**Hypersexuality responsive to phenobarbital in a male neutered DSH cat**

Theophanes Liatisa,b,\*, Giunio Bruto Cherubinib

a Small Animal Hospital, University of Glasgow, Glasgow, UK

b Neurology & Neurosurgery Service, Dick White Referrals, Six Mile Bottom, UK

\***Corresponding author:** Theophanes Liatis, DVM MRCVS Clinician in Veterinary Neurology, Small Animal Hospital, 464 Bearsden Rd, Bearsden, Glasgow G61 1BD, UK; [theofanis.liatis@gmail.com](mailto:theofanis.liatis@gmail.com)

**ABSTRACT**

A 6-year-old, vaccinated, male neutered domestic short-hair cat was presented due to polyphagia and hypersexuality manifested since he was adopted one-year ago. Clinical and neurological examination were unremarkable, including absence of scrotal testicles and penile spines, indicative of testosterone absence. Differential diagnoses included inadequate neutering and residual testicular tissue, cryptorchidism, adrenal hyperplasia/neoplasia (sex steroid-producing), urinary bladder neoplasia, infectious myelopathy/neuropathy (e.g. feline infectious peritonitis), temporal lobe epilepsy or primary behavioural disorder. Haematology, biochemistry and thyroid profile were almost unremarkable. Serology for FIV, FeLV and FCoV was negative. Serum pre-and-post-hCG stimulation testosterone ruled out cryptorchidism. Abdominal ultrasound revealed mildly enlarged colonic lymph-nodes, the cytology of which revealed mild reactive hyperplasia. Urinalysis and culture were positive to *Enterococcus* spp. Magnetic resonance imaging of the brain and spinal cord, and cerebrospinal fluid analysis were unremarkable. PCR in the cerebrospinal fluid for *Toxoplasma gondii*, FPV, FCoV and Bornavirus was negative. Attempts to treat hypersexuality as a behavioural disorder, urinary tract infection (co-amoxyclav), pain (meloxicam, diazepam) and anxiety (diazepam) were unsuccessful. Thus, phenobarbital was prescribed in a low dose (1 mg/kg PO q12h) which eliminated the episodes of polyphagia and hypersexuality without sedating. Phenobarbital withdrawal resulted in hypersexuality re-establishment. Two years later the cat remained episode-free on phenobarbital. Hypersexuality in male cats is characterized mainly by biting knap, mounting, pelvic thrusting, penile erection, coital intermission, ejaculation and/or masturbation. Temporal limbic structures play a significant role in the regulation of sexual arousal independently of testosterone. Hypersexuality has been associated with temporal lobe epilepsy in cats experimentally, whilst along with other clinical signs such as polyphagia it is an established condition in humans called Klüver-Bucy syndrome. This is the first report to describe phenobarbital as a successful treatment of hypersexuality and polyphagia in a male neutered cat, raising a suspicion of feline Klüver-Bucy syndrome and temporal lobe epilepsy origin of these signs.

**KEYWORDS:** feline; Klüver-Bucy syndrome; limbic seizures; polyphagia; temporal lobe epilepsy; phenobarbitone

**MANUSCRIPT**

**History and presenting complaints**

A 6-year-old, vaccinated, owner-owned, male neutered domestic short-hair cat was presented due to polyphagia and hypersexuality episodes manifested since he was adopted one-year before admission. Hypersexuality episodes were described as humping or biting knap of animate (e.g. owners, other household cats) or inanimate (e.g. towels) objects (Video 1), followed by pelvic thrusting and aftermath penile licking. No ejaculation was noticed. These episodes had a frequency of 20 times per day and a duration of 2-3 minutes each.

