

CASE REPORT

Diabetes mellitus and bilateral cataracts in a kitten

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An 18-week-old male domestic long-hair kitten was presented with a history of polyuria and polydipsia for several weeks. The general condition was unremarkable, but the kitten was considerably smaller than expected for the age and showed cataracts in both eyes. Serum glucose concentrations were persistently elevated and based on clinical findings and an elevated serum fructosamine concentration, a diagnosis of diabetes mellitus was established.

Diabetes mellitus is not commonly diagnosed in young kittens, nor are cataracts recognised as a frequent feature of this disease in cats. The cataracts progressed in spite of the insulin therapy and the kitten was euthanised 10 weeks after referral. Histopathological examination of the pancreas revealed few and small islets of Langerhans compared to the examination of pancreas from a healthy kitten of the same age. Histopathological changes in the eyes included cataracts affecting both cortex and nucleus.

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Diabetes mellitus is a common endocrine disease in adult, middle-aged to old cats (Panciera et al 1990, Thoresen & Grøndalen 1995). Cases of diabetes mellitus in young cats are rarely described (Woods et al 1994, Root et al 1995, Barnett & Crispin 1998). In dogs, diabetes mellitus is associated with a high incidence of bilateral cataracts that occur secondary to aberrations in carbohydrate metabolism, but this phenomenon is not a frequent feature of diabetes mellitus in cats. Feline cataract, when occurring, is almost always secondary to other eye diseases (Barnett & Crispin 1998).

In this report, a case of diabetes mellitus with concomitant bilateral cataracts in a kitten is presented and the quite uncommon features of the disease in this case are discussed.

Case history

An 18-week-old male domestic long-hair kitten, weighing 1.8 kg, was referred to The Norwegian School of Veterinary Science with a history of polyuria and polydipsia of 3 to 4 weeks duration. The kitten was small compared to the littermates,

although with normal body proportions (Fig 1). Its appetite had all along been normal. Vaccination status and worming protocol were fulfilled satisfactorily.

On clinical examination the kitten was alert and active. Body temperature was normal and pulse and respiration frequencies were within normal limits. The peripheral lymph nodes were palpably normal. Mucous membrane colour and capillary refill times were normal. No other abnormal clinical findings were found apart from the ocular changes.

Eye examination

The eyes were of normal size with no signs of intraocular inflammation. Direct and indirect pupillary light reflexes were normal in both eyes. Menace responses were inconsistent and difficult to evaluate. Intraocular pressures were 12 and 16 mmHg in the left and right eye, respectively. The cataracts in both eyes were immature and affected both nucleus and cortex (Fig 2a, b), but permitted examination of a normal fundus. During the following 2 months the cataracts progressed further, with the right eye most severely affected.

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Fig 1. Domestic long-hair kitten, 18-week-old. The kitten is smaller than expected but shows normal body proportions.

Clinical pathology

A blood sample was drawn from the jugular vein and a urine sample was collected by cystocentesis. Abnormal findings of clinical significance included glucosuria, hypocalcaemia and increased serum concentrations of alkaline phosphate, inorganic phosphate, glucose, and fructosamine.

The results of the haemogram, biochemical profile and urinalysis at the time of referral are listed in Tables 1, 2, and 3. The results of weekly serum fructosamine measurements during the 10 weeks treatment period are given in Fig 3.

Radiology

Left and right lateral and dorsoventral abdominal radiographs demonstrated no radiological abnormal findings.

Post-mortem examination

The kitten was euthanised 10 weeks after referral due to failure to respond to insulin treatment (Insulatard® NovoLet®, 0.8 IU/kg bw twice daily) and the progression of the bilateral cataracts. An autopsy was performed and the eyes were immediately removed and placed in 10% unbuffered formalin. The liver was enlarged and had a light brown colour with multiple, small red areas. Pancreas was of normal size and the middle portion had focal white spots with diameter up to 1 mm.

