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Polydipsia/polyuria syndrome in a Congo African grey parrot (*Psittacus erithacus erithacus*), a case report

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Abstract

Polydipsia/polyuria syndrome is not a common manifestation in birds. Hyperglycemia was found on plasma chemistry in an African Grey Parrot *(Psittacus erithacus)*, which was presented with polyphagia and self mutilation symptoms. Radiography demonstrated signs of mild renalomegaly, air sac involvement which proved to be due to mycoplasma spp. infection on bactriological examination. The polydipsia, polyphagia and polyuria were treated successfully by insulin regime but self mutilation around the neck was to be most consistent sign for 45 days since the bird presented to our Faculty's clinic. The exact origin of the Polydipsia/polyuria and mild hyperglycemia remains unclear but the history of long time topical cortone therapy might be involved.

Keywords: Polydipsia/polyuria syndrome, African grey parrot, Hyperglycemia, insulin

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Introduction

Polyuria presents in birds as a huge volume of urine with no changes in opacity and color. Crystalline urates more than usual may be seen but the stool portion of the droppings is often well formed. Polydipsia often accompanies polyuria because it is a consequence of the water and fluid loss.

There are a variety of conditions and diseases that can cause polyuria/polydipsia (PU/PD) in birds. In pigeons, it might be due to vaccination by paramyxovirus serotype 1 that it can cause severe PU/PD. In female birds that are about to lay eggs (especially in canaries) or those with possible peritonitis may have PU/PD as well (Brown and Sandersen, 2005). The behavioral history may reveal psychogenic polydipsia with secondary polyuria. Commonly the history includes information on the diet, its social and behavioral interactions and recent medications may help to point on these conditions.

Especially in the smaller psittacines, PU/PD has been observed in those consuming a larger part of their diet as extruded kibble. In these cases, lesions were limited to nonspecific tubular nephrosis and the condition showed to be reversible by feeding a non-pelletized diet for approximately 1–3 months (Martin *et al*., 2003).

There are a number of diseases or conditions that are associated with PU/PD syndrome in birds. The diseases include those of metabolic origin such as liver disease, kidney disease, diabetes mellitus, diabetes insipidus and those associated with diet such as dietary-induced polyuria, excess fruit consumption, psychogenic polydipsia, excess dietary sodium, calcium or vitamin D and excess dietary Protein. There are some medications that cause PU/PD as a sequel: diuretics, gentamicin and corticosteroids (Dorrestein, 1997). Finally, renal glucosuria due to excitement or fear when birds come to visit the avian veterinary hospital could be associated with PU/PD syndrome.

Case Presentation

A 6-yrea-old Congo African Grey Parrot (*Psittacus erithacus erithacus*) was presented to the Division of Avian Medicine of the Veterinary Faculty, Ferdowsi University of Mashhad, with PU/PD, polyphagia, mutilation, and exposed bare skin back on the neck, the head feathers were spared and appeared perfect and untouched.

The Bird was using its claws to perform self-mutilation vigorously (Figure.1B). The 456-g bird had been in the possession of the owner for 5 year and was fed a seed-based diet supplemented with vitamins and fruits. Diagnostic testing was performed by taking history and clinical examination of the patient.

The amounts of serum glucose, uric acid, AST, LDH, calcium, phosphorus, total protein, CPK and total protein were measured by commercial kit (Pars Azmoon, Iran) using an autoanalyser (Biotecnica, Targa 3000, Rome, Italy). The results of plasma chemistry showed mild elevation of glucose (520 mg/dl) and other parameters were within published reference ranges.

Wax paper was placed in the cage floor and the bird was allowed to void several droppings for analysis. The liquid urine has been aspirated with a 22g needle on a 1–3 ml syringe with minimal pressure as to not disrupt the cells or granular casts. The sample in the syringe held upright in a plastic rack for 5 minutes and then applying to the surface of a slide, cover slipped and the a drop of sedi-stain applied. The remaining sample was used for biochemical analysis with the simple dip strips (Urocolor, Standard Diagnostics, Korea). The results of urinalysis showed 3+ glucosuria (approximately 1000 mg/dl) by dip strips and no abnormal findings in the urine sediment. Water deprivation testing is considered when attempting to rule out unknown causes of PU/PD including central and nephrogenic diabetes insipidus and psychogenic polydipsia but unfortunately we have not access to the ingredients need to do this test.

Plain lateral and ventrodorsal whole body radiography was performed to evaluation the

avian liver and kidneys. Liver was within normal limit and renalomegaly was noted (Figure.2).

