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

Yusuf Abba1, Faez Firdaus Jesse Abdullah2*, 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

ARTICLE HISTORY

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.

**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.

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 supplying 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**


