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Case Report

Pregnancy Loss due to Partial Hydatidiform Mole in a Cat [1]

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Abstract

Molar changes of the placenta are exceptionally rare in animals and in the cat, only one case of partial hydatidiform mole in a stillborn kitten was reported. A 3-year-old female cat was referred for anorexia, vomiting, depression and vaginal discharge. Abdominal distention and pain were noted. Blood count abnormalities were also observed in the cat. A septated, enlarged uterus with anechogenic content was diagnosed on ultrasonographic examination. Ventral midline ovariohysterectomy was performed. An uneventful recovery was observed and total blood count was within normal range on day 3 following ovariohysterectomy. Partial hydatidiform mole diagnosis was made based on the presence of embryos, gross appearance and histopathological findings. This report reflects the clinical presentation and histopathological findings of a partial hydatidiform mole leading to pregnancy loss in the cat.

Keywords: *Feline, Partial hydatidiform mole*

Bir Kedide Kısmi Hidatidiform Mol Nedeniyle Gebelik Kaybı

Özet

Hayvanlarda plasentanın molar değişiklikleri son derece nadir olup kedilerde yalnızca ölü doğan bir yavruda kısmi hidatidiform mol olgusu bildirilmiştir. 3 yaşlı dişi bir kedi iştahsızlık, kusma, durgunluk ve vaginal akıntı şikayetleri ile getirildi. Abdominal gerginlik ve ağrı kaydedildi. Kedide ayrıca kan sayımı anormallikleri gözlendi. Ultrasonografik muayenede anekojenik içeriğe sahip, septumlu, genişlemiş uterus belirlendi. Ventral orta hattan ovaryohisterektomi yapıldı. Hızlı bir iyileşme gözlendi ve ovaryohisterektomiyi takiben 3. günde tam kan sayımı değerleri normale döndü. Embriyoların bulunması, makroskobik görünüm ve histopatolojik bulgulara dayanılarak kısmi hidatidiform mol tanısı konuldu. Bu raporda, bir kedide gebelik kaybı ile sonuçlanan kısmi hidatidiform mol olgusunun klinik görünümü ve histopatolojik bulguları tanımlanmıştır.

Anahtar sözcükler: *Kedi, Kısmi hidatidiform mol*

INTRODUCTION

Hydatidiform mole is a trophoblastic lesion characterized by a hydropic (ie, vacuolar) swelling of the chorionic villi and trophoblastic proliferation. It starts at the time of fertilization due to a defective union of the sperm and ovum, which causes an aberrant proliferation of trophoblastic tissue. Fluid filled and edematous placental villi are observed as grape-like structures. In the human, a molar pregnancy is defined as complete or partial depending on the way of formation and differences in histopathological features and karyotype. A complete mole is usually formed by union of anuclear ovum and a haploid (23X) spermatozoon, which then undergoes duplication [1]. The resultant tissue is entirely paternal in origin, usually with a 46XX karyotype. A few of complete moles arise from fertilization of empty ovum by two spermatozoa and they have a 46XY or 46XX karyotype [2]. Primary histopathological features of complete moles are significant atypia of trophoblastic cells and generalized trophoblastic hyperplasia and hydropic swelling. Fetal tissue is not identified in complete molar pregnancy [3].

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A partial mole is the result of fertilization of ovum by two spermatozoa or by one spermatozoon which reduplicates itself. Consequent genotypes are 69.XXY (triploid) or 92.XXXY (tetraploid), respectively [4]. The trophoblastic cells at the implantation site show mild atypia and only diffuse areas of trophoblastic hyperplasia and swelling are observed. Fetal tissue is identified in partial moles. A fetus that develops in a partial molar pregnancy is nonviable and exhibits congenital anomalies associated with triploidy [3].

Hydatidiform moles are rarely observed in animals and most of the reported cases are in cows [5-7]. In cats, only one partial hydatidiform mole in a stillborn kitten was reported [8]. The aim of this study was to describe the clinical presentation and histopathological findings of partial molar pregnancy in a cat.

