeding of feeds thus grown or improperly cured will all predispose to hypovitaminosis A. The chlorophyl content of a feed or roughage is a fairly reliable indication of its vitamin A value. The standard of comparison must, however, be fresh, green spring grass. Properly cured hays and hay meals will approach this standard. Examination of the ration fed and of feeding prac-

tices is very important in diagnosis of vitamin A deficiency.

Vitamin E has been shown to protect carotene against oxidation because it is itself an anti-oxidant. It has been so used in commercial feeds. Vitamin A has a beneficial effect on low ascorbic acid blood levels.

Physiology and Metabolism

Vitamin A is preferably administered orally, but can be given intravenously and may be absorbed through the skin. This vitamin is more readily absorbed from fats and oils, especially unsaturated ones. It is not absorbed from mineral oil. Absorption is associated with presence of bile salts, bile acids, and pancreatic lipase. It may be absorbed from a water emulsion. Vitamin A is readily absorbed in 5-6 hours and the provitamins A in 7-8 hours. Factors interfering with absorption of vtiamin A are: diarrhea, mineral oil in continued dosage, bile duct obstruction, congenital atresia of the bile duct, cirrhosis of the liver, hepatitis, pancreatic fibrosis, and intestinal obstruction.

In the body, the provitamins A are removed from the blood stream by the reticulo-endothelial system and stored largely in the liver, mainly in the Kupfer cells. These stores are gradually converted into vitamin A, and circumstantial evidence points to the liver as the place of conversion, although no direct proof is available.[1] Storage is largely in the liver. Vitamin A is carried in the blood stream in the serum portion, and in loose concentration with proteins. The blood level is somewhat independent of amounts stored in the liver and does not furnish an estimate of the level in the body until the estimate can be more accurely determined by observed symptoms.

Increased Demands

There is increased demand for vitamin A in all febrile conditions, in production of carcinomas, and in injection of carcinogenic compounds. Pregnancy and lactation also increase demand. Pathologic conditions may interfere with the interaction between blood levels of vitamin A and the liver content. Pneumonia, various infections, and tuberculosis of humans have demonstrated a reduced vitamin A blood level.[18] In fact, repeated determinations of blood levels have been used for their prognostic value.

Pathology

Before discussing specific species symptomatology and pathology of hypovitaminosis A, it is well to study the tissue changes produced by this condition in general. Among those demonstrated at present are: epithelial changes, testicular degeneration, continuous diestrum, urinary calculi, neurological lesions, nyctalopia, anophthalmia in new born pigs, stenosis of the optic canal, impaired tooth development during formative stages. The animal is found very susceptible to secondary bacterial invasion due to epithelial changes. Symptoms of inanition such as reduced growth rate, decreased activity of hematopoetic tissues, and retarded hair growth are found even as in most other vitamin deficiencies.

Epithelial Changes

The epithelial changes are considered the most characteristic of hypovitaminosis A as they appear at all ages and presumably in all vertebrates.[27] They have been demonstrated in man, monkey, horses, cattle, sheep, swine, goats, dogs, foxes, rabbits, chickens, turkeys, ducklings, game birds, guinea pigs, rats, and mice. Essentially the same organs are involved in all species although the order of response may vary slightly. There is a keratinizing metaplasia found in the salivary glands, respiratory tract, genito-urinary tract in both sexes, eyes, paraocular glands, corneal and palpebral conjunctiva, Harderian, intra-orbital, extra-orbital, and Meibomian glands.[26] Corneal ulceration is often demonstrated. The epithelium which has a secreting function in addition to protective function and whose functioning cells cannot divide is in general that which is replaced with a stratified keratinizing epithelium. The epithelium of the cornea, renal pelves, ureters, and bladder increases in growth rate and becomes hyperkeratotic in hypovitaminosis A.

The keratinized epithelium of each region returns to its normal function and morphology upon supplementation with vitamin A. This indicates that the stratum germinativum or similar structure retains its power to differentiate into the functional cells of the region during an entire period of metaplasia which is due to hypovitaminosis A.

General Changes

Changes in the teeth consist of an atrophy and keratinizing metaplasia of the enamel organ which is the primordium of the tooth. Following this atrophy there is a dysfunction of the odontoblasts or dentine formers. The end result is a malformed tooth which fails of eruption. This has been observed in rats. Horses present difficult dentition of permanent teeth in which hypovitamosis A may be a factor.