Tissue specimens were collected from various organs, including liver, kidneys and several sections of pancreas. The excised samples were fixed in 10% neutral buffered formalin, routinely

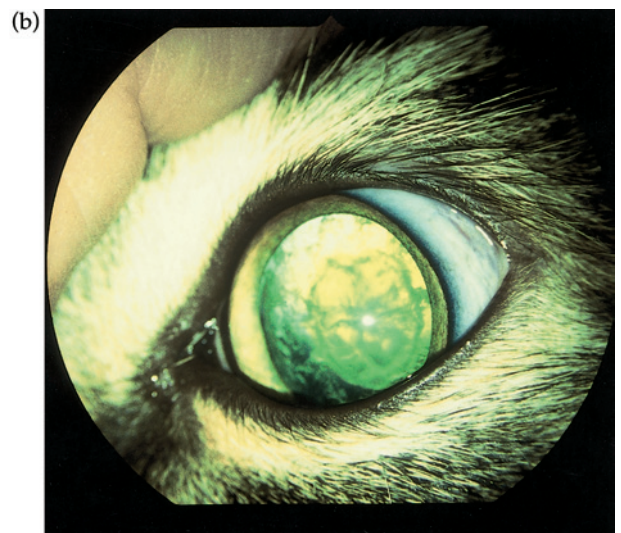


Fig 2a, b. Bilateral immature cataract in the 18-week-old kitten.

processed and embedded in paraffin. Sections were cut 3 to 5 µm thick and stained with haematoxylin and eosin (HE). Sections of pancreas were also stained with Congo red. Immunohistochemistry for insulin was performed on sections of pancreatic tissue. A guinea-pig polyclonal antibody against porcine insulin (DAKO A/S, Glostrup, Denmark) was used in an avidin-biotin complex (ABC) staining technique (Hsu et al 1981). Pancreas from a healthy 6-month-old cat was used as a positive control.

Few pancreatic islets were recognised in HE stained sections of the diabetic kitten (Fig 4) when compared with the control cat. The islet cells in the diabetic kitten showed degeneration with swollen, pale and vacuolated cytoplasm and peripherally located small nuclei (Fig 5).

Table 1. Haemogram

Parameter	Result	Reference range	Unit
RBC	6.6	(5.0–10.0)	T/L
HGB	99	(80–150)	g/L
HCT	0.32	(0.24–0.45)	L/L
MCV	48.1	(39–50)	fL
MCHC	311	(320–360)	g/L
RDW	13.9	(13.0–17.0)	%
HDW	16.8	(13.0–28.0)	g/L
PLT	Adequate	(190–400)	G/L
MPV	6.8	(5.5–8.5)	fL
WBC	7.3	(5.5–15.4)	G/L
NEUT	3.8	(2.5–12.5)	G/L
LYMP	3.1	(1.5–7.0)	G/L
MONO	0.1	(0–0.9)	G/L
EOS	0.3	(0–0.5)	G/L
BAS	0.0	(0–0.1)	G/L
LUC	0.0	na§	G/L

§Not applicable.

Table 2. Biochemistry profile

Parameter	Result	Reference range	Unit
AST	38	(0–60)	U/L
ALT	87	(0–75)	U/L
ALP	302	(0–90)	U/L
CK	505	(0–580)	U/L
Amylase	263	(0–800)	U/L
Protein (total)	71	(60–82)	g/L
Albumin	31	(25–39)	g/L
Globulins	40	(26–50)	g/L
Urea	8.3	(5.0–10.0)	mmol/L
Creatinine	75	(75–180)	µmol/L
Bile acids	10	(0–5)	µmol/L
Bilirubin (total)	1	(0–4)	µmol/L
Cholesterol	8.4	(1.5–6.0)	mmol/L
Glucose	23.5	(3.5–9.0)	mmol/L
Fructosamine	600	(146–271)	µmol/L
Inorganic phosphate	3.6	(1.0–2.8)	mmol/L
Calcium	2.9	(2.2–2.9)	mmol/L
Sodium	153	(150–165)	mmol/L
Potassium	3.4	(3.7–5.8)	mmol/L
Chloride	113	(112–129)	mmol/L

Amyloid was not present in the islets. The interstitial tissue of pancreas had multifocal infiltration with lymphocytes, neutrophils and histiocytes and moderate proliferation of fibrous tissue (Fig 4). The changes were most prominent close to the pancreatic ducts. Hyperplasia of duct epithelium was present in some areas.

