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Report On The Death Of Gilbert's Potoroo Female #17

by Jackie Courtenay
for the Gilbert's Potoroo Recovery Team

April 23, 1997

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(9412)
GIL

#18613

Background

On 19th March 1997, Female #17 was found dead on the floor of the cage when the cage was entered to obtain the bowls for feeding. The animal had been dead some hours as she was cold and was in rigor mortis. Female #17 was brought into the colony with her large pouch young (Female #27) in April 1997. Her current young, Female #29 was at heel but unweaned. Female #27 was an old animal as indicated by the extreme degree of wear on her molars.

The animal was taken immediately to the District Vet. (Kevin Ellard) at Agriculture WA for post mortem. The body weight of the animal was down about 160 g from the last handling 2 weeks earlier, although some of this loss could be attributed to dehydration following death.

Treatment of Unweaned Young

The young-at-heel was initially brought home for hand rearing. She was kept at home for five days housed in a modified cat trap with a hot water bottle (well wrapped up) for warmth. The ideal milk for rearing small Macropodoids "Wombaroo" was unavailable in Albany and it was felt that starting her on one milk and then changing after 2 or 3 three days when the Wombaroo arrived from Perth would cause unnecessary dietary disturbance. A decision was made to feed her "Divetalact" a lactose free veterinary milk which has been used routinely to raise baby woylies with no ill effect. Milk was offered at 4-6 hourly intervals and she was supplied with a dish of food to eat ad libitum (this included apple, dried currants, rolled oats, carrot, and mushroom) and fresh water. After four days she was accepting the teat well and also eating the solid food offered and had gained 7.5 grams of weight, and now weighed 301 g. However, she was showing signs of scouring (even though the milk was made up at half strength) and distress at being confined to such a small cage when she was used to 10m x 3m. Examination of past trapping data indicated that Male #6 had been brought into the colony without his mother at an even smaller size (286.5g) and had survived the enforced weaning. It was therefore decided to return the female to her cage and to check her for movement every day and monitor her weight gain weekly. She was moved in with the old Female #4 on 27th March and although her weight has fluctuated between about 290g and 300g throughout this period, she now weighs 303.3g (as of 19.4) and appears to be doing well. Both animals are checked nightly to ensure they are moving around and weekly monitorings will be continued in the short term.

Post Mortem Results

The post mortem indicated that the dead animal had a severe build up of oxalate crystals in the kidneys. While this has not been observed in the Potoroos at Healesville, it has been reported frequently in both wild and captive koalas (see Kevin Ellard's report), and in various other marsupial taxa such as Scaly tailed possum and Swamp wallaby[1]. In none of the previously reported cases has the cause of the condition been discovered. Possible causes are excessive oxalate in the diet, inadequacy of Vitamin B6 (which assists in the metabolism of oxalate), spoilage of food, excessive Vitamin C or metabolic failure of unknown cause (although old age combined with the pressure of lactation may be a factor in this case).

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[1] Ellis, Copland and Gaynor 1983 Oxalate toxicity in Scaly tailed possum, Patagonian Cavey and Swamp wallaby. *J. Wildlife Diseases* 19(3):290-293.

Actions

(1) Assaying of Diet for Oxalate levels.

Two full meals (one with banana and mushroom, and one with sweetcorn and bean) were prepared and couriered on ice to the Agriculture WA Laboratories in South Perth. The meals were assayed for oxalate content to determine if the diet contained excess oxalate. Full results will be provided in a veterinary report (to come), however, the results given verbally were that the diet contained no oxalate at all. Excessive dietary oxalate can therefore be ruled out as a factor.

(2) Dietary Modifications

Discussions with Healesville revealed that their Potoroo diet had been modified since the Gilbert's Potoroo diet was developed. A copy of their new diet was obtained and some modifications made to the Gilbert's Potoroo diet. New foods were introduced one at a time over a period of two weeks and in addition to the normal diet. These include:

- Addition of oranges, pears and sprouted wheat

Pears were discontinued after a few days because although the animals ate them readily, piles of diarrhoea were found in Cages 1 and 4 after the pears were introduced (the other foods had either not been introduced or the animals had not eaten them). No diarrhoea has been found since pears were discontinued. Oranges were eaten by some animals but not others and the wheat sprouts were hardly touched by any animal. Whole wheat berries soaked in water but not sprouted are being trialed as an alternative.

- Dog cubes are now being given as a daily food item (1 or 2 cubes per day) rather than ad libitum as it was feared they could be a possible source of "spoilage" moulds (and hence oxalate) in the diet. Other foods were given fresh each day and only the freshest foods are given to the Potoroos so other sources of "spoilage" fungi are unlikely
- Meal worms have been introduced as a twice weekly supplement to increase the fat and B vitamin content of the diet (following both the new Healesville diet and the recommendation of Kevin Ellard). The animals are eating these in small quantities.

