

DOI: 10.5455/javar.2015.b78

OPEN ACCESS

CASE REPORT

Biochemical and pathological findings of pregnancy toxemia in Saanen doe: A case report

Yusuf Abba¹, Faez Firdaus Jesse Abdullah^{2,*}, Eric Lim Teik Chung², Muhammad Abubakar Sadiq³, Konto Mohammed³, Abdinasir Yusuf Osman², Nurakmaliah binti Rahamat Rahmat², Ismasyahir Abdul Razak⁴, Mohd Azmi Mohd Lila¹, Abdul Wahid Haron² and Abdul Aziz Saharee²

¹Department of Veterinary Pathology and Microbiology, Faculty of Veterinary Medicine, Universiti Putra Malaysia, 43400 Serdang, Selangor, Malaysia;

²Department of Veterinary Clinical Studies, Faculty of Veterinary Medicine, Universiti Putra Malaysia, 43400 Serdang, Selangor, Malaysia;

³Department of Veterinary Medicine, Faculty of Veterinary Medicine, University of Maiduguri, PMB1069, Borno State, Nigeria;

⁴Hospital of Veterinary Medicine, Faculty of Veterinary Medicine, Universiti Putra Malaysia, 43400 Serdang, Selangor, Malaysia.

*Corresponding author's e-mail: jesseariasamy@gmail.com

ABSTRACT

A pregnant Saanen doe aging 1.5-year and weighing 40 kg was presented to the Large Animal Unit of Universiti Putra Malaysia Veterinary Hospital with history of inability to stand up. Transabdominal ultrasonography of the animal revealed two live fetuses at late pregnancy. Blood examination revealed decreased lymphocyte numbers, and increased monocytes count. Biochemical analyses showed marked decrease in glucose level and elevated level of aspartate aminotransferase (AST) and creatine kinase (CK). The goat was administered with 200 mL 20% Dextrose (G-20), NaCl solution (at 1.3 mL/animal), Flunixine meglumine (at 2.2 mg/kg bwt), and Vitavet multivitamin (at 1 mL/10 kg bwt). The prognosis of the case was grave, and the goat was sacrificed in order to save the fetuses. However, the fetuses were died within 10 min. Necropsy of the doe showed pale, yellow and friable liver and congested lungs, while histopathological evaluation of the liver showed diffuse hepatic lipidosis. Pregnancy toxemia in doe can be prevented by providing proper nutrition.

Keywords

Biochemistry, pregnancy toxemia, hepatic lipidosis, histopathology

	?Т	\mathbf{IC}	I F	S	$\Gamma \cap$	RY	
771	<u>ч</u>			 .0.		TVT	

Received : 19 February 2015,Revised: 26 March 2015,Accepted : 26 March 2015,Published online: 9 April 2015.

INTRODUCTION

Various nutritional, metabolic, genetic, physiologic, environmental, health and/or management factors can influence the development of clinical disease like pregnancy toxemia, and all these should be addressed earlier for the prevention of onset the disease (Fthenakis et al., 2012).

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 (Brozos et al., 2011). 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 (Ermilio and Smith, 2011). 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 a negative energy balance, this increased mobilization may overwhelm the capacity of 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, thus increasing the susceptibility to pregnancy toxemia (Menzies, 2011).

History and Signalment

A one and a half years old pregnant Saanen doe weighing 40 kg was presented to the Large Animal Unit of the Universiti Putra Malaysia Veterinary Hospital after it was found as sternal recumbent in the farm for 5 days. The doe was inactive and inappetent. The doe was managed intensively and fed with napier grass, commercial pellets, soya by-products and theracalcium diets.

Physical examination

Upon physical examination, the doe had 5% dehydration, and was dull and depressed with a body condition score of 2. There was tachycardia, tachypneic and dyspnea. The rectal temperature was within the normal range, mucous membrane was pale and no urination or defecation was observed. The doe was unable to stand and remained on lateral recumbency.

