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YOLK COELOMITIS IN FIJI ISLAND BANDED IGUANAS (*BRACHYLOPHUS FASCIATUS*)

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Abstract: Yolk coelomitis is a major cause of death in captive sexually mature female Fiji Island banded iguanas (*Brachylophus fasciatus*) maintained by the Zoological Society of San Diego. The medical records, breeding histories, and pathology archival materials from this group were reviewed to study this health problem. From 1987 through 2004, deaths of nine of 21 adult females were due to yolk coelomitis. Most iguanas had a history of reproduction-related problems, which included reproductive failure, episodes of lethargy associated with ovarian activity, folliculostasis, ovostasis, and behavioral abnormalities. Most affected iguanas either were found dead or presented moribund and subsequently died or were euthanized. Clinical signs were nonspecific and included lethargy, cutaneous discoloration, and coelomic effusion. Yolk leakage in most cases was associated with the presence of large vitellogenic follicles undergoing atresia and resulted in coelomitis characterized by florid mesothelial proliferation.
Key words: Atresia, Fiji Island, follicle, iguana, reproduction, proliferative, yolk coelomitis.

INTRODUCTION

The Fiji Island banded iguana or Fijian banded iguana (*Brachylophus fasciatus*) is one of two endangered species of iguana that inhabit the Fiji Islands. These species are the most geographically isolated of all iguanids. Fijian banded iguanas are highly arboreal, and they are rarely encountered in the wild.[8] As of 1993, the wild population was estimated to be less than 10,000 individuals and this number considered to be in decline.[13] The major threats to the wild population are feral predators and habitat destruction.[13]

Fijian banded iguanas are maintained in captivity by 14 North American zoological institutions. Females lay their first clutch at approximately 16 to 18 mo of age; however, fertility remains low until iguanas are 3 to 4 yr old.[15] Captive females may lay eggs any month of the year, but most clutches are laid between April and July in San Diego, California.[15] Although some animals occasionally lay two clutches per year, a single annual clutch is more common.[15] Clutch size ranges from two to six eggs, with most females producing three to five eggs.[15]

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The Zoological Society of San Diego (ZSSD) has maintained a successful breeding colony of Fijian banded iguanas since 1987. Since 1991, nine mature female Fijian banded iguanas at the ZSSD have been diagnosed with yolk coelomitis characterized by extensive mesothelial proliferation covering the reproductive tract and other visceral organs. The hyperplastic, reactive mesothelium had atypical cytological features and resembled granulosa cells of vitellogenic follicles undergoing atresia and reactive ovarian surface epithelium. Thus, confident tissue identification and orientation were difficult in some cases, and initial differential diagnoses included neoplasia based on cytology and surgical biopsies.

Yolk coelomitis is a well-recognized problem and a significant cause of death in captive adult female lizards. There are, however, very few published reports and little attention has been given to the pathogenesis(es) behind reproductive disorders in reptiles. This series of cases prompted a retrospective study of yolk coelomitis in Fijian banded iguanas that included clinical and breeding histories, necropsy, microdissection of ovaries from archived specimens, histopathologic evaluation, immunohistochemistry, and transmission electron microscopy. The objectives of this study were to 1) identify any consistent or predictive abnormalities in reproductive activity, 2) characterize the ovarian histologic lesions, and 3) evaluate the morphologically similar reactive mesothelium and ovarian granulosa cells for diagnostically useful immunohistochemical and ultrastructural features.

MATERIALS AND METHODS

Breeding pairs and unpaired adults were housed in 1.5- × 1.5- × 1.8-m screen enclosures, and they were provided with branches for climbing, regular misting as a water source, and frequent outdoor access to natural sunlight. Animals were maintained at 26.7°C to 31.1°C during the day and 22.2°C to 26.7°C at night. Large covered nest boxes (minimum, 46.0 × 46.0 × 46.0 cm) constructed from Tupperware™ (Tupperware, Wooster, Ohio 44691, USA) containers were used. A 10.0-cm square hole was provided in the top for an entrance. Nesting substrate consists of well-packed potting soil, and containers were filled to within 5.0 cm of the lid.