(3) Sampling of animals to test kidney function

On 23th March 1997 blood samples (of varying volumes) were collected by Kevin Ellard from 7 of the captive animals to test for renal function in other animals. Without established "normals" the results are difficult to interpret, however the values obtained fell within the range of values for domestic animals. An attempt was made to collect blood from the animals at East Firebreak to obtain "normal" values, however, no samples of sufficient size were obtained. Blood samples from wild caught animals will be obtained when possible to establish these values.

Conclusions

No firm conclusions can be drawn as to the cause of the oxalate build up in Female #17. The complete absence of oxalate in the diet indicates that a dietary source can probably be ruled out. The diet itself was found to be oxalate free on assay, and only the freshest food has always been used for the animals so the presence of spoilage moulds is unlikely. While Vitamin B6 deficiency can also result in a similar condition to that seen, other symptoms associated with Vitamin B6 deficiency were not observed (see Kevin Ellard's comments). Nevertheless, the diet is being modified slightly to increase dietary Vitamin B6.

In the absence of an obvious dietary source, it seems most likely that the condition occurred as a result of metabolic breakdown caused by lactational stress in an old animal.

Interim Veterinary report attached

Kevin Ellard
Wildlife Veterinary Services
649 Lower King Road
Albany WA 6330

Dr Jackie Courtney
CALM
120 Albany Highway
Albany WA 6330

Dear Jackie

RE: Post Mortem Report on Female Potoroo, Two Peoples Bay Albany.

Please find outlined below an interim report from David Forshaw, Veterinary Pathologist Agriculture WA, together with my conclusions relating to recent post mortem examination of the female potoroo. It must be stressed that any suggestions made at this stage are not conclusive and a significant degree of background research must be made before any firm conclusions are made.

At this stage the cause of death in this animal must be presumed to be renal failure caused by an accumulation of oxalate crystals within the kidneys. This condition is commonly reported within veterinary literature and as outlined in David's report it has a number of causes.

It is interesting to note that both Perth Zoo and Yanchep National Park had a similar condition occur within their koala colonies during 1993. In addition to this 'oxalate nephrosis' was identified in 4 koala during a necropsy survey of 235 free ranging and captive NSW koalas between 1980-1988. An actual cause within these colonies was never accurately determined. I shall investigate this further.

Clark et al. (Veterinary Toxicology, 1981) reports that oxalate poisoning may cause a number of different conditions within mammals, these being renal nephrosis, hypocalcaemia, central nervous disorders and intravascular haemolysis. Although renal nephrosis was the major histological finding oxalate induced hypocalcaemia may upset calcium metabolism sufficiently to interfere with milk production and bone growth in lactating animals. Considering the reproductive history, age and postmortem findings in this animal this condition must also be suspected.

In this case the renal changes suggest that the condition has been caused by small quantities of oxalate being ingested over a number of weeks.

David has outlined in his report possible sources of oxalate in the diet and the association of oxalate nephrosis with some fungi, this does not need to be repeated here. He also states that vitamin B6(Pyridoxine) deficiency has been linked with oxalate nephrosis in the past. As with most B group vitamins, B6 is produced by green plants and many microorganisms contained within the fermenting gut of certain animals, including macropods. It should be noted that this animal showed none of the classical associated oedema and dermatitis often associated B6 deficiency. Smith et.al (Mammalian Biochemistry, 1983) also states that the requirement for B6 is linked with the protein level of the diet.

In the interim period I would suggest the following action:

- Collect serum from the remaining animals within the colony together with at least five wild animals to analyse renal function.
- Have the current diet analysed for oxalate content.
- Modify the range of the current diet to include animal protein (insects) and green leaf.

Prevention of oxalate poisoning in other mammals such as sheep may be achieved by supplying a good source of calcium within the diet.

In previous koala cases it was postulated that a disturbance of the gut microflora may have predisposed animals to this condition. It is therefore essential that any modifications to the diet are made gradually rather than abruptly.

Yours sincerely

Kevin Ellard.
Veterinary Surgeon.
March 26, 1997

AGRICULTURE WESTERN AUSTRALIA - ANIMAL HEALTH LABORATORIES

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BUNBURY: North Boyanup Road, Bunbury WA 6231 Tel:(097)806 100 Fax:(097)806 136
ALBANY: Albany Highway, Albany WA 6330 Tel:(098)928 444 Fax:(098)412 707

Your Ref: NOT SUPPLIED
Enquiries: MR D. FORSHAW
Date: 24-03-97

Case Number: A-97-109
Submission number:

MR K ELLARD
DVO ALBANY
ALBANY REGIONAL OFFICE
AGRICULTURE W.A.
ALBANY 6330

Owner:
JACKIE COURTENAY
CALM
120 ALBANY HWY
ALBANY

Classification:
DIAGNOSTIC MISCELLANEOUS - ALBANY

Date Received: 20-03-97
Species: MARSUPIAL - GILBERT'S POTO

REPORT ON LABORATORY SUBMISSIONS

Sample(s) Submitted: FRESH GUT, FIXED X 3, GLUT FIXED X 5, FRESHLIV KID, SWABS

Pathology Results

HISTORY:
Aged female Gilbert's Potoroo held in captivity at Two Peoples' Bay.
Found dead in enclosure.

