Follicular Stasis in Captive Chelonians, *Testudo* spp.

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**Abstract:** This paper reviews normal female chelonian reproductive endocrinology. Clinicopathological data, treatment regimes, and the outcome of 10 presumed cases of follicular stasis are described in captive chelonians, *Testudo* spp. presented over a period of 3 years. Endocrinopathy associated with follicular stasis and some potential causes of follicular stasis are explored.

**Key Words:** Chelonian, anorexia, endoscopy, ultrasonography, follicular stasis, proligestone, ovariectomy.

**INTRODUCTION**

Follicular stasis is where vitellogenesis fails to progress through to ovulation and oviposition, and consequently affects the well-being of the animal (Backues and Ramsay 1994). Follicular stasis appears to be relatively common in captive oviparous lizards (Divers 1996), but is not yet reported in captive chelonians. (Table 1.)

**Endocrinology of normal reproduction in the female tortoise**

*Progesterone*

- In reptiles, a distinct corpus luteum is formed following ovulation, and available evidence suggests that it secretes progesterone (Bentley 1998; Kuchling 1999b).

- There is evidence that progesterone is one of the major steroids synthesized by the chelonian ovary (Klicka and Mahmoud 1977), and there is evidence that the corpus luteum of the chelonian is capable of synthesizing progesterone (Klicka and Mahmoud 1972; Klicka and Mahmoud 1973; Chan and Callard 1974; Callard and others 1976).

- Progesterone was shown to completely inhibit ovulation and reduce pituitary size, oviduct size, and follicle size in the turtle, *Chrysemys picta* (Klicka and Mahmoud 1977).

- Negative feedback inhibition of the release of a pituitary derived trophic hormone is suggested by Bentley (1998). Bentley further proposes that estrogens and progesterone can influence the hypothalamic release of reptilian gonadotropins in a manner similar to mammals.

- A potential physiological role of progesterone in the regulation of clutch size and maintenance of gravidity has been proposed (Klicka and Mahmoud 1977). In *Sceloporous cyanogenys*, Callard and others (1972) propose that progesterone prevents follicular development by both direct action on the hypothalamus and possibly peripherally, resulting in inhibition of vitellogenesis.
• Rostal and others (1998) found that progesterone levels in the Galapagos tortoise, *Geochelone nigra*, displayed a sharp surge during the mating period that coincided with ovulation.

• However, Licht (1984) suggests that various chelonians, including the green sea turtle, *Chelonia mydas*, do not maintain postovulatory levels of progesterone as expected and therefore the role of progesterone in chelonians may vary between species.

**Estrogen**

• In reptiles, estrogen stimulates vitellogenesis, the production of lipoprophosphoproteins by the liver, and their incorporation into the egg (Callard, and others 1972; Licht 1979; Duvall and others 1982; Kuchling 1999b).

• Ovarian maturation and follicular growth in the Galapagos tortoise coincides with elevations in estradiol levels (Rostal and others 1998).

• Ovarian maturation and follicular growth in *Dermochelys coriacea* follows an initial elevation in estradiol levels (Rostal and others 1996).

**Testosterone**

• In *Geochelone nigra*, testosterone levels were elevated during the mating period immediately prior to ovulation. This rise was presumed to relate to receptivity of female (Rostal and others 1998). A similar rise in serum testosterone is observed in *D. coriacea* (Rostal and others 1996) and *Lepidochelys kempii* (Rostal and others 1997). However, both these species demonstrate unique elements of nesting behavior [such as *arribada* and yolkless eggs], and these in turn may relate to unique reproductive endocrinology.

**Gonadotropins**

• The development of the ovary, its secretion of steroid hormones such as estrogen and testosterone, and ovulation, appear to be controlled by follicle stimulating hormone (FSH) or a nonspecific gonadotrophin. Pregnant mare serum gonadotrophin (PMSG), which has primarily FSH activity, was shown by Klicka and Mahmoud (1977) to promote chelonian ovarian growth.

• Mammalian FSH has been shown to induce ovulation in several species of lizards and Bentley (1998) assumes that an endogenous gonadotrophin has this effect in most reptiles. Licht and Papkoff (1974) specifically demonstrated this in *Chelydra serpentina*.

