

Raptor Myopathy

P.A. McKinney
Wildlife Protection Office, Dubai
PO Box 27942
United Arab Emirates
Email : peatvet@emirates.net.ae

ABSTRACT

Exertional myopathy is seen in falcons used for the sport of falconry in the United Arab Emirates. Diagnosis is based on clinical signs and elevated plasma creatine kinase (CK) levels.

In this study the case histories of 44 falcons with abnormally high plasma (CK) levels were assessed. Results indicate that myopathy is associated with strenuous exercise in unfit falcons, post capture trauma and muscle catabolism secondary to disease and malnutrition.

INTRODUCTION.

Extensive research has been carried out on exertional myopathy in horses. It is common in thoroughbreds and can be affected by temperament, sex, age, diet, exercise routines. Management that minimizes excitability may be the most effective way of controlling exertional myopathy. (MacLeay (1999)) In horses exercise myopathy may exist as two separate entities. One is associated with limited exercise and is usually seen in the more nervous excitable horses. The other is seen following protracted exercise such as endurance rides. The latter type is associated with other metabolic changes such as electrolyte loss, fluid depletion and is seen more in hot weather rather than cool. (Pascoe 1994) Intra-erythrocytic potassium levels may play a role in the development of the disorder in horses (Bain 1990).

In falcons the pectoral muscles and the supracoracoideus muscles are the main muscles used in flight (Fox 1995). Myopathy in falcons results in reduced ability to fly i.e. a noticeably reduced range of wing movement and a lack of flying stamina. Severe cases show a temporary grey to green discoloration of the urate portion of the mites.

In the early stages of training excitable falcons are more likely to develop myopathy due to excessive beating from the glove. Plasma creatine kinase (CK) and aspartate transaminase (AST) levels rise following exertional myopathy in horses. (Valberg 1993). Similar changes occur in falcons.

The aim of this study was to identify the incidence, clinical signs and predisposing factors associated with myopathy, in captive hunting gyr cross-bred falcons in the United Arab Emirates.

METHODS

289 gyr cross-bred falcons (*Falco.rusticolus x f.peregrinus* and *f.rusticolus x f. cherrug*) were presented to the Al Safa Falcon Clinic for a routine veterinary examination. This included endoscopic examination of the respiratory tract, whole body radiography ,haematology and plasma biochemistry analysis . All falcons were anaesthetized with isoflourane administered via a facemask.Blood was collected from the ulnar vein within two minutes of induction of anaesthesia, and placed in lithium-heparin tubes for biochemical analysis at the Central Veterinary Research Laboratory .Dubai.

Although muscle biopsies would prove invaluable in the diagnosis of myopathy,none were performed in this study.

Diagnosis of myopathy was based on clinical signs and elevated plasma CK values.

A proportion of the birds were untrained, highly excitable falcons which were prone to excessive bateing from the handler's glove in the initial stages of training.

Other falcons were considered trained and acclimatized to humans. The normal range of creatine kinase for birds of prey has been reported to be 100-800 U/L with elevations seen following exercise and various diseases.(Cooper 2002).

Gyrfalcons were found to have a mean plasma CK value of 402 sd 163. Although the sample study was small (n=12) (Samour 2000). Ranges of plasma CK values of 357-850U/L for peregrines(*F peregrinus*) and 355-651U/L for saker falcons(*falco cherrug*) have been reported.(Lumeij 1996).The gyrfalcon hybrids in this study were gyr-peregrine and gyr-saker hybrid falcons.

Forty four falcons with the highest plasma CK levels were selected. An attempt was made to identify predisposing factors which may have played a role in the development of the myopathy.

The falcons were classified as;

- Newly acquired falcons ie in the initial stages of training or recently taken from an aviary
- habituated or trained falcons ie in training more than one month
- Falcons with concurrent disease cases.eg aspergillosis, amyloidosis, pododermatitis.

RESULTS

Post capture myopathy ,strenuous flying(bateing) in the early stage of captivity and overtraining resulted in myopathy. Muscle catabolism secondary to malnutrition or disease resulted in moderate increases in plasma CK levels.

Of 289 gyrfalcon cross-breds which were blood sampled

25 % of samples had CK levels over 1000u/l . 15% had CK levels over 2000U/L.The range of CK values was 1119-6584U/L.

Table 1 . Assessment of 44 samples with CK values between 1119-6584U/L.

History	Distribution of samples n=44
New falcons –untrained	43.2%
Trained falcons-overtraining	43.2%
Concurrent diseases	13.6%

One confiscated wild caught gyrfalcon had been wrapped in a towel for five days had a CK of 12700 U/L. This was not included in the data as it was a unique case. The myopathy was so severe that the falcon could not move the wings for a period of three days. It made an uneventful recovery.

Discussion

Creatine kinase is found in skeletal muscle, cardiac muscle and nervous tissue. Clinical elevations of CK in birds are primarily attributed to changes in skeletal or cardiac muscle.

Muscle wasting, vigorous physical activity, rough handling and irritating injections can all cause marked elevations of this enzyme. CK elevates and declines most rapidly followed by AST (Fudge 1997).

Serum concentrations of CK and AST have been used indicators of capture myopathy in birds mallards (*Anas platyrhynchos*) (Dabbert 1993). Plasma CK activity was described as an indicator of muscle damage in migratory shorebird species (Guglielmo 2001).

