SOUTH AFRICAN MEDICAL JOURNAL SUID-AFRIKAANSE TYDSKRIF VIR GENEESKUNDE

Cape Town, 3 May 1969 Volume 43 No. 18 Deel 43 Kaapstad, 3 Mei 1969

VAN DIE REDAKSIE : EDITORIAL

SALARIS DIFFERENSIASIE

Die Federale Raad van die Mediese Vereniging van Suid-Afrika het gedurende 'n vergadering wat in Julie 1968 in Pietermaritzburg gehou is die volgende besluit geneem:

> 'That Federal Council considers that salary differentiation among full-time medical personnel should only be on the basis of grading, service and merit. It urges the authorities to give sympathetic consideration to removing the present source of friction existing regarding the differential salary structure existing between White and non-White doctors.'

Sy Edele die Minister van Gesondheid, dr. Carel de Wet, het so pas in the Parlement aangekondig dat gelyke salarisse vir Blanke en nie-Blanke geneeshere nooit oorweeg kan word nie aangesien dit in stryd is met die verklaarde beleid van die Regering.

Die Mediese Vereniging het reeds dikwels in die verlede gebots met verskillende owerhede, en sulke botsings sal sekerlik weer in die toekoms voorkom solank as wat die Vereniging sy self-opgelegde taak om te waak oor die belange van sowel die mediese professie as die publiek met opregtheid voortsit. Maar tot dusver was die geskille wat besleg moes word meesal van sodanige aard dat dit nie prinsipiële sake aangeraak het nie. Die lang stryd wat die Mediese Vereniging moes voer in verband met die Wet op Mediese Skemas het nie gegaan oor die wenslikheid al dan nie om mediese skemas te beheer nie. Dit het gehandel oor die detail wat in die Wet vervat is, want die Vereniging was dit nog altyd met die owerhede eens dat sulke wetgewing wel nodig is.

Deur onderhandeling kon ons nog altyd in die verlede meeste van ons probleme oplos. Dit is wel waar dat ons soms ons stemme moes dik maak om die gewenste uitwerking te kry, maar dit het steeds nie gehandel oor werklik fundamentele beleidsverskille nie.

Nou het ons vir die eerste keer te doen met 'n heel ander probleem. Die besluit van die Vereniging se Federale Raad is reëlreg in stryd met die Regering se onwrikbare standpunt. Dit gaan nie hierdie keer om onderhandelings nie; nie om arbitrasiekostes en onregverdige klousules nie. Dit gaan om 'n kop-aan-kop botsing en iemand sal moet wyk. Dit is nou nodig dat daar weer dringende besprekings met oop gemoedere plaasvind. Die Mediese Vereniging is nie 'n politieke liggaam nie en dit mag dit ook nooit word nie. Die lede is te uiteenlopend van denke dat dit ooit inderdaad moontlik sou wees, en in ieder geval sou enige sweem van politieke kleur 'n totale ineenstorting van die Vereniging se struktuur tot gevolg hê.

Dus wat staan ons nou te doen? Reeds sedert die vergadering in Maritzburg is die lede van die Uitvoerende Komitee druk besig om pogings aan te wend om die besluit van die Federale Raad ten uitvoer te bring. Daar is voortdurend onderhoude gehou met alle belanghebbendes en met die regerings- en provinsiale verteenwoordigers, en die onderhandelings het reeds vrugte afgewerp; weliswaar nie grootse resultate nie, maar darem iets.

Nou het die owerheid viervoet vasgesteek. Waarheen nou? Daar is net drie weë vir die Vereniging oop. Ons kan as eerste moontlikheid die stryd gewonne gee en aan ons nie-Blanke kollegas sê, 'Mille regrets'. Ons het ons bes gedoen, maar ons het voor 'n muur te staan gekom en die besluit van die Federale Raad is nie uitvoerbaar nie. Of as tweede alternatief kan ons aanhou met onderhandelings om dan altans die beste moontlike differensiasie te verkry en die gapings nie al te groot laat bly nie. Dit sal nie 'n uitvoering van die besluit van die Raad daarstel nie, maar dit sal ten minste 'n kompromis uitmaak. En derdens kan ons die stoute skoene aantrek om na die Regering te gaan en te vra vir 'n heroorweging van die verklaarde beleid.

Laasgenoemde sal kwaai sake wees; daaroor moet ons geen illusies hê nie. Hoe versigtig en korrek dit ookal gedoen word en met alle voorsorg teen 'n politieke atmosfeer, sal daar tog elemente wees wat dié houding sal uitbuit en sal beskryf as 'n konfrontasie aangaande die land se ontwikkelingsbeleid. Dit sal egter geensins die geval wees nie. 'n Versoek deur die Vereniging vir ewewaardige besoldiging van sy lede sal slegs te doen hê met betaling vir gelewerde dienste en het in der waarheid niks te doen met die siening in verband met afsonderlike ontwikkeling nie.

