Clinical study of experimentally induced vitamin E and selenium deficiency in Awassi ewes and their newborn lambs

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Accepted: 31 /1 / 2012

Summary

Experimental induction of vitamin E and selenium deficiency by deficient diet was carried out on Awassi ewes and their newborn lambs. The clinical signs were characterized by sudden death in 4 lambs out of 14 lambs in deficient group and other lambs showed a variable signs included inability to suckle, arched back, weakness, dullness, emaciation and recumbency. Serum selenium and vitamin E levels of these lambs were 0.01 ppm and 0.34 mg/L respectively. The clinical signs in ewes included loss of body weight and loss of wool, Weakness, dullness and recumbency. Serum selenium and vitamin E levels of these ewes were 0.02 ppm and 0.61mg/L respectively. It was concluded that vitamin E and selenium are essential antioxidants and their deficiency exposes the sheep industry to many serious losses.

Keywords: vitamin E, selenium, awasi ewe, deficiency.

دراسة سريرية لنقص فيتامين E والسلنيوم المستحدث في النعاج العواسية ومواليدها

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الخلاصة

تم أستحداث نقص فيتامين E والسلنيوم في الأغنام العواسية ومواليدها من خلال اعطاء عليقة غذائية لاستحداث التنقص الغذائي. هلك 4 حملان من 14 حملا ,والحملان الباقية عانت من صعوبة في الرضاعة ,تقوس الظهر ,الضعف ,الخمول,الهزال ,والرقود.وكان تركيز السلينيوم وفيتامين E في مصل هذه الحملان0.01 جزء بالمليون و 0.34 ملغم/ لتر على التوالي.وفي النعاج شملت الاعراض السريرية فقدان الوزن ,سقوط الصوف,الضعف ,الخمول,والرقود .وكان تركيز السلينيوم وفيتامين E في مصل هذه النعاج 0.02 جزء بالمليون و0.61 ملغم / لتر على التوالي.تم الأستنتاج بأن فيتامين E والسلينيوم هي مضادات أكسدة ضرورية ونقصها يسبب خسائر كبيرة في صناعة الاغنام.

الكلمات المفتاحية: - فيتامين اي ,سلينيوم , النعاج العواسية, نقص غذائي.

Introduction

Animals affected with vitamin E and selenium deficiency may be found in sternal recumbency and unable to stand collapse and die suddenly without any premonitory signs within a few days (1). Pregnant ewes deficient in vitamin E and/or selenium may increase the incidence of stillbirth progeny, or weak lambs, which only survive for a few days before dying because of acute heart failure (2). Skeletal and cardiac muscle necrosis has been reported, and clinical signs can range from stiffness and lameness to acute death, usual age of occurrence is 1 to 4 wk (3). In sheep, the congenital form affects stillbirth or weak neonatal lambs which die soon after birth where as the delayed form affects lambs aged from 3 weeks to 4 months (4). The main locomotors clinical signs range from mild stiffness to recumbence and reluctance to move eventually combined with respiratory distress (5).

White Muscle Disease primarily affects young lambs which may be stillborn or born weakly and die within a few days often from acute cardiac arrest after physical exertion. The general presenting symptoms include weakness, stiffness, rapid respiration and deterioration of muscles. Leg muscles are usually affected first and overt signs can range from mild stiffness and discomfort (often mistaken for joint-ill) to recumbency, reluctance to move or even collapse when driven (6, 7 and 8). The aim of this study was to evaluate the clinical signs of experimentally induced vitamin E and selenium deficiency in Awassi ewes and their newborn lambs.

Materials and Methods

Twenty one Awassi ewes and their newborn lambs from State Board of Agricultural Research / Ministry of Agriculture were used. The weight of ewes ranged between 48 to 55 kg and age ranged between 18 to 26 months. The deficient group includes 14 ewes and the control group includes seven ewes. Ultrasound scanner was used to check the uterine health of the ewes. Estrus synchronization was scheduled. The study lasted for 10 months started on 1/3/2011, and ended on 1/12/2011**.** Induction of selenium and vitamin E deficiency done by feeding a diet consisted of cod liver oil 3%, ground corn 0.5 kg/ animal, discolored bad quality hay ad lib and water was offered all the time (9).Feeding of this deficient diet lasted for three months (the last two months of gestation and one month after birth).The deficiency was diagnosed according to the clinical signs and the decrease in levels of selenium and vitamin E than normal. The control group was allowed the regular feeding program adopted in the state board of agricultural research. No minerals or vitamins were added to the deficient diet.