Mortality records, pathology reports, medical records, and breeding histories were reviewed for deaths of female Fijian banded iguanas occurring at the ZSSD from 1985 through 2004. Yolk coelomitis cases were identified based on gross and histopathologic diagnosis of intracoelomic yolk leakage associated with inflammation and mesothelial hyperplasia. Formalin-fixed tissues were processed for each case by routine methods to yield 5-μm paraffin wax sections that were stained with hematoxylin and eosin. Sections of ovary were evaluated for stage of follicular development and presence of intraovarian and intracoelomic yolk leakage. Special attention was given to morphology of granulosa cells, ovarian surface epithelium, and coelomic mesothelium. Select sections of ovary and the proliferative coelomic mesothelium from two Fijian banded iguanas were stained with low molecular (acidic) weight (AE1) and high molecular (basic) weight (AE3) cytokeratins (Biogenex, San Ramon, California 94583, USA). Ovary from a green iguana (*Iguana iguana*) with yolk leakage and a normal green iguana ovary and oviduct were used as controls based on previous experience with these antibodies.[27] Sections of the wall of an atretic follicle, which included granulosa cells, and the proliferative coelomic mesothelium of one Fijian banded iguana were further evaluated by transmission electron microscopy at the California Animal Health and Food Safety Laboratory.

RESULTS

Mortality review and clinical cases

Over the 19 yr of this retrospective study, 32 sexually mature Fijian banded iguanas have been submitted for necropsy, 21 of which were female. Nine of these females were diagnosed with yolk coelomitis. These cases included one individual (8) suspected to be the offspring of hybrid parents (*B. fasciatus*/*B. vitiensis*). The ages of affected animals ranged from 1 yr, 11 mo to 8 yr, 5 mo. With the exception of the single 2 yr old, the remaining animals were of prime reproductive age (4–8 yr). Other causes of mortality in mature animals included enteritis, septicemia, enteric volvulus and torsion, pentastomiasis, entamoebiasis, recurrent cloacal prolapse, neoplasia, congenital anomalies, and idiopathic pulmonary hemorrhage.

The most common clinical presentations of yolk coelomitis were dark discoloration of the skin and lethargy ($n = 6$) and coelomic distention ($n = 3$). Five iguanas had no medical history before the episode of presenting signs and death, and previous problems in an additional animal were limited to a minor traumatic injury. Four iguanas were found dead with no premonitory signs (1, 6, 8, and 9). Most affected iguanas that were alive upon presentation died shortly thereafter, or they were euthanized due to poor prognosis (4 and 7). One iguana died from complications during an attempted ovariohysterectomy (5). Hematologic and biochemical data at the time of presentation were available for three animals. Because reference intervals are not available for Fijian banded iguanas, data were assessed using ranges reported in green iguanas (given in brackets).[12] Case 1 had hypercalcemia (30.0 mg/dl [10.8–14.0 mg/dl]) and hyperphosphatemia (20.4 mg/dl [2.8–9.3 mg/dl]), elevated aspartate aminotransferase (AST) activity (90.0 U/L [7.0–102 U/L]), hypoglycemia (69.0 mg/dl [105–258 mg/dl]), and hyperuricemia (14.0 mg/dl [0.9–6.7 mg/dl]). A complete cell count could not be performed due to clot formation; however, phagocytized bacilli were observed within many monocytes. Case 3 had elevated phosphorus (15.0 mg/dl), hyperuricemia (9.5 mg/dl), hypoproteinemia (2.6 g/dl [4.9–7.6 g/dl]), hypoalbuminemia (1.2 g/dl [1.5–3.0 g/dl]), and monocytosis ($5.7 \times 10^3/\mu l$ [$0.4–2.3 \times 10^3/\mu l$]). Case 7 had mild hyperuricemia (7.2 mg/dl), hypoproteinemia (2.3 g/dl), hypoalbuminemia (1.1 g/dl), and severe leukocytosis ($114.0 \times 10^3/\mu l$ [$8.2–25.2 \times 10^3/\mu l$]) characterized by heterophilia ($84.4 \times 10^3/\mu l$ [$0.6–6.4 \times 10^3/\mu l$]) with a left shift ($11.4 \times 10^3/\mu l$) and toxic changes, as well as monocytosis ($11.4 \times 10^3/\mu l$).

Reproductive histories

Breeding histories and any reproduction-related medical history were obtained for all affected iguanas (Table 1). All iguanas were housed in pairs with males and all but three had produced clutches in previous years. Exceptions included a 2-yr-old nulliparous female, which likely was in her first year of reproductive maturity, and two nulliparous older females, which is unusual for this species in the

Table 1. Fiji Island banded iguanas with yolk coelomitis: age, reproductive tract status, breeding history, and reproductive abnormalities.