Wat ons ookal doen en welke van die drie rigtings ons ookal inslaan, sal die besluit van die Federale Raad moet uitgaan. Die volgende vergadering van die Raad is net om die draai en dit is te duur om 'n spesiale vergadering te belê. Ons kan net so wel die twee maande wag. Ons wil intussen 'n beroep op ons nie-Blanke kollegas doen om kalm en rustig en met dieselfde geduld wat hulle tot dusver aan die dag gelê het te wag vir die uitspraak van die Federale Raad. Dan doen ons die ding op die korrekte en verstandige manier sonder om die waardigheid van ons professie te skaad en sonder om te waag dat die polemiek op die ou end in die pasiënt se skoot beland. Want daarteen moet ons waak. Ons onderhandelings geld die owerhede en die professie, en dit is nie die skuld van die pasiënt nie. Onder geen omstandighede mag ons ooit toelaat dat die dienste wat ons lewer op enige wyse aangetas word nie. Dan het ons dadelik verloor. Daar mag geen vinger na ons gewys kan word nie.

HYPEROSMOLAR NON-KETOTIC DIABETIC COMA

The syndrome of depression of consciousness in a diabetic with hyperosmolarity of the serum without ketosis, yet usually responding promptly to insulin and fluid replacement, continues to excite interest for the following reasons: Its recognition may be difficult, so that it is not diagnosed until too late; the pathogenesis of the clinical state remains a mystery; if hyperosmolarity is the injurious factor, just how does it work? Likewise, the absence of ketosis is unexplained and controversy

exists concerning the best fluid to use for replacement. The condition is not uncommon and is frequently fatal.

Although to Sament and Schwartz[1] goes the credit of furthering interest in this syndrome by publishing an account of their case in 1957, its occasional occurrence had long been recognized. At least in the 1880s it was known that not all patients in diabetic coma had acidotic breathing and ketonuria, and several such exceptions were reported.[2]

The recognition of the syndrome in the uncomplicated case is not difficult. There is usually a history of acute diabetic symptoms such as thirst, polyuria and weakness; consciousness is clouded, urine loaded with glucose and the blood sugar high. Acetest is negative in the plasma or rapidly becomes so. The absence of ketones in the urine is not sufficient to make the diagnosis; one must also demonstrate a lack of ketones in the plasma or a lack of acidosis, or at most an insignificant degree of these. Occasional difficulty arises in patients presenting with neurological features. Diagnosis may be delayed in patients who develop the condition while being treated for other serious diseases, largely because of the absence of the warning of ketonuria which gives a clue to danger in ordinary diabetic crises. It must be suspected in a non-diabetic who suddenly develops 4+ glycosuria or in a known diabetic whose level of consciousness declines and who exhibits increased polyuria.

The presence of acidotic breathing and biochemical metabolic acidosis does not exclude the hyperosmolar syndrome provided ketosis is not present or is minimal, as can be readily judged by performing the nitroprusside test (or Acetest) on the plasma or serum. In patients with neurological features a high CSF sugar may suggest the diagnosis.

The patient's age is usually over 40 years, and diabetes has previously been mild or non-existent; frequently patients are obese. However, a few cases in young people have been described; in an 18-month-old child in 1954;[3] in an 11-year-old, previously non-diabetic girl taking prednisone;[4] in a 22-year-old long-standing, ketosis-prone, juvenile-type diabetic after pituitary stalk section;[5] and in a 16-year-old wasted Bantu youth.[6] Chronic pancreatitis and carcinoma of the pancreas have been present in some instances,[7,8] while autopsy has revealed acute pancreatitis in others.[9-12] Severe burns in an apparently non-diabetic individual may lead to the hyperosmolar syndrome during convalescence, possibly related to excessive carbohydrate ingestion.[13,14] Both peritoneal and haemodialysis have also been implicated.[15,16] The diuretic Hygroton may have been a precipitating factor in 2 cases,[17] and thiazide diuretics in others.[2] Yet other individuals have developed the hyperosmolar syndrome while on glucocorticoids in pharmacological doses.[4,12]

Seizures of various sorts are frequent, and irritability and violence may occur even in stuporous patients. This facet of the hyperosmolar syndrome was clearly emphasized by Maccario and co-workers,[18] who detailed the cerebral discharge phenomena in 7 patients. This complication does not occur in ketoacidosis, in which the ketone bodies suppress neuronal discharge, producing a flaccid stillness with depressed muscle tone and reflexes.

Blood-sugar levels in this syndrome are not always excessively high, but the serum osmolality is probably invariably above 350 mOsm./litre. The blood glucose is usually highly sensitive to insulin, but this cannot be relied on[8]—one patient required 13,720 units of insulin.[6] It would seem reasonable to start treatment with the standard dose of soluble insulin used in diabetic keto-acidosis, but subsequent blood-sugar levels must be carefully observed and further insulin doses accurately adjusted to avoid hypoglycaemia.