CASE HISTORY

A 3.2 kg, 3-year old, living indoor/outdoor female cat was referred with a history of anorexia, vomiting, depression and vaginal discharge for 3 days. On clinical examination, rectal temperature was 38.2°C and small amount of sero-sanguineous vaginal discharge was observed. Abdominal distention and pain were noted on abdominal palpation. Complete blood count (Exigo EOS Vet Analyzer, Sweden) revealed mild lymphocytosis (7.8×10^9/L; reference values: 1.0-7.0×10^9/L), monocytosis (1.1×10^9/L; reference values: 0.2-1.0×10^9/L), increased mean corpuscular hemoglobin concentration (39.3 g/dL; reference values: 31.0-38.5 g/dL) and thrombocytopenia (188×10^9/L; reference values: 200-500×10^9/L). Serum biochemistry (Erba XL 600, Mannheim, India) revealed no abnormalities. A septated, enlarged uterus with anechogenic content was diagnosed by ultrasonography (ProSound II 5 MHz sector; Aloka, Tokyo, Japan). No embryonic/fetal structure was observed *(Fig. 1A)*. Serum hormone concentrations were determined by electrochemi-luminescence immunoassay using an autoanalyser (Cobas6000 C601; Roche Diagnostics, Mannheim, Germany). Peripheral blood levels of estradiol, progesterone, follicle stimulating hormone and testosterone were <5.00 pg/mL, 2.92 ng/mL, <0.10 mIU/mL and 0.03 ng/mL, respectively. Ventral midline ovariohysterectomy was performed under general anesthesia. Anesthesia was induced with propofol (2% propofol, Fresenius Kabi GmbH, Austria) at approximately 6 mg/kg/minute intravenously and maintained with isoflurane (Isoflurane USP® Adeka, Turkey) delivered in 100% oxygen at a flow rate of 500 mL/kg/min through an Ayre›s T-piece. The vaporiser was initially set at 2% isoflurane.

Postoperatively, the cat received meloxicam (0.2 mg /kg sc, *q* 24 h for 2 days; Maxicam, Sanovel, Turkey), metoclopramide hydrochloride (2 mg/kg iv; Metpamid, Sifar, Turkey) and amoxicillin-clavulanic acid (20 mg/kg sc, *q* 12 h for 5 days; Synulox®, Pfizer, Turkey). An imminent recovery was observed and complete blood count was within normal range on day 3.

The uterus contained a huge number of clear viscous fluid filled sacs with dimensions differing between 0.7-4.5 cm at all six placental sites *(Fig. 1B)*. Estradiol, progesterone, follicle stimulating hormone and testosterone levels in mole fluid were 9.60 pg/mL, 0.04 ng/mL, 0.47 mIU/mL and 0.06 ng/mL, respectively. Embryos of 6-8 mm length were observed at each placental site. Pregnancy age was calculated to be around eighteen days based on the length of the embryos [9]. Conceptus fluids were totally resorbed *(Fig. 2)*. Corpora lutea were observed on both ovaries. Both ovaries contained numerous 1-5 mm in length serous fluid filled cysts. Material for histopathological examination was fixed in 4% buffered formalin, embedded in paraffin and stained routinely with haematoxylin and eosin. Partial villi distention by edema was observed. Affected villi were hydropic and enlarged with irregular scalloped borders. Small nonmolar villi were also evident. Hydropic villi were lined by uniform trophoblastic cells, a few of them showing atypia *(Fig. 3A)*. Some enlarged villi had a central cavity (cistern) containing mucinous matrix. Trophoblastic inclusions in the villous stroma were observed *(Fig. 3B)*. Trophoblastic cells were focally and mildly proliferated and an increased number of endometrial glands, lymphocytes and decidual cells were evident. Vacuolation of the trophoblast cytoplasm and picnotic nuclei were noted focally. Erythrocytes in villous capillaries were observed