Changes in the osseous and nervous systems are observed. When vitamin A deficiency is established at a sufficiently early age, skeletal growth is retarded a considerable period of time before the rate of increase in weight is materially affected. The central nervous system continues growth at a normal rate until weight becomes stationary or decreases. This results in overcrowding the cranial cavity, overcrowding the spinal canal, and mechanical damage with subsequent irregular nerve degeneration in the central and peripheral nervous systems. Rate of growth rather than age is the governing factor in this condition. Mature individuals will not usually develop these lesions.

Nyctalopia or interference with visual adaptation to dark is an early symptom of vitamin A deficiency. Vitamin A and the protein, retinine, combine to form rhodopsin or visual purple. Rhodopsin is a pigment of the rods that breaks down upon exposure to light of low intensity and initiates a nerve impulse. Lack of vitamin A in sufficient quantities results in decreased formation of rhodopsin during subsequent darkness and consequently hinders dark adaptation.

Species Symptomatology

With an understanding of the pathological picture produced by hypovitaminosis A, the symptoms and lesions exhibited by various species form a more understandable pattern. These will now be discussed for each species including both symptoms demonstrated and lesions produced as they are applicable to that species.

Horses demonstrate nyctalopia, lacrymation, corneal keratinization, respiratory symptoms, reproductive difficulties, capricious appetite, and progressive weakness with death occurring quite regularly.[7] Scaling of the periople of hoof and a regular fading of area below the coronary band in pigmented hooves are symptoms of hypovitaminosis A observed in equines. Accompanying this fading is a defective hoof growth.[9] This is frequently seen in horses during wintering on rations which are deficient in vitamin A or in stable fed horses. Dentition disturbances and improper formation of permanent teeth may also be produced by this deficiency although this has not been demonstrated experimentally in the horse to date.

Ruminants

Symptoms of hypovitaminosis A in ruminants are: Nyctalopia, corneal opacity and ulceration, mottled appearance of tapetum lucidum and nigrum,[14] convulsions, anascarca with swelling of legs and other parts of the body, nasal discharge, unpliable skin, dry pityriasis, failure of reproduction, panting and drooling in hot weather, sensitiveness to heat, loss in weight, anorexia and death in severe advanced cases.[22] These symptoms develop in varying periods depending upon body reserves which in turn depend upon age and amount of carotene contained in feed. Young animals are more rapidly depleted than old. Blindness without classical symptoms of deficiency may develop in young due to stenosis of the optic foramen.

A greater amount of carotene is required for maintenance of health than for fattening cattle. However, recent studies have demonstrated development of typical vitamin A deficiency symptoms in cattle fattened on a balanced ration containing 11-14 pounds of yellow corn and oat straw as roughage.[20] This empha-

sizes the value of green leafy hay and silage of good quality in the ration of fattening cattle. It was previously thought that yellow corn provided sufficient carotene to supply the needs of fattening cattle.

There apparently is some connection between urolithiasis and vitamin A deficiency in goats.[22] This is thought to result from the desquamation of cornified epithelial cells from the kidney and ureters which are found in large masses in the urinary tract. These may act as nuclei for uroliths. Extension of this finding to other species except the rat has not been conclusively demonstrated. Microscopic examination of bovine kidneys from A deficient cattle revealed degenerative changes which were most extensive in the proximal convoluted tubules.[11] Some renal inflammatory changes were observed characterized by accumulations of lymphocytes and some macrophages with tubular atrophy. Some cicatrization was evident but keratinizing metaplasia was rare.

Reproductivity

Bulls and cows may lose reproductive ability in advanced vitamin A deficiency but this is not necessarily permanent.[9] Cows will usually conceive and later abort or give birth to weak or blind calves which fail to survive in most cases.

Vitamin A has been used in treatment of acetonemia with apparently excellent results in some cases but there is evidence that uncomplicated cases of ketosis in cows have normal blood plasma carotene and vitamin A levels.[23] The oral administration of vitamin A in dosages of 1 to 4 million I.U. over periods of as long as 3 weeks had no beneficial effect in these cases.

