

Fetal anasarca in an Abyssinian kitten

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Abstract

Case description

A four-year old Abyssinian queen was presented to a reproductive specialist for dystocia.

Clinical findings

Physical examination and history of the queen was consistent with Stage II labor that has not progressed normally. Transabdominal ultrasound revealed an abnormal fetus with visible fluid accumulation in the thorax, abdomen and subcutaneous space, with no detectable heartbeat. Heart rates of the other two visible fetuses were measured and were found to be low or not detectable. Hematology and biochemistry analysis of the queen revealed mild decrease in the mean corpuscular hemoglobin concentration, mild monocytosis and mild elevation of creatinine kinase.

Treatment and outcome

Cesarean section was performed soon after admission. Three kittens were born. The first kitten had no heartbeat and appeared grossly abnormal; significantly larger than the other two kittens, with marked accumulation of fluid subcutaneously, ascites and pleural effusion. Two remaining kittens required intensive neonatal resuscitation but survived and were discharged on the same day with the queen.

Clinical relevance

Fetal anasarca or hydrops fetalis has been reported in humans, dogs and ruminants but there have been no reported cases in the domestic cat.

Keywords: Anasarca, queen, cesarean, fetus, hydrops fetalis, feline

Introduction

Fetal anasarca, more commonly referred to as hydrops fetalis in human medicine, is defined as the accumulation of abnormal quantities of subcutaneous fluid in the fetus, with or without visceral cavity fluid.¹ Fetal anasarca has been described in sheep,²⁻⁴ cattle,⁵ buffalo,⁶ goats,^{7,8} and humans⁹ with limited reports in canines.^{1,10,11} In canines, a predisposition of brachycephalic breeds, including pugs, French and English bulldogs, has been established.¹ One or several pups in a litter may be affected, with the remainder born clinically normal. The pathophysiology of anasarca in non-human species is broad and poorly defined. A number of processes, including cardiac malformations, infectious processes, trauma, genetic abnormalities, abnormal lymph node or kidney development, teratogens, immune mediated causes, placental and umbilical disorders have been suggested in the etiology. The functional disturbance associated with these processes can create an imbalance between plasma oncotic and hydrostatic pressure and/or lymph flow, resulting in interstitial fluid accumulation.¹² In all species in which the abnormality is described the prognosis for the fetus is grave and dystocia is common, which has associated risk for the dam. While anecdotally fetal anasarca is understood to occur in felines, to date there are no case reports in the literature. The aim of this case report is to provide a detailed case description of fetal anasarca in an Abyssinian kitten to establish a database and contribute to the understanding of the pathophysiology of the condition in cats.

Case report

History and physical examination

A four-year-old Abyssinian queen was presented for dystocia. Stage II labor commenced four and one half hours prior to presentation, with production of placental fluid but with no evidence of any kittens. The queen had a body condition score (BCS) of four out of nine. She had a previous

pregnancy two years prior, and this litter had been born clinically normal and had not required veterinary intervention.

Physical examination of the queen was consistent with Stage II labor with slight tachypnea (36 breaths/min) and tachycardia (160 beats/min) noted, but was otherwise unremarkable. Reproductive examination revealed a swollen vulva and a soft and dilated vagina with sanguinous discharge. No kitten was palpable on digital vaginal examination. Transabdominal ultrasound revealed three kittens: two visible on the left side of the abdomen, and one on the right. The caudal-most kitten on the left side had no observable heartbeat and fluid accumulation was visible in the thorax, abdomen and subcutaneous space. The cranial kitten on the left had a visible heart beat with a heart rate of 150 beats/min. The kitten on the right had no sonographically visible heartbeat.

Procedure

A cesarean section was performed under general anesthesia promptly after admission. General anesthesia was induced with alfaxalone IV to effect to facilitate intubation, followed by maintenance with isoflurane and 100% oxygen. Midline laparotomy was performed to expose the uterus and an incision was made at the base of the left uterine horn. The first kitten removed was large, edematous and malformed in appearance and lacked an audible heartbeat. The two remaining live kittens were significantly bradycardic with severe respiratory depression and required intensive neonatal resuscitation with supplemental oxygen. The uterus was closed using a continuous Utrecht suture pattern, followed by a three-layer closure of the abdomen. After fetal removal, the queen was given oxytocin (2 IU IV during surgery and 2 IU SC in recovery), and meloxicam (0.1 mg/kg SC). Recovery from anesthesia was uneventful.

