Clinical Communication

Pregnancy-related diabetes mellitus in two dogs

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Abstract

CASE SUMMARIES: Two cases of diabetes mellitus occurring in bitches in association with pregnancy are reported. In the first case, a bitch with suspected acromegaly developed diabetes mellitus within 2 weeks of the due date. Despite insulin therapy, euglycaemia was not achieved. Two live, small pups were delivered by elective Caesarean section but died within 2 days. Signs consistent with acromegaly resolved but diabetes mellitus was permanent in the bitch. In the second case, diabetic ketoacidosis was diagnosed 2 days after Caesarean section was performed due to dystocia. The pups delivered died within 5 days. The bitch recovered fully from diabetes mellitus within 2 weeks and has remained euglycaemic without insulin for a period of at least 18 months.

CLINICAL RELEVANCE: These two cases demonstrate that diabetes mellitus can occur in association with pregnancy in dogs, that diabetic ketoacidosis can occur during transient diabetes mellitus in dogs, and suggest that acromegaly may occur during pregnancy-related dioestrus in dogs. The scarcity of previous reports of this nature, however, suggests that such cases are unusual.

Lack of prompt resolution of hyperglycaemia may result in secondary diabetes mellitus becoming permanent. Management should focus on immediate insulin therapy or ovariohysterectomy to minimise this risk. Even mild hyperglycaemia should not be ignored during pregnancy. The insulin antagonistic effects of pregnancy, stressful illness, surgery and dystocia can be enough to result in diabetic ketoacidosis in the absence of permanent insulin deficiency. Maternal hyperglycaemia may contribute to adverse fetal outcomes in dogs but further study is required regarding the nature of the risk.

KEYWORDS: Diabetes mellitus, pregnancy, acromegaly, ketoosis, dog, gestational diabetes mellitus

Introduction

Diabetes mellitus can arise as a consequence of insulin resistance associated with other disease states, or drug administration. This association is recognised in dogs with hyperadrenocorticism (Ling et al 1977), in dioestrus (Eigenmann 1981), with acromegaly (Eigenmann et al 1983; van Keulen et al 1996) and glucagonoma (Gross et al 1990), or following administration of drugs such as glucocorticoids (Campbell and Latimer 1984) and progestagens (Eigenmann et al 1983). The hormonal changes during late pregnancy in dogs also cause profound insulin resistance, and uptake of glucose by peripheral tissues in response to insulin was reduced by 30% of values during anoestrus (Connolly et al 2004). Yet, although its occurrence is mentioned in authoritative texts (Feldman and Nelson 2004a), reports of diabetes mellitus during pregnancy in dogs are scarce (Foster 1975; Johnston et al 2001b).

Transient diabetes mellitus is very uncommon in dogs and when it occurs it is usually associated with insulin antagonism from disease or drug therapy (Feldman and Nelson 2004a). Transient diabetes mellitus associated with ketoacidosis was reported in a dog with stressful concurrent illness, and treated with glucocorticoids (Edwards 1982). Dioestrous bitches with diabetic ketoacidosis may also sometimes have resolution of insulin-requiring diabetes mellitus following successful therapy and ovariohysterectomy (Feldman and Nelson 2004b).

With the rare exception of functional pituitary tumours, excessive production of growth hormone in canine acromegaly is derived from the mammary gland, where it is induced by either progesterone or synthetic progestagens (Selman et al 1994b; Mol et al 1995). As a consequence, signs of acromegaly are noted in a consistent relationship with dioestrus or administration of progestagens, although they may wax and wane through several heat or treatment cycles before diagnosis (Eigenmann et al 1983). Typically, signs begin 3–5 weeks after oestrus in non-pregnant bitches (Eigenmann et al 1983), and include respiratory stridor, increased soft tissue mass, formation of skin folds, fatigue, enlargement of inter-dental spaces, polyuria and polydipsia, and hyperglycaemia (Eigenmann and Venker-van Haagen 1981). Diabetes mellitus occurs commonly in acromegaly because growth hormone causes profound glucose intolerance and insulin resistance (Eigenmann et al 1983; Selman et al 1994a). The fact that concentrations of progesterone during pregnancy in the bitch are similar to those during non-pregnant dioestrus (Concannon et al 1975) suggests that acromegaly should be equally likely in pregnant bitches, yet reports of its occurrence in pregnancy could not be found.

