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Clinical case of pregnancy toxaemia and its therapeutic management in a she goat: A case study

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Abstract
A pregnant doe was presented with primary complaint of inappetence and recumbency from last 3 days. The most prominent abnormality during clinical examination was weakness in both hind limbs presented by difficulty in standing. History and abdominal ballotment confirmed that the doe were about 4.5 months pregnant. Typical signs of hypocalcemia ("S" shaped posture, sternal recumbency) were seen. Besides that, ketone bodies were also detected from the urine (3+ or 8mmol/L). The doe was aggressively treated for pregnancy toxemia with administration of 1 liter of 20% dextrose and 5 liter of normal saline BID IV for 3 days. 30ml of glycerine (Enerdyna®) was also given orally BID for 3 days as glucose precursor. Along with aggressive fluid therapy supportive treatment was giving which includes flunixin meglumine @ 2.2mg/kg BW IM OD, methylcobalt (Tribivet M®) @ 2ml IM OD, Phosphorus (Tonophos®) @ 3ml IM OD and antibiotic (Intacef®) @ 10mg/kg BW OD for 3 days. In addition, 100 ml of calcium borogluconate (Qualidrop®) was given slow intravenous (i/v) infusion and 50 ml calcium supplement was given orally 5 days. However, the doe's condition started to deteriorate on Day 3 and kidding was induced by giving 5ml of dexamethasone (Dexona®) intravenously on the same day and two live kids were born after 40 hours of induction. The doe's condition improved tremendously and was kept for oral calcium and glucose therapy for a week thereafter. The goat recovered uneventfully.

Keywords: Doe, clinical management, dexamethasone, pregnancy toxaemia

Introduction
Pregnancy toxemia, also known as "twin-lamb" disease, is a metabolic disorder of pregnant small ruminants, caused by an abnormal metabolism of carbohydrates and fats, which occurs at the final stage of pregnancy [1]. Obese ewes or does carrying multiple fetuses are at higher risk to develop the disease because of the limited space for adequate intake of feed [2]. Rapid fetal development at the late gestation causes rapid mobilization of the fat stores to assure adequate energy. The liver also increases gluconeogenesis to facilitate glucose availability to the fetus. However, in the negative energy balance (NEB), this increased mobilization may overcome the capacity of the liver resulting in hepatic lipidosis. At the same time, ketone bodies are being produced and accumulated, which eventually leads to excessive ketone bodies in blood circulation, and thus increasing the susceptibility to pregnancy toxemia [3].
Pregnancy toxemia is also evaluated as metabolic disorder that occurs in does during the late stage of gestation. Under these conditions, the doe's body is depleted of carbohydrates that are used to produce glucose or sugar. When this condition occurs, rumen capacity decreases as the uterus expands to handle the growth of one or more foetuses [4]. Doe's that have low energy levels are more susceptible to toxemia. This low energy level is caused by a sudden increase in nutritional demands as a result of rapid fetus development[5]. The body of the doe that has multiple foetuses and being fed with low energy feed will utilise alternative sources of energy by producing glucose from other non-carbohydrate substances such as fat to facilitate glucose to the foetuses [6]. When the body mobilizes more fatty tissue, ketone bodies are produced and are released into the blood causing an increase in hepatic fat accumulation leading to hepatic lipidosis.
Obese does carrying multiple foetuses are at higher risk because of the limited rumen space for adequate intake of feed [7]. In severe outbreaks, morbidity can reach up to 20% with a mortality rate of 80% of affected animals [8].
Early clinical signs of pregnancy toxaemia in ewes and does include inappetance, listlessness, aimless walking, muscle twitching, fine muscle tremor, opisthotonos, and grinding of teeth.

As the condition progresses, blindness, ataxia and finally recumbency, coma and death will occur in animals that are not diagnosed and treated with the condition [9, 10].