The animals in the deficient group and the control group were watched at a regular daily basis. Vitamin E in serum was estimated according to (10) by spectrophotometer while selenium in serum was estimated by flameless atomic absorption (11). Blood without anticoagulant was collected directly from the jugular vein at the time when clinical signs of the deficiency occurred and centrifuged to harvest the serum which then stored at -20ċ until being analyzed. Statistical analysis was conducted using ready – made statistical design statistical package for Windows Integrated Student Version (SPSS) (12).

Results and Discussion

Lambs in the deficient group showed the clinical signs of the deficiency within the first three days of life (two months after feeding deficient diet to their dams) the serum selenium levels of the lambs reached 0.01 ppm, while the serum selenium level of the lambs (borne to control ewes) was 0.45±0.03 ppm. Serum vitamin E concentration in the deficient lambs group reached 0.34±0.05 mg/L as compared with 2.27±0.19 mg/L of control group (table 1) with a significant differences at $P<0.05$ across treatments.

The clinical signs in deficient lambs were characterized by sudden death in 4 lambs out of 14 lambs and lambs which remained live showed a variable signs included inability to suckle as showed in figure (1) , arched back as showed in figure (2), weakness, dullness, emaciation and recumbency as showed in figure (3) .

The postmortem changes in the dead lambs were characterized by paleness and atrophy of thigh muscle and paleness of heart muscle as showed in figures (4 and 5).

In ewes of deficient group the clinical signs of the deficiency were apparent almost in all animals and appeared three months after the ewes started to be fed the deficient diet and when serum selenium levels reached 0.02 ppm, compared with serum selenium levels in control group was 0.47±0.3 ppm. and serum vitamin E levels reached 0.61 mg/L compared with serum vitamin E levels in control group 2.72±0.19 mg/L (table1) with a significant differences $P<0.05$. The clinical signs in deficient ewes included loss of body weight and loss of wool as showed in figures (6 and 7). Weakness, dullness and recumbency were also found as showed in figures (8 and 9).

Table (1) Selenium and Vit E levels in serum of ewes and their newborn lambs

Group	Parameter	Serum selenium (ppm)	Serum vitamin E (mg/L)
Deficient group (Ewes)		0.02 ± 0.00 A	0.61 ± 0.09 A
Control group (Ewes)		0.47 ± 0.30 B	2.72 ± 0.19 B
Deficient group* (Lambs)		0.01 ± 0.00 A	0.34 ± 0.05 A
Control group (Lambs)		0.45 ± 0.03 B	2.45 ± 0.16 B

Deficient group: n=14, Control group: n=7, * Four lambs died, Values represent means ± SE, Different capital letters mean significant (P<0.05) results between different groups.

