Medical Memoranda

Three Diabetic Mongol Idiots

Diabetes mellitus has not so far been reported in mongol children, and it is at least of interest to record briefly 3 cases I have been asked to treat (?) in the last ten years. However much one might consider such therapy wasted and displaced, this has not been the parental attitude, and I have had the opportunity of seeing the response these patients made to insulin treatment over some years. As all 3 cases were typical mongols I shall make no detailed reference to their physical and mental mongol characteristics.

Hundreds of detailed studies of these idiots have been published, including a few recent investigations of their metabolism. Schachter (1941) found no chemical blood abnormalities in his own and other reported cases, but does not mention the blood sugar. O'Leary (1931) and Bixby (1940) have published a series of glucose-tolerance tests which gave normal results. The occurrence of diabetes in these 3 cases therefore seems fortuitous and the two conditions in no way aetiologically related. It should be noted that each child had a distant relative who was diabetic.

DETAILS OF 3 CASES

Case 1.—Female, fifth and last child of healthy family (one grandfather died from diabetes); developed thirst polyuria and pruritus vulvae in 1931 at the age of 10. Given 4 units b.d. irregularly at first, and glycosuria and ketosis were heavy; 10 units b.d. by 1933, when I first saw the child. The urine then contained 4 to 6% of sugar, moderate ketosis was present, and I reluctantly advised more insulin. Some 20 units b.d. were given for the next 9 years, and this miraculously maintained an active and well-nourished condition, with no occurrence of either serious diabetic symptoms or hypoglycaemia. Menstruation has occurred irregularly since the age of 15. In February, 1942, she had 5 convulsions at irregular intervals, and was again brought to me. From their timing these were clearly hypoglycaemic attacks, and they have been stopped by reducing the insulin to 16 units b.d. When tested the urine was sugar- and ketone-free at the expected times, but contained a moderate cloud of albumin; a blood-sugar estimation showed 244 mg. per 100 c.cm. Her general health and nutrition at this age (21) were excellent, but she had developed bilateral lens opacities: from an opaque central nuclear opacity water cleavages radiated out in spokes to the periphery of the lens, where coronary opacities were present in a complete circle. This pattern was quite different from the usual cataracts described in mongol idiots, and was presumably diabetic in origin. In every way her response to insulin has been that of a normal child, but her physical and mental characters remain typically mongolian.

Case 2.—Girl aged 12, first living child. One elderly first cousin of the mother had diabetes. Diabetes developed acutely at 5 years of age, with thirst, polyuria, and heavy glycosuria. For 18 months restricted diet alone had been used, but for 5 years insulin had been given with an almost normal diet and a dosage of 6 to 16 units b.d., varying with the urine tests. The parents had little difficulty in controlling the sugar, and ketosis had remained absent. The eyes were normal, and physical diabetic complications were absent, but there was widespread alopecia. The parents were harassed by the care necessary, and particularly by sleepless habits, but were devoted to the child's welfare.

Case 3.—Male; one grand-aunt diabetic in a large family. Patient developed acute diabetic symptoms at the age of 10. I then found 5% of glycosuria and heavy ketonuria, but a blood-sugar test was impossible. Early coma was imminent, and at the mother's request I sent a nurse to their home to establish a diet-and-insulin routine. Response was immediate, and 2 years later the child was well on 14 and 8 units of insulin. Since then the case has been untraceable, as the family has moved and the practitioner in charge died and left no records.

These patients responded as other children to insulin treatment—one for 10 years and another for 6 years; the third could not be traced after 2 years. Their control was not unduly difficult. This treatment made no difference to the mongol state, and I regard its efficacy with mixed feelings.

London, W.