• Bentley (1998) reports that pituitary FSH and luteinizing hormone (LH) have both been identified in reptiles and negative feedback by estrogen on the pituitary gland release of gonadotropins is proposed by Bentley (1998).
Potential factors influencing chelonian vitellogenesis and ovulation

- Light intensity and photoperiod - day length and its rate of change (Bartholomew 1959; Peaker 1969)
- Ambient temperature and its rate of change (Peaker 1969; Licht 1972; Licht 1984; Duval and others 1982; Bentley 1998)
- The influence of pheromones on female chelonians, such as those released from male mental glands (Rose 1969; Winokur and Legler 1975)
- The act of mating (Kuchling and Razandrimamilafinjarivo 1999)
- Further factors such as rainfall, moisture, humidity, nutrition and food supply (Licht 1984; Duval and others 1982)
- Potentially influenced by endogenous factors such as the balance of progesterone, testosterone and estrogen and/or thyroid hormones; as in avian species (Millam 1977)

Is follicular stasis in chelonians a genuine pathological condition?

To date a diagnosis of chelonian follicular stasis remains presumptive and it is therefore a diagnosis of exclusion. This underlines the importance of a full clinical examination and a thorough assessment of the anamnesis. As follicular stasis is a chronic disease, all efforts should be made to rule out other concurrent disease.

It is unclear if follicular stasis is merely a secondary sign of other health problems, or a specific condition in its own right. There is an apparent correlation between prolonged folliculogenesis, hepatic lipidosis, chronic anorexia, and nonspecific illness. The author believes that follicular stasis is a condition in its own right. It relates to an endogenous, physiological need for partial or complete induction of ovulation through mental gland or other pheromones and contact (mating, butting or biting) from a male tortoise. Therefore, a failure of induced ovulation by restricted exposure to suitable male tortoises results in follicular stasis and disease.

Prolonged duration of reproductive arrest while at an advanced stage of follicular development, visualization of a large number of advanced follicles within each ovary, and prolonged anorexia, all suggest follicular stasis. However, there is limited reproductive data available to define what is “normal” with respect to ovarian cycles in captive female chelonians. This is especially true of Testudo spp. It is unclear that it is valid to extrapolate hormonal and other reproductive findings from different chelonian species. Ultimately, until there is a larger database of “normal” reproductive physiological data for each species encountered, the diagnosis/condition of stasis must remain presumptive. This paper hopes to reveal data that might help to validate follicular stasis as a plausible diagnosis in future, especially with respect to captive Testudo spp.

DIAGNOSIS

History/Anamnesis

All aspects of husbandry and nutrition should be investigated. Cases presented to this author have invariably been isolated, mature, female tortoises, often they have been provided with low standards of nutrition and husbandry. Follicular stasis appears to be a chronic disease. Signs might arise months after onset of the disease; consequently this disease does not appear to show strong seasonality. Cases have been presented in summer, autumn, winter, and spring.
Clinical signs

Lethargy, anorexia, abdominal distension, and weight gain have been reported in lizards with follicular stasis (Backues and Ramsay 1994; Divers 1996). Cases eventually diagnosed as in follicular stasis, are generally presented with prolonged anorexia and absence of feces. The keeper commonly reports total anorexia and an absence of feces for periods of 3-7mo.

Clinical pathology

Differentiation between periods of normal follicular development and follicular stasis is not possible using a single blood sample. Trends over time must be represented and an adequate database of normal information in captive Testudo spp. is not yet available.

Hypercalcemia (elevations in total calcium), hyperalbuminemia, and elevations in total protein occur in females undergoing vitellogenesis. It is possible that ionized calcium levels are simultaneously subnormal.

- Hypercalcemia occurred in 8/10 cases
- Hyperalbuminemia occurred in 7/9 cases
- Elevated total protein occurred in 7/9 cases
- Alkaline phosphatase was elevated in 8/8 cases
- Moderate anemia occurred in 7/7 cases
- Leukopenia occurred in 5/7 cases
- Heteropenia occurred in 5/7 cases

Cases that have become chronically anorexic, are in concurrent liver failure, or have concurrent metabolic bone disease may not show all of these changes. These findings presumably relate to hyperestrogenism and/or starvation. If the follicular stasis is uncomplicated, other hematological and biochemical parameters may be within normal ranges.