Case no.	Age	Follicle stage[a]	Oviduct status[b]	Breeding history	Previously diagnosed folliculostasis/ ovostasis	Malaise associated with reproductive cycle	Abnormal eggs (unshelled or slugs)
1	5 yr, 4 mo	A	SE	Nulliparous	No	No	No
2	4 yr, 1 mo	A	NE	Multiparous	No	No	No
3	4 yr, 7 mo	LV, A	NE	Nulliparous	No	Yes	No
4	8 yr, 5 mo	A	SE, UE	Multiparous	Ovostasis	No	Yes
5	5 yr, 7 mo	LV, A	SE	Multiparous	Ovostasis	No	Yes
6	1 yr, 11 mo	LV	SE	Nulliparous	No	No	No
7	6 yr, 4 mo	A	NE	Multiparous	No	Yes	Yes
8	6 yr, 8 mo	A	NE	Multiparous	No	No	Yes
9	8 yr, 5 mo	LV, A	NE	Multiparous	Folliculostasis	Yes	Yes

[a] A, atretic follicles; and LV, late vitellogenesis.
[b] SE, shelled egg; UE, unshelled egg; and NE, no eggs.

experience of one of us (JK). One iguana (2) had produced a single clutch, and it had an unremarkable reproductive history before death. Of the six iguanas that had laid eggs, reproduction-related abnormalities were documented in five (4, 5, 7, 8, and 9). Two iguanas (4 and 7) failed to lay eggs in the previous year. One of these animals (7) had a period of lethargy that may have been associated with reproductive activity, and the other (4) had undergone two salpingotomies 5 yr before death to treat egg stasis. The latter animal had only produced abnormal unshelled eggs since the salpingotomies. Two iguanas (8 and 9) had a history of persistent digging after oviposition, including one animal (9) that exhibited lethargy associated with follicular and egg development for three consecutive years. This iguana produced three small clutches in a single season 2 yr before death and a single unshelled egg the season before death. In addition, resorption (atresia) of vitellogenic follicles was documented by ultrasonography in this animal during a season in which no eggs were produced and during an episode of lethargy. An additional iguana (5) had a history of egg stasis attributed to kyphosis of the caudal vertebral column. Three iguanas had produced unshelled eggs or ''slugs'' in previous years (4, 8, and 9). Only one affected iguana (4) had produced fertile eggs that resulted in offspring; however, eggs from an additional animal were never incubated due to captive management protocol.

Figure 1. Fijian banded iguana. The entire coelomic cavity is filled with yolk debris and serosal surfaces are covered by a thick layer of reactive mesothelium. Bar = 1.0 cm.

Gross and histopathologic examination

On gross examination, the coelomic cavities of all affected iguanas contained various amounts of globular yellow-to-white friable material, consistent with yolk, immersed in opaque, yellow, often red-tinged fluid (Fig. 1). Similar material was adhered to coelomic viscera. The serosal surfaces of multiple organs, including the ovaries, oviducts, liver, lungs, pericardium, spleen, and pancreas, as well as the body wall, were multifocally or diffusely covered by yolk debris overlying thick mats of hyperplastic mesothelium that measured 0.1 cm to 0.5 cm in thickness and had a prominently villous surface. Extensive adhesions between organs and the body wall were noted in most cases. The ovaries contained follicles in various vitellogenic and previtellogenic stages of development, including one to multiple large red–yellow or dark orange late vitellogenic or atretic follicles. Collapsed vitellogenic follicles that seemed to be ruptured were observed