Two cases of diabetes mellitus in bitches occurring in association with pregnancy with contrasting presentations and maternal outcomes are reported here.

Case 1

A 6-year-old pregnant Labrador Retriever was presented for investigation of facial swelling 14 days before her expected due date (EDD). The bitch had delivered nine viable pups 3 years previously, but despite regular cycling since then had failed to con-
A 6-year-old female Siberian Husky that had delivered five viable pups 1 year previously was presented with a primary complaint of dystocia. Following the birth of a single, live pup no further abdominal contractions were observed. The bitch was pale and mildly tachycardic (heart rate 150 beats per minute). There was no response to two injections of 10 IU oxytocin (Ethical Agents, South Auckland, NZ) administered intramuscularly 1 h apart. A Caesarean section was performed and a further three live pups were delivered 5 h after the birth of the first pup. Due to continued haemorrhage at the sites of placental attachment and because there were no future plans to breed the bitch, an ovariohysterectomy was performed at the same time. Four of the pups weighed approximately 450 g, similar to the five pups of the bitch’s previous litter which ranged from 450 to 538 (mean 491) g, and one pup was small, at 257 g. All pups appeared strong and were vocalising at birth. The bitch recovered from anaesthesia well and displayed an interest in the pups, and was sent home in the care of the owner.

Two days later, the bitch was presented severely ill with diabetic ketoacidosis. The dog had lost weight since surgery and was weak and unable to stand, and there was vomiting, melena, tachypnoea, tachycardia, pyrexia and severe dehydration. Clinical pathology tests were performed in-house (Vet Test and QBC Vet Autoreader; IDEXX, Neuchatel, Switzerland). There was severe hyperglycaemia (glucose 36.75, reference range 4.28–6.94 mmol/L), a high urea (29.2, reference range 2.5–9.64 mmol/L), and a creatinine of 160 (reference range 44–159) µmol/L. There were mild elevations in hepatic enzyme activities (alanine aminotransferase 242, reference range 5–51 U/L; alkaline phosphatase 179, reference range 14–134 U/L), and a normal concentration of bilirubin (<2, reference range 0–15 µmol/L). The concentration of cholesterol was elevated at 10.72 (reference range 2.84–8.27) mmol/L. The total concentration of calcium was 2.06 (reference range 1.98–3.00) mmol/L, and concentrations of phosphorus was 4.13 (reference range 0.81–2.19) mmol/L. Amylase activity was normal at 504 (reference range 500–1,500) U/L. The concentration of total protein was 54 (reference range 52–82) g/L, and that of albumin was 26 (reference range 22–39) g/L. A severe leucocytosis (total white cells 39.1, reference range 6.0–16.9 x 10⁹/L) was present. Blood glucose concentrations were measured during the terminal management of both pups, but in neither case was a value ascertainment before dextrose had been administered. The blood glucose concentration was 3.5 mmol/L in one pup, and ranged between 2.2 and 4.2 mmol/L in the other pup.

Post-operatively, pre-insulin blood glucose values were measured twice daily for 3 weeks, and 12-h glucose curves were performed on Days 2, 4, 7, 14, 21, 28 and 35. During this period, doses of insulin were increased by 0.1–0.2 IU/kg in response to persistent hyperglycaemia during a glucose curve, and decreased whenever a blood glucose of <5.5 mmol/L was demonstrated (on three occasions). Doses of insulin ranged between 0.3 and 0.7 IU/kg during this period. Despite these adjustments, insulin was unable to be withdrawn, and 18 months later the bitch remains well-controlled on a dose of 0.4 IU/kg.

By 2 months post-operatively, the facial swelling, skin thickening, excess folds and respiratory stridor had resolved.