Wilkinson (2000) suggests the disease may also be characterized by acidic urine (Innis 1997) and raised blood and urine β-hydroxybutyrate [Ketosis] (Christopher and others 1994), as these changes are consistent with catabolism in herbivores.

Cholesterol and triglyceride levels may become elevated during vitellogenesis (Derikson 1979). Normal levels are poorly defined. However, similar elevations might also occur during hepatic lipidosis, which appears to be intimately associated with follicular stasis, and numerous other conditions causing anorexia in chelonians.

Estrogen, testosterone and other hormone assays may prove to be of future use.

Ultrasonography

Data on the ultrasonography assessment of normal annual follicular cycles of UK captive female Testudo spp. is limited. Several breeding and nonbreeding females have been monitored using serial ultrasonography. During late autumn and spring, moderate numbers of follicles (generally less than 20), 15-22mm in diameter, are considered to be normal in mature female Testudo spp. Where the numbers of medium sized follicles (5-15mm), and large follicles (15-22mm), become in excess of
50, follicular stasis or ovulatory failure should be suspected. If sequential ultrasonography in *Testudo* spp., over several weeks, demonstrates >20 mature ovarian follicles, 15-22mm diameter, follicular stasis is suggested. In cases of follicular stasis, some follicles may not seem to progress to ovulation, but might regress and become resorbed.

Ultrasonography supported the diagnosis in 4/5 cases diagnosed. One animal did not have adequate anatomical access to the prefemoral acoustic window to allow scanning.

**Endoscopy**

Endoscopy examination of mature females in follicular stasis may reveal ovaries with 20-50 follicles, 15-22mm diameter. It is not easy to differentiate between normal folliculogenesis and stasis from a single examination. An accurate count of developed coelomic follicles is often impractical.

Endoscopy supported the diagnosis in 8/10 cases.

**Exploratory coeliotomy**

Exploratory coeliotomy in cases of follicular stasis will reveal a coelomic cavity packed with 50-200 follicles, 15-22mm diameter. It is hard to differentiate between normal ovarian activity and follicular stasis without further supportive history and evidence that follicular progression was absent.

**TREATMENT AND PREVENTION**

Follicular stasis has been reported to regress in lizards (Backus and Ramsey 1994), but it is unknown if spontaneous resolution might also occur in chelonians. Surgical and medical treatments utilized all appear to have both advantages and disadvantages. These include improvements in environment, nutrition, and social interactions wherever possible, and specific medical or surgical treatment aimed at resolving inappropriate ovarian activity.

**Improvements in environment**

Photoperiod and temperature management should be assessed and formalized. All chelonians should be given a suitable annual photo period cycle (Jones 1978).

**Improvements in nutrition**

Potential nutritional disorders should be identified and managed. Hepatic lipidosis is a consistent finding in cases of reproductive stasis. Cases should be managed as though concurrent hepatic lipidosis is present.

**Social interactions**

Tortoise ovulation may be induced by the presence of a suitable male tortoise. Discourage keepers from maintaining mature tortoises as isolated females. It is unknown if a meeting with a male tortoise, or a mating, will stabilize female reproduction. Females that are mixed with appropriate males every 2-3yr may maintain normal reproductive function better than those that are not.
Medical treatment

Rosskopf and Woerpel (1983) used medoxyprogesterone acetate at a dose of 22mg/kg body weight to suppress ovarian activity in the Californian desert tortoise, Geochelone agassizii, and 20mg/kg by Bennett (1998). Frye (1991) suggests that twice yearly dosing with a mammalian dose of medoxyprogesterone acetate will completely suppress reptilian follicular activity.

Proligestone (Delvosterone; Intervet) has been given at 20mg/kg. It successfully reduced follicular activity in a Testudo hermanni diagnosed as having follicular stasis.

No adverse effects are described in the literature as a result of medical suppression of chelonian follicular activity using progestogen, but adverse effects seem plausible. Cases suspected to have follicular stasis are also likely to have hepatic lipidosis. The return of a large amount of follicular material to a compromised liver, following progestogen therapy and successful follicular resorption, may be detrimental to health. Potential interactions between insulin and progesterone might result in diabetes. In some cases of follicular stasis in Testudo spp., repeated progesterone therapy has not stimulated follicular regression. Subsequent ovarioectomy was required.

