Hypospadius in Two Male Cats

Case 1

The patient is a 6-month-old Abyssinian cat. He is the product of an outbred mating and was first examined for routine vaccination and determination of gender at 12 weeks of age. His small size, 2.2 lbs (1kg), necessitated magnification of the perineal area to determine the status of this visibly peculiar region.

The penile and perineal urethra was ventrally split its entire length. The scrotum was bifid, with each laterally displaced sac containing a normal size for age testis. The crus of the penis was ventrally dystoped forming congenital chordae. The urethral meatus was on the ventral midline immediately beneath the rectum. There was a urorectal septum composed only of mucous membrane, and there was no cutaneous covered septum separating the rectum from the urinary tract.

The external genitals as they existed were enveloped in folds of skin that began at the three o’clock position of the rectum, extended ventrally down on the right side to the small remnant of the penis. The skin fold then directed dorsally on the left side to the nine o’clock position of the rectum. Within the skin folds were the exposed mucous membrane covered structures. The rectal ring was intact 360 degrees, and both the rectum and the urethra were neurologically and functionally normal. Figure 1 shows the cat at 6 months of age.

The cat was neutered, at 6 months of age, via a routine surgical approach through each scrotum. Normal appearing testes were removed. No surgical correction of the defects was attempted. Laparotomy to locate paramesonephric ducts (persistent Mullerian ducts), or ovaries, was not performed, nor was chromosomal analysis done. A genetic history was taken and it was non-revealing. There was no inbreeding evident within the pedigree.

Case 2

The patient is a 1-year-old feral Domestic Shorthaired tomcat. He was presented by the owner for routine vaccination and neutering. His external genitalia consisted of a bifid scrotum, each sac containing a testis, the penis and prepuce.
widespread in the sinusoids with marked involvement of perilobular areas (Fig. 3). Prominent reduction of lymphocyte numbers and replacement by tumor cells were noted in the spleen. The neoplasms appeared to completely efface the normal lymph node architecture. Since these histopathological findings suggested involvement of granulocytic and monocytic lines, a final diagnosis of suspected myelomonocytic leukemia was made.

Discussion

Myelomonocytic leukemia is characterized by a neoplastic clone of undifferentiated cells having both myelogenous and monocytic features. Granulocytes and monocytes are derived from the common stem cell in the bone marrow (granulocyte-monocyte colony-forming-unit) and it is at this level that maturation arrest and clonal proliferation are thought to occur in myelomonocytic leukemia. The present case had many features similar to those of the feline myelomonocytic leukemias reported heretofore: an elderly patient, nonregenerative anemia, thrombocytopenia, hypercellular bone marrow, and proliferation and infiltration of tumor cells in the lymph nodes and visceral organs. The leukocyte counts were generally between 25,000 and 50,000/μl in the previously reported canine and feline cases, whereas the present cat exhibited severe leukopenia especially during the early period of this disease. Since marked depletion of metamyelocytes, band forms, and mature neutrophils were seen in the bone marrow, maturation arrest might have occurred in the present case. Because a moderate number of mature neutrophils were seen in the visceral organs, we could not clearly explain about the cause of leukopenia. In this case, there were many rubricytes in the bone marrow and some of them exhibited erythroagglutination. Therefore, erythroagglutination may be one of the causes of non-regenerative anemia. Most cases of myelomonocytic leukemia had severe infiltration of blast cells in the bone marrow, but this case did not exhibit severe infiltration of blast cells in the bone marrow.

There was markedly proliferation and infiltration of tumor cells in the spleen. Thus, the spleen was regarded as a major extramedullary site of tumor cell proliferation.

The tentative diagnosis of this case as lymphoblastic leukemia was made on the basis of findings of blood and lymph node aspiration smears. Histologic examination gave us the final diagnosis as myelomonocytic leukemia. However, more detailed examinations such as cytochemistry and electron microscopy are required to clarify cell types.

