

Transient cataracts in a diabetic child with hyperosmolar coma

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Temporary lens opacities in diabetic coma were noted by Lawrence, Oakley, and Barne (1942) and thought to be common if the lens was carefully examined. This does not appear to have been frequently discussed, but Sheldon and Pyke (1968) showed pictures of opacities in the lens of a girl aged 10 years, which cleared rapidly with treatment of the diabetic coma. Neuberg, Griscom, and Burns (1958) found only six documented case reports to show acute development and complete reversal of diabetic cataracts and four cases in which the development was unknown but clearing occurred completely.

Because only Lawrence and others (1942) noticed complete opacification of the lens in diabetic coma, we thought it valuable to report this case of transient complete opacification of both lenses, complicating hyperosmolar, hyperglycaemic, hypernatraemic coma in a diabetic child of nearly 4 years old; this type of diabetic coma is rare in childhood (McCurdy, 1970).

Case report

This normal baby girl was first admitted soon after her second birthday with diabetic precoma. After rehydration, she was stabilized on a diet containing 110 g. carbohydrate and 8 units of soluble insulin in the morning and 4 units at night.

One month before her 4th birthday she was re-admitted with a history of "strange behaviour", anorexia, polyuria, polydipsia, and misery, but without diarrhoea or vomiting, which had been present for 1 week.

Examination

She was very confused, agitated, unco-operative, and unresponsive, but moving all four limbs. The eyes were sunken and the tongue dry and furred, but the elasticity of the skin was not abnormal. The plantar reflexes were extensor but no localizing neurological signs were noted. The fundi and lenses were normal. Blood pressure 100/75 mm. Hg; pulse rate 160/min. with a poor pulse volume; respiration 40/min., but not typically acidotic.

Laboratory examination

Plasma urea 136 mg./100 ml.; plasma sodium 168 mEq/l.; plasma potassium 4.0 mEq/l.; plasma chloride 133 mEq/l.; plasma bicarbonate 16 mEq/l.; true blood glucose 2,000 mg./100 ml.; plasma ketones present but not measured.

Urine contained 2 per cent. glucose and ketones ++.

Lumbar puncture normal apart from 300 mg./100 ml. sugar.

Her weight was 15.1 kg., which was 1.1 kg. more than the weight recorded in the out-patients department 2 weeks earlier.

Diagnosis

Diabetic hyperosmolar dehydration.

Treatment

In the first 6 hours 60 units of soluble insulin were given, half intravenously, with 360 ml. normal saline and 150 ml. half normal saline with 2.29 per cent. Dextrose containing 25 mEq potassium/litre. Ampicillin 250 mg. 6-hrly and phenobarbitone 30 mg. 8-hrly were also given. The child became very stiff with opisthotonos 3 hours after admission but improved with 10 mg. intravenous diazepam and this was repeated approximately every 8 hrs.

Progress

7 hrs after admission the true blood glucose was 80 mg./100 ml.; blood urea 54 ml./100 ml.; bicarbonate 23 m Eq/l.; other electrolytes remained the same. By this time only 90 ml. urine had been passed and none in the past 4 hrs. In view of the anuria and development of opisthotonos 70 ml. 20 per cent. mannitol was given intravenously. This produced a diuresis and the rate of intravenous drip continued at 25 ml./hr, which is approximately the daily requirement for a child of this age. When the low blood sugar was recorded, 10 ml. 50 per cent. dextrose was given every 2 hrs, but the Dextrostix always showed a blood sugar approximately 100 mg./100 ml.

20 hrs after admission both lenses were completely opaque and no red reflex could be seen at all. The electrolyte picture at this time was blood urea 42 mg./100 ml.; plasma sodium 165 mEq/l.; potassium 3.0 m Eq/l.; chloride 123 mEq/l.; bicarbonate 27 mEq/l.; true blood glucose 155 mg./100 ml. Intravenous fluid was continued at the same rate and 4 hrs later a red rim could be seen around the lens opacity.

29 hrs after admission the lens had a dense stellate central area, which 3 hrs later had fragmented and was noticed to be involving both the anterior and posterior cortex (Figure). 24 hrs after the first noticed, there were just a few opacities scattered in the lenses and some of these were in the form of bubbles. 12 hrs later (36 hrs after the onset of the opacities) the lenses were completely normal. Throughout this period of 36 hrs the electrolytes, urea, and sugar did not change significantly from the figures last noted.