Diagnostic plan and Results

Transabdominal ultrasonography was performed to determine viability of fetus and stage of pregnancy, and revealed two viable fetuses at full term pregnancy. Blood was collected in EDTA and plain tubes for complete blood count and biochemical analyses. Hemogram showed lymphopenia, monocytosis and eosinopenia, while biochemistry revealed hypocalcemia and severe hypoglycemia (decrease about 30 folds). Aspartate aminotransferase (AST) and creatine kinase (CK) levels were markedly elevated by 16 and 36 folds, respectively. There was a slight hypoglobulinemia and hypoproteinemia.

Management plan

The doe was stabilized with intravenous infusion of 200 mL of Dextrose (G-20) as glucose replacement followed by rehydration infusion of 1 L of NaCl solution. Flunixin meglumine (at 2.2 mg/kg bwt) was also given intravenously to provide analgesic effect, and 4 mL of Vitavet (at 1 mL/10 kg bwt) was administered intravenously as vitamin supplements. The above treatments were given once because the clinician decided to sacrifice and perform cesarean section at the same time due to grave prognosis as the

doe started showing neurological signs. The clinician managed to save 2 full term kids but unfortunately both of them died 10 min after the procedure.

Gross pathological findings

Post-mortem examination showed a cachectic and dehydrated carcass with minimal subcutaneous and visceral fat. The liver was pale yellow, slightly swollen and friable. The lungs were severely congested with an evidence of frothy exudation from the cut surface of the trachea (**Figure 1 a, b, c, d**). Cut sections of the liver, lung and kidney were collected in 10% buffered formalin, processed, sectioned and stained with H&E for histopathological examination.



Figure 1: Gross photographs showing (a) Cachectic carcass with low fat cover (b) Pale, friable and enlarged liver (c) Congested lung (d) Froth in tracheal lumen.

Histopathological findings

Section of the liver showed moderate lipidosis with fat globules distending the hepatocyte nuclei to the periphery. There was congestion in the lung with mild edema of the interstitial spaces. Hyperplasia of mucosa associated lymphoid tissue was observed close to the bronchiole. Mild interstitial lymphocytic infiltration resulting in slight interstitial thickening was also observed (**Figure 2a**, **b**).

DISCUSSION

Pregnancy toxemia is a metabolic disorder with high mortality rate and occurs in twin-bearing ewes (does) in late gestation (Schlumbohm and Harmeyer, 2008). This case report was in agreement with Schlumbohm et al. (2008), where the doe had 2 fetuses. Environmental stress or chronic illness that result in weight loss, depressed appetite, and a negative energy balance; all these lead to alterations in insulin-glucagon ratio (Edmondson and Pugh, 2009). The environmental stress in this case includes the competition for feed that creates stressful condition to the pregnant doe.



Figure 2: Photomicrograph of the (a) liver showing fatty change, $H\&E \times 200$ (lipidosis) in hepatocytes (b) lungs showing lymphoid hyperplasia, mild interstitial congestion and infiltration with edema, $H\&E \times 100$.

We observed that there was a marked decrease in glucose level (severe hypoglycemia) in the dam. Maternal hypoglycemia is a characteristic symptom of pregnancy toxemia and has been attributed to an increase in glucose uptake by the twin-bearing uterus (Schlumbohm and Harmeyer, 2008). However, according to Smith and Sherman (2009), the blood glucose levels are variable; severe hypoglycemia or terminal marked hyperglycemia both are possible. Thus, measurement of blood glucose concentration can support the diagnosis (Mavrogianni and Brozos, 2008) of this condition. Cortisol-induced changes in the hemogram (neutrophilia, lymphopenia, eosinopenia) and evidence of dehydration (elevated hematocrit and total protein) can be expected. The cortisol-induced changes in the hemogram is significant in this case, however, there is no elevation of total protein. AST and CK showed marked elevation by 16 and 36 folds, respectively. This might be attributed to the muscle inactivity and a possible hepatic involvement. There slight hypoproteinemia was due а to hyperglobulinemia, which might be due to immune suppression.