**Clinical findings and differential diagnoses**

Clinical and neurological examinations were unremarkable. Absence of scrotal testicles and penile spines was noticed. Differential diagnoses for a neutered male cat with hypersexuality included (a) inadequate neutering and residual testicular tissue, (b) cryptorchidism, (c) adrenal hyperplasia/neoplasia (sex steroid-producing), (d) urinary bladder neoplasia, (e) infectious myelopathy/neuropathy (e.g. feline infectious peritonitis), (f) temporal lobe epilepsy or (g) primary behavioural disorder. Differential diagnoses for polyphagia in cats included (a) primary polyphagia (e.g. central nervous disease affecting the satiety center, psycogenic, stress, introduction of a more palatable diet) or (b) secondary polyphagia (e.g. endocrine diseases, exocrine pancreatic insufficiency, parasitism, neoplasia, portosystemic shunts, medications etc.).

**Diagnostic investigations**

Haematology, serum biochemistry and thyroid profile (total thyroxine) were unremarkable. Serology for FIV, FeLV and FCoV was negative. Alpha 1 - acid glycoprotein (AGP) levels were elevated (1004 μg/ml, RI: <500 μg/ml). Serum resting (<0.3 nmol/L, reference intervals [RI]: 0 - 7.6 nmol/L) (Johnston et al., 1996), as well as post-hCG stimulation testosterone levels (<0.3 nmol/L, RI: 17.3-41.6 nmol/L) (Johnston et al., 1996) were low, suggesting absence of cryptorchidism. An abdominal ultrasound was then performed to investigate for retaining testicles and evaluate the adrenal glands, and it revealed mildly enlarged colonic lymph nodes, the cytology of which was compatible with mild reactive hyperplasia. Urinalysis and urine culture were suggestive of urinary tract infection by *Enterococcus* spp. Following that, a magnetic resonance imaging (MRI) of the central nervous system was decided for investigation of hypersexuality/polyphagia of neurologic origin. A low-field MRI (Hitachi Lucente 0.4 T) of the brain was performed, including T2, T2-fluid-attenuated inversion recovery (FLAIR), T2\*, and T1 pre- and post-contrast sequences, and of the lumbar spinal cord, including T2, T2-Short-tau inversion recovery (STIR), T1 pre- and post-contrast sequences. MRI of both brain and spinal cord was unremarkable.

Cerebellomedullary cisternal cerebrospinal fluid (CSF) was unremarkable (total protein 13 mg/dl, RI: 8 - 36 mg/dl); total nucleated cell count <1 /μL, RI: 0-8 /μL). PCR in the CSF for infectious diseases, including *Toxoplasma gondii*, FPV, FCoV and Borna disease virus (BDV), were negative.

**Treatment, outcome and follow-up**

Initially, co-amoxiclav 20 mg/kg PO q12h, meloxicam 0.1 mg/kg PO q24h and diazepam 0.3 mg/kg PO q8h were given for a 2-week course as antibiotic, analgesic and bladder relaxant/anxiolytic respectively. Although this therapy treated the underlying urinary tract infection, it failed to cease hypersexuality/polyphagia.

Following the urinary tract infection treatment, attempts to treat the cat’s hypersexuality/polyphagia as a behavioural disorder were chosen. Specifically, modifications of the household to provide extra space, privacy, entertainment (e.g. toys) and hygiene (e.g. additional litter trays) (Heath and Wilson, 2014) were made, in addition with application of pheromone diffuser (Feliway ® Classic Diffuser, CEVA, UK). None of them changed the sexual activity of the cat.

Then, antiepileptic treatment was decided, and phenobarbital 1 mg/kg PO q24h was initiated for a 2-week course which resulted in episode frequency reduction to 10 per day. Subsequent increase of phenobarbital of the same dosage from q24h to q12h ceased completely the episodes for a month. Then, phenobarbital gradual withdrawal was decided with an aim to evaluate potential episode recurrence. When phenobarbital was withdrawn and the cat was medication-free, the episodes returned completely back to the initial frequency of 20 per day. At last, phenobarbital 1 mg/kg PO q12h was reintroduced and the cat remained hypersexuality/polyphagia episode-free for a total of 2 years, until the time of writing. During all this period, sedation, ataxia or polyuria/polydipsia were not noticed, and the owners considered that the cat was lively as always. Phenobarbital serum levels were evaluated 1-month post phenobarbital re-introduction and appeared to be lower than the therapeutic levels (11 mg/l, RI: 15-30 mg/l).