The most logical fluid to use for rehydration would seem to be ½ N saline.[2,8,12,19-22] With higher concentrations there is a clear risk of hypernatraemia and hyperchloraemia, while with lower tonicity the risk of haemolysis might arise. Water by stomach tube is a possible alternative.[23] In ketoacidosis the stomach is typically atonic and acutely dilated and should be kept empty, but this may not apply to the non-ketotic state. Bicarbonate or lactate is not indicated, except for the former in the case of lactic acidosis. In the presence of gross hyperglycaemia the use of sugar-containing fluids seems to be irrational, since this must delay reduction of the hyperosmolarity, which we believe to be the damaging influence. The danger of giving sugar was demonstrated well in Kolodny's patient[22] who was able to drink initially, was given orange juice containing sugar, and within 15 minutes suffered repeated generalized convulsions followed by total coma.

Fluid must be given rapidly, since dehydration is often extreme—patients may need up to 16 litres for full rehydration, which can be monitored by serum osmolality readings. Rehydration alone did not restore consciousness in one patient with viscous blood, who received 6 litres of ½ N saline over 3 hours with no insulin and without alteration in his comatose state, which rapidly improved after 200 units were injected in addition to the continuation of the infusion.[3] On the other hand, intravenous fluid alone was satisfactory in another reported case.

It would appear that potassium should be given even more urgently than in keto-acidosis. It is now our plan to give neutral potassium phosphate (25 mEq.) with the first litre of fluid, as soon as we have the report that the initial serum level is not raised. If at all possible the serum potassium level should be measured hourly. If this is not possible, at least an hourly ECG should be obtained.

It is clear that the hyperosmolar syndrome is not rare, though in certain circumstances it may be easy to miss the diagnosis. Neurologists, neurosurgeons, and other surgeons especially in burns units should be aware of the condition, as well as internists and general practitioners. We really do not know the reason for the rapid rise in blood sugar that occurs, except for the rare cases associated with burns and with dialysis. The existence of the latter certainly indicates that hyperglycaemia *per se* can produce the necessary CNS damage. The very similar condition caused by acute hypernatraemia in non-diabetics suggests that the induced hyperosmolarity is the damaging factor, rather than excess of any individual solute. Just how this works is uncertain. Not all bloods appear viscous or even show haemoconcentration, while in keto-acidotic coma the degree of hyperosmolarity is frequently as great as in many fatal cases of the non-ketotic syndrome. It thus seems very peculiar that the mortality rate in severe keto-acidosis should be so low, when there is ketosis, acidosis and hyperosmolarity, whereas with hyperosmolarity alone the death rate is far higher!

The most widely accepted reasons for the lack of ketosis seem to be the presence of some insulin plus the great hyperglycaemia. But keto-acidotic coma occurs in plenty of normally mild, maturity-onset diabetics under stress, and in children as an initial event, who later remit spontaneously and with just as high blood-sugar levels. We delude ourselves if we think it is that simple.

REFERENCES

1. Sament, S. and Schwartz, M.B. (1957): S. Afr. Med. J., **31**, 893.
2. Sølusteen, P., Olsen, V. V. and Hansen, E. L. (1968): Acta med. scand., **184**, 83.
3. Ehrlich, R. M. and Bain, H. W. (1967): New Engl. J. Med., **276**, 683.
4. Kumar, R. G. (1968): Lancet, **1**, 48.
5. Kolodny, H. D. and Sherman, L. (1968): J. Amer. Med. Assoc., **203**, 461.
6. Seftel, H. C., Goldin, A. R. and Rubenstein, A. (1967): Lancet, **2**, 1043.
7. Bergoz, R. and Hausser, E. (1964): *Ibid.*, **1**, 116.
8. Jackson, W. P. U. and Forman, R. (1966): Diabetes, **15**, 714.
9. Ward, F. G. (1963): Lancet, **1**, 450.
10. Davidson, A. I. G. (1964): Brit. Med. J., **1**, 356.
11. Halmos, P. B. (1966): *Ibid.*, **2**, 685.
12. Schwartz, T. B. and Apfelbaum, R. I. *in* Schwartz, T. B., ed. (1966): *Year Book of Endocrinology, 1965-66*, p. 165. Chicago: Year Book Medical Publishers.
13. Rosenberg, S. A., Brief, D. K., Kinney, J. M., Herrera, M. G., Wilson, R. E. and Moore, F. D. (1965): New Engl. J. Med., **272**, 931.
14. Bailey, B. N. (1960): Brit. Med. J., **2**, 1783.
15. Potter, D. J. (1966): Ann. Intern. Med., **64**, 399.
16. Boyer, J., Gill, G. N. and Epstein, F. H. (1967): *Ibid.*, **67**, 568.
17. Morét, Von B. (1965): Dtsch. med. Wschr., **90**, 1136.
18. Maccario, M., Messis, C. P. and Vastola, E. F. (1965): Neurology (Minneap.), **15**, 195.
19. Bradley, R. F. (1965): Med. Clin. N. Amer., **49**, 961.
20. Matz, R. and Drapkin, A. (Correspondence) (1966): Lancet, **1**, 1101.
21. Rosen, H. and Glick, S. M. (Correspondence) (1966): *Ibid.*, **1**, 1101.
22. Tyler, E. H. (1968): Amer. J. Med., **45**, 485.
23. Gigon, J. P. (1963): Path. et Biol., **12**, 1191.
24. Hayes, T. M. and Woods, C. J. (1968): Lancet, **1**, 209.