Diagnostic testing

All three kittens born were female. The two live kittens weighed 84.9 g and 89.9 g, respectively, and both were anatomically normal.

Necropsy examination of the deceased kitten was performed just after surgery in the hospital. The deceased kitten (Figure 1 and 2) weighed 102.9g after the abdominal fluid was inadvertently removed prior to weighing (estimated to be approximately 107 g with fluid). The following gross abnormalities were noted: the skin and subcutis had generalized edema with a “jelly-like” consistency; the peritoneal cavity contained a large volume of serosanguinous fluid; and the liver was bright red in color with marked rounding of the lobes (Figure 2). The kidneys and gastrointestinal tract appeared grossly normal. The pleural cavity contained serosanguinous fluid, but the heart and lungs were grossly normal, with no evidence of pericardial effusion. The brain did not have a formed structure, and the brain tissue was poorly formed and spontaneously exteriorized following craniotomy. The placental tissue appeared grossly normal.

Blood was taken from the queen for analysis. In-house blood glucose was 9.0 mmol/L on handheld glucometer, and blood ketones were 0.0 mmol/L. A Feline Total Annual Health Profile was sent for analysis to IDEXX Laboratories. Notable abnormalities on hematology were a very mildly decreased mean corpuscular hemoglobin concentration (MCHC) of 280g/L (reference range 282-333g/L) and a mild monocytosis of $1.0 \times 10^9/L$ (reference range $0.0-0.6 \times 10^9/L$). The only abnormality on biochemistry was a moderately elevated creatinine kinase (CK) of 725 U/L (reference range 64-400 U/L).

Formalin fixed samples of placenta, heart, and lungs were submitted for gross examination and histopathology. Histopathology results showed that the placenta had multifocal necrosis with infiltration by lymphocytes, plasma cells, macrophages, and neutrophils. The pulmonary airways and alveoli contained lymphocytes, macrophages and neutrophils, with an interstitial leukocytic infiltration. The myocardium had interstitial edema with leukocytic, predominantly lymphocytic, infiltration. There was concurrent mediastinal edema and lymphocytic infiltration. These results were consistent with placentitis, with fetal bronchopneumonia, myocarditis, and mediastinitis. These findings indicate an infectious process, possibly a low-grade bacterial infection.

Outcome

The remaining clinically normal kittens were assisted to nurse on the queen and were discharged the same day of presentation. At 14 days after surgery the kittens and queen were doing well.

Discussion

Hydrops fetalis is a result of deranged fluid homeostasis. In humans, the underlying pathophysiology is broadly divided into two categories: immune mediated and non-immune mediated.¹² Immune mediated hydrops fetalis is understood to occur due to Rhesus gene incompatibility whereby a Rhesus-D (Rh)-D negative mother delivers a (Rh)-D positive fetus and develops autoantibodies against the (Rh)-D positive blood type. Any subsequent pregnancy with a (Rh)-D positive fetus, in the presence of the maternal autoantibodies, results in severe hemolytic anemia in the fetus, as well as hypoproteinemia and multi-cavitary effusion.¹³ Advancements in biomedical science have greatly reduced the incidence of immune mediated fetal anasarca in humans, with non-immune causes of hydrops fetalis accounting for 85-90% of all cases.¹⁴⁻¹⁶ Non-immune hydrops fetalis in humans can be attributed to a variety of factors including teratogens, cardiovascular abnormalities, congenital fetal abnormalities, placental insufficiencies, infection, and miscellaneous causes.¹⁷ In non-immune mediated hydrops fetalis in humans, four mechanisms as per Starlings' forces have been identified. These include an increase in hydrostatic capillary pressure, a reduction of intravascular osmotic pressure, obstruction of lymphatic flow, and damage to peripheral capillary integrity. Over 80 conditions associated with these four mechanisms have been identified to cause hydrops fetalis in humans.¹⁸