**Discussion**

Hypersexuality is extremely frequent or suddenly increased libido, reported also as *nymphomania* (female) or *satyriasis* (male). Hypersexuality in male cats is characterized by biting knap, mounting, pelvic thrusting, penile erection, coital intermission, ejaculation and/or masturbation (Hart, 1974; Hart and Leedy, 1985; Beaver, 2003a). Urine spraying or marking could be considered as sexual in origin, however it is most commonly a social behaviour (Neilson, 2003). Sexual aggression is a rare manifestation of hypersexuality in cats usually more common in intact or neutered male cats (Curtis, 2008), and it ischaracterized by mounting the owner's limb, grabbing the skin, initiating pelvic thrusting, and growling possibly resulting in human injury (Curtis, 2008). Feline nymphomania is characterized by posturing, licking the vulva, vocalizing, rolling on the ground, head rubbing, mounting, or rubbing the anogenital region against the floor (masturbation) and it is a relatively common variation of female sexual behavior, especially in Siamese and Persian cats (Beaver, 2003b). Hypersexuality can be manifested in entire (physiological or pathological), but also in neutered male and female individuals (pathological).

Sexual behaviour in cats originates by the limbic system, and particularly the septal area and the amygdala (De Lahunta, et al. 2015). Additionally, spinal cord segments, spinal nerves, and autonomous (sympathetic) nervous system contribute to the sexual function of male cats. Specifically, the second sacral nerve and the sympathetic fibers of the hypogastric nerve contribute to erection, whilst the lumbosacral spinal cord segments and the pudendal nerves contribute to ejaculation (Beaver, 2003a).

Clinical evaluation of hypersexuality in a male neutered cat should include (a) exhaustive palpation of the abdomen and inguinal region for retaining testicular tissue evaluation (e.g. inguinal), (b) penile spines evaluation, (c) penile evaluation (e.g. erection, priapism etc.). Penile spines consist of a clinical indicator related to the presence of testosterone in male cats. They start to appear at about 12 weeks of age, and reach full size in the adult, and they regress by 6 weeks post neutering. Penile spines regress by 6 weeks after postpubertal castration (Aronson and Cooper, 1967). Therefore, complete absence of such penile spines is expected in a castrated cat. Single resting testosterone is not a reliable biomarker of testicular dysfunction in the cat due to the episodic, pulsatile nature of its secretion, and therefore undetectable concentrations can be a common finding even in entire male cats (Johnston et al., 1996). Stimulation of maximal testosterone secretion by administration of hCG or GnRH permits a more reliable assessment of testosterone reserve of the testicle or other testosterone-producing tissue (Johnston et al., 1996). Abdominal ultrasound is imperative in order to evaluate the existence of retaining testicle (cryptorchidism) as well as the condition of adrenal glands (e.g. adrenal tumours).

Hypersexuality has been reported in cats as a clinical sign of several non-neurological non-behavioral diseases. Male and female neutered cats with hyperadrenocorticism manifested hypersexuality, which was attributed to the excessive sex steroid production (Boag et al., 2004; Muschner et al., 2018). Oestrus behaviour or urine spraying and aggression attributed to sex-steroid producing adrenocortical carcinomas have been reported in female and male neutered cats respectively (Millard et al., 2009; Meler et al., 2011). Urine spraying and increased sexual behaviour has been observed in feline neutered individuals with secondary hyperandrogenism as a result of functional ectopic testicular tumour (male) (Rosen and Carpenter, 1993) or ovarian remnant syndrome (female) (Jones et al., 2019). Priapism has been reported in cats as a result of post-castration complication, trauma, suspected thoracolumbar spinal cord injury or FIP penile granuloma (Swalec and Smeak, 1989; Gunn-Moore et al., 1995; Rota et al., 2008). Sudden-onset persisting mounting episodes has been associated with urinary bladder carcinoma in an older cat (Houpt, 1997). It is important also for the clinician during history-taking not to confuse the urine spraying with pollakiuria. Pollakiuria (increased frequency of urination) is a clinical sign of lower urinary tract disease (e.g. urinary tract infection, idiopathic cystitis etc.). In case of doubt, investigations for lower urinary tract disease should be initiated. Misinterpretation of hypersexuality has occurred in entire male cats which manifested persistent mounting with intense, prolonged pelvic thrusting but no intromission. This is believed to be a result of the formation of a hair ring around the base of the glans penis. The caudally directed penile spines some-times collect hair from the female’s perineum, which may not have been removed by normal grooming. In other tomcats, the problem might be due to improper pelvic orientation, which usually results from lack of experience (Beaver, 2003a).