Training new falcons involves habituation with the falconer or rehabilitator. Very excitable falcons in the early stages of captivity constantly bate from the falconers fist or from the block to which it is attached. The flight muscles become fatigued and myopathy may develop resulting in a rise in CK levels.

In the wild, falcons tend to stoop at prey in contrast to the short, explosive bursts of speed seen with accipiters.

In UAE traditional training methods require the falcon to fly at quarry from the glove. The hunt may be much more protracted than stoops in nature and may result in muscle fatigue and damage.

Vitamin E deficiency has been associated with muscular disorders in birds (Bennett, R.A. (1994), (Jensen 1992). Captive peregrines fed unsupplemented Japanese quail were found to have lower circulating plasma alpha-tocopherol than their wild counterparts. Increasing the Vitamin E content of the quail food resulted in plasma optimal vitamin E levels in the falcons (Dierenfeld 1989). Further research is required into the vitamin E status of captive falcons in the UAE.

One of the major side effects of exertional myopathy in endurance horses is nephropathy. In man acute renal failure (ARF) is an important complication of rhabdomyolysis (Watanabe 2001). The myoglobin released from muscle breakdown is excreted in large quantities via the kidney. This can lead to toxic renal damage. It is not known if this phenomenon occurs in falcons but considering the extreme physical nature of falconry in the hot climate of UAE, care should be taken to prevent myopathy.

Myopathy is a significant disorder of captive, hunting falcons in the United Arab Emirates. Prevention of myopathy by minimizing post-trapping myopathy and prevention of over exertion in training, are important. These principles also apply to wild injured falcons presented for care and rehabilitation.

ACKNOWLEDGEMENT

I would like to thank HH.Sheikh Mohammad Bin Rashid Al Maktoum ,Crown Prince of Dubai. Dr Ali Ridha,Dr Wernery,Dr Kinne of the Dubai Central Veterinary Research Laboratory.

Dr A. Sharma and Dr L.Belwal of the Emirates falcon Hospital Dubai.Dr Laco Molnar ,Abu Dhabi Wildlife Research Centre and all the local falconers of Dubai

References

Bain F.T.,Merritt .A.M(1990) Decreased erythrocyte potassium concentration associated with exercise –related myopathy in horses.J Am Vet Med Assoc.1990 Apr 15;196(8):1259-61

Bennett.R.A (1994) In Avian medicine .Principles and Applications. Ch 28 Neurology. p732. Harrison,Harrison,Ritchie.Wingers Publications.

Cooper .J.E(2002) In Birds of Prey-health and Disease 3rd Edition .Methods of Investigation and Treatment. p60 Blackwell science.

Dabbert ,C.B.,Powell K.C (1993) Serum enzymes as indicators of capture myopathy in mallards(*Anas platyrhynchos*) j Wildl Dis Apr;29(2):304-309

Dierenfeld,E.S.,Sandfort,C.E.,C.E &Satterfield,W.C(1989) Influence of diet on plasma Vitamin E in captive peregrine falcons. J.Wildl.Management.53(1):160-164.

Fox. N(1995) The Wing Structure . p31 In Understanding the Bird of Prey. Hancock House Publishers Ltd UK

Fudge,A.M(1997) In: Avian Medicine and Surgery .eds Altman .B.,Clubb. .L.,Dorrestein. .M.,Quesenberry.K . Ch.11 Avian Clinical Pathology-Hematology and Chemistry p 150-151 W.B.Saunders Co.

Guglielmo C.G.,Piersma T,Williams.T.D .A sport-physiological perspective on bird migration:evidence for flight induced muscle damage . J Exp Biol Aug ;204(Pt15):2683-90

Jensen.J.M.,Johnston.J.H.,Weiner,S.T(1992) In Husbandry and medical management of ostriches,emus and rheas.p24 ISBN 0-9626069-8-7 Wildlife and Exotic Animal Teleconsultants.USA

Lumeij. J. T(1996) In :BSAVA Manual of Raptors Pigeons and Waterfowl. Ed Beynon.P.H., Forbes.N.A & Harcourt –Brown N.H. Ch 7.Biochemistry and sampling. Appendix 8.1 Haematology and Clinical Biochemistry Values. p 75.BSAVA UK.

MacLeay J.M.,Sorum S.A,Valberg S.J,Marsh W.E,Sorum M.D.(1999)Epidemiologic analysis of factors influencing exertional rhabdomyolysis in Thoroughbreds. Am J Vet Res. Dec 1999;60(12):1562-6

Pascoe.R (1994) Exertional Rhabdomyolysis.In Differential Diagnosis of Diseases of Horses.Series B.No 19.p 106-107 University of Sydney Post Grad Foundation in Veterinary Science

Samour J(2000) In :Avian Medicine .Blood chemistry values for selected birds of prey.p350.Mosby.

Valberg,S.J & Hodgson,D.R(1996) Disease of muscle.In Large Animal Internal medicine.2nd edn.Ed B.P Smith.St Louis,Mosby-Year Book,p 1489

Watanabe.T(2001) Rhabdomyolysis and acute renal failure in children. *Pediatr Nephrol*.2001 Dec;16(12):1072-5