The pathophysiology of fetal anasarca in domestic animals remains largely undetermined when compared to human medicine. Proposed causes of anasarca in domestic species include hereditary predisposition due to autosomal recessive genes,¹⁹ abnormal lymph node development,²⁰ minute virus in canines,²¹ congenital renal dysfunction,²² and many others. The mechanisms of fluid accumulation in the subcutis and visceral cavities in fetal anasarca, as described by Starling's principles, is likely comparable to that seen in humans. The present case details placentitis, with fetal bronchopneumonia, myocarditis and mediastinitis consistent with a low-grade bacterial infection. Both infectious processes, and placental insufficiency/disease have been postulated as potential underlying causes for fetal anasarca in domestic species. This may provide one possible explanation for the present case, however may also be an incidental finding in a kitten which may have been deceased in utero for >12h. In human reports, a wide variety of infectious agents have been associated with fetal anasarca.¹² Infections have been shown to preferentially target the fetal bone marrow, the myocardium, and vascular endothelium.¹² Subsequent congestive heart failure, anemia, fetal sepsis, anoxia, endothelial cell damage, and increased capillary permeability result in the fetal anasarca.²³

The most likely route of infection in this case report would have been hematogenous spread or ascending placentitis. This therefore raises the question why not all of the kittens in the litter were affected, when in fact the remaining two kittens were anatomically normal, resuscitated successfully, and discharged the same day with the queen. Furthermore, the queen had been clinically healthy throughout pregnancy.

An important point to note is that the affected kitten was the caudal-most kitten in its respective uterine horn so this may fit the pattern of an ascending placentitis. A limitation of the present study was that external gross pathology and complete post mortem by a registered veterinary pathologist was not performed. It is plausible the fetus with anasarca may have had underlying congenital defects, which may have been the primary cause of the anasarca, or perhaps increased susceptibility to infection and subsequent fluid derangements.

Fetal anasarca, as in the present case, can result in dystocia. In canine breeds defined as having an increased risk for fetal anasarca, namely bulldogs, it has been suggested that preparturient diagnosis (transabdominal ultrasound) and planned cesarean section may help reduce risk for both the bitch and the remaining pups.¹¹ Cats are, in general, at low risk for dystocia, reported at 5.8%.²⁴ This is one possible explanation for the lack of published literature in feline anasarca. It is likely that if the queen did not have an obstructive dystocia she may have not presented to the veterinary hospital.

The clinopathological findings of the queen in the present case report are most likely unremarkable. There was a moderate elevation in creatinine kinase, which may be explained by prolonged obstructive dystocia, or may be insignificant. The mild clinopathological findings in the queen would not account for the fetal anasarca.

Conclusions

Fetal anasarca is well described in human medicine and in several domestic species, notably ruminants and, to a lesser extent, canines. While anecdotally it is thought to occur in felines, there is presently no published literature. To the authors' knowledge this is the first reported case of anasarca in a kitten, suggesting while an uncommon condition in cats, it can occur and may result in obstructive dystocia in the queen.

Learning points

Fetal anasarca is a common cause of dystocia in mammals.

Theoretically fetal anasarca can occur in any mammalian species, however there are no published reports documenting its occurrence in felines.

Fetal anasarca in this Abyssinian kitten is believed to have occurred due to placentitis.

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Figure 1. Fetal anasarca in a stillborn female Abyssinian kitten.

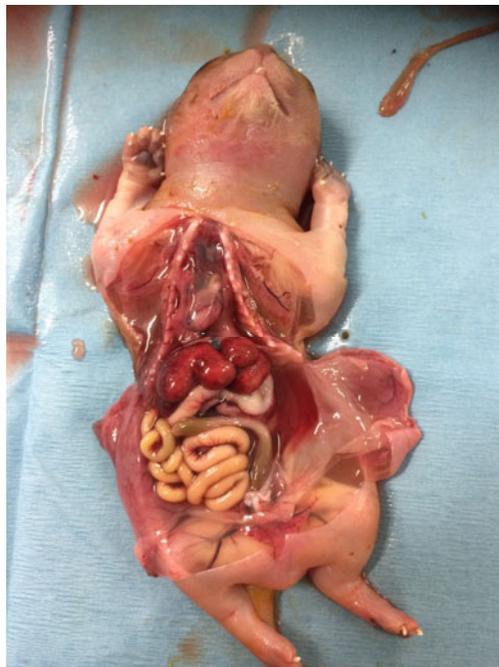


Figure 2. Postmortem examination of a kitten with fetal anasarca showing fluid in pleural, peritoneal and subcutaneous spaces.

(Editor's note: Photographs in this paper are available in color in the online edition of *Clinical Theriogenology*.)