Hypersexuality and polyphagia have been reported as clinical manifestations of limbic system disorder in cats (Delahunta et al., 2015). Experimental studies in male cats showed that amygdala lesions cause decreased selectivity, that is the propensity to perform sexual mounts on less appropriate objects (Aronson and Cooper, 1979). Polyphagia, hypersexuality, aggression, hyperthermia or hypothermia and seizures have been reported in experimental studies with rhinencephalic lesions in cats (Schreiner and Kling, 1953; Green et al., 1957; Katahira and Tsukahara, 1974). Although debatable, feline hypersexuality has been proposed as a manifestation of temporal lobe epilepsy (limbic seizures) (Andy, 1977). On the other hand, hyposexuality has been reported to be an alternative manifestation of temporal lobe epilepsy in experimental conditions in cats (Feeney et al., 1998).

In 1937, Klüver and Bucy described hypersexuality as a part of a clinical syndrome in monkeys after bilateral temporal lobectomy (Lanksa, 2018). Currently, Klüver-Bucy syndrome (KBS) is a clinical syndrome caused by disturbances of temporal portions of the limbic system, including amygdala, that interface with multiple cortical and subcortical circuits to modulate emotional behavior and affect (Lanksa, 2018). KBS usually consists of three or more of the clinical features below: (a) hypersexuality, (b) hyperorality (marked oral examination of objects with licking, sucking, chewing movements, and placing of non-food objects in the mouth), (c) placidity (loss of fear response), (d) hypermetamorphosis (compulsory urge to respond to visual objects), (e) dietary changes (polyphagia, pica, coprophagia), and (f) visual agnosia (e.g. prosopagnosia). (Lanksa, 2018; neo). Hypersexuality in humans may include ictal or postictal improper sexually oriented remarks, exhibitionism, attempts at touching the genitals of others, solicitation of sex, masturbation, or attempting sex with inanimate objects (Lanksa, 2018). Additionally, other sexual behaviour alterations such as interictal hyposexuality, interictal transvestism, exhibitionism and fetishism have been described in humans with temporal lobe epilepsy (Blumer, 1970; Cogen et al., 1979; Ellison, 1982). The most frequently described clinical features in both adults and children with KBS are hypersexuality and polyphagia/hyperorality (most author cannot distinguish between these two) (Gasquoine1 2020). Several causes have been reported to affect the temporal lobes bilaterally resulting in KBS in humans (Table 1) (Lanksa, 2018). Viral encephalitis, severe traumatic brain injury, post-hyponatraemia myelinolysis, adrenoleukodystrophy, systemic lupus erythematosus and bithalamic infarction are common causes of KBS in adults, whilst in children the most common cause revealed to be encephalitis by herpes simplex virus (Gasquoine, 2020).Most autopsied cases of KBS have shown extensive lesions involving the medial, inferior, and anterior temporal cortex bilaterally (Blumer, 1970; Cogen et al., 1979; Ellison, 1982). Two neuropathological mechanisms for KBS in humans have been proposed: (a) direct bilateral injury to amygdala and other mesiotemporal limbic structures (septal area, hypothalamus) and (b) disconnection of those limbic structures from frontal lobe structures (Gasquoine, 2020). Hypersexuality has been proposed to be a result of a combination of lesions to the abovementioned structures, as restricted lesions (e.g. in bilateral amygdalas) do not typically result in hypersexuality. It is unclear though, why a lesion at these brain regions cause hyper- and not hyposexuality (Gasquoine 2020). Compulsive eating, recognized as polyphagia, has been attributed to disconnection of the hypothalamus from orbitofrontal cortex (Ahmed et al., 2014). Nevertheless, many cases involving bilateral frontal and mesiotemporal damage do not exhibit hypersexual behavior and therefore KBS is considered quite infrequent (Gasquoine 2020).