NECROPSY FINDINGS:

PM by Kevin Ellard 19/3/97
Body weight 778g. Moderate condition. Moderate autolysis.
Liver - 25g, spleen - 2g, heart - 6g, kidney - 8g. GIT - 77g, lungs and trachea - 5g.
Distictly pale and possibly enlarged kidneys.
Liver mottled with some rounding of margin edges edges.
Spleen appears enlarged, otherwias normal.
Heart appears enlarged, otherwise normal.
Adrenals appear pale and enlarged, approx 8mm long, dark cream colour.
Lungs appear non deflated possibly emphysematous
Long bones and ribs appear thin + brittle cortices however normal appearance is not known.
Good deposits of fat around the kidney.
Full stomach with area of congestion towards the pylorus.

Histology.
Kidney - Massive dilation of cortical tubules, most of which contain large deposits of birefringent crystalline material. In the medulla, quit just below the pelvic epithelium, there are a number of large cystic structures, some of which contain similar crystalline material.
Crystals are also present in the interstitium and in both the

interstitum and the tubules there is a inflammatory response which consists of purely multinucleate giant cells in some foci and as mixed polymorph/mononuclear infiltrates in other areas. There is patchy moderate interstitial fibrosis associated with multifocal mixed cell inflammatory infiltrates.
Tissue - Advanced autolysis. Glandular tissue with closely packed acinar structures but no evidence of ducts.
Skeletal muscle x 2 - NSA.
Pancreas - Advanced autolysis, NSA.
Bone marrow - NSA.
Heart x 4 - NSA.
Trachea - NSA.
Adrenal - NSA.
Brain x 8 - NSA.
Liver x 2 - NSA.
Lung - Well developed peri bronchial lymphoid follicles.
Spleen x 2 - Extensive diffuse extra medullary haemopoiesis.
Mesenteric node - Disorganised structure with follicles deep in medulla and sinusoids extending to the capsule. NSA.
Oesophagus, stomach, colon, small intestine x 4 - NSA. Autolytic.
Urogenital tract, colon, lymph node - NSA.
Uterus x 4 - NSA.
Ovary x 2 - NSA. Both copora lutea and developing follicles present.

DIAGNOSIS:
1. Renal tubular necrosis, subacute, diffuse, severe with crystal deposition consistent with oxalate.
2. Interstitial nephritis, patchy, chronic, active, severe.
COMMENT:
I will confirm the identification of the crystals with a specific stain for oxalates. This process has been going on for some time, possibly weeks.

There are three recognised mechanisms resulting in excess oxalate excreted by the kidney;

1. Excessive intake of oxalic acid or its precursors.
2. Increased intestinal absorption of oxalate.
3. Increased endogenous synthesis of oxalate.

Kidney failure itself can lead to hyperoxalaemia but oxalate excretion is reduced and crystalluria is not seen.

Taking each of these mechanisms and examining it from the point of

view of the potoroos:

1. Certain drugs and chemicals such as ethylene glycol commonly used as anti-freeze are metabolised to form oxlate but can be ruled out as possible sources in this case.
There are no obvious sources of oxalate but possible sources to rule out include;
a. oxalate accumulating plants of the Chenopodiaceae and Oxalidaceae (the commonest plants implicated in cases of oxalate poisoning in livestock in the south coast area is Chenopodium alba or "goosefoot" or "mintweed").
b. Oxalate producing fungi of foodstuff. Many of the common spoilage fungi eg Aspergillus, Mucor, Penicillium, Rhizopus, have been recorded to produce high levels of oxalates. On hay and silage, the gross appearance can be normal in feed which has had high oxalate levels.
c. High oxalate containing food in the diet. In humans, cocoa, tea, parsley, spinach, pepper and nuts are listed as possible sources of high oxalate levels.
d. Vitamin B6 deficiency causes hyperoxaluria and renal damage in the cat, rat and humans. Sources of vitamin B6 in the diet are
e. Large doses of vitamin C can result in hyperoxaluria in humans and has been reported in a goat.

2. The pathogenesis of hyperoxaluria with gastrointestinal disease is poorly understood but in humans, enteric disease is the most common cause of hyperoxaluria. Clinically there has been no mention of gastrointestinal disease and in humans, there is usually a malabsorption syndrome resulting in chronic weight loss. The histological examinations of the gut of the potoroo were hampered by autolysis and enteric disease cannot be ruled out in this animal.

3. In humans, there are two rare inherited disorders of oxalate metabolism. These usually shows up in early childhood but death may not occur until early adulthood. An equivalent disease is possible in potoroos but would seem highly unlikely.

Yours faithfully
MR D. FORSHAW
VETERINARY OFFICER

Cost Advice Summary. (INVOICE will be sent if total charge > $0.00)

Lab	Service	Quantity	Discount Value	%	Reason	Charge
HIA	SLIDE PREPARATION	11	143.00	0		143.00

PTA HISTOPATHOLOGY	1	28.00	0	28.00

Total value: 171.00 Total charge: 171.00

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