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CONGENITAL TEMPORARY DIABETES MELLITUS

It is well known that infants rarely develop diabetes, but when they do their diabetes is usually of the ordinary insulin-sensitive type with a proneness to ketosis. Insulin by injection is required for the patient's lifetime, and in fact the disease appears basically not to differ from the diabetes that starts in later childhood or early adult life. It is less well known that there is a second type of infant diabetes, apparently occurring from or even before birth, which is also insulin-sensitive, but which is not accompanied by ketosis and which goes on to eventual total spontaneous recovery. Although this would normally be considered a very unusual type of diabetes indeed, Professor Hutchison and his co-workers are able to describe 4 cases seen by themselves. Apparently the first recognized case occurred in the son of a doctor who described it in 1852. The child died after a short period with polyuria, polydipsia, glycosuria and emaciation.

The 4 cases described by Hutchison and his colleagues were diagnosed between the ages of seven and seventeen days. Each of them showed marked hyperglycaemia and glycosuria, with rapid loss of weight and dehydration in spite of adequate intake of food and fluid. There was no diarrhoea, vomiting, or ketosis. All four patients responded readily to insulin; in one case tolbutamide was tried and failed to have any effect. All patients recovered completely; one within 28 days, another not for 18 months.

In no case was there a family history of known diabetes. All 4 infants had birth weights below 5½ lb., although 3 were born at or after full term. The authors remark that postmaturity with low birth weight has been previously observed in other reported cases. The authors were further impressed by the appearance of their diabetic infants at the stage of dehydration. They noted a peculiar pallor together with a lined, aged appearance which was associated with a remarkably alert facies. They consider that this contrasts with the usual appearance of the semi-comatose and glazed-eyed state in severely dehydrated infants. They remark on the difficulty of recognizing polyuria in a newborn infant, and hence the importance of considering a possible diagnosis of neonatal diabetes in such small newborn infants as would fit the above description.

The highest blood-sugar recordings in their 4 cases ranged from 700 to 1,300 mg. per 100 ml. Twentyfour-hour specimens of urine were analysed for 17 ketosteroids and 17 hydroxycorticosteroids, and in one case there seemed to be some increase in the output of 17 hydroxycorticosteroids above normal. During insulin therapy in 2 of the patients a round, full-moon-faced appearance developed, which was highly reminiscent of Cushing's syndrome or of the infants of diabetic mothers. The published photograph of case 2 shows this appearance extremely well.

Although the authors' 4 patients all recovered from the point of view of their diabetes, so that even glucose-tolerance tests became normal, 3 of them developed cerebral abnormalities. Two became mentally retarded, while a third was considerably below the level of intelligence of her parents and in addition suffered from a spastic monoplegia and had epileptic seizures associated with focal abnormality in the electro-encephalogram. In two of these children frequent hypoglycaemic episodes occurred during the early stage of their insulin therapy, and these might possibly account for the brain damage. In the third case, however, hypoglycaemic attacks apparently did not occur; it is therefore not clear why brain damage should have developed. Cerebral sequelae had not been recorded in earlier cases. This might have been because the period of follow-up in most instances had been short. The follow-up period in the authors' 4 cases ranged from 6 months to 9 years.

There seems to be little or no information as to the aetiology or pathogenesis of this strange and interesting diabetic state. The authors here quoted have no theories to put forward. The condition would certainly appear to be very different from any other known variety of diabetes, and it would be extremely interesting to know the full life histories of the people concerned — especially whether any vascular disorder of diabetic type develops. The authors end by remarking that this disorder may not be as excessively rare as would appear from the literature, because of the difficulty of diagnosis, and they suggest that increased knowledge of its existence may lead to more frequent recognition.

Hutchison, J. H., Keay, A. J. and Kerr, M. M. (1962): Brit. Med. J., 2, 436.

DIE HANTERING VAN PASIËNTE MET AKUTE VERGIFTIGING

Alle gevalle van akute vergiftiging is mediese noodgevalle. Die behoud van sy lewe of die intree van sy dood hang in die geval van elke ongelukkige slagoffer in 'n groot mate af van hoe spoedig behandeling ingestel word. In ligte vergiftiging sou dit moontlik kon wees om met minder spoed op te tree sonder om die pasiënt te benadeel — en tog kan selfs ligte vergiftiging soms verrassend vinnig vererger. In die meeste gevalle moet die diagnose gemaak word sonder die hulp van tegniese middels of spesiale prosedures — wat in elk geval tydrowend is. Daar moet ook vasgestel word dat die betrokke geval wel 'n geval van vergiftiging is en nie iets anders nie. 'n Foutiewe diagnose in hierdie opsig kan noodlottige gevolge hê.