There are many reports on treatments for leukemia of lymphoid origin in veterinary oncology, but treatments of leukemia of monocytic or myeloid origin are limited. G-CSF is a cytokine that is critical in the proliferation and differentiation of hematopoietic cells. The hG-CSF is commercially available and induces a marked leukocytosis with a mature neutrophilia and monocytosis in the dog. It is also effective in alleviating the neutropenia seen in cyclic hematopoiesis. Unfortunately the long-term use of heterologous factors are not possible because of the development of neutralizing antibodies, which also may inhibit the function of endogenous growth factor. Therefore, canine G-CSF (cG-CSF) has recently been used on an experimental basis. However, there are very few reports on the treatment with hG-CSF or cG-CSF in cats. In this case, hG-CSF was administered to stimulate granulocyte proliferation, but no improvement was noted. Since there is the possibility that hG-CSF increases granulocytic tumor cells, the treatment of leukemia patients with hG-CSF is problematic. On the other hand, supportive care with prednisolone was temporarily effective and alleviated clinical signs.

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were split ventrally. The remnant of the penis was ventrally dystoped. Only the penis and prepuce were nonfused. There was a hairless cutaneous cover over the urorectal septum providing a distinct separation of the rectum from the urethral meatus. The meatus was located at the proximal end of the nonfused penile urethra (Fig. 2).

The young tomcat was neutered via a routine scrotal approach. The testis appeared normal. Laparotomy or chromosomal analysis was not performed. The cat was released to its closely bred neighborhood cat population following neutering.

Traditional embryology teaches that the conditions described in this case represent a failure of the urogenital folds to unite or fuse on the ventral midline. The external genitalia in the male arise from the mesonephric (Wolffian) duct system. The ventral fusion of the urogenital folds results in the approximation of the genital swellings that contain the testes and form the scrotum. The fusion also create a tubular urogenital groove that becomes the urethra, penis, and associated structures. Failure of the folds to unite results in hypospadias and scrotal displacement as seen in these cases. Descriptive names exist that describe the anatomical location of the urethral defect such as anal hypospadias, perineal, scrotal, glandular penis, or penis hypospadias. The descriptive names themselves imply varying degrees of severity and dysfunction resulting from the nonfusion of the urogenital fold.¹²

Urogenital deformities similar to these cases have been seen in other intersex animal species that are pseudo or true hermaphrodites. Sensitive aged male fetuses' exposed to androgen antagonists or estrogenic compounds, fetal tissues that are resistant to male hormonal potentiation and male fetus' that fail to produce enough androgens, all represent other possible causes of urogenital fold fusion failure.

Hypospadias can occur in both male and female genders, however the ratio is skewed 15 to 20:1, male to female.⁵ In dogs, a polygenic origin of hypospadias is hypothesized. The Boston Terrier may have a slightly higher incidence of the anomaly.¹ In a study of canine hypospadias an incidence of sixty-six dogs out of 2.2 million hospital/clinical events was observed over a twenty year period. Obviously this malady is rare in dogs.³

Hypospadias in goats and sheep is considered an intersex condition, genetic or environmental etiologies are hypothesized only.³ Studies in laboratory animals have created hypospadias by exposing sensitive aged fetuses to progestational compounds, androgen antagonists and Vitamin A deficiency.⁶

In human fetuses' both estrogenic and progestational compounds are known to induce hypospadias. The anomaly is not rare in humans. In fact, hypospadias has a spontaneous incidence of 160 to 800 cases per live male births. As a result, there is no shortage of physicians and surgeons skilled in human hypospadias.³⁰

**Discussion**

These young male cats' multiple urogenital defects rendered the external genitals to have an anatomical appearance that resembled an avian cloaca, in Case 1. In Case 2, the defects were less dramatic and created only minor visual and functional disturbance. There are no reports in the veterinary literature pertaining to these urogenital defects of the domestic feline. This is the first instance of these particular anomalies being reported. Hypospadias may receive little attention because of the minimal financial impact of the anomaly, the minor social consequence it has, and ambiguity of the pathogenesis of the defect. These cats obviously are removed from the genetic pool, being unable to reproduce, and are typically surgically sterilized.

In these cases the cause of the failure of fetal sex hormones to elaborate normal external genital development would be conjectural only. These patients would be described as intersex tomcats, male pseudohermaphrodites. The result being a variant degree of feminization. Patient 1 is living and functioning well at this time. Patient 2 had been lost to follow up.

**REFERENCES**


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**FIG. 2 – Photograph of unanesthetized Case 2 patient. The hairless septum is clearly evident, as is the large bifid scrotum and penile deformity. Self-soiling due to the anomaly is minimal.**