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Surgical management of follicular cyst concurrent with Cystic Endometrial Hyperplasia (CEH) in a dog

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Abstract

A 3-year-old intact female Indian spitz was presented to Veterinary Gynaecology and Obstetrics, VCC, RIVER with the history of post-mating vaginal bleeding and hyporexia for one month. Physical examination revealed profuse vaginal bleeding and elevated rectal temperature (103.2°F). Exfoliative vaginal cytology predominantly showed superficial cells. Ultrasonography identified uterine endometrial cysts ranging from 3.0 to 3.5 mm, uterine horn diameters of 7.0 and 7.5 mm and a right ovarian cyst measuring 9.0 mm while the left ovary appeared normal. Haemato-biochemical parameters were well within the physiological range. The animal was initially treated for five days with antibiotics, oral stypitics and subsequently underwent ovariohysterectomy. Before surgery, measured serum anti-müllerian hormone (AMH) was 0.44 ng/ml which decreased to 0.13 ng/ml by day 10 post surgery. Similarly, the serum estrogen concentrations declined from 27 to 3 pg/ml. Histopathology confirmed cystic endometrial hyperplasia characterized by haemorrhagic endometrium and hyperplastic glands. Post-operative care with Tab. Amoxicillin and Potassium clavulanate @ 15mg/kg BID, Pantoprazole @ 1mg/kg SID for 10 days and Serratiopeptidase @ 10 mg/kg SID for 3 days, resulted in an uneventful recovery.

Keywords: Canine, cystic endometrial hyperplasia, hormonal assay, ultrasonography

1. Introduction

Cystic endometrial hyperplasia (CEH) is a common reproductive disorder of intact female dogs, particularly in middle-aged to older bitches and is characterized by excessive proliferation of the endometrial lining with cystic dilatation of uterine glands, thereby predisposing the uterus to secondary infections such as pyometra (Jelínková *et al.*, 2022) [5]. The pathogenesis of CEH primarily involves repeated hormonal stimulation during successive estrous cycles, wherein progesterone promotes endometrial glandular secretion, decreases myometrial contractility and suppresses local uterine immunity, thereby facilitating endometrial hyperplasia and bacterial colonization (Rosas *et al.*, 2021) [9]. Follicular cysts are thin-walled, fluid-filled ovarian structures that may be unilateral or bilateral and are hormonally active, frequently producing elevated estrogen concentrations that lead to prolonged estrus behaviour, vulvar swelling, serosanguinous vaginal discharge, alopecia, hyperkeratosis, cystic endometrial hyperplasia and an increased risk of mucometra or pyometra (Johnston *et al.*, 2001) [6]. Persistent follicular cysts arising from anovulatory follicles result in prolonged estrogenic stimulation, and hormonal imbalance, which further enhances endometrial proliferation and accelerates the development of CEH (Gupta *et al.*, 2023) [4]. Although medical management using progesterone receptor antagonists or prostaglandins may be attempted in breeding animals, these therapeutic approaches are often associated with limited success and high recurrence rates; therefore, ovariohysterectomy remains the definitive curative treatment, as it eliminates both the diseased uterus and the ovarian source of hormonal imbalance while preventing life-threatening complications such as pyometra (Fieni., 2014) [2]. Furthermore, elevated and variable serum anti-Müllerian hormone (AMH) concentrations observed in bitches with ovarian cysts reflect persistent ovarian activity, supporting the use of AMH as a valuable biomarker for assessing ovarian status and identifying cystic ovarian disorders in dogs.

Materials and Methods

Case history and observation

A 3-year-old intact nulliparous female Spitz dog, weighing 14.1 kg, was presented to the Small Animal Unit, Department of Veterinary Gynaecology and Obstetrics (VGO), Veterinary Clinical Complex, RIVER, with a history of pro-estral bleeding lasting for 15 days, after which the dog was mated, followed by continuous post-mating vaginal bleeding. The dog also exhibited inappetence for one month and prolonged, persistent estrus behaviour. Physical examination revealed vulvar swelling, increased vaginal bleeding and an pyrexia (103.2°F).