Sections stained for insulin with the ABC procedure in the diabetic kitten (Fig 6a) showed positive reactivity in a few islet cells, compared with the control cat where more cells stained positive for insulin (Fig 6b). Microscopic examination of the liver and kidneys showed fatty degeneration of centrolobular hepatocytes and

Table 3. Urinalysis (sample collected by cystocentesis)

Parameter	Result	Reference range
Specific gravity	1.028	(1.035–1.060)
Color	Pale	na§
Turbidity	Clear	na§
pH	6.5	(5.5–7.0)
Glucose	Positive	(Negative)
Ketones	Negative	(Negative)
Bilirubin	Negative	(Negative)
Protein	Negative	(Negative)
Occult blood	Negative	(Negative)
WBC/hpf*	2	(0–3)
RBC/hpf*	3	(0–3)
Casts	Negative	(Negative)
Crystals	Negative	(Negative)
Bacteria	Negative	(Negative)
Culture	Negative	(Negative)

*High power field.

§Not applicable.

renal epithelial cells of the Henle's loop. Histopathological changes in the eyes were limited to the lens. There was evidence of migration of lens epithelial cells beneath the surface of the posterior capsule, and in an area of capsular wrinkling fibrous metaplasia was present (Fig 7). The cortical changes were dramatic and include membranolysis of lens fibre cells and liquefaction of lens proteins. The anterior capsule and subcapsular cortex showed equatorial vacuolisation and degeneration of the lens epithelium, with associated subcapsular cortical membranolysis and liquefaction (Fig 8).

Discussion

The problems identified during the initial database collection in this case includes polyuria and polydipsia, glucosuria, small but proportional body size, bilateral cataracts, hypocalaemia and significantly increased levels of serum alkaline phosphate, inorganic phosphate, glucose, and fructosamine.

In this case, the presented problem list justifies the suspicion of diabetes mellitus. The single diagnostic criterion for diabetes mellitus is persistent hyperglycaemia. However, one single demonstration of hyperglycaemia might be due to several other causes than diabetes mellitus. Especially in cats, stress and drug induced short-term hyperglycaemia is a well-known

phenomenon (Feldberg & Symonds 1980, Hsu & Hemborough 1982, Weiland et al 1984, Opitz 1990, Crenshaw et al 1996).

Measurements of serum fructosamine (glycated serum proteins) and glycated haemoglobin are increasingly used to replace plasma glucose concentration to diagnose diabetes mellitus and to monitor the response to insulin treatment in several species (Baker et al 1983, Salemans et al 1987, Akol et al 1992, Reusch et al 1993, Thoresen & Bredal 1996). These measurements are not affected by transient changes in glucose concentration. An initially, pre-treatment elevated serum fructosamine concentration usually verifies a persistent hyperglycaemia. Another major factor that may affect the serum fructosamine concentration, besides the average serum glucose concentration is the serum albumin concentration (Dolhofer & Wieland 1980, Iberg & Flückiger 1986). In this kitten protein status was normal, implying that the elevated serum fructosamine concentration was caused by a persistent alteration in plasma glucose concentration only.

