Diabetic Acidosis and Acute Renal Insufficiency

Acute Tubular Necrosis Treated by Dialysis

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CASE HISTORY

A seventeen-year-old girl with no prior history of diabetes was admitted to a local hospital on March 8, 1963, in coma. She did not appear to be dehydrated. Blood pressure was 112/68 mm. Hg. and blood sugar was 724 mg. per 100 ml. Urinalysis revealed a 4 plus glycosuria and acetonuria. The CO_2 combining power was 2.5 mEq./L. During the next 20 hrs. she received intravenously 5,000 ml. of 5 per cent dextrose mostly in saline, and 660 U. Regular Insulin but no sodium lactate or sodium bicarbonate. Blood sugar level decreased to 144 mg. per 100 ml., whereas the CO_2 increased only to 8.5 mEq./L. Although somewhat improved, she remained mentally confused and became oliguric. On the third day after admission, blood urea nitrogen content was 58.9 mg. per 100 ml. Because of the persistence of the confusion, and a stiff neck, a lumbar puncture was performed; the opening pressure was 600 mm. H_2O. The spinal fluid was slightly blood tinged, and a diagnosis of a subarachnoid hemorrhage was considered.

She was transferred to the Cleveland Clinic Hospital on March 11, 1963, for further therapy. She was confused and overhydrated. Blood pressure was 150/60 mm. Hg. Initial laboratory studies revealed: blood hemoglobin, 10.6 gm. per 100 ml.; cell volume, 32; blood sugar, 245 mg. per 100 ml.; blood urea, 204 mg. per 100 ml.; serum sodium, 115; serum potassium, 2.7, and serum CO_2 content, 8.2 mEq./L.; and venous blood *p*H, 7.25. The urine was clear; analysis revealed 1-plus glycosuria, 4-plus proteinuria, and a trace of acetone. Salicylate could not be detected in the blood.

In view of the possible presence of subarachnoid hemorrhage, the use of peritoneal dialysis was elected rather than hemodialysis, since even with regional heparinization, a rise in clotting time may occur during hemodialysis. A peritoneal dialysis was performed for approximately 12 hrs. The dextrose concentration was 2.5 per cent. There was a considerable clinical improvement after correction of overhydration, removing 5,750 ml. of fluid.

However, during the next seven days her condition deteriorated, and the blood urea increased to 246 mg. per 100 ml. A ten-hour peritoneal dialysis was performed, and the blood urea was 210 mg. per 100 ml., with CO_2 content of 13.8 mEq./L. at the termination of dialysis. A renal biopsy showed acute tubular necrosis. It was decided not to continue the peritoneal dialysis because of the hazard of peritonitis, as there was abdominal pain with the second dialysis. She was then dialyzed with the Kolff disposable twin-coil kidney on the tenth and twelfth days after admission to the hospital. The patient improved, the daily urine volume had increased to 1,000 ml. twenty days after the onset of coma (figure 1).

Recovery was delayed by complication of staphylococcal enteritis. On the fifty-second day after admission, the blood urea level was 32 mg. per 100 ml., with creatinine clearance of 99 ml. per minute. Diabetes was controlled with 16 U. Ultralente Insulin and 8 U. Regular Insulin in addition to a special diet.

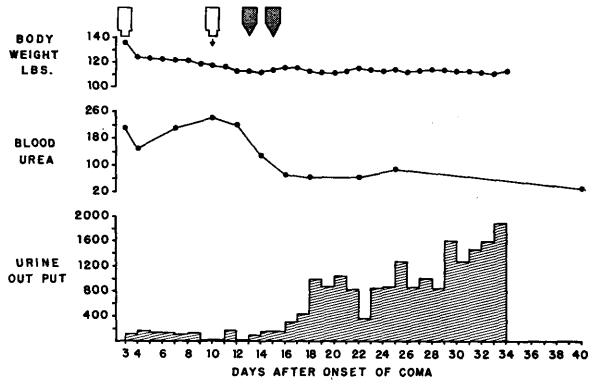

FIG. 1. The clinical record of a seventeen-year-old girl in whom acute tubular necrosis developed as a complication of severe diabetic acidosis. Note that the blood urea content expressed in milligrams per 100 ml. is lower after the hemodialysis (shaded form) than after the peritoneal dialysis (white inverted "bottles") although the latter was effective in correcting the initial overhydration. The diuretic phase is late and the urine volume (expressed in milliliters per 24 hrs.) not large, the highest value being 1,880 ml. thirty-four days after the onset of the diabetic coma.

COMMENT

Since this patient was not severely dehydrated and hardly in shock, the prolonged period of acidosis was probably the major factor in the development of the acute tubular necrosis. Aoyama and Kolff[1] reported a similar case of diabetic acidosis and concomitant acute renal failure. In that case renal failure was believed to be due to shock and severe acidosis aggravated by the

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use of large amounts of normal saline alone without sodium lactate or sodium bicarbonate. Bernstein, Foley, and Hoffman[2] postulated that the persistent renal dysfunction, after correction for the dehydration and electrolyte imbalance in patients with diabetic acidosis, is a mild form of tubular necrosis due to renal ischemia. The rare occurrence of acute renal failure may be due to the rapid response to treatment of the diabetic acidosis.[3] The prolonged period of acidosis in our two patients could have been shortened if sodium lactate or sodium bicarbonate had been added at the onset of electrolyte therapy. Peritoneal dialysis proved to be effective in correcting the overhydration, and the hyperglycemia resulting from the hypertonic dextrose used for osmotic fluid removal was easily controlled with insulin. The uremic symptoms were relieved more effectively by the artificial kidney.

ACKNOWLEDGMENT

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Special Article

Convocation in Toronto

An Address

Randall G. Sprague, M.D., Rochester, Minnesota

Mr. Chancellor, Dean Bladen, Members of the Faculty, Ladies and Gentlemen:

The great honor which you have just given Professor Hoet, Doctor Lawrence and me is deeply appreciated. I feel that this distinguished University has complimented me far beyond what I had any right to expect, and I have asked myself in which of my various guises I should address this Convocation. For me, here in Toronto, the answer comes easily. I prefer to appear before you simply as one who has had diabetes since boyhood, who because of insulin has lived to attain the status of a "senior diabetic," and who wishes to express his gratitude. Not only would I like to express my own appreciation but also that which I know is felt by Doctor Lawrence, as well as by an untold number of diabetics all over the world who live because of insulin.

It was once said that the age of one with diabetes is properly reckoned as the sum of his chronologic years and the years of his diabetic life. Using this method of calculation, and disregarding for the moment any question of its validity for the three individuals who are being honored this evening, I find that I am now exactly a century old, Doctor Lawrence is my senior by some thirteen or fourteen years, and Professor Hoet, without benefit of the diabetes factor, is a relative youngster. It is indeed beneficence redoubled that the University of Toronto, the birthplace of insulin, the very institution which enabled two of us to live and work, should now honor us for our work in diabetes.

THE GREAT DISCOVERY

In this restless, changing world great universities do many things through education and research to advance the well-being of man. Many of the contributions are subtle in character. Others are dramatic in their immediate humanitarian effect. The discovery of insulin by Banting and Best[1] in 1921 was one of these. Since the day in January, 1922, in Toronto General Hospital when Dr. Walter R. Campbell and Dr. Almon A. Fletcher gave Leonard Thompson the first injection

From the Section of Medicine, Mayo Clinic and Mayo Foundation, Rochester, Minnesota.

Presented at a Special Convocation at the University of Toronto on July 20, 1964, to confer honorary degrees upon Prof. J. P. Hoet, Dr. R. D. Lawrence and Dr. Randall Sprague.