Treatment

The main objective of treatments in pregnancy toxaemia is to increase the formation of glucose for utilization at tissue level, and increase the usage of ketone bodies to prevent acidosis and electrolytes disorder. However, treatments with drugs are only effective in an early disease diagnosis where prognosis is always grave if the animals start showing irreversible neurological injuries [11]. Euthanasia or removal of foetuses should be considered in advance stages of the disease or in heavily pregnant doe that does not response well to treatment due to the high glucose demand [12]. Treatment of pregnancy toxaemia is usually unsuccessful and prevention is the key in reducing the occurrence of this disease [13]. This manuscript describes the clinical approach in management of pregnancy toxaemia in a doe. This clinical case management highlighted that institution of correct treatment at the right time was able to give better prognosis and success.

History and Clinical Observations

A doe weighing 45 kg during last trimester of pregnancy was presented with primary complaint of inappetance and recumbency for a week (Figure 01A). The doe was managed intensively and was fed with cut herbs, pellets, brewer's grains and supplemented with calcium suspension and a liver tonic. Further the vaccination and deworming status were not up-to-date. The doe was presented on sternal recumbency with body score of 3 out of 5. Clinical examination revealed pyrexia 39.8°C, dull depressed, lethargy, staggering gait and weakness of both the hind limbs.

Result and Discussion

History revealed that the goat is anorectic from last 5-7 days and after 5-7 days of anorexia the goat become recumbent. Clinical evaluation revealed pyrexia, dull and depressed, abdominal ballotment confirmed that the doe was pregnant and carrying viable foetuses that were about 4.5-months old. Typical signs of hypocalcemia ("S" shaped posture, sternal recumbency) were seen on physical examination of goat (Figure 01 B). Besides that, ketone bodies were also detected from the urine (3+ or 8mmol/L). Based on the history, clinical signs and diagnostic work-ups, the doe was diagnosed with pregnancy toxaemia. The doe was treated vigorously for pregnancy toxaemia with 1L of Dextrose 20% (d-20) and 5L of 0.9% NaCl, administered intravenously BID for 3 days as glucose replacement and rehydration respectively. 30mL of glycerine (Enerdyna®) was also given orally BID for 3 days as glucose precursor. Flunixin meglumine 2.2mg/kg was administered intramuscularly SID for 3 days as non-steroidal anti-inflammatory. In addition, 100mL of calcium borogluconate (Qualidrop®) was given slow intravenous (i/v) infusion and 50mL calcium supplement was given orally 5 days and to stimulate body metabolism respectively. One vial (2ml) of methylcobalt (Tribivet M®) was administered intramuscularly SID for 3 days as a nerve supplement, In addition to this, Phosphorus (Tonophos®) and antibiotic (Intacef®) SID for 3 days were provided as a supporting therapy. However, the doe's condition started to deteriorate on Day 3 and kidding was induced by giving 5ml of dexamethasone (Dexona®) intravenously on the same day and two live kids were born after 40 hours of induction (Figure 01B). The induction was possible only due to early diagnosis of the disease. The doe recovered fruitfully after the complete therapeutic approach for 3 days (Figure 02C).

The doe's condition improved tremendously and was kept for oral calcium and glucose therapy for a week thereafter. The doe was bright, alert and responsive with increase in appetite after 2 days of hospitalization.