4 days after admission the electrolytes had returned completely to normal and the blood glucose remained between 100 and 300 mg./100 ml. At this time the child regained consciousness and started talking. She has since made a complete recovery neurologically and now attends a normal school. 16 months later the lenses were absolutely clear on slit-lamp examination.

Comment

There is no doubt that on admission this patient showed the characteristics of a severe case of hyperosmolar, nonketotic diabetic coma, as described in children by Rubin, Kramer, and Drash (1969). The calculated serum osmolality

$$\left[2 (\text{serum Na mEq/l}) + \frac{\text{blood sugar in mg./100 ml.}}{18} \right]$$

was 447 mOsm/l., compared with the normal in childhood of 297 ± 2 (Rubin and others, 1969). Although ketones were present in the urine and blood, they were not sufficient to increase the unidentified ion gap beyond the normal of 23 mEq/l. Unlike most of the cases described by Rubin and others (1969), this child was neurologically normal, had previously been diagnosed as diabetic, and recovered with no residual disability.

Treatment of this condition is difficult in adults (McCurdy, 1970) and in children (Rubin and others, 1969), and, with our lack of experience, we relied upon the principles we use to treat hypernatraemic dehydration in infants—*i.e.* restoration of vascular volume with normal saline or plasma and slow replacement of intracellular electrolytes and water

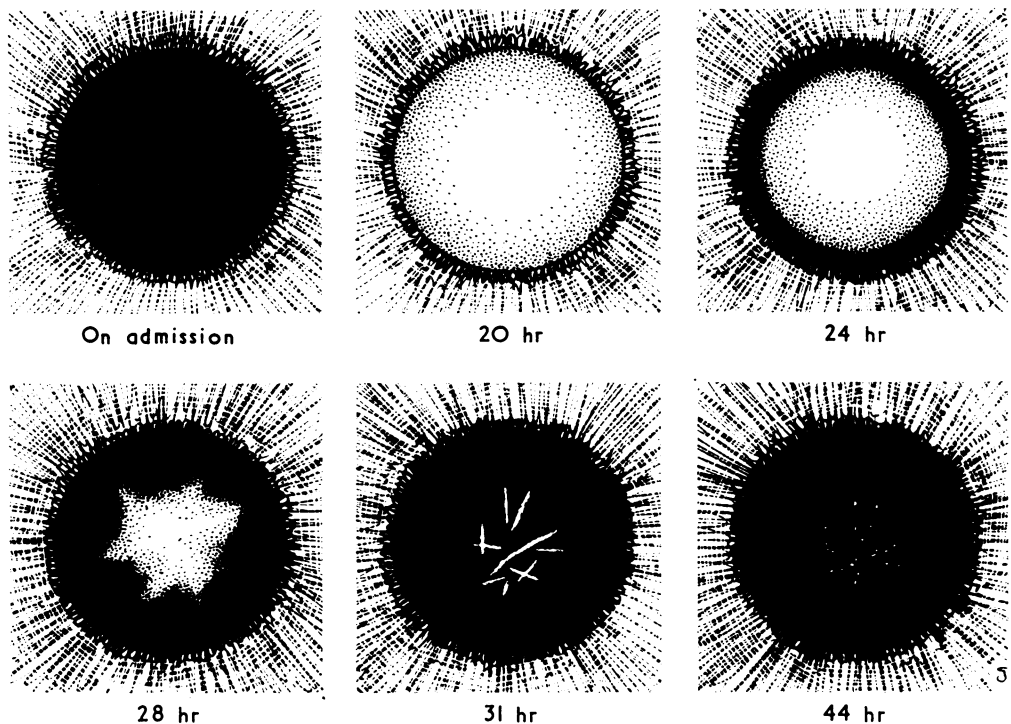


FIGURE *Appearances of lens related to time after admission*

by giving the daily requirements of fluid containing at least 50 mEq/l. of sodium. The use of lower concentrations of saline causes a rapid reduction of serum osmolality and cerebral oedema, possibly leading to permanent neurological damage (Finberg, 1969). This child developed opisthotonos and generalized stiffness and these were probably signs of cerebral oedema due to reduction of serum osmolality, caused by the rapid drop in the true blood glucose. This cerebral oedema was treated successfully by diazepam and mannitol.