Chronically elevated serum fructosamine concentrations in this kitten for the 10 weeks treatment period reflect a continuing persistent hyperglycaemia in spite of the insulin administration. Verifying that the kitten received the insulin in a proper manner, and that the insulin was stored as recommended, the results indicate insulin resistance. This may result from problems occurring before the interaction of insulin with its receptor, at the receptor, or at steps distal to the interaction of insulin and its receptor (Ihle & Nelson 1991). Pre-receptor problems reduce free metabolically active insulin concentration and include increased insulin degradation and insulin-binding antibodies. Circulating insulin-binding antibodies were not measured in the present case. In dogs and cats, receptor and post-receptor abnormalities are usually caused by obesity or a disorder causing excessive secretion of a diabetogenic hormone, ie, acromegaly and hyperadrenocorticism. Obesity was certainly not the case, and no other disease involving diabetogenic hormones was tested for. In the cat, acromegaly is caused by a pituitary tumour that secretes excess growth hormone. In the present case, no clinical signs indicative of acromegaly were present and acromegaly is less likely to be suspected in a cat that lacks typical conformational alterations. In cases suspected to suffer from acromegaly, a definite diagnosis requires documentation of increased baseline

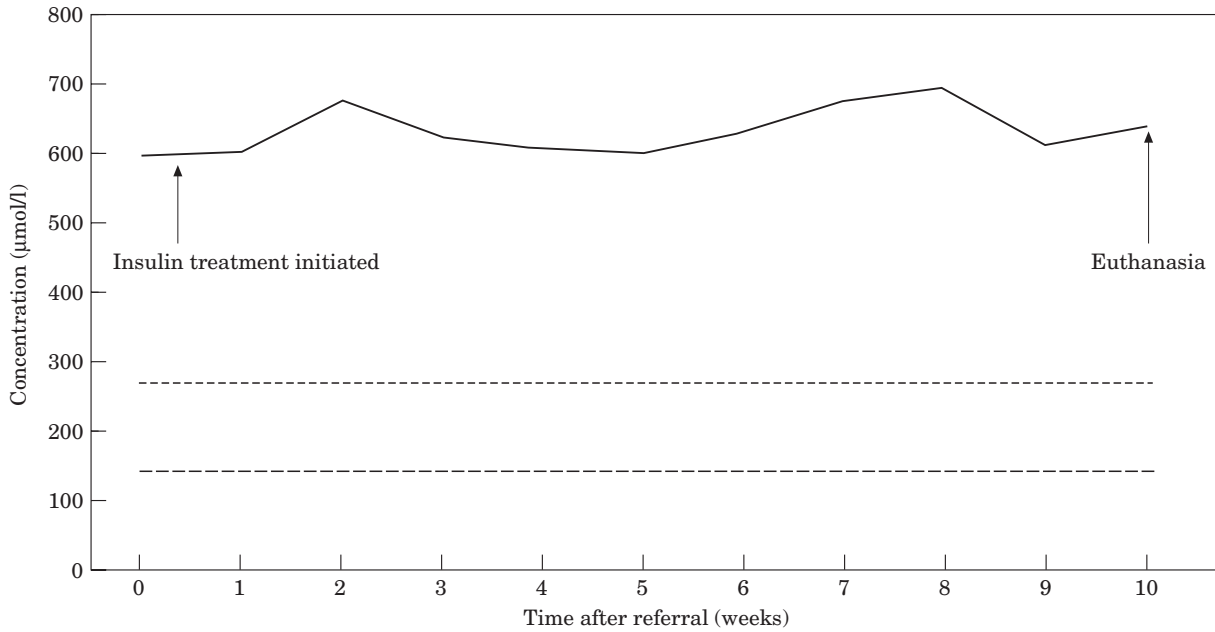


Fig 3. Serum fructosamine concentrations (—) measured weekly during the 10-week treatment period. Upper reference limit, - - -; lower reference limit, - - -.