Diagnosis

An accurate diagnosis of CEH relies on a combination of

clinical assessment and advanced diagnostic tools: Exfoliative vaginal cytology predominantly showed superficial cells. While haematological analysis revealed to be normal. On Doppler ultrasonographic examination, the uterus revealed multiple anechoic areas in the endometrium. The uterine horn diameters measured 7.0 mm and 7.6 mm, suggesting the absence of pyometra. Endometrial cysts measuring 3.0 mm, 3.5 mm, and 3.0 mm (Fig.1a) were observed and the uterine wall thickness was recorded as 2.0 mm. The resistive index (RI) in the middle uterine artery was 0.84 (Fig.1b). Additionally, the right ovary exhibited a follicular cyst measuring 9.0 mm (Fig.1c).

Grading

Table 1: Histopathological grading evaluates the microscopic severity of endometrial hyperplasia and cyst formation, whereas ultrasonographic grading assesses *in vivo* uterine morphology and echogenicity, enabling clinicians to correlate imaging findings with histopathological severity and guide therapeutic decision-making

Classification System	Grade/Severity	Characteristic Features
Histopathological Classification	Mild CEH	Mild endometrial hyperplasia with few cystic glands.
	Moderate CEH	Hyperplastic endometrium containing multiple cystic glands.
	Severe CEH	Marked endometrial hyperplasia with numerous cysts; frequently associated with secondary bacterial infection (pyometra).
Ultrasonographic Classification (Rahul Ram, 2024) [8]	Grade A	Anechoic uterine contents without visible endometrial cysts.
	Grade B	Anechoic uterine contents with a few small endometrial cysts.
	Grade C	Hyperechoic uterine contents, hypertrophied endometrium, and multiple large cysts.
	Grade D	Hyperechoic uterine contents with an irregular or atrophied endometrium exhibiting extensive cystic changes.

Biomarkers

Anti-Mullerian Hormone (AMH) and estrogen are valuable biomarkers, as their serum concentrations are significantly elevated in dogs with follicular cyst concurrent with CEH, aiding in early detection and monitoring.

Therapeutic & Surgical Management

Initial stabilisation of the dog involved Tab. Augmentin® @ 15 mg/kg BID, Tab. Pantoprazole @ 1mg/kg SID, and Tab. Etamsylate @ 10 mg/kg BID for 5 days. Later, it was decided to perform ovariohysterectomy. The surgical site (mid ventral) was shaved and aseptically prepared. The animal was premedicated with Inj. Atropine sulphate @ 0.04 mg/kg body weight (bwt), S/C, and Inj. Tramadol @ 4 mg/kg bwt, S/C. Sedation was achieved with Inj. Diazepam @ 0.5 mg/kg bwt, I/V. Anaesthesia was induced and maintained using Inj. Xylazine and Ketamine in a 1:2 combination administered intravenously. The animal was positioned in dorsal recumbency and a sterile surgical drape was placed. As per standard procedure, the mid ventral laparotomy was performed and the uterus along with the ovaries was gently exteriorized through the incision. The ovarian end was held, and the suspensory ligament was carefully broken-down using fingers. At the right ovarian end, a follicular cyst measuring approximately 9.0 mm in diameter was observed (Fig.2) and as an incidental finding, a parovarian cyst measuring 0.7 cm was noted adjacent to the left ovary. Haemostats were placed below and above the ovary. Ligatures were applied using absorbable suture material (size 1-0 catgut), followed by placement of a second ligature and trans fixation. The cut end of the ligatures was checked for bleeding. After confirming haemostasis, the stump was gently released back into the abdominal cavity. The same procedure was repeated for the contralateral ovary. The cervical end was ligated, and the individual uterine arteries were transfixed. The muscle layer along with the peritoneum was closed in a Ford interlocking

suture pattern using vicryl size 1. The subcutaneous layer was closed using 2-0 PGA in a modified simple continuous pattern, to encourage proper apposition of the skin edges. The skin was closed using a horizontal mattress suture pattern from the cranial to the caudal direction. Postoperatively, the dog was administered Tab. Augmentin® @ 15 mg/kg BID for 10 days and Tab. Pantoprazole @ 1 mg/kg SID for 10 days. Supportive therapy included Inj. Meloxicam @ 0.2 mg/kg SID for 3 days and Tab. Serrasil forte® @ 10 mg/kg SID for 3 days for wound healing and pain management. An Elizabethan collar was applied to prevent self-mutilation. The dog was regularly monitored for complications, and surgical wound dressing was performed using 5% povidone iodine solution. The animal had an uneventful recovery without complications on day 10 post surgery.