**Case 2**

A 6-year-old female Siberian Husky that had delivered five viable pups 1 year previously was presented with a primary complaint of dystocia. Following the birth of a single, live pup no further abdominal contractions were observed. The bitch was pale and mildly tachycardic (heart rate 150 beats per minute). There was no response to two injections of 10 IU oxytocin (Ethical Agents, South Auckland, NZ) administered intramuscularly 1 h apart. A Caesarean section was performed and a further three live pups were delivered 5 h after the birth of the first pup. Due to continued haemorrhage at the sites of placental attachment and because there were no future plans to breed the bitch, an ovariohysterectomy was performed at the same time. Four of the pups weighed approximately 450 g, similar to the five pups of the bitch’s previous litter which ranged from 450 to 538 (mean 491) g, and one pup was small, at 257 g. All pups appeared strong and were vocalising at birth. The bitch recovered from anaesthesia well and displayed an interest in the pups, and was sent home in the care of the owner.

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represented a modification of that recommended by Feldman and Nelson (2004b).

Additional therapy comprised broad-spectrum antibiotics, anti-emetics and gastrointestinal protectants for symptomatic control of gastrointestinal signs. After 36 h of intensive therapy, intermediate-acting porcine insulin (Caninsulin; Intervet) was commenced at an initial dose of 0.5 IU/kg. The insulin requirement of the bitch gradually declined and insulin was able to be discontinued 7 days later. Intravenous fluids were continued for 7 days, at which point the bitch was well enough to be discharged. Haematological and biochemical screening performed at a commercial laboratory 11 days after admission with ketosis showed only mild abnormalities and resolution of leucocytosis, resolution of elevated concentrations of urea, improvements in the activities of liver enzymes, a mild non-regenerative anaemia (haematocrit 32.0, reference range 37.0–55%) and thrombocytosis (928, reference range 200–500 x 10^9/L). Intermittent vomiting and inappetence gradually resolved over 3 weeks. The bitch remains euglycaemic 18 months later and had gained weight, and there had been no glucosuria or other clinical signs of illness.

All four puppies were managed at home by an experienced breeder but died within 5 days of birth.

Discussion

Although not confirmed by assessment of growth hormone concentrations, Case 1 presented with signs that could be consistent with acromegaly, of increased skin thickness and prominent skin folds around the face, neck, and shoulder area, respiratory stridor, and development of diabetes mellitus (Eigenmann and Venker-van Haagen 1981). Because mammary production of growth hormone is known to be stimulated by progesterone, and progesterone concentrations are similar during pregnancy and non-pregnant dioestrus in dogs (Concannon et al 1975), it is surprising that acromegaly occurring during pregnancy is not more frequently reported. Growth hormone concentrations were elevated in only 1/45 pregnant dogs tested (Eigenmann et al 1983), however the ages of those pregnant bitches were not reported. Bitches are generally used for breeding at a younger age than the mean age at which those authors reported acromegaly was seen (mean 8.5 ± standard error 0.5 years), and therefore acromegaly might be less likely to be seen during pregnancy. The occurrence of considerable inter-dog variation in the magnitude of increase in growth hormone (Eigenmann and Venker-van Haagen 1981; Selman et al 1994a) may also mean that certain individuals are more predisposed to developing acromegaly than others.

In Case 2, diabetes mellitus was not diagnosed until after delivery, but the history of polydipsia suggests its presence for some time during late pregnancy. Most dogs with diabetes mellitus have had consistent signs for a period of at least 2 weeks (Ling et al 1977). Just as with acromegaly, diabetes mellitus might be expected to occur with equal frequency during pregnant and non-pregnant dioestrus in the dog since both progesterone and growth hormone cause insulin resistance (Eigenmann and Rijnberk 1981). However, the scarcity of published cases of diabetes occurring during pregnancy suggests its occurrence is uncommon. Indeed, peripheral insulin resistance during pregnancy may be adaptive, giving priority for glucose to fetal and placental tissues during a period in which the demand for glucose is more than doubled (Connolly et al 2000), and reducing the likelihood of hypoglycaemia which may develop during fasting, or as a result of the insulin response during a low-carbohydrate meal (Kooistra and Okkens 2002). In the non-pregnant dioestrus dog, despite lower insulin resistance (McCann et al 1988, non-peer-reviewed) this fetal drain of glucose is not present, perhaps predisposing non-pregnant dogs to develop diabetes more frequently than pregnant ones.