Deficiency at Birth

It has been demonstrated that carotene and vitamin A do not cross the placental filter in appreciable amounts and therefore the newborn individual is dependent almost entirely on external sources for vitamin A activity. It is also known that colostrum milk has about 10 to 100 times the vitamin A activity of milk obtained after the first few days of lactation. This accentuates necessity for feeding colostrum milk and for adequate provision of vitamin A to the maternal parent during pregnancy.

This factor undoubtedly operates in most of the species but particular study has been made of it in the dairy calf.[3 13 15 16 17 25] Nyctalopia, papillary edema, and constriction of the optic nerve causing blindness have been observed in calves deficient in vitamin A or its precursor. Degenerative and inflammatory conditions in kidneys, enteritis, persistent diarrhea, degeneration and necrosis in liver, and degenerative changes in the testicles have been observed in this condition. Metaplasia of epithelial tissues is not consistently observed.

It has been shown that one causative factor in diarrhea of calves is the deficiency of vitamin A with which they are always born. The newborn calf has about 4 micrograms of vitamin A per 100 cc. of blood and requires 15 micrograms per 100 cc. of blood is an adequate level.[17] Supplying this deficiency along with niacin and ascorbic acid has reduced economic and individual losses from this condiiton considerably in territories under observation. Niacin apparently aids in absorption of vitamin A in the newborn calf.

Swine

Observations on experimental deficiency of vtiamin A in swine have demonstrtaed the following: Initial anorexia, inappetance and roughened hair coat, slight exophthalmus, lacrymation, xerophthalmia, blindness, cocking of the head, stiff and awkward gait.[4 5] Soon after the abnormal gait is noticed nervous symptoms appear and consist of convulsions, running movements while lying down, and audible complaint. These convulsions terminate with twitching limbs and exhaustion. Posterior paralysis and even loss of control of all legs is observed. Lesions at necropsy revealed a rather constant catarrhal gastritis, enteritis, and congesion of the lungs. Recent experiments demonstrated that hogs suffering from vitamin A deficiency showed marked instability and loss of equilibrium along with extreme nervousness.[21] There was a swaying, uncertain circling, and finally

loss of balance, falling, and struggling to arise. These symptoms were considered as differing from previously reported posterior paralysis. The first symptoms in these cases appeared in about 140 days from the onset of a deficient ration.

Estrous Cycles

In contrast to other species estrous periods in swine apparently become abnormally long and more frequent in some individuals but others fail to breed entirely.[8] Vulvar congestion may persist in gilts as if they were in continuous estrum. Sows on moderate deficiency of vitamin A, if bred, may abort or resorption of feti take place. If feti are carried full term they may be born weak or die shortly after birth. Embryonic injuries and developmental defects have been observed in this condition. Anophthalmia, optic stenosis, accessory ears, subcutaneous cysts, palatoschisis, cheiloschisis, faulty kidney development, and malformed hind limbs are reported.[5]

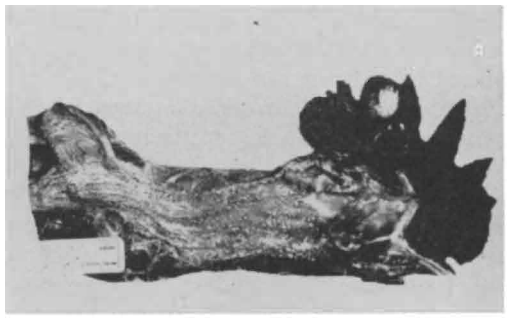
Chicks

Clinical symptoms of hypovitaminosis A are exhibited in chicks as a cessation of growth at three to four weeks of age, drowsiness, incoordination, staggering gait, emaciation, and ruffled plumage.[2] Yellow pigmentation is lacking in breeds normally demonstrating this pigmentation and comb and wattles are usually pale. Eye symptoms may develop but often the chick dies previous to this time.

The susceptibility of chicks to this deficiency depends in the first two weeks of life on the adequacy of the ration fed the maternal parent. This has a definite bearing on total chick mortality in these first two weeks. If the chick has suffifficient vitamin A to last these first two weeks in his tissues then he will survive a deficient vitamin A ration for four to seven weeks.

Adult birds become emaciated, weak, and exhibit a marked decrease in egg production and length of time between clutches. Hatchability of eggs decreases, embryonic mortality increases, and eye symptoms develop, demonstrating in advanced cases a caseous exudate.