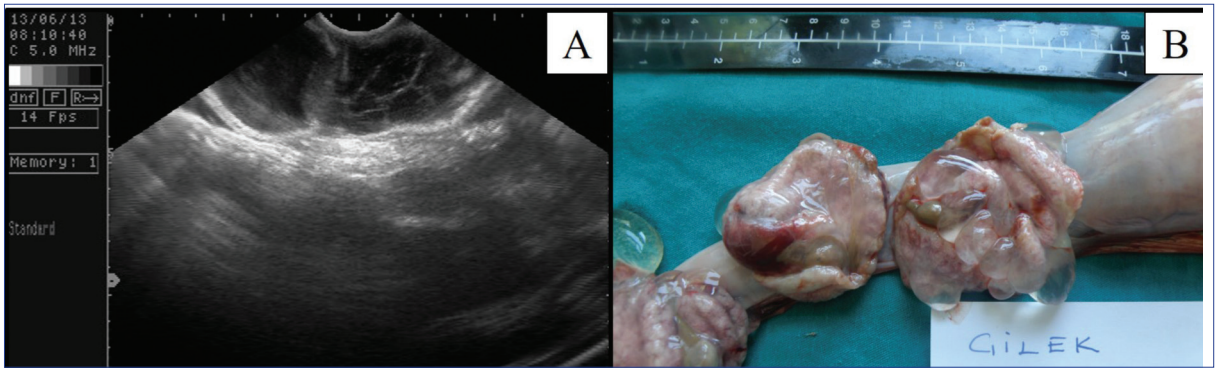

Fig 1. Ultrasonography (A) and macroscopy (B) of partial hydatidiform mole

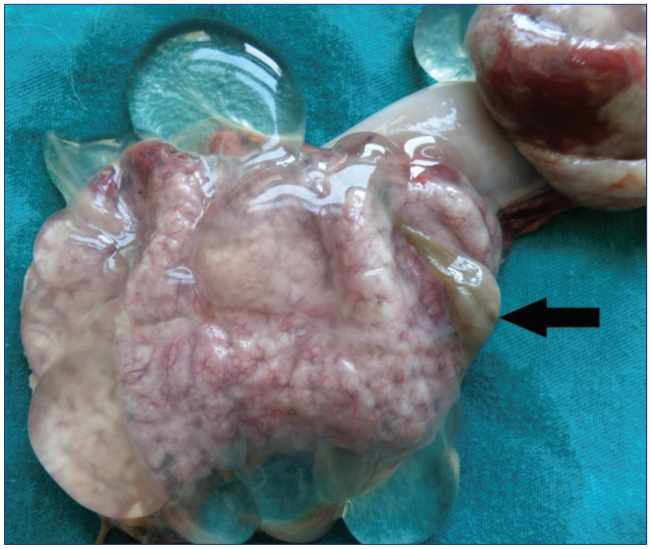

Fig 2. Partial hydatidiform mole-placental changes and embryo *(arrow)*

In addition, trophoblastic inclusions are rarely observed in complete moles, while atypia of trophoblasts are often present [4]. In the current case trophoblastic inclusions were observed frequently and atypia of trophoblasts were less pronounced. Collectively, most of the histopathological features of partial mole in the human were observed in the current case.

Differential diagnosis of hydatidiform mole in the human includes non-molar hydropic abortion with villous edema. In hydropic abortion, villous hydrops is not grossly visible, as it was in our case, rather; it is microscopic and limited. In hydropic abortion, villous shape is round and small; trophoblastic inclusions are usually absent and infrequent small cisterns do not cause gross villous enlargement. Beckwith Wiedemann syndrome or placental angiomatous malformation sometimes mimic partial mole, however, trophoblast proliferation is not observed in these abnormalities [4].

