ABSTRACTS

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Cortisol profile and clinical evaluation of canine neonates exposed antenatally to maternal corticosteroid treatment

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OBJECTIVES AND METHODS: Prenatal administration of corticosteroids is related to fetal maturation in many animal species, as well as in premature infants. However, little has been done to recognize its effect in canine perinatology. Therefore, the objective of the present research was to investigate whether maternal corticosteroid treatment can improve neonatal vitality and alter maternal and neonatal cortisol profile. We allocated 6 bitches and their neonates (delivered by cesarean section) into two groups: control group (CONT) – maternal administration of saline solution (0.9% NaCl) at 55 days of gestation (n = 3) and betamethasone group (BETA) – administration of a single dose of 0.5 mg/kg maternal body weight of betamethasone (Celestone®) at 55 days of gestation (n = 3). Maternal serum cortisol was measured before injection, 1 hour after treatment and daily until parturition. Two neonates per female were examined by Apgar score (1) and body temperature at birth and 5, 60, 120 and 240 minutes postpartum. Blood was collected for serum cortisol concentration from puppies at birth and 120 minutes postpartum. Data were analyzed by ANOVA and LSD at p ≤ 0.05. This study was approved by the Bioethics Committee of the College of Veterinary Medicine - USP.

RESULTS: Neonates from CONT group were born at 63 days of gestation, while BETA neonates had to be urgently delivered at 58 days (as betamethasone treatment induced precocious whelping 72 hours after administration). All neonates presented unsatisfactory (<7) Apgar score at birth without statistical difference between groups. However, newborns from BETA group showed lower Apgar score (9±0.3) when compared to CONT group (10±0) at 240 minutes postpartum. CONT neonates reached ideal Apgar score earlier (60 minutes) than BETA subjects (120 minutes postpartum). Hypothermia was verified at 5 minutes postpartum in both groups. A gradual increase in body temperature occurred amongst periods of observation, without a statistical difference between groups. Statistical difference was found in cortisol levels only at birth (CONT: 3.85 ± 0.56 µg/dL and BETA: 0.17 ± 0.06 µg/dL), result that may be associated with the influence of the corticosteroid treatment on the hypothalamic-pituitary-adrenal axis (HPA). No difference was noticed among periods of analysis within the same group. Maternal cortisol levels differed only 24 hours after betamethasone or saline injection (CONT: 14.1 ± 2.6 µg/dL and BETA: 1.1 ± 0 µg/dL). Moreover, BETA group exhibited a gradual decrease in cortisol levels from treatment until parturition (pre-treatment: 10.3 ± 1.7µg/dL; post-treatment: 5.4 ± 1.3 µg/dL and delivery day: 1.1 ± 0 µg/dL). Similarly to BETA neonates, this decline in maternal cortisol may be attributed to the suppressive effects of betamethasone on HPA axis. No correlation (r=0.48; p=0.16) was verified between maternal cortisol at the delivery day and neonatal cortisol levels at birth.

CONCLUSION: Maternal administration of betamethasone at 0.5 mg/kg induced premature labor without causing clinical negative effects in canine preterm neonates. Corticosteroid treatment led to labor onset and fetal maturation, especially of vital organs. In addition, maternal cortisol concentration had no effect on fetal profile and vice-versa, whereas betamethasone showed a marked influence on neonatal cortisol levels during the first hour of life.


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