Treatment included 0.9% NaCl (2 ml IV), lactated Ringer's solution (50 ml/kg SC q12h), insulin (NPH) (0.2 U/kg IM q12h), low-carbohydrate, low-sugar diet, Enrofloxacin (15 mg/kg IM q24h) and haloperidol (0.2 mg/kg PO) to reduce self-mutilation behavior. After 2 weeks glucose level return to low normal limit (180 mg/dl) and the owner had not any complaints from polyuria/polydipsia and polyphagia but the bird continued scratching the back of neck by claws sometimes. We put rosary beads with glue on his claws (Figure 1(B and C)) and continued treatment by Diazepam (2 mg/120 ml drinking water for 10 days). To date, the bird is in normal behavior and life.

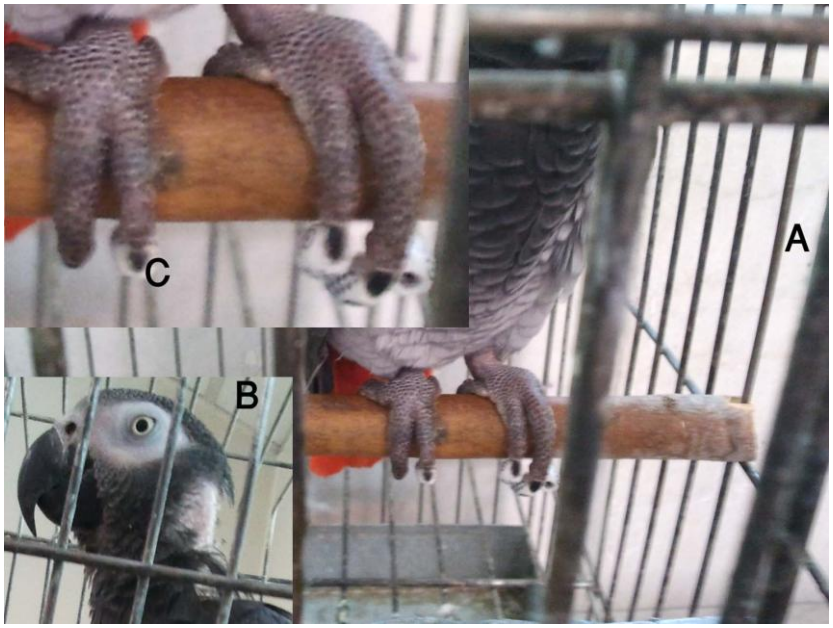

Figure 1. Bared area of self-mutilation(B), Rosary beads placed on claws to avoid self-mutilation mechanically(A and C).

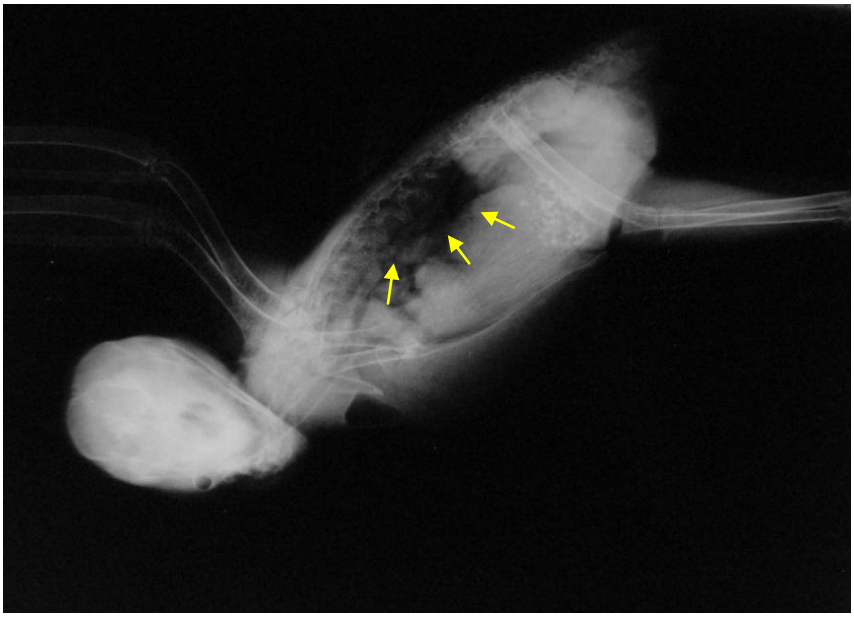

Figure 2. Whole body lateral radiographic image showing renomegaly (arrows: swollen kidneys).