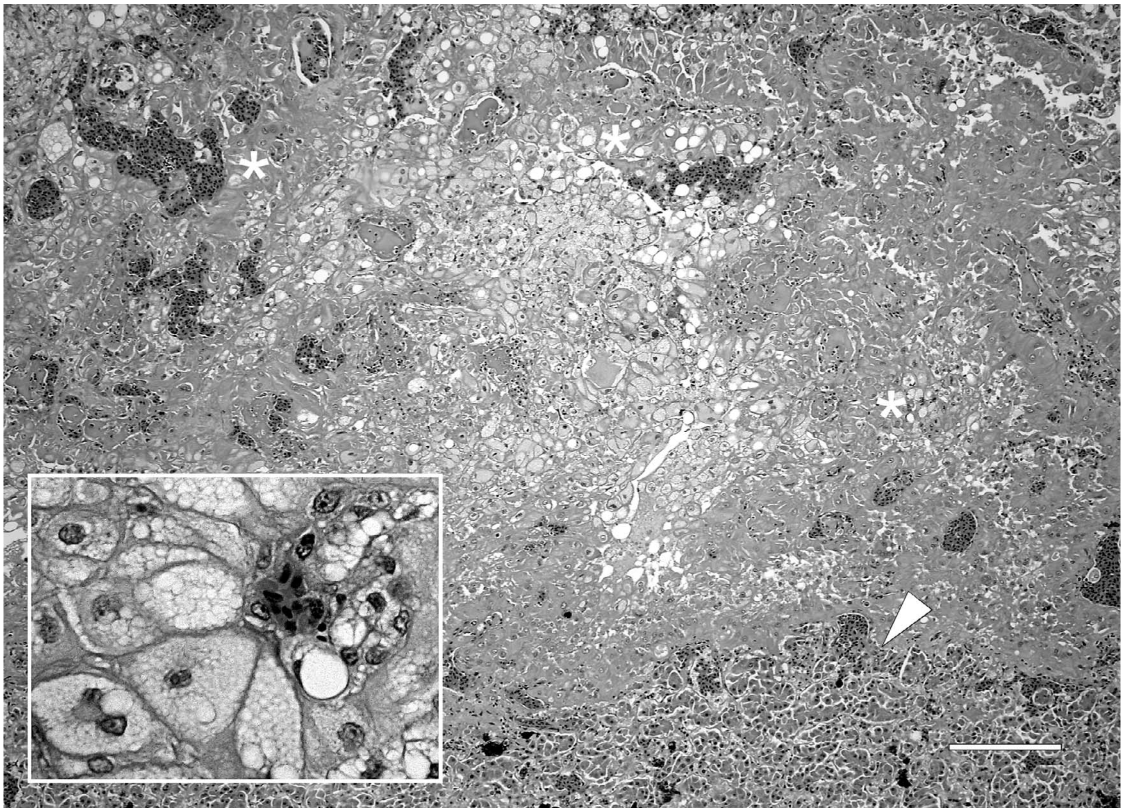

Figure 2. Fijian banded iguana, liver: the capsular surface of the liver (white arrowhead) is covered by a thick layer of reactive, hyperplastic mesothelial cells (asterisks). In the inset, the cells palisade around supportive vessels and binucleated cells are observed. Hematoxylin and eosin. Bar = 100 μm (20 μm in inset).

in seven cases. Eggs were observed in the oviducts of four iguanas (1, 4, 5, and 6), and the oviducts in some of these cases were thickened and congested. None of the eggs were ruptured. Two iguanas were in poor body condition, and one animal had concurrent pulmonary pentastomiasis. Secondary bacterial coelomitis was documented in five cases (1, 4, 6, 8, and 9). Bacterial isolates (aerobic only) included *Salmonella* sp., *Salmonella arizonae*, *Acinetobacter baumanii*, *Klebsiella pneumoniae*, *Klebsiella oxytoca*, *Pseudomonas aeruginosa*, and *Aeromonas* sp. Infections included mixed Gram-negative species in two cases (8 and 9).

Histologically, serosal surfaces of most coelomic visceral organs, especially the liver, spleen, pancreas, mesentery, and body wall, were covered by hyperplastic, hypertrophic mesothelium that palisaded or formed thick sheets and lined papillary projections supported by a thin fibrovascular stroma (Fig. 2). The mesothelial cells seemed to pile into multiple layers, and they were characterized by moderate-to-abundant eosinophilic to vacuolated cytoplasm, distinct cell borders, and oval-to-round euchromatic nuclei with distinct nucleoli. Scattered amphophilic to eosinophilic globules of various sizes, consistent with yolk, were within the cell cytoplasm or on the tissue surface. Various degrees of anisocytosis and anisokaryosis, as well as binucleated cells, were observed in some areas. Examination of numerous sections did not reveal any evidence of tissue invasion. Multifocally, moderate numbers of inflammatory cells, including macrophages, heterophils, and lymphocytes infiltrated the supporting stroma and proliferative mesothelium. Although five cases yielded positive bacterial cultures, bacteria only were observed in two cases (4 and 9).

Within the ovary, large follicles filled with yolk globules and lakes of eosinophilic fluid and lined by stratified or pseudostratified layers of vacuolated granulosa cells, consistent with follicular atresia, were observed in eight cases (all except case 6) (Fig. 3). Available ovary sections were very limited for case 6; thus, follicular atresia may have been missed in this animal. In all cases, extrafollicular free yolk