KBS has been reported in one single dog with pica and other behavioural problems but no hypersexuality, diagnosed with bilateral symmetrical necrosis of the amygdalas, parahippocampal gyri and extratemporal cortices as a result of prolonged status epilepticus (Hasegawa et al., 2005). It should be noted, also, that *catamenian seizures* should not be confused with KBS. It is known that sexual hormones might play a role in epiletic activity. *Catamenian seizures* are described as a pattern of seizures that changes in severity (e.g. clusters) during particular phases of the menstrual cycle, wherein estrogens are proconvulsant, increasing the neuronal excitability; and progesterone is anticonvulsant, enhancing GABA-mediated inhibition (Verrotti et al., 2012).

In cats, other than epileptic in origin, hypersexuality could be a physiological activity or a manifestation of a behavioral disorder. Around 10% of castrated cats retain behaviors associated with intact tomcats (as a learnt behavior), thus mating can be present indefinitely after castration (Beaver, 2003a). It has been suggested that hypersexuality in cats can be a stereotypic behavior due to environmental, husbandry or welfare factors (e.g. colony of cats, cat rescue centre), in which occasion it should not be related with temporal lobe pathology (Michael, 1961). Sporadic cases of hypersexuality in male neutered cats have been attributed to behavioural disorder. Two male neutered cats with excessive mounting have been reported, one of which was treated with amitriptyline reducing the intensity and frequency of this behavior (Houpt, 1997). Amitriptyline is a tricyclic antidepressant that decreases *libido* causing, particularly in men, impotence and ejaculation disorder (Chen et al., 2018). In another male neutered cat with hypersexuality, fluoxetine and pheromone diffuser (Feliway® Classic Diffuser, CEVA, UK) were used as treatment. The hypersexuality episodes decreased temporarily, however the owners did not comply at the environmental modifications (e.g. enrichment of environment etc.) and therefore the episodes recurred (Guardini et al., 2013). Fluoxetine is a selective serotonin reuptake inhibitor, which has been reported to reduce hypersexual disorders in humans (Naficy et al., 2013). Amitriptyline or fluoxetine have been reported to treat urine spraying as a sole sign in cats (Neilson, 2003). F3 facial pheromone of the cat is included in the abovementioned pheromone diffuser (Feliway® Classic Diffuser, CEVA, UK). F3 facial pheromone of the cat shows interesting efficacy in decreasing urine marking, scratching, improvement in feeding, scanning, and playing (Pageat and Gaultier, 2003). The mechanism of F3 facial pheromone is unknown, however it is hypothesized that it induces some modifications in both the limbic system and the hypothalamus regulating as such the emotional status and the way the animals react (Pageat and Gaultier, 2003). In particular, F3 facial pheromone provides antagonism between facial and urine marking, and anxiolytic effects (Pageat and Gaultier, 2003). On the other hand, hypersexuality in entire male cats has been well controlled with subcutaneous implants of deslorelin acetate (Suprelorin®, Virbac, UK) (Palm et al., 2013), as its goal is to reduce testosterone. In the current case, fluoxetine or other antidepressants were not tried. Environmental modifications and pheromone diffuser did not provide clinical improvement. Nevertheless, low-dose phenobarbital seemed to cease completely the episodes, whilst withdrawal of it resulted in episode recurrence. Phenobarbital is a barbiturate antiepileptic drug effective for both generalised tonic-clonic seizures and focal seizures in humans of all ages (Goldenberg, 2010). Phenobarbital potentiates inhibitory neurotransmission by increasing the duration of time that GABA-mediated chloride channels remain open and reduces neurotransmitter release from nerve terminals, probably through its effect on calcium channels, as well as it diminishes excitatory neurotransmission by reducing the effects of glutamate (Goldenberg, 2010). Phenobarbital has not widely been used for KBS treatment in humans, as carbamazepine is the treatment of choice (Goldenberg, 2010). Carbamazepine is dibenzazepine anticonvulsant widely used in human medicine, however it is not spread in veterinary practice due to its short half-life in dogs, the lack of evidence in canine and feline epilepsy and the dramatic vomiting side effects (Plumbs, 2018). Nevertheless, it has been effectively used in feline aggression (Plumbs, 2018). Therefore, in our case it is difficult to confirm whether phenobarbital might have acted as an antiepileptic drug ceasing ictal or postictal hypersexuality, or as a sedative calming the abnormal hypersexual behavior of the cat. The prevalence of sedation as a side effect of phenobarbital administration in epileptic cats has been recently reported to be 89% (Marsh et al., 2020). However, the owners of the cat of the current report did not notice any change to his usual activity to justify a possible sedation. Additionally, sedation would be an unlikely adverse effect of phenobarbital in this cat given the low phenobarbital serum levels, making the cessation of the abnormal behavior of this cat less likely to be a result of a potential sedation. The simultaneous response of clinical signs and the low serum level of phenobarbital could be a result of either idiosyncrasy of the cat and possible sensitivity to the drug or cessation of the suspected seizures by a low dose as they resemble a focal complex seizure rather generalised. In humans, the treatment of KBS is often difficult and unsatisfactory (Lanksa, 2018). Treatment includes behavioural management and carbamazepine, however other medications have been also tried: mood-stabilizing agents (e.g. valproic acid, gabapentin), selective serotonin reuptake inhibitors, propranolol, leuprolide, antipsychotic agents (e.g. haloperidodl) Leuprolide, a potent inhibitor of gonadotropin secretion and testicular steroidogenesis) improved sexually aggressive and inappropriate behaviours. (Lanksa, 2018).