Pregnancy toxaemia can be divided into four categories [14]. 1) The first category is primary pregnancy toxaemia where inadequate nutrition such as poor quality feed or period of fasting that occurs in a doe. 2) The second category is fat doe pregnancy toxemia which is due to over-conditioned during early gestation then the animal suffer a nutritional decline during late gestation. 3) Third is severely under-conditioned does due to lack of feed after drought or after heavy flood. 4) Lastly is secondary pregnancy toxemia due to concurrent disease such as parasites, poor dentition, or lameness. In this present case, the doe may be suffering from category (1) and (4) pregnancy toxaemia where poor quality of feed was fed throughout the gestation period and the doe was having lameness during the late gestation period. Clinical signs such as anorexia, depression and separation from the herd are the first step in diagnosis of pregnancy toxaemia in a pregnant doe which were the primary complaint in the present case [5]. As the disease progressed, animal will start showing neurological signs include depression, tremors, star-gazing, ataxia, circling, teeth grinding and blindness due to encephalopathy that result from hypoglycaemia [15]. In the final stage without proper diagnosis and treatment, affected animal will be in recumbency and coma where the prognosis at this stage is very poor with low survivality rate [5]. Next, laboratory diagnosis of serum betahydroxybutyrate (BHB), glucose, calcium, potassium parameter and urine ketone level can be performed to further diagnose pregnancy toxaemia [16]. BHB is one of the ketone bodies found from metabolism of fat but was not measured in this case because urine ketone concentrations are more sensitive and specific compared to serum BHB [11].

Fig 1: Doe before kidding in lateral recumbency (A) and after kidding typical hypocalcemic posture (S shaped posture) (B)

Fig 2: Doe showing the signs of recovery after two days of treatment (C)

In cases of pregnancy toxemia, there will be hypoglycemia where the glucose level is less than 2mmol/L and urine ketone

level will be elevated more than 0.7mmol/L which was both observed in the present case. The main therapeutic plan in pregnancy toxaemia is to restore the glucose metabolism and dehydration status, which were the main focus in this case [17]. In the present case, the doe was promptly administered dextrose and fluid therapy to replace the glucose and electrolyte loss, as well as to rehydrate and at the same time to flush out the ketone bodies. Oral replacement of glucose with 30 to 60 mL of propylene glycol twice a day as glucose precursor also must be given [15, 18]. In addition, propylene glycol also inhibits rumen bacteria to prevent production of volatile fatty acid [13]. Similarly, as it was in this case, caesarean is highly recommended in advanced stages of the disease or in heavily pregnant doe that do not respond well to treatment due to the high glucose demand [10]. There are few important points to be noted in the control and prevention of pregnancy toxaemia; high energy supplement feed such as 0.5-1 kg of cereal grain such as corn, oats, barley or combination should be given during the final months of gestation [14].

Besides that, free choice of trace mineral salt and clean water should always be provided ad-libitum to the animal [5]. Animals need to be regrouped based on the stage of pregnancy and evaluation body condition scores (BCS). BCS of 2.5 to 3.5 should be achieved one month before kidding and 2.0 to 2.5 at kidding [19]. Addition of high-energy feeding to thin does with BCS of less than 2 should be performed and revaluation of the condition can be done every 2 weeks [20]. All these can be achieved with good herd health management emphasising on the nutrition, reproduction, housing, and welfare of the animals which will eventually help prevent disease development and outbreaks in order to reduce economic losses [21]. In summary, this present case reports the clinical approach in the management of a clinical case of pregnancy toxaemia in a doe, where prompt treatment at the right time was able to give better prognosis to the dam and the foetuses.

Conclusion

Pregnancy toxaemia is a metabolic disease of small ruminants and susceptibility is higher in dam with twin pregnancy. Negative energy balance between dam and foetuses predisposes the condition. Pregnancy toxaemia results from disruption of the glucose homeostatic mechanism in response to increased nutritional demands of rapidly developing foetuses. Early treatment is very crucial to save the life of the dam and the foetuses. Treatment is benefited from early investigation, which includes timely diagnosis and immediate administration of energy sources to affected animals.

Induction of kidding/lambing in dam is helpful in fruitful recovery of pregnancy toxaemia if the condition of animal deteriorates severely.