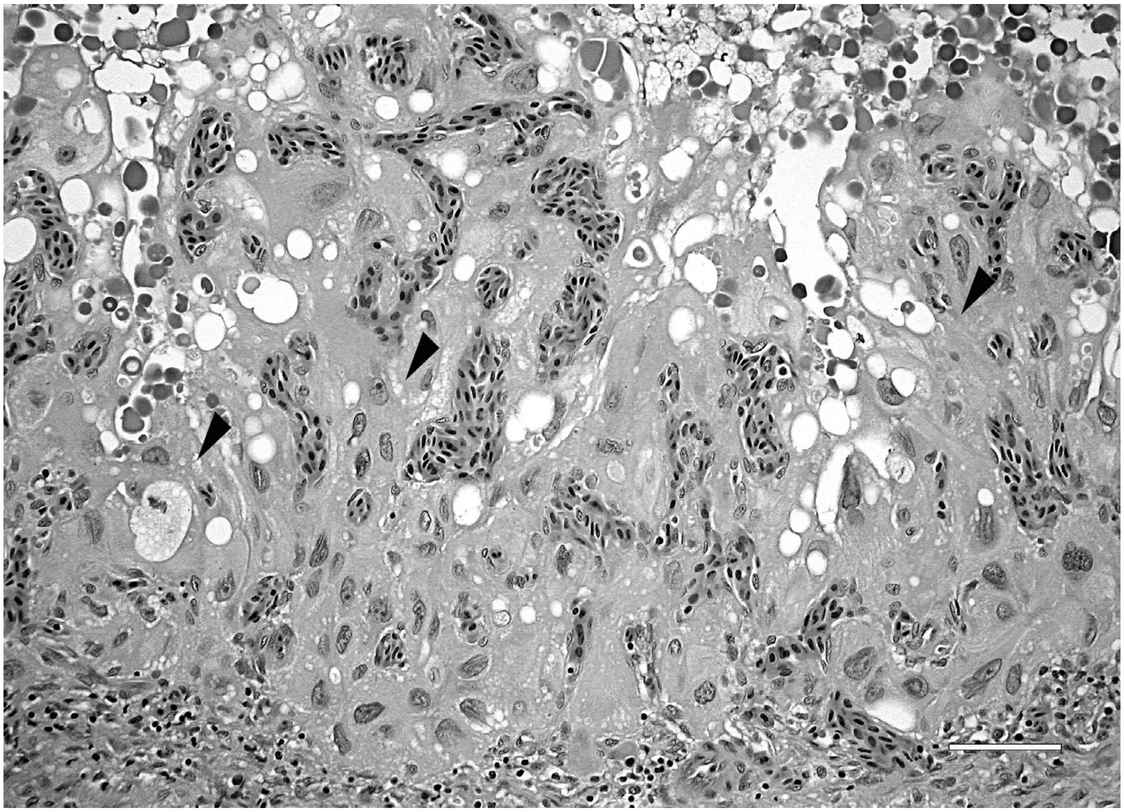

Figure 3. Fijian banded iguana, ovary: a vitellogenic follicle undergoing atresia is lined by hypertrophic, vacuolated granulosa cells (black arrowheads) that are phagocytizing yolk globules. Note the abundant yolk (dark globular material) in the upper margin of the image. Hematoxylin and eosin. Bar = 55 μm.

was present within the ovary, and the associated ovarian surface epithelium was hyperplastic and underwent a transition from simple flattened or cuboidal epithelium to layers of large vacuolated cells. These latter cells resembled both the proliferative coelomic mesothelium and the vacuolated granulosa cells. At high magnification or in tissue sections devoid of architectural orientation, the granulosa cells, hyperplastic ovary surface epithelium, and proliferative mesothelium often were indistinguishable (Fig. 4). Furthermore, it also was difficult to distinguish these cell types from macrophages containing phagocytized yolk, especially cells that were exfoliated into the body cavity or ovary.

Immunohistochemistry and transmission electron microscopy

The only major immunohistochemical differences in the examined cell populations were in the expression of AE1 cytokeratins. The ovarian surface epithelium and mesothelium of the Fijian banded iguanas and the green iguana control were strongly and diffusely immunoreactive for AE1. In contrast, vacuolated granulosa cells in green iguana were negative for AE1 and only very lightly, multifocally immunoreactive in Fijian banded iguanas. All four cell types in the green iguana were strongly immunoreactive for AE3 cytokeratins, as were the mesothelium and ovarian surface epithelium in Fijian banded iguanas. Fijian banded iguanas differed from the green iguana in that granulosa cells were less immunoreactive for AE3.

The ultrastructural features of the granulosa cells of vitellogenic follicles undergoing atresia were very similar to the proliferative mesothelium, and they did not assist in confidently distinguishing the two cell types. Shared features of these cell types included surface microvilli, many prominent desmosomes, numerous mitochondria, and abundant globular cytoplasmic material consistent with yolk. The microvilli of the adjacent granulosa cells seemed to interdigitate more extensively than the mesothelium; however, the significance and consistency of this finding are unknown.