Limitations such as absence of electroencephalography during the episodes, the practical difficulty to identify the epileptic or behavioral origin of clinical signs (e.g. hypersexuality), and the vulnerability of cats to manifest behavioural changes when the environment does not meet their criteria, contribute to the inability to confirm that the cat of the present report manifested a limbic seizure as a part of temporal lobe epilepsy and KBS. Nevertheless, the co-manifestation of hypersexuality and polyphagia and the successful response of both to phenobarbital, could raise a suspicion that both of them could be of epileptic origin and this cat might have manifested KBS and temporal lobe epilepsy.

**Summary**

This case report describes a male neutered cat with hypersexuality and polyphagia that could be a manifestation of KBS and therefore attributed to temporal lobe epilepsy. Hypersexuality episodes and polyphagia completely ceased with low-dose of phenobarbital without concurrent side effects. A behavioural disorder (stereotypy) could not be completely ruled out. As a consequence, phenobarbital might be a useful therapeutic modality for feline patients with intensive and frequent episodes of hypersexuality accompanied or not by polyphagia, when other non-epileptic aetiologies have been exhaustively ruled out.

**Acknowledgments**

N/A

**Ethical considerations**

The authors declare no ethical concerns. The owners consented to the publication of this report.

**Conflict of interest**

Authors declare no conflict of interest.

**References**

Ahmed, R. M., Irish, M., Kam, J., van Keizerwaard, J., Bartley, L., Samaras, K., Hodges, J.R., Piguet, O., 2014. Quantifying the eating abnormalities in frontotemporal dementia. Journal of the American Medical Association: Neurology 71, 1540–1546.

Andy, O.J., 1977. Hypersexuality and Limbic System Seizures. Pavlov. J. Biol. Sci. 12, 187–228.

Aronson, L.R., Cooper, M.L., 1967. Penile spines of the domestic cat: Their endocrine‐behavior relations. Anat. Rec.157, 71 – 78.

Aronson, L.R., Cooper, M.L., 1979. Amygdaloid hypersexuality in male cats re-examined. Physiol. Behav. 22, 257 – 265.