Thyroid hormone appears necessary for normal ovarian regression in birds. Supplementation in thyroidectomized starlings induced photorefractoriness and induced gonadal regression (Millam 1997). Therefore it is plausible that thyroid function may also influence efficacy of proligestone therapy in chelonians. The relationship between testosterone levels and ovulation is also poorly understood, and it is similarly plausible that circulating levels of other hormones such as testosterone might influence proligestone efficacy.

A conclusive outcome regarding the efficacy of proligestone has not yet been determined, but proligestone appears better able to suppress ovarian activity in early cases or in juveniles reaching puberty, and it may therefore prove a suitable drug to prevent the condition, especially in isolated juvenile females.

Surgical treatment

Follicle aspiration was suggested as a method of treating pre-ovulatory egg binding in a green iguana, Iguana iguana, by Orosz and others (1992). The high probability of complications such as egg peritonitis, as well as anatomical restrictions that result from the chelonian shell, make follicle aspiration unsuitable in most presentations of follicular stasis in chelonian.

Backus and Ramsey (1994) suggested ovarioectomy for the treatment of follicular stasis in lizards. Assuming there is no intention of breeding, several authors including Backus and Ramsey (1994), Raiti (1995), and Divers (1996), describe ovarioectomy/ovariosalpingectomy as the treatment of choice in isolated, anorexic, female lizards. Ovariosalpingectomy in tortoises has been described (Holt 1979; Müller and others 1989; Bennett 1993; Divers 1997). It appears to be a suitable method of managing utero-ovarian disease in chelonians. However it should be remembered that there might be potential long-term complications, such as osteoporosis, associated with spaying chelonians. Long-term follow up studies have yet to be performed on chelonians undergoing ovarioectomy.

All surgical cases in this series (excluding the single death) showed significant improvement following ovarioectomy. All required a prolonged period of nursing of 6-12mo. All cases improved and ate in the spring following surgery. These tortoises were not hibernated the winter after spaying.
Esophagostomy tube feeding was necessary in 1 case for 5 mo. Recovery in many cases of apparent follicular stasis treated by ovariectomy has been prolonged when compared to similar iguana cases. This may relate to the chronic nature of these cases.

DISCUSSION

Potential causes of follicular stasis

Absence of environmental cues

- Environmental cues such as hibernation, photoperiod, and thermoperiod may influence folliculogenesis (Peaker 1969; Licht 1972; Licht 1984; Duvall and others 1982; Bentley 1998). It is possible that an appropriate territory and nesting area must be present before ovulation may occur. Animals that are not permitted to follow an annual cycle of changing photoperiod and changing daily ambient temperatures, may lose their natural breeding cues and fail to ovulate. Jones (1978) gives photoperiod tables appropriate to Testudo spp.

- In the UK, the weather is unsuitable for most reptiles. Consequently, it is customary to keep tortoises indoors in enclosures with supplementary heat and light, especially during spring and autumn. It is possible that under the influence of a human waking cycle, breeding cues are deranged.

Loss of social cues

- Ovulation may be induced, in some way, by the presence of a male tortoise. This could involve pheromones, male courtship behavior such as butting and biting, or the act of mating. Follicular stasis could be the result of a failure of induced ovulation. Regular socialization of females with an appropriate male tortoise might reduce the prevalence of follicular stasis. A similar hypothesis is proposed by Backues and Ramsay (1994) in oviparous lizards. Galbraith (1993) implies that chelonians retain the ability to perform fertile ovulations some time after a successful encounter with a male in some species.

- Both Jessop (2000) and Wilkinson (2000) believe that some cases of follicular stasis have been previously isolated females experiencing temporary contact with males (e.g., whilst the keeper is on holiday). These cases appear to have gone into follicular stasis when returned into isolation once more.

- Isolated female chelonians might revert to a physiological state requiring induced ovulation, but, following successful contact with a male, may then ovulate unaided for several years once more.

Chronic nutritional disease, systemic illness, and old age

- Follicular stasis may be a secondary condition, where female tortoises are maintained inappropriately, suffer from any chronic illness, or endocrinopathy.