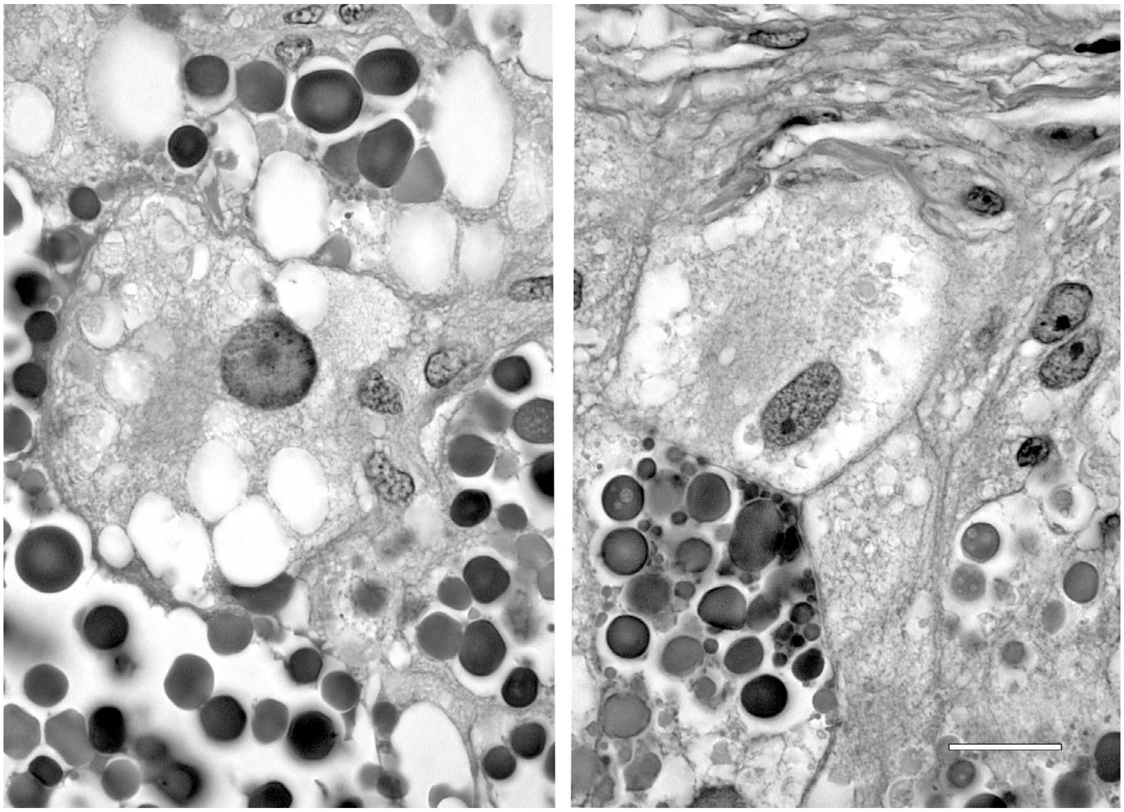

Figure 4. Fijian banded iguana: granulosa cells from a vitellogenic follicle undergoing atresia (left) and reactive mesothelial cells (right) are compared. These cell types are highly similar and have abundant vacuolated cytoplasm, similar nuclear morphology, and some contain phagocytized yolk droplets. Hematoxylin and eosin. Bar = 25 μm.

DISCUSSION

Yolk leakage resulting in proliferative coelomitis and secondary bacterial infection is the leading cause of death in sexually mature female Fijian banded iguanas at the ZSSD. The clinical signs in those animals that presented alive were nonspecific, although reproductive tract disease certainly was a primary consideration in these adult females, especially those with coelomic effusion. The hematologic and biochemical abnormalities also were relatively nonspecific. Hypercalcemia and hyperkalemia observed in case 1 were comparable with that observed in clinically healthy gravid green iguanas.[12] Notably, this was the only animal that had eggs in the oviducts for which biochemical data was available. All cases had some degree of elevated uric acid concentration, which was most severe in case 1. Uric acid concentration may have been elevated in cases 1 and 3 due to secondary septicemia and associated renal injury. Dehydration also could have contributed to uric acid elevation in all cases. The hypoproteinemia and hypoalbuminemia observed in cases 3 and 7 may have resulted from the high-protein coelomic effusion.