The presence of ketonuria at the same time as severe hyperglycaemia in Case 2 supports a diagnosis of diabetic ketosis rather than pregnancy ketosis, in which ketonuria without glucosuria is found, and blood glucose concentrations are low (Irvine 1964; Jackson et al 1980). Although the clinical presentation of Case 2 was more typical of diabetic ketoacidosis than diabetic ketosis (Ling et al 1977; Parsons et al 2002), acidosis was not confirmed and the clinical presentation could also be consistent with concurrent diabetic ketosis and severe acute primary gastrointestinal or pancreatic disease. Signs of severe anaemia, pyrexia and leucocytosis suggested such complicating disease. The anaemia and disproportionate elevation of urea compared to creatinine were thought to be the result of gastrointestinal bleeding. The haematocrit of healthy pregnant bitches at term can range from as low as 26 (mean 30.6)% (Concannon et al 1977), and the amount of blood lost in the gastrointestinal tract may not have been as substantial as the haematocrit of 15.3% may otherwise have indicated. Surgical losses during ovariohysterectomy are likely also to have contributed to the low haematocrit at this time. Although concentrations of amylase were not elevated, the possibility of pancreatitis remains, since amylase is frequently not elevated in pancreatitis (Strombeck et al 1981). Whatever the cause, such a severe concurrent illness could contribute to insulin resistance and ketogenesis by increasing secretion of counter-regulatory hormones such as catecholamines and cortisol, already likely to have been promoted by the stress of dystocia and surgery. Pancreatitis could additionally cause transient or permanent damage to islets during the pathological process, reducing their capacity to secrete insulin. These factors would have added to ketogenic factors normally present in late pregnancy such as high endogenous concentrations of progesterone and potentially also high concentrations of growth hormone. Antagonism of the actions of insulin, promotion of fatty acid mobilisation (Diehl and Wheeler 1992) and stimulation of the release of glucagon (Gerich et al 1973) by such counter-regulatory hormones facilitates ketogenesis. When not balanced by tissue uptake and utilisation of ketones, as is normally enabled by insulin (Alberti 2001), severe ketogenesis results.

Persistence of insulin-requiring diabetes mellitus after resolution of secondary diabeticogenic factors in Case 1 suggests that, whether or not underlying pancreatic pathology pre-existed, there had been further irreversible destruction of islets. The metabolic state of diabetes mellitus can contribute to destruction of beta-cells through the effects of glucose and lipid toxicity (Imamura et al 1988; Lee et al 1994). These effects are dependent on the duration and severity of hyperglycaemia. Decreasing concentrations of blood glucose to below 14 mmol/L will therefore minimise the risks of progression of destruction of pancreatic islets; however, this is difficult to achieve at the best of times in dogs let alone in the face of the severe insulin resistance of pregnancy. Very high doses of insulin are likely to be required and intensive monitoring of blood glucose concentrations needed. Immediate
termination of the pregnancy by ovariohysterectomy may be a more pragmatic approach to achieving good glycaemic control in the bitch. Regardless, given the risks of future dioestrous-related diabetes mellitus, ovariohysterectomy should be performed before the next oestrus.

The possibility that the predisposition for diabetes is inherited must also be considered. Although the bitch in Case 2 remained euglycaemic without insulin therapy some 18 months after the episode of ketosis, diabetes mellitus may redevelop, especially if other factors causing insulin resistance are not well controlled.