- Most females with signs consistent with follicular stasis are poorly maintained. Most are kept without adequate heat, photoperiod, balanced nutrition, and with limited exposure to male tortoises. Many may be very old and beyond useful breeding age.
CONCLUSIONS

Follicular stasis appears to be a relatively common condition of isolated mature female *Testudo* spp. in the UK. It should be considered one potential cause of prolonged anorexia in those mature female tortoises prevented from regular exposure to suitable males.

Often mature female chelonians in a state of apparent follicular stasis will have failed to pass feces for periods in excess of 3mo. Occasionally, hind limb function will be poor. Typical clinicopathological features may include hypercalcemia, hyperalbuminemia, elevated total protein, moderate anemia, leukopenia, heteropenia, lymphopenia, and elevated alkaline phosphatase. Ultrasonography and endoscopy may reveal the presence of large numbers of well-developed coelomic follicles that fail to either regress or ovulate with time.

Ovariectomy would presently appear to be the most effective form of treatment, especially in a nonbreeding situation. An effective medical treatment requires further investigation. Future consideration should be given to the suppression of reproductive function in isolated, captive, females at, or immediately after, puberty.

Chelonians in a state of prolonged follicular stasis suffer a major metabolic insult. In many cases significant recovery is either unlikely, or at best it will be prolonged. Early intervention would therefore appear beneficial but chronic cases merit a guarded prognosis.

Impaired hind limb function may not improve in the immediate post treatment period. Several cases have remained unable to make voluntary hind limb movements 2-3yr post treatment despite an otherwise good recovery.

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REFERENCES*


Lewis W. 2000. Personal Communication. Southbeech Veterinary Surgery


*not all references cited in text; some are provided for additional reference*
<table>
<thead>
<tr>
<th>Presented Species</th>
<th>Species</th>
<th>Weight</th>
<th>Presenting signs</th>
<th>Others in contact</th>
<th>Previous reproduction</th>
<th>Hematology</th>
<th>Biochemistry</th>
<th>Radiology</th>
<th>Endoscopy</th>
<th>US</th>
<th>Follicles are</th>
<th>Medical treatment</th>
<th>Surgical treatment</th>
<th>Management</th>
<th>Meds</th>
<th>Other Outcome</th>
<th>(Time post op)</th>
<th>Other Information</th>
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<tbody>
<tr>
<td>Ben H</td>
<td>F. whitei</td>
<td>2885g</td>
<td>Prolonged anorexia</td>
<td>Weak hind limbs</td>
<td>Never laid eggs</td>
<td>Borderline anemia</td>
<td>Leukopenia</td>
<td>Large FB</td>
<td>Yes</td>
<td>Yes</td>
<td>Follicles present</td>
<td>Spayed 05/10/98</td>
<td>Liver - lipidotic</td>
<td>E-tube 4 mo</td>
<td>Soloxine</td>
<td>Ate 08/04/99</td>
<td>6 mo</td>
<td>Weak hind limbs</td>
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<tr>
<td>Tortie R</td>
<td>T. hermanni</td>
<td>1850g</td>
<td>Prolonged anorexia</td>
<td>Death of in contact</td>
<td>Never laid eggs</td>
<td>Borderline anemia</td>
<td>Hypercalcemia</td>
<td>NAD</td>
<td>NAD</td>
<td>Yes</td>
<td>Follicles present</td>
<td>Spayed 02/12/98</td>
<td>Liver - lipidotic</td>
<td>Peri-operative antibiotics and analgesics</td>
<td>Metacam</td>
<td>Ate mid 27/04/99</td>
<td>5 mo</td>
<td>Poor vision persisted</td>
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<td>Timml F</td>
<td>T. graeca</td>
<td>2055g</td>
<td>Prolonged anorexia</td>
<td>Isolated 30+ yr</td>
<td>Never laid eggs</td>
<td>Borderline anemia</td>
<td>Hyperalbuminemia</td>
<td>NAD</td>
<td>NAD</td>
<td>Yes</td>
<td>Follicles present</td>
<td>Spayed 22/08, 28/09 and 20/10</td>
<td>Liver - lipidotic</td>
<td>Critical Care Formula</td>
<td>Soloxine</td>
<td>Ate 28/02/00</td>
<td>3 mo</td>
<td>Appears normal</td>
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<td>MP</td>
<td>T. hermanni</td>
<td>1730g</td>
<td>Prolonged anorexia</td>
<td>Isolated 16 yr</td>
<td>Never laid eggs</td>
<td>Borderline anemia</td>
<td>Hyperalbuminemia</td>
<td>NAD</td>
<td>NAD</td>
<td>No</td>
<td>Follicles present</td>
<td>Spayed 20/11/99</td>
<td>Liver - lipidotic</td>
<td>Proligestone</td>
<td>Metacam</td>
<td>Died ~1 week</td>
<td>N/A</td>
<td>Viral stomatitis?</td>
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<td>HGO P</td>
<td>T. graeca</td>
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<td>Prolonged anorexia</td>
<td>Absence of feces</td>
<td>Isolated 24+yr</td>
<td>Borderline anemia</td>
<td>Hyperalbuminemia</td>
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<td>NAD</td>
<td>No</td>
<td>Follicles present</td>
<td>Unspayed</td>
<td>Liver - lipidotic</td>
<td>Peri-operative antibiotics and analgesics</td>
<td>Soloxine</td>
<td>Ate 08/04/99</td>
<td>6 mo</td>
<td>Weak hind limbs</td>
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Table 1 continued. Clinical data from 10 cases of follicular stasis in *Testudo* spp.