Most iguanas had a history of abnormalities related to the reproductive cycle, including reproductive failure, lethargy associated with follicular development, and abnormal nesting behavior. One iguana had two previous salpingotomies for treatment of egg stasis; however, there was no evidence that oviductal eggs were the source of yolk leakage in any case. There is insufficient data to indicate that any specific anomalies in reproductive activity are predictive for yolk coelomitis, and two animals had no previous problems. Given the apparent rapid progression of this disease and high case fatality rate, close monitoring of sexually mature females of "high-risk" species, such as Fijian banded iguanas, is clearly indicated. At the ZSSD, females are now examined if oviposition does not occur within 180 days of copulation.

Although epidemiologic studies have not been performed, the potential for health problems related to reproductive activity in mature female lizards, especially varanids and iguanids, is well known in the herpetoculture and reptile medical communities.[19,26]. Noninfectious disorders include ovostasis or egg-binding and follicular stasis, either of which may result in yolk leakage and associated coelomitis. As seen in the Fijian banded iguanas, septic oophoritis and coelomitis are common sequelae, and they are contributing causes of death (pers. obs.). Xanthomatosis also may be a sequela of intracoelomic yolk leakage in geckos.[7] Follicular stasis is an ill-defined clinical entity that is most frequently reported in green iguanas.[1,19,28] The clinical presentation is one of a depressed, often anorectic female with abundant vitellogenic follicles. Diagnosis is based on the clinical impression of delayed regression of follicles and concurrent clinical signs.[1,19] However, even in relatively common species, such as green iguanas, follicular stasis has yet to be adequately correlated with objectively measurable disorders of reproductive physiology, and careful assessment and characterization of the ovaries often is neglected. Evaluation of ovarian follicles is not simply of academic interest because some features may provide insight into clinically relevant aspects of reproductive physiology, and they may eventually offer evidence of an underlying pathogenesis.

In most of the iguanas of this study, yolk leakage was associated with large vitellogenic follicles undergoing atresia. Notably, in one of the few published reports of follicular stasis, 10 cases in green iguanas were described and the ovarian histologic findings, although difficult to compare in the translated text and published photomicrographs, also seemed to represent follicular atresia.[28] Yolk leakage in reptiles often is attributed to burst follicles; however, the underlying mechanism of such rupture has not been addressed. Obviously, physical injury can cause follicular rupture; however, evidence or history of trauma often is lacking. An alternative explanation is that rupture occurs as the result of follicular atresia, a hypothesis that is supported by known aspects of avian and reptilian physiology. In birds, causes of intracoelomic yolk leakage include internal ovulation (disorder in which the ovum is not received by the oviduct and remains within the coelomic cavity), internal laying, and bursting atresia of vitellogenic follicles.[23] Internal ovulation has not been documented in reptiles, and it may be missed due to the often advanced state of associated coelomitis, rupture of free ova ovulated into the coelomic cavity, and lack of correlative ovarian histology, i.e., presence of corpora lutea, also referred to in reptiles as postovulatory follicles.[25] Atresia of large vitellogenic follicles, including follicular bursting, has been documented in reptiles and warrants further investigation.[2,3,24]

Follicular development in most nonmammalian vertebrates is different from that of most mammals in that maturation involves a period of increasing ooplasmic volume and nutrient reserves (yolk) referred to as the vitellogenic stage.[16] Follicular atresia, a common process in vertebrate ovaries, is defined as the death and resorption of a follicle and associated oocyte without ovulation, and it may occur at any stage of development, including late vitellogenesis.[16,24] Atresia of vitellogenic follicles, also known as large follicle atresia, is well documented in a variety of nonmammalian vertebrates.[17,24] The causes, functions, and significance of this physiologic process are poorly defined, and they may differ across taxa.[10,24] Large follicle atresia is a proposed means of regulating clutch size in birds and at least some species of reptiles.[9–11,20,21] This process likely is hormonally controlled, and it is hypothesized to be influenced by a host of factors, including environmental cues and maternal nutritional status.[11,20] Thus, there is some evidence that vitellogenesis may be a critical point at which these factors influence whether large follicles ovulate or undergo atresia; however, large follicle atresia and its role in reproduction requires further study.[4]

Resorption of the yolk of vitellogenic follicles occurs either within an intact follicle or by rupture of the follicle, referred to as ''bursting atresia.'' As implied in the term, bursting atresia occurs when atretic vitellogenic follicles rupture and release yolk into the ovarian lacunae, and, in some cases, the coelomic cavity.[5,6,22] Although not mentioned in the veterinary literature, bursting atresia is a natural physiologic process, and it has been documented in many different species of birds and in a few species of reptiles.[2,3,6] It is unknown what factors determine whether a follicle is resorbed intact or bursts. The proportion of vitellogenic follicles that undergo atresia, as well as other factors, may dictate whether the process results in uncomplicated resorption of yolk or clinical disease. Yolk coelomitis resulting from large follicle or bursting atresia is observed in some lines of laying hens and turkeys, rapidly growing or obese birds, and in hens that are subjected to fasting or dietary restrictions (Davis, pers. comm.). Further studies are needed to investigate the occurrence of bursting atresia in different reptilian species, especially ''normal'' atresia rates in vitellogenic follicles and correlation with intracoelomic yolk leakage.