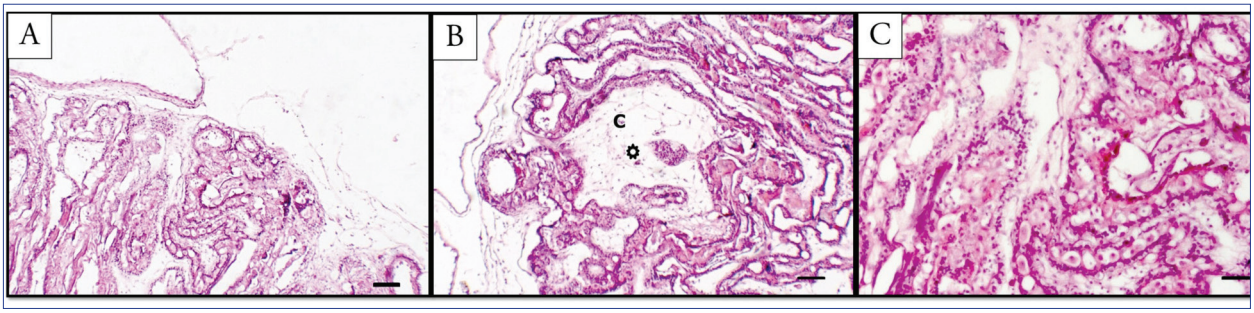

Fig 3. Histopathological findings in partial hydatidiform mole. **A:** Hydropic villi with irregular shape and scalloped borders, bar = 250 µ; **B:** Cystern (C) including mucinous matrix, trophoblastic inclusion *(asteriks)*, bar = 450 µ; **C:** Decidual cells with clear vacuoles, focal lymphocytic infiltrations, villious edema, bar = 300 µ

(Fig. 3C). Based on these histopathological findings and previously reported criteria [4], a diagnosis of a partial hydatidiform mole was made. Histopathology of both ovaries revealed numerous follicular cysts lined by a single layer of cuboidal epithelial cells.

DISCUSSION

Hydatidiform moles which are relatively common in the human and occur in approximately 1 in every 1500 pregnancies. Moles can be complete or partial depending on their gross appearance, histopathology and karyotype [1]. In this case, partial hydatidiform mole diagnosis was made based on the presence of embryos, gross appearance and histopathological findings. Embryo-fetal development usually observed in partial moles is absent in complete moles. In contrast to generalized villi edema with cistern formation and diffuse hyperplasia of trophoblastic tissue in complete moles, villous edema is limited and trophoblastic hyperplasia is focal in partial moles. As a result, some small non-molar villi are also observed in partial moles. These features were clearly evident in the current case.

Our findings are in accordance with gross morphology and histopathology of the only case report in the cat, which describes a stillborn kitten with a triploid karyotype [8]. Authors reported vesicular edematous villi distension, focal mucous stromal degeneration, growth of the trophoblast epithelium and focal cytoplasmic vacuolations of the trophoblastic cells which we also observed. In contrast, atypia of trophoblasts were less pronounced in our case.

Partial moles are usually triploid in karyotype. They contain one maternal and two paternal sets of genes as a consequence of reduplication of the paternal haploid set from a single spermatozoon. In rare cases, partial moles arise from dispermic fertilization of the ovum [10]. In the case with stillborn kitten [8], it was shown that a haploid egg was fertilized by two spermatozoa, resulting in triploid karyotype. Unfortunately, we were not able to perform cytogenetic analysis. However, in our case all six embryos were affected and it seems unlikely that all embryos resulted from dispermic fertilization which is a random event.

Although fluids were totally resorbed, embryos were

not decomposed which shows that embryonic viability was lost recently. In the case with the stillborn kitten, it was reported that some parts of the placenta resembled normal and other parts showed abnormalities. It was also reported that the birth weight of the affected kitten was lower than other newborn kittens of the same breed [8]. In the current case, placentae were highly affected and contained higher numbers of cysts. The severity of the placental lesions might have contributed to different pregnancy outcomes in two cases, with more pronounced placental deficiency and earlier termination of pregnancy in our case. In addition, pregnancy was lost when blood progesterone was above baseline levels, therefore luteal deficiency does not seem to be a contributing factor in termination of pregnancy.

It is interesting to note that uterine enlargement, bloody vaginal discharge, vomiting and blood count abnormalities observed in the current case are among traditional clinical symptoms of hydatidiform mole in the human [10]. The proliferating trophoblastic tissue seems to be the reason for uterine enlargement and vaginal discharge in this case, because uterine interplacental sites were not affected. In early human pregnancy, hCG is primarily produced by the syncytiotrophoblast and excessive amounts of hCG, observed in most cases of hydatidiform mole, leads to medical problems including anemia and hyperemesis gravidarum. In addition, hyperstimulation from increased hCG may cause ovarian enlargement and theca lutein cysts [3]. Although similarities exist between clinical presentations, it should be noted that ovarian cysts we observed were of follicular type and feline trophoblasts do not produce hCG.

In this report, clinical presentation and histopathological findings of a partial hydatidiform mole leading to pregnancy loss in the cat were described.

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