Pathological lesions demonstrated in adult birds are mainly the keratinizing metaplasia, particularly of the respiratory and esophageal musoca and an accamulatiou of ureates in the renal tubules,



Courtesy of Beister, et al.

Lesions of Vitamin A Deficiency in the chick.

ureters, and visceral organs. The metaplasia is evidenced by a caseous exudate and an enlargement of the various glands, particularly evident in the esophagus. Early in the deficiency a seromucoid mass of water clear exudate can be found in the nasal turbinates and manipulated by pressure out of the nodules and cleft palate. Other species of poultry and wildfowl demonstrate lesions and symptoms somewhat similar to chickens.

Symptoms demonstrated in dogs include: Xerophthalmia, decreased rate of gain, rough coat and desquamation of skin, conjunctivitis, lacrymation, keratitis, ulceration of cornea, and at times keratocele. There is also anorexia, muscular incoordination and paralysis. There may be inattentiveness due to deafness.[12] This condition has been suspected of predisposing to uroliths in dogs.[6] It is an important factor in predisposing to infectious bacterial dermatitis in the dog.

Foxes

Experimental deficiency of vitamin A in foxes has resulted in demonstration of nervous disturbances prior to development of other symptoms.[24] These occurred about 8 weeks after placing on a

deficient ration and consisted of trembling, twisting of neck as a chicken watching a hawk, periods of whirling and coma at ca. 2-4 months. The coma would last for 5-15 minutes and usually followed periods of excitement. Xerophthalmia was observed in three fox pups between 18 and 27 months after initiation of low vitamin A ration. Other general vitamin A deficiency symptoms were also observed. Attempts to cure the nervous deficiency symptoms failed; two foxes still demonstrating nervous symptoms one year after being placed on a nutritionally complete ration.

Diagnosis

Diagnosis of hypovitaminosis A is most valuable when it is made in the early manifestations of the condition. At this time economic losses can be forestalled and a valuable service rendered livestock owners. Perhaps the complaint will be that the animals are not doing as well as they should. Decreased rate of gain is one of the symptoms which is often found in this deficiency. An inquiry into the previous management of the group in question is essential; the ration fed to young, ration fed to maternal parent, and condition of the ingredients are important. The ration ingredients should be checked as to possible carotene or vitamin A content. Were roughages grass green? Was there any possible content of carotene consuming agents such as meat and bone meal, tankage, or skimmilk in the ration? Actual observation of ration ingredients is more satisfactory than interrogation.

Diagnostic Lesions

Symptoms and lesions are quite characteristic of this condtiion. The keratinizing metaplasia, nyctalopia, and corneal involvement all point to this deficiency. In testing farm animals for nyctalopia, which is one of the earlier symptoms, they can be exercised in the yard near dusk to observe their recognition of obstacles placed in their way. Another method is driving them into a barnway in which has been placed an obstruction and noting their recognition of this object. Use of the ophthalmoscope is a diagnostic aid which is valuable but this must be done in the dark and also after the animal has been in the dark for a period of time.[13] [14] Demonstration of keratotic epithelial cells in smears from corneal, vaginal, and other available areas is a valuable diagnostic aid.

Laboratory Tests

Laboratory tests to determine either the carotene content or the vitamin A content of tissues are usually chemical or physical as biological tests require extended periods of time. They consist of antimony trichloride color reactions and the spectrophotometric method.[18] The spectrophotometric method is not widely used. The antimony trichloride color reaction depends on the development of a transitory blue color which a chloroform solution of vitamin A forms when antimony trichloride is added. The intensity of this blue color can be read by visual comparison with copper sulfate standards or with the aid of a photoelectric colorimeter.[10] The latter reduces human error and makes readily possible both carotene and vitamin A determination but requires additional equipment. The blood level of vitamin A is physiologically more constant than that of carotene and therefore is of more aid in diagnosis. However, blood carotene below certain levels will indicate hypovitaminosis A. Blood for determination may be treated with potassium oxalate and kept refrigerated for 24 hours without materially affecting the determination.

Clinical interest lies in the blood levels below which vitamin A deficiency is indicated. Most studies have been made on cattle and calves. These values for mature cattle are 20 to 50 micrograms of blood plasma carotene per 100 cc. of blood and for calves the critical blood vitamin A value is 15 micrograms per 100 cc. of blood.