A high risk of adverse fetal events, including abortion, small unthrifty pups and overly large pups (macrosomia), has been described in diabetic bitches (Feldman and Nelson 2004c). Vascular effects of diabetes may reduce placental blood supply, contributing to abortion or poorly-grown pups, and macrosomia may result from the anabolic effects of fetal hyperinsulinaemia, a response to excessive delivery of glucose, amino acids and fatty acids to the fetus from the maternal circulation. In a study involving bitches with experimentally-induced diabetes mellitus, higher numbers of stillborn pups, with no gross abnormalities, were born to six diabetic bitches than to six control bitches (Kliegman et al 1983). The birthweights of the pups of diabetic dogs were slightly greater than those of controls when controlled for the size of the litter. A tendency to high birthweight of pups was also reported in a colony of diabetic Golden Retrievers (Johnson et al 1987). Excessive fetal growth is the most frequent complication of gestational diabetes mellitus in humans (Metzger et al 2002). In the two cases presented here high birthweight was not present even though poor maternal glycaemic control was demonstrated in Case 1 and suspected in Case 2. The risks and types of adverse fetal outcomes may depend on the degree and duration of hyperglycaemia in the dam, and whether the onset of diabetes mellitus predates organogenesis and placentation.

The pups of diabetic bitches may also be predisposed to hypoglycaemia after birth, when cessation of maternal delivery of fuels is not immediately matched by a reduction in insulin secretion by the fetus (Feldman and Nelson 2004c). However, limited experimental evidence suggests that a predisposition to hypoglycaemia is not likely in the pups of diabetic bitches in the absence of other predisposing factors. Although elevated concentrations of insulin were present in pups of diabetic dogs at birth, hypoglycaemia did not occur even when such pups were fasted (Kliegman et al 1983); hepatic glycogen stores were elevated, and glycogenolysis occurred at increased rates immediately after birth (Kliegman and Miettinen 1983), suggesting a degree of hepatic insensitivity to the effects of hyperinsulinism. Although gluconeogenesis is initially attenuated, normal rates are attained by 24 h of age (Kliegman et al 1983). A blood glucose concentration as low as 2.2 mmol/L was measured in one pup from Case 1, despite glucose supplementation, a value below the reported range of 2.8–7.0 mmol/L for canine neonates of between 1 and 3 days of age (Hoskins 2001). Several other factors, however, may have precipitated hypoglycaemia in that pup from Case 1, including hypothermia, hypoxia, or rejection by the bitch; small or underweight pups, as this was, may be especially susceptible (Atkins 1984).

In the cases presented here, the loss of 100% of the pups in both litters is unusual (Blunden 1986) and suggests that the metabolic state of the dams may have been a contributing factor. This cannot be assumed, however, since several other risk factors were also present, including low birthweight (Lawler and Evans 1995), dystocia (Gaudet 1985), emergency Caesarean section (Moon et al 2000), and maternal illness. It is not possible to identify the factors involved in the neonatal deaths in these cases because of a lack of detailed investigation.

These two cases demonstrate that diabetes mellitus can occur in association with pregnancy in dogs and that diabetic ketoacidosis can occur during transient diabetes mellitus in dogs. The clinical findings in Case 1 also suggest that acromegaly can occur during pregnancy-related dioestrus but it may be uncommon during pregnancy because of the difference between the usual age of onset of this condition and the age that dogs are usually bred. Diabetes mellitus may be less common during pregnancy than non-pregnant dioestrus because of the net glucose drain by the fetus, which balances the profound insulin resistance of pregnancy. Transient diabetes is uncommon in dogs, but the insulin-antagonistic effects of stressful illness, surgery, dystocia and pregnancy were sufficient to result in diabetic ketosis in the absence of absolute insulin deficiency in Case 2.

Diabetes mellitus should be included as a differential diagnosis in all pregnant dogs with polyuria and polydipsia. Even mild hyperglycaemia should not be ignored during pregnancy as, despite their insulin-resistant state, normal bitches maintain euglycaemia during pregnancy (Connolly et al 2000). Persistence of diabetes after resolution of insulin antagonism in Case 1 suggests destruction of pancreatic islets, which could have been the result of poor metabolic control. Either control of blood glucose concentration to below 14 mmol/L or immediate termination of the pregnancy via ovariohysterectomy may have prevented this. Maternal hyperglycaemia may contribute to adverse fetal outcomes in dogs but further study is required to determine the nature of the risk.

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