Reversible pupillary miosis during a hyperglycaemic episode: case report

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Summary. A young female patient presented with non-ketotic hyperglycaemia, pupillary miosis and clouding of consciousness. The initial blood glucose was 83.3 mmol/l; there was no metabolic acidosis. After insulin therapy and improved diabetic control, pupillary function returned to normal. There

was no other associated somatic or autonomic nerve dysfunction.

Key words: Pupillary miosis, non-ketotic hyperglycaemia.

In recent years many authors have studied pupil size and motility in diabetic subjects [1–5]. Hreidarsson [4] concluded that the resting pupil size in darkness decreased with increasing duration of diabetes. This would place the abnormally small pupil as an integral part of the long-term diabetic syndrome. Metabolic derangement has also been associated with a markedly reduced ability to maintain miosis in continuous light [3].

Acute reversible pupillary changes have been described in Type 1 (insulin-dependent) diabetes during phases of poor metabolic control and redilation time was prolonged from 2.26 to 2.90 s [3]. These changes were related to diabetic control and may be analogous to the well established reversible functional change in the somatic neuro system in early diabetes. We report a patient showing acute reversible pupillary changes in the non-ketotic hyperglycaemic state.

Case report

A 34-year-old single black lady walked into the Emergency Department of the Touro Infirmary complaining of weakness, blurred vision and visual hallucinations for 3 days. An accurate history could not be obtained, but there appeared to be no history of polydipsia, polyuria, nausea, vomiting or weight loss. Her past history revealed that 18 months ago she had been treated for hypertension for 2 months with 20 mg furosemide/day. History obtained from her immediate family excluded previous diabetes, drug intake of any kind during the

past year, or abnormal blood pressure readings within the past 18 months.

Her general physical examination upon admission demonstrated clouded sensorium and pinpoint pupils. Vital signs were: blood pressure: 130/95 mmHg; pulse rate: 78 beats/min; temperature: 37 °C; respirations: 16 breaths/min; height: 175 cm; weight: 75 kg.

Naloxone (1 ml) was given intravenously with no effect. Laboratory results on admission included blood glucose: 83.3 mmol/l; sodium: 139 mmol/l; potassium: 3.6 mmol/l; chloride: 76 mmol/l; and carbon dioxide: 30 mmol/l. Urinary analysis showed a specific gravity of 1.025; glucose 2+; protein 1+, but was negative for ketones.

The patient was admitted to the Intensive Care Unit and intravenous insulin treatment was started with a pump delivering 10 U insulin/h, and the glucose value was estimated every 3 h. On day 2 after admission, the patient required 40 U insulin. Her biochemical picture had improved remarkably with a blood glucose level of 15.5 mmol/l; there was no proteinuria and her creatinine clearance was 100 ml/min. The patient's sensorium, however, remained clouded. Eye examination revealed the pupils to be equal and reactive to light with a diameter of 5 mm.

The patient maintained a blood glucose level ranging from 11.11 to 15.00 mmol/l on 35 U insulin/day over the first 3 days in hospital. Her insulin requirement dropped to 10 U/day on days 4 and 5 of admission. On day 4 after admission, the patient's level of consciousness improved and she related that she was amnesic for

Fig. 1. a and b. The state of the pupils of a 34-year-old black lady with non-ketotic acute hyperglycaemia: **a** Pupillary miosis upon initial presentation; **b** pupils 1 day after onset of treatment

the past 3 days. The patient was discharged from hospital on 10 U insulin/day after 8 days in hospital without the development of complications. Her blood glucose level on discharge was 5.5 mmol/l.

Results

The initial bedside clinical assessment revealed an uncorrected visual acuity of 20/40 for each eye. Tension with a Schiottz tonometer (Storz, St. Louis, Missouri, USA) demonstrated intraocular pressures of 14 and 18 mmHg for the right and left eyes, respectively. No conjunctival injection was noted and the corneas were clear. Both pupils were miotic and measured 1 mm in diameter with questionable reaction to light (Fig. 1 a). The severe miotic state of the pupils precluded clear documentation of the pupillary reaction to near, direct and consensual stimuli. The patient's accommodative status could not be assessed, but she did demonstrate 10 diopters of convergence to near stimuli. Her extraocular muscles had full motion with intact fields of gaze.

On day 2 after admission, her eye examination revealed pupil size to be equal, round and reactive to light with a diameter of 5 mm (Fig. 1 b). Near, direct and consensual pupillary reflexes were normal and all eye versions were intact. Her visual acuity was 20/20 in each eye and refraction revealed her eyes to be ametropic. Fundus examination was within normal limits.

Discussion

Focal neurological deficits in non-ketotic acute hyperglycaemia is a relatively recent concept first demonstrated in 1968 by Maccario [8]. He concluded that hyperglycaemia can cause metabolic derangements that could lead to focal somatic reversible neurological dysfunction. Pupillary changes could be present, but according to Maccario they were not reversible and therefore not related to the hyperglycaemia, but rather to long-standing diabetes.

Since that time, further reports on patients with non-ketotic hyperglycaemia have suggested that the various

focal neurological signs described might indicate brain stem dysfunction as well as autonomic nervous system disturbances. [3, 6–10]. Yet the first and only mention of reversible autonomic nervous system disturbances and specific pupillary changes were mentioned by Hreidarsson [3]. His report, however, involved short-term Type 1 diabetic patients in whom he documented reversible prolongation of redilation time with poor control. However, the miosis in these subjects was only moderate and could not be sustained. Our case report supports Hreidarsson's conclusion that reversible changes in the autonomic nervous system do occur with poor metabolic control.

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