Beaver, B.V., 2003a. Male feline sexual behavior. In: Beaver BV, editor. Feline Behavior. 2nd ed. Saunders, Missuri, pp. 164-181.

Beaver, B.V., 2003b. Female feline sexual behavior. In: Beaver BV, editor. Feline Behavior. 2nd ed. Saunders, Missuri, pp. 182-211.

Blumer, D., 1970. Hypersexual episodes in temporal lobe epilepsy. Am. J. Psych. 126, 1099 – 1106.

Boag, A.K., Neiger, R., Church, D.B., 2004. Trilostane treatment of bilateral adrenal enlargement and excessive sex steroid hormone production in a cat. J. Small. Anim. Pract. 45, 263 – 266.

Chen, L.W., Chen, M.Y., Lian, Z., Lin, H.S., Chien, C.C., Yin, H.L., Chu, Y.H., Chen, K.Y., 2018. Amitriptyline and Sexual Function: A Systematic Review Updated for Sexual Health Practice. Am. J. Mens. Health. 12, 370–379.

Cogen, P.H., Antunes, J.L., Correll, J.W., 1979. Reproductive function in temporal lobe epilepsy: the effect of temporal lobectomy. Surg. Neurol. 12, 243 – 246.

Curtis, T.M., 2008. Human-Directed Aggression in the Cat. Vet. Clin. North. Am.: Small. Anim. Pract. 38, 1131 – 1143.

De Lahunta, A., Glass, E., Kent, M., 2015. Nonolfactory Rhinencephalon: Limbic System. In: De Lahunta A, Glass E, Kent M, editors. Veterinary Neuroanatomy and Clinical Neurology. 4th edn. Elsevier Saunders, St Louis,pp: 470-474.

Ellison, M., 1982. Alterations in sexual behavior in temporal lobe epilepsy. Psychosomatics 23, 499 – 509.

Feeney, D.M., Gullotta, F.P., Gilmore, W., 1998. Hyposexuality Produced by Temporal Lobe Epilepsy in the Cat. Epilepsy 39, 140 – 149.

Gasquoine, P.G., 2020. Case study on the neuropsychology of hypersexual behavior. Act. Nerv. Super. https://doi.org/10.1007/s41470-020-00068-y

Goldenberg, M.M., 2010. Overview of Drugs Used For Epilepsy and Seizures Etiology, Diagnosis, and Treatment. P. T. 35, 392 – 415.

Green, J.D., Clemente, C.D., De Groot, J., 1957. Rhinencephalic lesions and behavior in cats. J. Comp. Neurol. 108, 505 – 545.

Guardini, G., Mengoli, M., Mariti, C., Zilocchi, M., Gazzano, A., 2013. Hypersexuality in a neueterd male cat towards his owner. ECAWBM Conference Proceedings 2013, IVBM, Lisbon, pp. 119.

Gunn-Moore, D.A., Brown, P.J., Holt, P.E., Gruffydd-Jones, T.J., 1995. Priapism in Seven Cats. J. Small. Anim. Pract. 36, 262 – 266.

Hart, B.L., 1974. Normal Behavior and Behavioral Problems Associated with Sexual Function, Urination, and Defecation. Vet. Clin. North. Am. 4, 589 – 606.

Hart, B.L., Leedy, M.G., 1985. Neurological Bases of Male Sexual Behavior. In: Adler N, Pfaff D, Goy RW, editors. Reproduction: Vol.7 (Handbook of Behavioral Neurobiology), Springer, Boston, MA, pp. 373 – 410

Hasegawa, D., Nakamura, S., Fujita, M., Takahashi, K., Orima, H., 2005. A Dog Showing Klüver-Bucy Syndrome-like Behavior and Bilateral Limbic Necrosis After Status Epilepticus. Vet. Neur. Neurosurg. J. 7, 1 – 14.

Heath, S., Wilson, C., 2014. Canine and Feline Enrichment in the Home and Kennel: A Guide for Practitioners. Vet. Clin. North. Am.: Small. Anim. Pract. 44, 427 – 449.