<table>
<thead>
<tr>
<th>Presented</th>
<th>Toby R 21/10/99 Spur thigh (white's)</th>
<th>Elizabeth 01/09/97</th>
<th>Toby L N/A</th>
<th>Big BR N/A</th>
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<td>Species</td>
<td><em>T. graeca</em> mature</td>
<td><em>T. hermanni</em> mature</td>
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<td>N/A</td>
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<td>Weight</td>
<td>3620</td>
<td>2125</td>
<td>1880</td>
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<td>Presenting signs</td>
<td>Declining appetite (3yr) Oxytocin induced lay, 9/96 Anorexia Sept/Oct</td>
<td>Prolonged nasal discharge Poor appetite anorexia from 2/99 Annual male contact 2 wks/yr Contact stopped 94.</td>
<td>Owner thought blind</td>
<td>Male was present Isolated 40+ yr</td>
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<tr>
<td>In contact data</td>
<td>Isolated 40+ yr</td>
<td>(Other died '87)</td>
<td></td>
<td></td>
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<tr>
<td>Previous reproduction</td>
<td>Never laid eggs</td>
<td>Never laid eggs</td>
<td>Laid eggs last yr</td>
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<td>Hematology</td>
<td>Not done</td>
<td>Marked toxic heterophils</td>
<td>Lymphopenia. Reactive lymphocytes</td>
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<td>Biochemistry</td>
<td>Hypercalcemia</td>
<td>Hypercalcemia</td>
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<tr>
<td></td>
<td>Hyperalbuminemia</td>
<td>Hyperalbuminemia</td>
<td>Hypercalcemia</td>
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<td></td>
<td>Hypouricemia</td>
<td>Raised urea, creatinine</td>
<td>Hyperalumininemia</td>
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<td></td>
<td>Hyperphosphate</td>
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<tr>
<td>Radiology</td>
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<td>Slight radiodensity R ovarian quadrant in</td>
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<td>US</td>
<td>NAD</td>
<td>endoscopic confirmation</td>
<td>endoscopic confirmation</td>
<td>endoscopic confirmation</td>
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<td>Follicles are &gt;10mm</td>
<td>Spayed 5/11/99</td>
<td>Spayed 17/3/99</td>
<td>Spayed 30/09/97</td>
<td>Spayed Nov 99</td>
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<tr>
<td>Treatment</td>
<td>Liver lipidotic/ fibrotic (histo)</td>
<td>Liver not biopsied</td>
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<td>Management</td>
<td>Environmental only</td>
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<td></td>
<td></td>
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<td></td>
<td></td>
<td></td>
<td>Recovery seen 2mo post op</td>
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<tr>
<td>Outcome</td>
<td>Ate 8/11/99 3days</td>
<td>Ate 27/4/99 5-8wk</td>
<td>Ate 7/11/97 38 d</td>
<td>Ate ~ 10d post op</td>
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