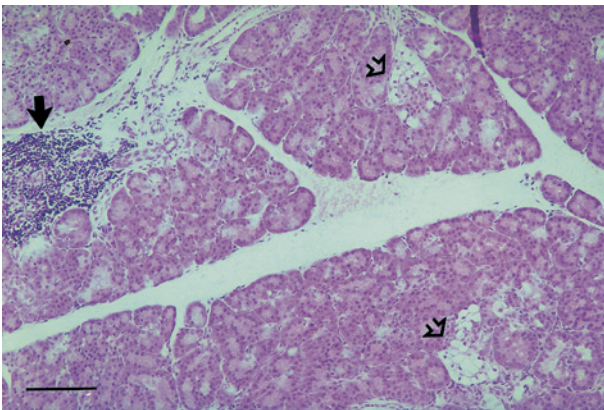


Fig 4. Pancreas, 18-week-old kitten with diabetes mellitus. Pancreatic islets contain several vacuolated cells (open arrows). Focal interstitial aggregates of leucocytes (black arrow) are present. Haematoxylin and eosin. Bar=100 µm.

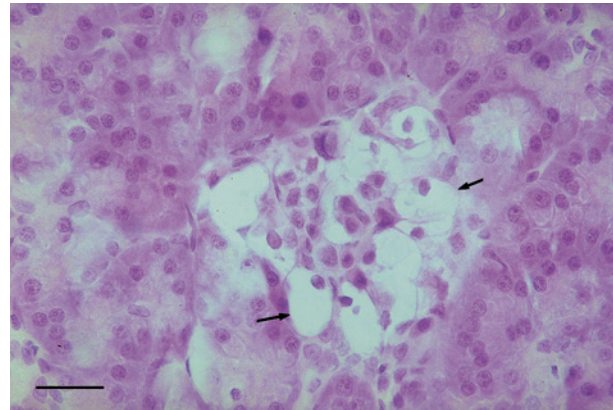


Fig 5. Pancreas, 18-week-old kitten with diabetes mellitus. Higher magnification of a pancreatic islet with degeneration. Some islet cells have pale, vacuolated cytoplasm and peripherally located, small nuclei (arrows). Haematoxylin and eosin. Bar=25 µm.

serum growth hormone or IGF-1 concentrations. However, due to lack of consistent clinical signs and that such measurements are not readily available, they were not performed.

Hyperadrenocorticism is rare in the cat (Watson & Herrtage 1998). Without any clinical signs suggestive of the disease, further testing was not done.

Cataracts were present in both eyes. Signs of other eye diseases were not observed. Primary cataract is an infrequent entity in the cat. Cataract associated with multiple ocular defects, including microphthalmia, occasionally occurs in cats (Peiffer & Gelatt 1974), but in the present

case the eyes were considered to be of normal size with no concomitant ocular defects. Primary congenital cataracts and suspected hereditary cataracts have been reported (Peiffer & Gelatt 1975, Schwink 1986, Rubin 1986), but are considered uncommon in domestic cats. Diabetic cataract has been described as rare in the cat and is certainly much less common than in the dog, and of slower onset. Reports on diabetic cataract in very young cats are few. Barnett & Crispin (1998) reported diabetic cataract in a 22-week-old Chinchilla cross Persian male kitten, and James (1960) reported cataract as one of the findings in