Houpt, K.A., 1997. Sexual behavior problems in dogs and cats. Vet. Clin. North. Am.: Small. Anim. Pract. 27, 601 – 615.

Johnston, S.D., Root, M.V., Olson, P.N.S., 1996. Ovarian and testicular function in the domestic cat: clinical management of spontaneous reproductive disease. Anim. Reprod. Sci. 42, 261 – 274.

Jones, S.A., Owens, S.L., Birchard, S.J., 2019. Hyperandrogenism associated with an ovarian remnant in a spayed female cat. J. Fel. Med. Surg. Open. Rep. 5, 2055116919885698.

Katahira, K., Tsukahara, S., 1974. Hypersexuality following unilateral amygdalectomy in the cat. Fukushima. J. Med. Sci. 20, 67 – 69.

Lanska, D.J., 2018. The Klüver-Bucy Syndrome. Front. Neurol. Neurosci. 41, 77 – 89.

Marsh, O., Corsini, G., Van Dijk, J., Gutierrez-Quintana, R., De Risio, L., 2020. Prevalence and clinical characteristics of phenobarbitone-associated adverse effects in epileptic cats. J. Fel. Med. Surg. 2, 1098612X20924925.

Meler, E.N., Scott-Moncrieff, J.C., Peter, A.T., Bennett, S., Ramos-Vara, J., Salisbury, S.K., Naughton, J.F., 2011. Cyclic estrous-like behavior in a spayed cat associated with excessive sex-hormone production by an adrenocortical carcinoma. J. Fel. Med. Surg. 13, 473 – 478.

Michael, R.P., 1961. "Hypersexuality" in Male Cats without Brain Damage. Science 134, 553 – 554.

Millard, R.P., Pickens, E.H., Wells, K.L., 2009. Excessive production of sex hormones in a cat with an adrenocortical tumor. J. Am. Vet. Med. Assoc. 234, 505 – 508.

Muschner AC, Venzon Varela F, Hazuchova K, Niessen, S.J., Gomes Poppl, A., 2018. Diabetes mellitus remission in a cat with pituitary-dependent hyperadrenocorticism after trilostane treatment. J. Fel. Med. Surg. Open. Rep. 4, 2055116918767708.

Naficy, H., Samenow, C.P., Fong, T.W., 2013. A Review of Pharmacological Treatments for Hypersexual Disorder. Sexual Addiction & Compulsivity 20, 139 – 153.

Neilson, J.C., 2003. Feline house soiling: elimination and marking behaviors. Vet. Clin. North. Am.: Small. Anim. Pract. 33, 287 – 301.

Pageat, P., Gaultier, E., 2003. Current research in canine and feline pheromones. Vet. Clin. North. Am.: Small. Anim. Pract. 33, 187 – 211.

Palm, J., Balogh, O., Reichler, I.M., 2013. The clinical use of Deslorelin acetate (Suprelorin®) in companion animal medicine. Europ. J. Comp. Anim. Pract. 23, 28 – 32.

Plumb, D.C., 2018. Plumb’s Veterinary Drug Handbook. 9th edn. Pharma Vet Inc, Wisconsin.

Rosen, D.K., Carpenter, J.L., 1993. Functional ectopic interstitial cell tumor in a castrated male cat. J. Am. Vet. Med. Assoc. 202, 1865 – 1866.

Rota, A., Paltrinieri, S., Jussich, S., Ubertalli, G., Appino, S., 2008. Priapism in a castrated cat associated with feline infectious peritonitis. J. Fel. Med. Surg. 10, 181 – 184.

Schreiner, L., Kling, A., 1953. Behavioral Changes Following Rhinencephalic Injury in Cat. J. Neurophysiol. 16, 643 – 659.

Swalec, K.M., Smeak, D.D., 1989. Priapism after castration in a cat. J. Am. Vet. Med. Assoc. 195, 963 – 964.

Verrotti, A., D’Egidio, C., Agostinelli, S., Verrotti, C., Pavone, P., 2012. Diagnosis and management of catamenial seizures: a review. Int. J. Womens. Health. 4, 535 – 541.