Figure (1) shows a lamb which is unable to suckle

Figure (2) shows a lamb with an arched back

Figure (3) shows dullness and recumbency

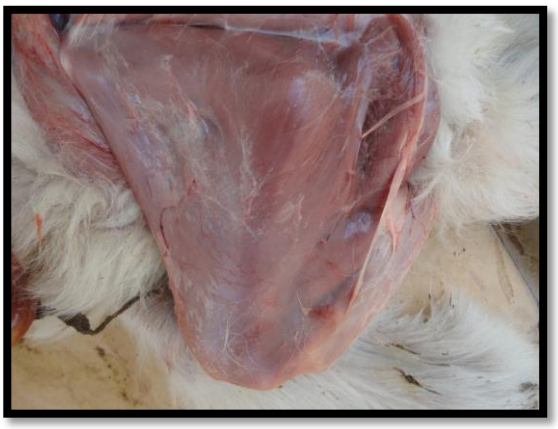

Figure (4) shows paleness and atrophy of thigh muscle

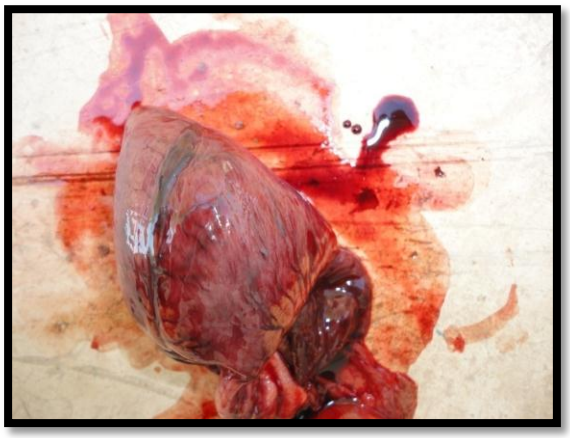

Figure (5) shows paleness of heart muscle

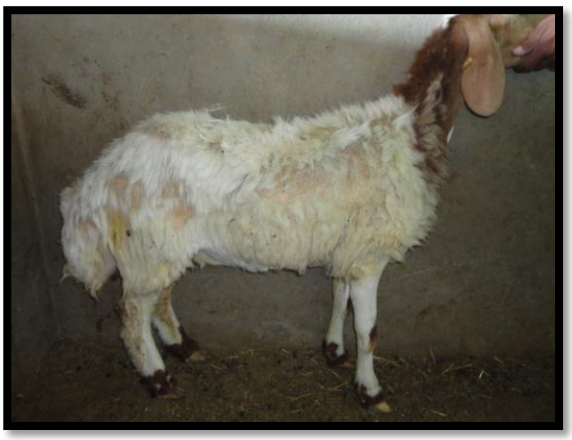

Figure (6) shows loss of body weight and loss of wool

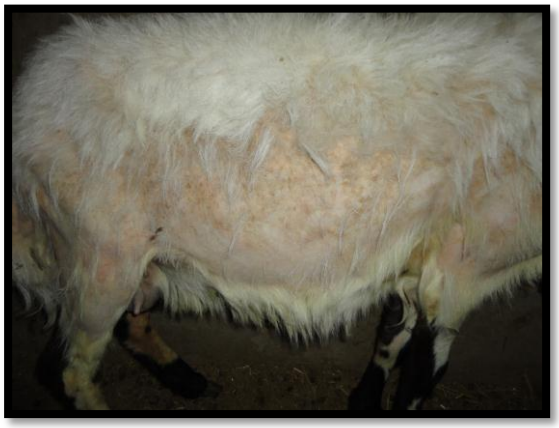

Figure (7) shows loss of body weight and loss of wool

Figure (8) shows weakness, dullness and recumbency

Figure (9) shows recumbency

The clinical signs of the deficiency in this study were characterized by sudden death in 4 lambs out of 14 lambs in the deficient lambs borne to deficient dams, these results were in agreement with (1, 2, 3 and13) mentioned that animals affected with vitamin E and selenium deficiency may be found in sternal recumbency and unable to stand, collapse and die suddenly without any premonitory signs within a few days.

The expected cause of the death is the acute cardiac arrest and this was supported by Hamliri, (2) who reported that pregnant ewes deficient in vitamin E and/or selenium may increase the incidence of stillborn progeny, or weak lambs, which only survive for a few days before dying because of acute heart failure. Other 10 lambs in deficient group which remained live showed a variable signs included inability to suckle, arched back, weakness,

dullness, emaciation and recumbency. The postmortem changes in the dead lambs were characterized by paleness and atrophy of thigh muscle and paleness of heart muscle.

These results were in concert with results of Menzies *et al.*, (6) who reported that lambs affected by white muscle disease that the main presenting complaints were sudden deaths, as well as ill thrift, weakness, and painful ambulation in many of the lambs, gross findings included generalized atrophy of skeletal muscles, pallor and occasional white streaking of skeletal muscles.

In this study clinical signs of ewes included loss of body weight, decreased milk production and loss of wool, weakness, dullness and recumbency were also found, these results were agreed with (7,8 and 13) who reported that animals affected with nutritional muscular dystrophy with involvement of cardiac muscle show acute signs that include recumbency, respiratory distress, and death, while in skeletal muscle involvement hunched appearance, stiff gait, and overall poor production are a predominant clinical manifestations and some adult animals continue to eat, but others are dysphagic because of involvement of the tongue. In conclusion feeding cod liver oil mixed with ground corn for two months during pregnancy and one month after parturition was an effective method of producing an experimental vitamin E and selenium deficiency in Awassi ewes and their newborn lambs.

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