R. D. LAWRENCE, M.D., F.R.C.P.

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Chronic Meningococcal Septicaemia

The following case of chronic meningococcal septicaemia is recorded because it differs in some respects from the syndrome described in recent literature. Stott and Copeman (1940) analysed 17 cases out of a total of 27 seen in France among members of the B.E.F., and the 4 cardinal symptoms are given as recurrent tender skin lesions, headache, muscle and joint pains, and joint effusions, associated with intermittent or relapsing fever. All these cases occurred during an outbreak of cerebrospinal fever, and, as the authors point out, meningococcal septicaemia becomes common whenever meningococcal meningitis is prevalent in a community. The following case was of a sporadic type, and there was no history of contact with cerebrospinal fever.

CASE HISTORY

The patient, a well-built athletic airman aged 25, was admitted to an emergency hospital on Sept. 15, 1941, with a pyrexial illness diagnosed by the station medical officer as acute rheumatism with Aschoff's nodes; this diagnosis was probably influenced by a history of acute rheumatism 4 years previously. The present condition started on Sept. 12, when he felt shivery, complained of pains in the legs and back, and developed what he described as "painful lumps on the left arm." He was kept in the station sick-quarters for observation, but pyrexia accompanied by the painful lumps on the left arm persisted, so he was transferred to hospital. For the first 24 hours after admission he was afebrile, with no signs or symptoms apart from 4 painful papules on the extensor surface of the left arm. The lesions were oval in shape, varied from a quarter to half an inch in length, were dusky red in colour, and in appearance closely resembled "blind boils." The largest lesion was directly over the olecranon process and was very tender. For the next 4 days the patient felt quite well, but had an evening rise of temperature to 99° F. The painful lesions on the arm disappeared, leaving no trace of discoloration of the skin.

On the ninth day of illness he developed a temperature of 102° F. and complained of a slight pain in the left mammary region, where a raised red oval papule about half an inch in length was found just below the nipple. This was very tender on pressure, and disappeared within 48 hours. His temperature for the next 5 days began to swing up to 102° F. every night; he developed no further cutaneous lesions and was, except for fever, completely symptomless. A leucocyte count at this time showed 10,700 cells per c.mm., 76% polymorphs. A blood culture was sterile after 72 hours' incubation. Following this period of pyrexia he was afebrile for 48 hours, after which his temperature again rose each evening to 102° F. During this second febrile period, which was unaccompanied by any skin lesions, a blood culture resulted in the isolation of a pure growth of Group I meningococcus. He was immediately given sulphapyridine by mouth, 1 g. four-hourly, and the temperature returned to normal within 2 days; there was no further rise of temperature during the rest of his stay in hospital. Sulphapyridine was discontinued after 10 days. The dosage was 1 g. four-hourly for 48 hours, then 1 g. thrice a day for the rest of the period, making a total of 36 g.

COMMENTARY

Col. J. C. Kennedy (1926) reported a series of cases in which he emphasized the importance of the rash as a diagnostic feature and described it as a recurrent erythema multiforme distributed sometimes over the whole of the body but showing a predilection for the limbs. The meningococcus was actually recovered from the centre of the nodule in one of his cases. Stott and Copeman (1940) suggested that chronic meningococcal septicaemia occurring sporadically is not rare, and that the disease presents a characteristic clinical picture which enables bedside diagnosis to be made with ease.

It is submitted that chronic meningococcal septicaemia may not always present such a typical clinical picture, particularly when isolated cases occur. In this case, for example, there was very little constitutional upset at the beginning; there was no headache, only very slight muscular pains lasting for two days, and no effusions into the joints. The most interesting difference was, however, the scarcity of the skin lesions and the absence of any fresh outcrop of lesions in the second bout of pyrexia, during which time repeated scrutiny of the patient revealed no further lesions of any kind. There were never any lesions on the shins, and there was no enlargement of the spleen.

I am indebted to Dr. E. C. Benn for permission to publish this case and to Dr. G. M. Bonser for the pathological diagnosis.

A. A. DRIVER, M.D., D.P.H.,
Resident Medical Officer at an Emergency Hospital.

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