The antimony trichloride color reaction can also be used to determine content of vitamin A and carotene in the liver at necropsy. These tests render a feeling of positiveness to diagnosis and are valuable

aids. However, the symptoms are available earlier and should be used on conjunction with the history to evolve an early diagnosis and one which allows for correction of the condition prior to heavy losses.

Prevention

The prevention of hypovitaminosis A lies in feeding those feed substances which are known to have relatively satisfactory amounts of this vitamin and that have been properly handled in curing, storage, processing, and during the feeding process. An important fact to remember is the instability of carotene to oxidation, light, high temperatures, and the presence of products of animal origin in the mixed feeds. Grass green properly cured hays, green pastures, high quality ensilage, sweet potatoes, pumpkins, carrots, and green vegetables are feeds which supply this vitamin. There is increasing evidence that yellow corn does not furnish adequate amounts, particularly if it has been in storage.

Requirements

Vitamin A requirements have been found quite uniform in relation to body weight in horse, cattle, sheep, swine, dog, rabbit, rat, and man. These animals require 25 gamma or 40 I.U. of beta carotene per kilogram of body weight daily. This requirement can also be furnished by 4 gamma or 20 I.U. of vitamin A per kilogram of body weight per day. One gamma is equal to .001 of a milligram. Recommended nutrient allowances established by National Research Council are valuable references giving specific amounts of vitamin A as well as other vitamins and nutrients needed for various species.[19]

There are commercially available two sources of vitamin A; various fish liver oils and solutions of carotene. The fish liver oils contain largely natural vitamin A.

Pure vitamin A has demonstrated some toxic symptoms in the rat consisting of rapid loss of weight, general hemorrhage, bone fragility, and death. Dosages ellicting this response were those in excess of 100,000 U.S.P. units. A U.S.P. unit equals one international unit (I.U.) which is defined as 0.6 gamma beta carotene in coconut oil and hydroquinone.

Interesting observations of symptoms demonstrated by dogs continuously fed rations containing feeds high in vitamin A content have recently been reported.[62] Diarrhea was consistently observed in these cases. This was experimentally shown using concentrates of vitamin A as a constituent of the ration. Liver was one of these feeds. This condition was attributed to an acceleration of metabolism to a point where it causes diarrhea.

Dosage Levels

Provitamin A or carotene can be fed in unlimited amounts with little danger except excess yellow pigmentation. Both carotene and vitamin A are readily absorbed in eight hours or less from oral administration and this is the preferred method although it can be given parenterally and also is absorbed through the skin. Parenteral administration would be indicated where absorption was interferred with or there was evidence of liver damage. Liver damage in the patient would require the preparations containing vitamin A itself. In newborn calves 25,000 I.U. vitamin A daily have been recommended for the first ten days followed by 5,000 I.U. daily for the next 20 days per calf along with niacin, ascorbic acid, and vitamin D if necessary. The recommended daily allowance mentioned previously would give a guide as to the dosage levels which should be considerably greater where deficiencies exist.

Conclusions

In conclusion, hypovitaminosis A has been shown to be a specific problem in disease control which has a specific method of prevention and treatment. The main use of vitamin A preparations lies in treatment of cases which exhibit a deficiency in this accessory food substance. Its use in other conditions uncomplicated by hypovitaminosis A has not been conclusively proven beneficial. However, it must be recognized there are many borderline cases in judging results from its use in individual cases. Vitamin A is

thus best regarded as a specific nutrient element which is preferably furnished the animal by natural feeds but must otherwise be furnished with available pharmaceutical preparations.

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Ornithosis, a disease of pigeons quite similar to psittacosis, is widely prevalent in the United States and is thought may be responsible for many sporadic cases of primary, atypical pneumonia in man. Levinson, Gibbs and Beardwood report (J. Am. Med. Assn, 12-23-44) six such cases in Philadelphia. All had been exposed to pigeons.

Beef cattle in this country now number 17 million more than in 1938 and the trend is still upward. For two years the U. S. Department of Agriculture has been urging increased marketing of beef cattle, but without noticeable effect. With the cessation of the war-time demand for beef, the cattle industry seems to be in for some difficulties.