In the non-diabetic child a relationship between hypernatraemia and hyperglycaemia is now clearly established (Stevenson and Bowyer, 1970). Jung and Done (1969) believed that many cases of "transient diabetes" in the newborn were in fact not diabetic at all, but that the biochemical picture was due to insufficient dilution of the infant feeds. Burman and Glaspole (1964) attributed the combination of hyperglycaemia and hypernatraemia to an excessive carbohydrate intake as treatment for gastroenteritis. Stevenson and Bowyer (1970) suggested that, when this combination occurred in gastroenteritis, the release of glucose was secondary to the hypernatraemia and there is experimental evidence to support this (Sotos, Dodge, and Talbot, 1962; Nitzan and Zelmanovsky, 1968). The association between hyperglycaemia and hypernatraemia has also been noted in heatstroke and this is then attributed to a stress reaction (Monteleone and Keefe, 1969). Whatever the mechanism, the association of hyperglycaemia in non-diabetic infants certainly occurs and in a case reported by Jung and Done (1969) the blood sugar was as high as 970 mg./100 ml. There may then be a vicious circle in hyperosmolar nonketotic diabetic coma, whereby the hypernatraemia further exaggerates the hyperglycaemia. This may account for the extremely high blood sugars which have been observed and for the danger of giving too much insulin (McCurdy, 1970).

By the time complete opacification of the lenses was noticed in our patient, the blood urea had fallen from 136 to 42 mg./100 ml., and the bicarbonate had risen from 16 to 29 mEq/l. The true blood glucose had fallen from 2,000 to 155 mg./100 ml. and the calculated serum osmolality from 447 to 339 mOsm/l. The major changes obviously took place in the sugar and the serum osmolality and these changes may well be related to the development of the cataracts. Experimentally, if the lens is exposed to a high concentration of sugar in the aqueous, the intracellular concentration of polyols increases significantly (van Heyningen, 1959) and this accumulation is significantly greater in younger than older animals (Kinoshita, Merola, and Tung, 1968). When the sugar is glucose, sorbitol and fructose, normally present in the lens, increase greatly. When the sugar is galactose, as in most experimental work, then dulcitol is found in the lens. Thus both osmolality of the lens and aqueous are increased. In our patient, treatment produced a sudden, large decrease in the osmolality of the serum, and so of the aqueous, leading to hydration of the lens. If, as is likely, the lens still retains an increased sugar content, the osmotic swelling of the lens is increased further. This in turn damages the sodium and potassium pumps in the lens (Kinoshita, Merola, and Hayman, 1965). These pumps normally maintain a high potassium and a low sodium level inside the lens and are essential to maintain a high concentration of negatively charged macromolecules (Duncan and Croghan, 1969). The electropotential differences across individual lens fibres and across the lens capsule have an active ion-pumping effect which is reduced in a hydrated lens (Sperelakis and Potts, 1959). Sodium therefore accumulates, and with it more water, and the potassium concentration decreases. The aminoacid pump also becomes defective and protein metabolism can be interfered with through decreased diffusion of amino acids from the aqueous into the lens (Dische, 1968). If the cataract is transient, as in our patient, it is unlikely that actual coagulation of the lens protein has occurred. An alternative and more likely explanation is suggested by Philipson (1969): in the hydropic lens of experimental galactose cataracts, he found vacuoles and irregular interfaces between differing protein concentrations in the lens, and measurement of the angular distribution of scattered light supports his theory that light scattering is the cause of the apparent opacification of the lens.

It is thus possible to see that, in our patient, the reduction in serum and aqueous osmolality after a period of hyperglycaemia could readily lead to lens hydration and an influx of sodium, exaggerated by the hypernatraemia. The hydration of the lens, if prolonged, would cause loss of transparency through interference with the protein metabolism of the lens fibres. In the transient cataracts in our patient, it is possible that light scattering by the swollen lens fibres may be a more likely explanation.

Many of the acute cataracts in diabetes, discussed by Lawrence and others (1942) and by Neuberg and others (1958), occurred after treatment was begun, and may be related to changes in hydration and blood sugar. Only three cases (Lawrence and others, 1942; Alt, 1906; Neuberg and others, 1958) took only hours to develop, and only two (Lawrence and others, 1942; Sheldon and Pyke, 1968) returned to normal in a matter of hours. Neither of these two patients had the complete opacification of the lenses that we observed and in neither are the biochemical details given. Our patient's cataracts appeared to be the result of a very unusual combination of factors.

Summary

A child was admitted to hospital with hyperosmolar diabetic coma. 20 hours after

admission both lenses were completely opaque but 36 hours later they were completely normal again. The mechanism of this phenomenon is discussed.

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