Om mee te begin is 'n goeie en volledige geskiedenis van die grootste belang. Die pasiënt mag of mag nie gewillig of in staat wees om met die dokter te praat nie,

of hy mag nie die waarheid vertel nie. Daar kon byvoorbeeld 'n poging tot selfmoord gewees het. Dit is dus noodsaaklik dat inligting ook ingewin word van naasbestaandes of enige ander persone wat iets mag weet van die omstandighede waaronder die vergiftiging voorgekom het. Kennis van die pasiënt en sy omstandighede kan dus van groot betekenis wees, maar dit is nie altyd beskikbaar nie. Dit is nie waarskynlik dat die dokter wat die pasiënt eerste sien in die hospitaal hom persoonlik sal ken nie. Die oorsaak van die vergiftiging mag bekend wees, maar die getuienis daaroor mag misleidend wees. Die hele geskiedenis van die geval moet opgeteken word, aangesien dit vir almal van belang is — veral vir die mediese beampte wat met die behandeling moet voortgaan en ook omdat dit later mag nodig wees vir medies-geregtelike doeleindes.

Fisiese ondersoek moet baie deeglik wees. Die benarde en verontrustende toestand waarin die pasiënt mag verkeer, moet die dokter nie daarvan weerhou om noukeurig en presies te dink terwyl eerstehulp-behandeling toegepas word nie. Bewyse van lokale inmenging of van prikkelende middels of bytstowwe spreek gewoonlik vanself. 'n Uitslag op die vel mag subakute of akute vergiftiging aandui. 'n Onvaste gang, rusteloosheid, stupor, of koma mag tekens wees van gestelsvergiftiging, maar daar moet baie noukeurig ondersoek ingestel word om die moontlikheid van hoofbesering of verhoogde binneskedelse druk uit te skakel. Al die vroeë tekens van akute vergiftiging kan nie volledig hier bespreek word nie, maar baie van hulle sal in die loop van die gewone kliniese ondersoek aan die lig kom. Die volgende voorbeelde kan onder meer as illustrasies dien:

'n Pienk verkleuring kan in gevalle van sianied- of koolstofmonoksied-vergiftiging voorkom, en 'n baie donker sianose in methemoglobinemie as gevolg van analien-vergiftiging. Edeem van die longe kan veroorsaak word deur inaseming van prikkelende stowwe en deur 'n hele aantal middels. Edeem kan natuurlik egter ook 'n komplikasie wees van baie siektes, veral kardiovaskulêre siektes en toestande van bewusteloosheid. Die kardiovaskulêre stelsel word nie dikwels alleen aangetas in akute vergiftiging nie, maar die hart moet in elke geval goed ondersoek word vir aanduidings van hartsiekte. 'n E.K.G.-ondersoek moet in die geval van elke bewustelose pasiënt gemaak word en in diegene met abnormale polse en bloeddruk wat deur die gif veroorsaak word. Halfuurlikse rekords van die polssnelheid, bloeddruk, en die respirasie is noodsaaklik as daar akute perifere versaking is soos wat mag voorkom by erge vergiftiging. Die reuk van die asem mag diagnostiese waarde hê, soos byvoorbeeld in vergiftiging met gas of in ketose. Die ondersoek van braaksel en stoelgange met betrekking tot hul voorkoms en chemiese samestelling mag belangrik wees. Die urine moet altyd ondersoek word met die oog op die uitskakeling van asperien- of barbituurvergiftiging. Dit is baie belangrik om die onderbuik te betas sodat retensie van urine ontdek kan word. Sommige gifstowwe irriteer die niere en veroorsaak teerheid in die lende, pyn in die blaas, en hematurie. Gifstowwe wat die beenmurg aantas kan purpura, anemie en leukopenie veroorsaak. Twee bloedmonsters moet dus geneem word — 15 ml. om te stol sodat chemiese toetse op die serum uitgevoer kan word, en 10 ml. met 'n stollingsteenmiddel vir seltellings en ander doeleindes.

Middels en gifstowwe tas alle dele van die senuweestelsel aan sodat neurologiese simptome dikwels by vergiftiging aangetref word. Die meeste gestelsgifstowwe het 'n uitwerking op die bewussyn deur direkte inwerking op die sentrale senuweestelsel of deur sekondêre veranderinge van die respiratoriese of sirkulasiefunksies. Sentrale versteurings kan ook op ander maniere tot uiting kom, bv. duiseligheid, naarheid, tinnitus, disorientasie, opgewondenheid, slaperigheid; en veranderde reflekse en pupilreaksies mag aanduidings wees van vergiftiging.

Daar is 'n groot aantal ander tekens en simptome wat van belang mag wees by die differensiële diagnose van vergiftiging. As vergiftiging eers oorweeg word, behoort die regte diagnose in verreweg die meerderheid van gevalle gemaak te word. Aangesien daar daagliks meer middels op die mark kom, word hierdie probleem progressief moeiliker.

Graham, J. D. P. (1962): *The Diagnosis and Treatment of Acute Poisoning*. London: Oxford University Press.