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Severe Hypoglycemia in a Patient with Chronic Renal Failure due to Amyloidosis

Dear Sir,

The cause of hypoglycemia in a patient with chronic renal failure (CRF) is a difficult clinical problem since it may be due to various causes, the first thought of is uremia itself. Here we describe a case with severe hypoglycemia in a patient with CRF.

A 26-year old male patient with end-stage renal disease was admitted to our medical intensive care unit (MICU) because of upper gastrointestinal (GI) bleeding. He had systemic amyloidosis, diagnosed on rectal biopsy in 1984, secondary to seronegative rheumatoid arthritis since 1982. He had been on a regular hemodialysis program three times weekly since 1985. On admission his blood pressure was 180/100 mm Hg, pulse 104/min, body temperature 36.2°C and physical examination was unremarkable with the exception of a systolic murmur on the mesocardium. Laboratory examination revealed: hemoglobin 8.5 g/dl; blood urea nitrogen 74 mg/dl; creatinine 6.9 mg/dl; Na concentration 136 mgEq/dl; K concentration 4.2 mEq/dl; Ca 8.5 mg/dl on calcium supplement, and P 5.4 mg/dl. GI bleeding was successfully managed with medical therapy and 2 units of packed red blood cells were transfused. During his hospital stay, his plasma glucose levels were noted to be low, with fasting venous blood glucose concentrations of 40 and 45 mg/dl on two separate occasions, though he was asymptomatic. The patient denied any alcohol and drug abuse. Blood samples were taken for basal cortisol

and adrenocorticotrophic hormone (ACTH). ACTH stimulation test was done with 250 mg synthetic ACTH (Synacthen; Ciba-Geigy, Basel, Switzerland) administered intramuscularly and plasma cortisol was measured 30 min later. Thyroid-stimulating hormone (TSH), insulin and C-peptide levels were measured after an overnight fast and fasting for 72 h with simultaneous glucose determinations. After stabilization of his GI bleeding, the patient was discharged on his own wish as he refused any other tests while the results of the tests were pending. Two days after discharge he was taken to the emergency room by a family member, in deep coma and unable to respond to even painful stimuli. Blood pressure was 130/75 mm Hg, pulse rate 56/min, respiratory rate 12/min with shallow breaths and axillary temperature 35°C. Physical examination was otherwise unremarkable. Blood glucose was 13 mg/dl, the other blood biochemistry results being no different from the previous ones. He was started on intravenous glucose infusion and admitted to the MICU. Cranial computed tomography was also done to exclude any intracranial pathology which did not reveal any pathology. He was intubated and mechanical ventilation was started. He needed about 5–8 g/h glucose infusion to be able to maintain blood glucose levels within reasonable limits. Basal cortisol was 14.88 µg/dl (n = 5–25 µg/dl) and the plasma cortisol level after stimulation with synthetic

ACTH was 16.78 µg/dl. As the expected increment should not be less than 6.8 µg/dl, the patient was diagnosed as having primary adrenal insufficiency, aggravated by an undetermined stress. ACTH, TSH, insulin and C-peptide levels were within normal limits, helping to exclude other possible causes like insulinoma and pituitary involvement. Prednisolone treatment of 7.5 mg/day was begun. His vital signs and blood glucose levels dramatically improved. He was able to respond to painful stimuli but he never resumed his previous level of consciousness and he died 10 days later in adult respiratory distress syndrome due to nosocomial pneumonia. Unfortunately autopsy was refused by the family.

It is difficult to definitely diagnose hypoglycemia in a patient with CRF since uremia itself can also be the cause. In our patient with unexplained hypoglycemia, the need for glucose replacement of <8–10 g/h suggests underproduction of glucose rather than overutilization as in cases of insulinoma, which was excluded in our case [1]. Since our patient had amyloidosis, a disease of multiorgan involvement including the endocrine glands [2–5], we thought adrenal insufficiency due to adrenal involvement with amyloid. Adrenal involvement is reported to be common at postmortem examinations, but amyloidosis is considered to be a rare cause of primary adrenocortical dysfunction [1]. In various studies it has been shown that clini-

cal adrenocortical hypofunction is present in about 42 [4] to 46% [2] of cases with amyloidosis due to various causes. Pituitary involvement in a patient was also reported [3]. In our patient, pituitary involvement was ruled out by demonstrating normal ACTH and TSH levels. This case shows that adrenal insufficiency, which is common in systemic amyloidosis, should be suspected in patients with amyloidosis, especially in patients presenting with hypoglycemic coma, and steroid replacement should be considered under acute stress.

References

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