At post-mortem, the carcass appeared dehydrated with poor subcutaneous tissue cover. The liver showed signs of fatty change. According to Ermilio and Smith (2011), the doe's liver could be enlarged and become yellow in pregnancy toxemia cases due to infiltration of fat into the hepatocytes resulting in decreased function. Here, we observed large fat globules within the hepatocytes, pushing the nucleus of the cell to the periphery. Management of this condition may involve correction of energy, electrolyte, and acid-base imbalances, as well as stimulating appetite and treating dehydration (Edmondson and Pugh, 2009). We instituted 20% Dextrose intravenously, followed by administration of NaCl solution in order to correct the dehydration. Propylene glycol can be administered (15-30 mL every 12 h) as a glucose precursor. In later stages of the condition, when the animal is recumbent, treatment must be aggressive in order to improve the prognosis of the case. Thus prompt treatments must be initiated immediately, and removal of the fetuses is crucial for the survival of the dam (Navarre and Pugh, 2002). In animals with signs of terminal stage of the condition (neurological signs, blindness, recumbency), treatment often leads to transient improvement of the general condition of the animal, which could subsequently deteriorate, with eventual death of the animal. In such cases, for welfare reasons euthanasia of affected animals would be recommended, even before instigation of treatment (Brozos et al., 2011).

CONCLUSION

Pregnancy toxemia is a metabolic disorder causing high mortality and economic loss to the farmers. The single most important factor for preventing pregnancy toxemia can be suppling of proper nutrition to the dam. In the present case, it should be viewed more as flock problem, as there has been a history of pregnancy toxemia in the farm.

ACKNOWLEDGEMENT

The authors wish to acknowledge En Nazim Razali Kanini, En Yap Keng Chee, En Ghazali Md Yusoff and En Apparau Somanaidu of University Veterinary Hospital (UVH), and Faculty of Veterinary Medicine Universiti Putra Malaysia for their technical support.

REFERENCES

- Brozos C, Mavrogianni VS, Fthenakis GC (2011). Treatment and Control of Peri-Parturient Metabolic Diseases: Pregnancy Toxemia, Hypocalcemia, Hypomagnesemia. Veterinary Clinics of North America: Food Animal Practice, 27: 106-107.
- Edmondson MA, Pugh DG (2009). Pregnancy Toxemia in Sheep and Goats. In Food Animal Practice 5th Edn.; pp 144-145.
- Ermilio EM, Smith MC (2011).Treatment of Emergency Conditions in Sheep and Goats. Veterinary Clinics of North America-food Animal Practice, 27: 105-106.

- Fthenakis GC, Arsenos G, Brozos C, Frangkou IA, Giadinis ND, Giannenas I, Mavrogianni VS (2012). Health Management of Ewes during Pregnancy. Animal Reproduction Sciences, 130: 200.
- Mavrogianni VS, Brozos C (2008). Reflections on the causes and the diagnosis of peri-parturient losses of ewes. Small Ruminant Research, 76: 77-78.
- Menzies PI (2011). Pregnancy Toxemia in Ewes: Hepatic Lipidosis: Merck Veterinary Manual. Merial: USA.
- Navarre CB, Pugh DG (2002). Diseases of the gastrointestinal system: Sheep and goat medicine, Saunders: Philadelphia.
- Schlumbohm C, Harmeyer J (2008). Twin-pregnancy increases susceptibility of ewes to hypoglycaemic stress and pregnancy toxaemia. Research in Veterinary Science, 84: 286.
- Smith MC, Sherman DM (2009). Nutrition and Metabolic Diseases. In: Goat Medicine, 2nd Edn.; pp 761. http://onlinelibrary.wiley.com (Accessed on November 01, 2014)



Under the terms of Creative Commons Attribution 3.0 Unported License