Hyperglycemia and Hyperosmolarity in a Brittle Diabetic with Thyrotoxicosis

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SUMMARY

A thirty-six-year-old female brittle diabetic presented in a confused state complaining of intense thirst. She had a twelve-year history of known diabetes mellitus but for the previous five years had been well controlled. Four years before admission she was found to be euthyroid with a PBI of 6.8 mcg. per 100 ml.

On examination she was dehydrated and without Kussmaul respirations. Blood sugar on admission was 1,590 mg./100 ml. and only a trace of acetone was detected in urine. Calculated serum osmolality was 365 milliosmols/liter. The patient responded well to 800 U. of Regular Insulin and eight liters of intravenous hypotonic solutions. However the pulse rate remained at 140/min. with no evidence of heart failure or infection; EKG showed sinus tachycardia. Evaluation of the patient's thyroid function revealed hyperthyroidism. I-131 uptake after twenty-four hours was 74 per cent. The glucocorticosteroids were found to be within normal limits.

Four months after receiving methimazole 40 mg./day the hyperthyroidism and diabetes mellitus were well controlled. DIABETES *19*:70-71, January, 1970.

The clinical syndrome of hyperglycemic hyperosmolar nonketotic coma is now a well recognized condition and has been described in a number of clinical situations. The "classical" form occurs in mild maturity-onset diabetics,[1,2,3] but has recently been described in juvenile diabetics,[4,5] associated with severe burns,[6] hemodialysis,[7] peritoneal dialysis,[8] glucocorticoid therapy,[9] following diphenylhydantoin administration[10] and acute pancreatitis.[11] Recently this syndrome was observed in a patient with brittle diabetes mellitus and thyrotoxicosis.

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CASE REPORT

Mrs. J.N., a thirty-six-year-old white woman, was admitted to the emergency room while in a confused state and complaining of intense thirst.

The patient had known diabetes of twelve years' duration controlled initially on diet and 15 U. of Lente insulin per day. Within three years her insulin requirement had risen to 35 U. per day, and she was given in addition chlorpropamide 250 mg. daily. Although the earlier years of her diabetes were marked by ketoacidosis and continual difficulty in management, for the previous five years she had been well controlled on 35-40 U. of Lente insulin daily without ketoacidosis.

In 1957 and 1963 she was evaluated for infertility and was found to be euthyroid. In 1963 a serum PBI level was 6.8 mcg. per 100 ml.

The present illness began two days prior to admission when the patient experienced nausea, vomiting and increasingly severe polyuria and polydypsia. She had not eaten for twenty-four hours, and a relative noticed the onset of confusion about four hours prior to admission. There was no history of urinary or respiratory tract infection, recent weight loss, nervousness, or irritability.

On examination the blood pressure was 110/70, pulse 160, regular and bounding in character, temperature 100° F. She was clinically severely dehydrated. The respiratory rate was 36/min. and it was consistently noted that the character of the respiration was not Kussmaul. The patient was confused and disoriented as to time and person. The skin was warm and flushed. There was no thyromegaly, the eyes were normal and the cardiovascular system, aside from the tachycardia, was normal with no evidence of heart failure. There was no tremor and the deep tendon reflexes were normal. The remainder of the examination was unremarkable.

Urinalysis revealed four plus glycosuria and only a trace of acetone. There was no proteinuria and microscopic examination was normal. The blood sugar on admission was 1,590 mg./100 ml. and no acetone was detected in the serum. (Ketones in serum and urine were tested for, using the Ames

Acetest tablet and by the qualitative Rothera Test.[12]) Blood urea was 75 mg./100 ml., serum bicarbonate 11 mEq./L. and sodium 134 mEq./L. Calculated serum osmolality was 365 milliosmols/liter.[13] The hematocrit was 47 per cent and white blood count 24,400 with 90 per cent polymorphonuclear cells.

The patient received 100 U. of Regular Insulin intravenously on admission, and two hours later no further acetonuria was detected, although the blood sugar was 1,464 mg./100 ml. Within twelve hours, after receiving a total of 800 U. of Regular Insulin and eight liters of intravenous hypotonic solutions the patient was fully conscious, well-hydrated with a blood sugar of 258 mg./100 ml., serum bicarbonate 17 mEq./L., urea 38 mg./100 ml., sodium 124 mEq./L., and potassium 4.5 mEq./L. The pulse rate remained at 140/min., however, without evidence of heart failure; the ECG showed sinus tachycardia.

The course over the next forty-eight hours was characterized by fluctuations in the patient's diabetic control. Blood sugar levels varied between 52 and 440 mg./100 ml. with one episode of ketoacidosis with serum acetone present in 1:8 dilution. This rapidly responded to appropriate insulin therapy.

Throughout this period the sinus tachycardia persisted. Chest X ray and repeated urine, sputum and cervical cultures were negative. Sleeping pulse was 92-100/min. and serum cholesterol 97 mg./100 ml. The tanned red cell uptake of tri-iodo-thyronine was 41.8 per cent (normal 26-35 per cent). The I-131 uptake was 47 per cent after three hours and 79 per cent after twenty-four hours. The urinary excretion of 17-ketosteroids was 5.7 mg./24 hrs. and the 11-hyroxycorticosteroids 3.4 mg./24 hrs. A diagnosis of thyrotoxicosis was made and the patient treated with methimazole, 10 mg. every six hours.

During the next fourteen days there was a gradual fall in pulse rate, stabilization of the blood sugar and reduction of insulin requirements. She was discharged weighing ninety-six pounds and her diabetes was controlled with 36 U. NPH insulin and chlorpropamide 125 mg. twice daily.

She was then closely followed as an outpatient. Four months later, while she was receiving the same dosage of methimazole, the fasting blood sugar levels remained between 90 and 150 mg./100 ml. Her weight rose to 110 lbs., the pulse rate was normal, and there were no further clinical features of hyperthyroidism. The PBI was 3.7 mcg. per 100 ml. and a repeated tanned red cell uptake of tri-iodo-thyronine was 28 per cent (normal 26-35 per cent).

CONCLUSION

A case report of hyperglycemia and hyperosmolarity in a young brittle diabetic is presented. Diabetic ketoacidosis has occurred in the course of thyrotoxicosis, but this is believed to be the first report of the hyperosmolar state associated with thyrotoxicosis.

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