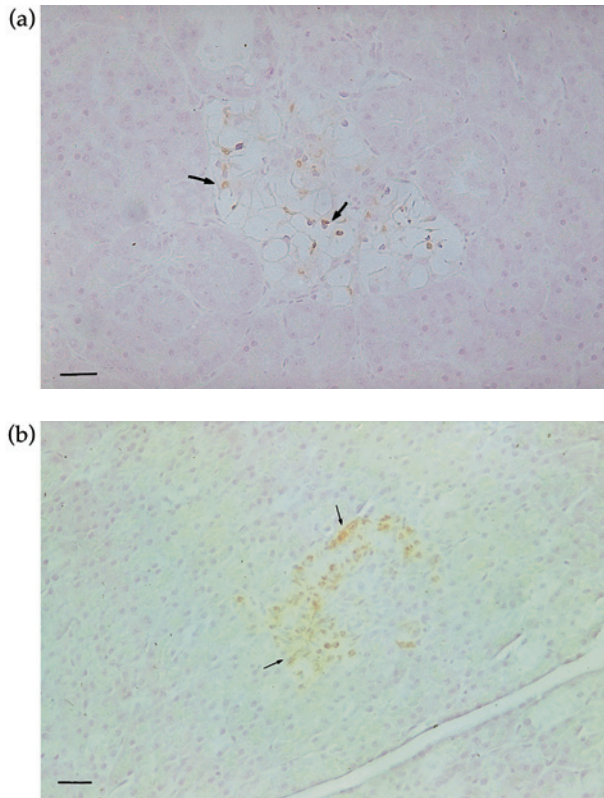


Fig 6. (a) Pancreas, of 18-week-old kitten with diabetes mellitus, stained for insulin. A few islet cells are positive for insulin (arrows). ABC staining technique, haematoxylin counterstain. Bar=25 µm. (b) Pancreas of a 6-month-old control kitten, stained for insulin. Several islet cells show reactivity for insulin (arrows). ABC staining technique, haematoxylin counterstain. Bar=25 µm.

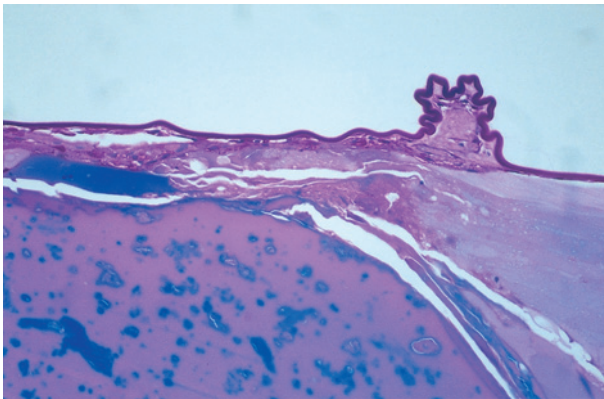


Fig 7. Posterior equatorial lens capsule showing capsular wrinkling, indicative of liquefaction and leakage of cataract proteins. Periodic acid-Schiff stain $\times 200$.

a 7-year-old domestic short-hair cat. Initial lens changes in diabetic cataract in cats, when occurring, are described as equatorial lens vacuoles (Glaze & Gelatt 1999). This is in accordance with the findings in the present case report. In a study by Taylor et al (1997) diabetic canine cataractous

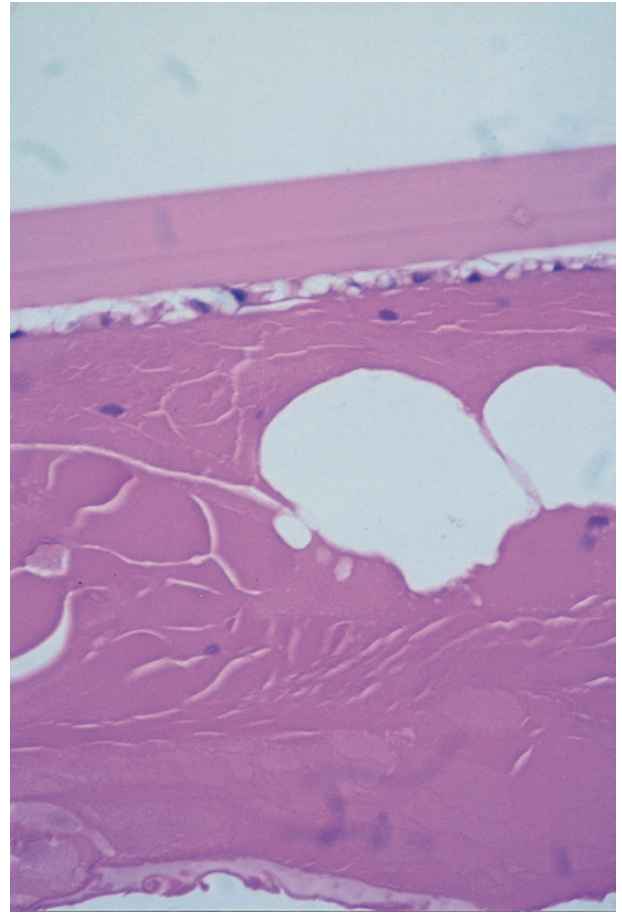


Fig 8. The anterior lens capsule and subcapsular cortex. Haematoxylin and eosin $\times 400$.