Discussion

Excess vitamin D3 promotes metastatic mineralization of viscera including the kidney in birds (Speer, 1997, Schoemaker *et al*., 1997 and Styles and Phalen,1998). This problem most commonly affects nestling parrots (Speer, 1997). Clinical signs may include polyuria/polydipsia, anorexia, crop stasis, and weight loss (Schoemaker *et al*., 1997 and Phalen, 2000) but according to history given by owner, there was not any evidence of excess vitamin D3 in our case. Gentamicin represents as a cause of nephrotoxicity in the birds and polyuria/polydipsia is often seen even at low doses (Frazier *et al*., 1995). There is a report in which Gentamicin (5 mg/kg intramuscularly every 12 hours for 7 days) led to profound polyuria/polydipsia in cockatoos (Eolophus sp) which persisted for 23 days after stopping treatment (Flammer *et al*., 1990). In falcons (Falco biarmicus) given gentamicin (5 mg/kg/d for 4 days), loss of balance, impaired vision, and muscle spasms were described (Speer, 1997). Uric acid excretion is largely independent of urine flow and therefore is unaffected by moderate changes in glomerular filtration (Chandra *et al*., 1985). . Elevations in uric acid (up to 20 mg/dL) may be seen with severe dehydration (Van Toor, 1984 Fudge, 1997, Styles and Phalen,1998), but uric acid does not increase significantly with renal disease unless there is extensive tubular damage (Lumeij,1990). These facts are supported with our findings in this bird because there was not any sign of dehydration, uric acid accumulation in feces and Urea nitrogen (BUN) elevation as indicator of dehydration in our case. BUN has little value in the detection of renal disease in most birds (Van Toor *et al*., 1984 and Speer, 1997), but BUN is a sensitive indicator of hydration. In the dehydratedbird, up to 99% of BUN is reabsorbed.

Polyuria may also occur with sepsis even when the pathogen does not directly affect the kidney (Lumeij and Westerhof, 1988 and Phalen, 2000). To identify the cause of sepsis and bacterial nephritis, blood culture is a much better test than urine culture (Speer, 1997). We isolated Mycoplasma spp. via tracheal swap from this bird and no bacteria in blood cultures. However, the role of the isolated bacteria is unclear.

Products such as psittacine hand-feeding formula and low-protein products have been recommended by many of researchers (Speer, 1997 and Lumeij, 2000). A diet restricted in protein and minerals also reduces the serum phosphorus levels, which may slow the progression of renal disease. Never the less, low-protein diets may lead to malnutrition, and low-protein diets are contraindicated in patients with advanced renal failure (Brown and Sandersen, 2005). Low dose non-steroidal anti-inflammatories may be used but with caution as their effect on the kidneys in birds is not understood. Dietary management is important but controversial in regards to protein levels. Current human and veterinary literature both supports and refutes protein restriction in patients with renal disease. The current recommendation is to provide adequate levels of protein.

The exact cause of PU/PD in this case was not clear but supportive treatment regime including: diet correction, insulin therapy, fluid therapy, using of broad spectrum antibiotic to prevent secondary bacterial infection, psychotherapeutic agents to prevent self-mutilation and aggressive behavior and finally simple physical barrier for correcting aggressive behavior during a period of time, all together could save the bird from further subsequences.

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گزارش یک مورد سندروم پرادراری/ پرنوشی در طوطی خاکستری آفریقایی مشهد، ایران

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چکیده

سندروم پرادراری/ پرنوشی در پرندگان معمول یک عارضه نمی باشد. در این گزارش یک طوطی خاکستری آفریقایی با افزایش گلوکوز خون ، پرخوری و آسیب رسانی به خود مورد بررسی قرار می گیرد. رادیولوژی علائمی از بزرگی کلیه ، درگیری کیسه های هوایی که در آزمایشات باکتری شناسی مشخص شد ناشی از درگیری با باکتری مایکوپلاسما بوده است را نشان داد. علائم بیماری شامل پرخوری ، پر نوشی و پر ادراری با استفاده از رژیم درمانی انسولین بخوبی کنترل شد اما علائم آسیب رسانی به خود بویژه در ناحیه گردنبرای مدت بیشتری ادامه یافت . علت اصلی پرخوری و پرنوشی و پر ادراری و هیپرگلیسمی متوسط پرنده مشخص نشد اما ممکن است ارتباطی با سابقه مصرف طویل المدت داروهای کورتیکوستیروئیدی داشته باشد.

واژگان کلیدی: پرنوشی، پرخوری، پر ادراری، هیپر گلیسمی، انسولین