Based on the presence of ruptured atretic follicles in several iguanas in this study and known features of reptilian physiology and similar phenomena in birds, the following pathogenesis for yolk coelomitis seen in Fijian banded iguanas and other lizard species is proposed: mature vitellogenic follicles fail to ovulate and undergo atresia; atretic follicles rupture, leading to intraovarian and/or intracoelomic resorption of yolk; and the resorptive capacity is overwhelmed, inciting proliferation of mesothelial cells, inflammation, and predisposing to secondary bacterial infection. Potential factors that may suppress ovulation include suboptimal environmental conditions, and abnormal behavioral stimuli and nutritional condition, all of which are critical concerns in captive husbandry. It was not possible to determine in this retrospective study whether the number of atretic follicles of affected iguanas was different from those animals with a normal reproductive cycle. Such a comparison is a potential area of further study.

The florid proliferation of the coelomic mesothelium in response to yolk leakage in Fijian banded iguana is striking, and other differentials that were initially considered included mesothelioma, ovarian surface epithelial tumor, and granulosa cell tumor. The proliferative cell population was identified as mesothelium and as a reactive response to free intracoelomic yolk based on signalment (mature females), histomorphology, immunohistochemistry, and consistent association with free intracoelomic yolk. Marked mesothelial proliferation is encountered in other reptile species, and it is not unique to Fijian banded iguanas (pers. obs.). The basis for the florid mesothelial response is unknown, and it may be influenced by the volume of yolk released and chronicity of the inciting event.

The morphologic similarity between granulosa cells of atretic vitellogenic follicles and reactive mesothelium is interesting, and it has been observed in several reptile species examined by one of us (BAS). The reason for morphologic similarity is unknown, but it may be due to a common cell of origin or convergent morphology stemming from the common role of phagocytosis of yolk and other debris. As noted in *Thamnophis sirtalis*, phagocytosis of yolk is performed by both the granulosa cells and cells of the coelomic and ovarian epithelium.[3] Proposed origins of granulosa cells include the coelomic epithelium (mesothelium), mesonephric cells, and cells derived from a central blastema.[14] There were differences in the expression of AE1 cytokeratins that may assist in distinguishing these cell types. Low molecular weight cytokeratins typically are expressed in simple epithelia, such as mesothelium and ovarian surface epithelium. These results contrast with those of a study of the lizard *Podarcis sicula* in which the ovarian follicular epithelium (granulosa cells) also was strongly immunoreactive for AE1.[18] This previous study, however, only examined previtellogenic follicles, and it is reasonable to speculate that changes in cytokeratin expression may accompany the dramatic morphologic changes that occur during development of squamate follicles. Transmission electron microscopy on archival specimens did not reveal morphologic features that confidently distinguished tissue types for diagnostic purposes. A limitation of this study was that all available specimens were fixed in 10% formalin rather than fixatives preferred for electron microscopy, which may have obscured ultrastructural features. Additional ultrastructural study of reactive mesothelium and granulosa cells of atretic follicles may reveal more subtle differences and would be an interesting component of ontologic studies.

Yolk coelomitis is a significant problem in captive reptiles, and it is an important cause of mortality in adult female Fijian banded iguanas, as well as at least one other endangered reptile, the Komodo dragon (*Varanus komodoensis*).[26] Since the time of this study, three addition cases of yolk coelomitis with mesothelial proliferation have been observed in Fijian banded iguanas at the ZSSD. Mortality in individuals of prime reproductive age impairs captive propagation and efforts to maintain genetic diversity. The veterinary literature on this topic is sparse, and no efforts have been made to identify specific causes or abnormalities in ovarian follicular development. Several areas require further study, including numbers of vitellogenic follicles that undergo atresia in normal and abnormal animals, hormone levels, and influences on atresia and ovulation in reptiles, and further definition of physiologic and pathologic aspects of follicular stasis. A basic understanding of normal physiology of yolk leakage and correlation with abnormalities in the follicular cycle may provide insight into underlying problems, diagnosis, and potential management solutions.

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