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References

1. Brozos C, Mavrogianni VS, Fthenakis GC. Treatment and control of peri-parturient metabolic diseases: Pregnancy toxemia, hypocalcemia, hypomagnesemia. Veterinary Clinics of North America: Food Animal Practice. 2011; 27:106-107.
2. Ermilio EM, Smith MC. Treatment of emergency conditions in sheep and goats. Veterinary Clinics of North America: Food Animal Practice. 2011; 27:105-106.
3. Menzies PI. Pregnancy Toxemia in Ewes: Hepatic Lipidosis: Merck Veterinary Manual. USA: Merial, 2011.
4. Kahn CM, Line S. The Merck Veterinary Manual 9th Edition. USA: Merial. 2005; 14.
5. Tinkler SH. Common Nutritional and Metabolic Diseases of Small Ruminants: http://c.ymcdn.com/sites/www.invma.org/resource/resmgr/2014 metabolic disease pdf, 2015.
6. Brozos C, Mavrogianni VS, Fthenakis GC. Treatment and Control of Peri-Parturient Metabolic Diseases: Pregnancy Toxemia, Hypocalcemia, Hypomagnesemia. Therapeutics and Control of Sheep and Goat Diseases. 2011; 27(1):105-113.
7. Jones M, Navarre C. Fluid Therapy in Small Ruminants and Camelids. Veterinary Clinical Food Animal. 2014; 30: 441-453.
8. Ismail ZAB, Al-Majali AM, Amireh F, Al-Rawashdeh OF. A metabolic profile in goat does in late pregnancy with and without subclinical pregnancy toxemia. Veterinary Clinical Pathology. 2008; 39(4):434-437.
9. Kahn CM, Line S. The Merck Veterinary Manual 9th Edition. USA: Merial. 2005; 14.
10. Abba Y, Abdullah FFA, Chung ELT, Sadiq MA, Mohammed K, Osman AY, *et al*. Biochemical and pathological findings of pregnancy toxaemia in Saanen doe: A case report. Journal of Advance Veterinary and Animal Research. 2015; 2(2):236-239.
11. Cal-Pereyra L, Gonzalez-Montana JR, Benech A, Acosta-Dibarrat J, Martin MJ, Perini S, Abreu MC *et al*. Evaluation of three therapeutic alternatives for the early treatment of ovive pregnancy toxaemia. Irish veterinary Journal. 2015; 68(25):1-7.
12. Lima MS, Pascoal RA, Stilwell GT. Glycaemia as a sign of the viability of the foetuses in the last days of gestation in dairy goats with pregnancy toxaemia. Irish Veterinary Journal. 2012; 65(1).
13. Olfati A, Moghaddam G, Bakhtiari M. Diagnosis, treatment and prevention of pregnancy toxaemia in ewes. International Journal of Advanced Biomedical research. 2013; 1(11):1452-1456.
14. Edmondson MA, Roberts JF, Baird AN, Bychawski S, Pugh DG. Theriogenology of Sheep and Goats. In: Pugh D.G., Baird A.N., eds. Sheep and Goat Medicine, 2nd Edition. Maryland Heights, MO: Elsevier-Saunders. 2012; 150-231.
15. Andrews A. Pregnancy toxemia in the ewe. In Practice. 1997; 19:306-312.
16. Ramin AG, Asri-Rezaie S, Macali SA. Evaluation on Serum Glucose, BHB, Urea and Cortisol in Pregnant Ewes. Medycyna Wet. 2007; 63(6):674-677.
17. Ermilio EM, Smith MC. Treatment of Emergency Conditions in Sheep and Goats. Veterinary Clinical Food Animal. 2011; 27:33-45.
18. Pugh DG. Sheep and Goat Medicine. Philadelphia: W.B. Saunders, 2002.
19. Cannas A, Pulina G. Dairy Goat Feeding and Nutrition, CAB International. 2008; 238-262.
20. Fthenakis GC, Arsenos G, Brozos C, Fragkou IA, Giadinis

ND, Giannenas I, *et al*. Health management of ewes during pregnancy. Animal Reproduction Science. 2012; 130:198-212.
21. Abdullah FFJ, Rofie AM, Tijjani A, Chung ELT, Mohammed K, Sadiq MA, *et al*. Survey of goat farmers' compliance on proper herd health program practices. International Journal of Livestock research. 2015; 5(11):8-14.