Results and Discussion

Ultrasonographic examination of the uterus revealed findings consistent with grade (b) cystic endometrial hyperplasia (CEH) (see Table 1), characterized by anechoic uterine contents and the presence of a few small endometrial cysts. Additionally, an ovarian follicular cyst was identified. These imaging findings were subsequently confirmed on histopathological evaluation, which established a diagnosis of moderate CEH. Microscopically, the uterine tissue showed cystically dilated endometrial glands, hyperplastic endometrial epithelium, a cyst wall lined by flattened epithelium and areas of haemorrhagic endometrium with cystic glands (H&E, ×100) (Fig.3). Preoperative hormonal analysis (day 0) demonstrated elevated estrogen levels (27 pg/ml) and detectable anti-Müllerian hormone (AMH) concentrations (0.44 ng/ml), indicating persistent ovarian activity. By the 10th postoperative day following ovariohysterectomy, both biomarkers showed a marked decline, with AMH decreasing to 0.13 ng/ml and estrogen to 3 pg/ml, confirming successful removal of hormonally active

ovarian tissue.

Follicular cysts in intact bitches develop due to persistent anovulatory follicles that continue to secrete estrogen, resulting in prolonged estrogenic stimulation of the endometrium and subsequent progression to CEH. The concurrent occurrence of a follicular cyst and moderate CEH in the present case supports this well-established hormonal pathogenesis. Similar clinical, ultrasonographic, and histopathological findings have been reported by Kumar *et al.* (2019) [7] and Devi *et al.* (2021) [1], who described follicular cyst-associated CEH in bitches with comparable diagnostic features. The elevated preoperative AMH concentration observed in this case aligns with the findings of Gál *et al.* (2024) [3], who reported higher and variable AMH levels in canine females diagnosed with ovarian cysts, reflecting ongoing ovarian follicular activity. The significant postoperative reduction in both estrogen and AMH further substantiates the endocrine origin of the uterine pathology and confirms complete surgical removal of ovarian tissue. Ovariohysterectomy was therefore performed as the definitive therapeutic intervention to eliminate the source of hormonal imbalance and prevent potential progression to pyometra. The favourable postoperative recovery, together with the marked decline in reproductive hormone levels, indicates successful surgical management. Overall, the present findings are in agreement with previously published reports describing similar diagnostic, hormonal, and histopathological trends in bitches affected with follicular cysts associated with cystic endometrial hyperplasia.

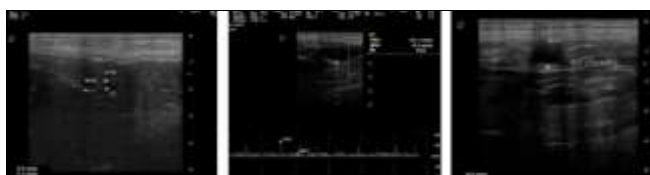


Fig 1a, 1b, 1c: Ultrasonographic examination (preoperative)



Fig 2: Follicular cyst



Fig 3: Histopathology-Hyperplastic epithelium and cystic endometrial glands (H&E x100)

Conclusion

The present case demonstrates that follicular cysts can act as an important hormonal trigger for the development of cystic endometrial hyperplasia in intact bitches. Early diagnostic interventions allow accurate assessment of disease severity. Surgical management proved to be an effective and definitive treatment in the present case.

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Conflict of Interest

Authors declare no conflict of interest.

Author Contributions

Sai Naga Vaishnavi: Conceptualization, case management, surgical procedure, data collection and recording, manuscript drafting, and revision. Manimaran V: Surgical assistance, postoperative case monitoring, and clinical support. G. Shalini and Hemalatha: Ultrasonographic evaluation, interpretation of imaging findings, and contribution to manuscript preparation. S Kantharaj: Corresponding author; supervision of the study, guidance throughout the work, critical review, editing, and correction of the manuscript. K. Murugavel: Overall guidance on the clinical procedure and academic supervision as Dean of the College.

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