lenses were completely opaque in both the cortex and nucleus. It has been reported that formation of diabetic cataract is mainly due to osmotic swelling and the rupture of lens fibre cells caused by reduction of the excess glucose via the hexose monophosphate shunt resulting in intracellular accumulation of sorbitol (Kinoshita 1965, Peiffer et al 1977). The question why diabetic cataracts in cats are so infrequent has not yet been answered. A possible explanation for the species difference may be related to differences in aldose reductase activity. Aldose reductase catalyses the reduction of glucose to sorbitol. Thus, diabetic cataractogenesis may be dependent not only on the amount of glucose available but the level of lens aldose reductase activity as well. Studies have shown that there is a species difference in lens aldose reductase levels corresponding with the susceptibility to form cataracts (Datiles & Kinoshita 2000). In addition to the osmotic effect of sorbitol, there is evidence that sorbitol also directly interacts with lens proteins (Basher & Roberts 1995), causing amino

acid modification and lens protein insolubility with ensuing cataract formation. Glycation may bring about a conformational change in the proteins such that the buried sulphhydryl groups become exposed (Whikehart 1994). One of the susceptible amino acids is lysine that can react with a carbonyl group to form what is known as a Schiff base. The most important sources of free carbonyl groups in cells are sugars; hence, the process is referred to as glycation. Should the Schiff base progress to form a more stable Amadori product, the end result will also be unfolding of proteins and cross-linking into aggregates. Aggregates arising from sugar-protein adduct formation have been called advanced glycation end products (AGEs) (Brownlee et al 1988). It has also been reported that the glycation makes the proteins more sensitive to oxidative stress caused by lowered pH and visible light (Kamei 1991). The fact that the insolubility of the lens proteins is accelerated by glucose in the presence of visible light suggests that light may enhance the effects of glucose on the lens protein aggregation and insolubilisation.

Insolubilisation of the lens proteins by glucose is also increased under acidic conditions. The pH level in blood is decreased in diabetic acidosis and glucose may therefore exhibit a more effective action to insolubilise the lens proteins in diabetic patients. Based on the case history and the clinical signs the cataracts were considered most likely to be secondary to diabetes mellitus.

Microscopic examination of pancreas showed few and atrophic islets, indicating that a hypoplasia may be present in addition to degeneration. Normally, the distribution and number of islets vary in different parts of pancreas and also with age (Bencosme & Liepa 1955, Jubb 1993, Xu et al 1999). However, sections from 10 different areas of pancreas were examined and only a few islets were identified, supporting the diagnosis of hypoplasia of pancreatic islets. In contrast, islets were more numerous in various parts of pancreas of the age-matched control.

The vacuolar degeneration, which was present in islet cells of this kitten, is in accordance with findings reported in diabetes in dogs and cats (Atkins et al 1988, Jubb 1993). Amyloid deposits in the islets were not observed in the present case, although amyloidosis of the islets is reported as a common feature of feline diabetes (O'Brien et al 1986). Findings consistent with a moderate interstitial pancreatitis were observed in the diabetic kitten, but the acinar,

exocrine part of pancreas was apparently normal. Therefore, it seems unlikely that an interstitial inflammatory reaction has contributed to the lesions of the islets. Diabetes mellitus in a kitten showing bilateral cataracts is uncommon. The pathological findings showed that this case of diabetes mellitus was apparently due to pancreatic islet hypoplasia. The development of bilateral cataracts is considered most likely secondary to diabetes mellitus.

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