CLINICAL INVESTIGATIONAND THERAPEUTIC MANAGEMENT OF HYPOGLYCEMIC SEIZURES IN PUG

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A male Pug of age 3 years (body weight 12 kg.) was presented to the Veterinary Clinical Complex, Nagpur Veterinary College, Nagpur with a history of anorexia, muscle twitching, blindness, and lateral recumbency for 2 days with a recurrent rate of seizures within a period of 10 to 15 min. The dog has mild to no response to pain stimulation (mild to no withdrawal reflex), pupils were dilated, and on complete blood count, leucocytosis was observed and random blood glucose revealed a blood glucose level of 28mg/dl (60-111mg/dl), calcium was within the normal range (60-110mg/dl) and with SpO₂ 87%. The case was treated with dextrose 10% I/V, injection ringers lactate I/V, dexamethasone I/M,injection vitamin B complex I/M bid for 3 days and oxygen supplementation was given.

Keywords: Hypoglycemia, Seizures, Muscle twitching,

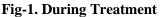
Hypoglycemic seizure is not a common disease in dogs. In this condition, there is a severe fall in blood glucose levels. clinically normal dogs, glucose is maintained within a narrow range of 60mg/dl to 111mg/dl (Smith, 2004), and clinical signs of hypoglycemia do not usually develop until glucose concentrations are <50 mg/dl (Koenig, 2009) and hypoglycemia are defined as glucose less than 50mg/dl. (Koenig, 2009) and (Nelson, 2014). Four mechanisms due to which hypoglycemia may arise: i) glucose and other substrates used in hepatic gluconeogenesis deficit diet; ii) sometimesin normal or neoplastic cells may cause an increase inglucose uptake and utilization due to an increase in demand or secondary to hyper-insulinism; iii) disturbance in hepatic glycogenolytic or gluconeogenic pathways; and iv) a deficiency of counter-regulatory hormones such as cortisoldue to endocrine abnormalities (Koenig, 2009) and (Nelson, 2014). Clinical signs may include altered mentation behavior such as seizures, muscle twitching, lethargy, collapse, incoordination, weakness, and impaired vision. These clinical

signs are mainly due to neuroglycopenia (cerebral hypoglycemia) (Nelson, 2014).

Materials and Methods

A male Pug of age three years was presented to the Veterinary Clinical Complex, Nagpur Veterinary College, Nagpur with a history of anorexia, muscle twitching, blindness, and lateral recumbency for two days with a recurrent rate of seizures within a periodof 10 to 15 min. On clinical examination, the dog was observed to have mild to no response to pain stimulation (no to slight withdrawal reflex). Pupils were dilated, and on complete blood count, leucocytosis observed. Random blood glucose revealed a blood glucose level of 28mg/dl, with Spo2 of 87% (normal->95%). The case was relatively confirmed for hypoglycaemic seizures based on history, clinical signs, and blood biochemistry. The case was treated with injection dextrose 10%, injection ringers (7ml/kg/hr), glucocorticoid lactate100ml dexamethasone1ml (0.5-1mg/kg) bid for 3days and oxygen supplementation.





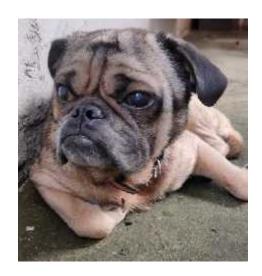


Fig-2. After Treatment

PRE AND POST-THERAPY HEMATOLOGICAL ANALYTES AND BIOCHEMICAL PARAMETERS

Table-1. Erythrogram Analytes

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Parameters	Day 1	Day 2	Day 3	Day15	Normal range		
TEC (× 10 6 /μl)	7.60	7.50	7.90	8.1	5.5-8.5		
Hb (gm/dl)	14.2	14.1	15.0	14.9	12-18		
PCV (%)	45.8	50	51.5	52	37-55		
MCV um ³	60.0	61	60	63	62-77		
MCH (Pcg)	18.7	18.9	19	23	21-26.2		
MCHC (gm/dL)	31.1	34.0	34.5	34.7	32-36		

Table-2. Leucogram Analytes

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Parameters	Day 1	Day 2	Day 3	Day15	Normal range			
WBC(/µL)	52200	36000	23000	15000	6000-17000			
Neutrophils(%)	62.5	63	66	70	62-80			
Lymphocytes(%)	24.5	22.6	18.2	15.2	10-28			
Monocytes(%)	13.0	10	7	5	3-9			
Eosinophils(%)	4.6	5.0	5.3	5.8	2-12			

Table-3. Random Blood Glucose

Glucose(g/dl)	28.0	104	134.2	105	70-120
Spo2 (%)	87-92	95-96	96	96-98	98-100

Results and Discussion

The dog has 28mg/dl of blood glucose in this case. That's why clinical signs were manifested in this case. In the current case the dog hasclinical manifestations like seizures, muscle twitching, lateral recumbency, loss of vision, and anorexia, hemo-biochemical parameters before initiation of treatment reveal leukocytosis, random glucose value was low, this is in accordance to the reports

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of Koenig, 2009 and Nelson, 2014. During therapy as well as the recovery period, all the parameters were restored within the normal range (Table 1)

The case was successfully treated with an injection of dextrose 10% after 1st day of administration, and there was a significant increase in glucose level and improvement in feed intake as also mentioned by Koenig, 2009 and Harmon, 2016. Glucocorticoids

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promote gluconeogenesis in the liver as also elucited by Kuo *et al.* 2015. In contrast, skeletal muscle and white adipose tissue decrease glucose uptake and utilization by antagonizing insulin response. Hence, in this case, dexamethasone is used.

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