known to be rare.[1,4] Among the four cases presented, only cases 1 and 2 should be considered clear cases of generalized insulin allergy; the problems observed in case 4 were probably related to tuberculosis. Both of our insulin-allergic patients had a history of penicillin allergy, which is reported to be frequent in such cases. They exhibited allergic reactions to purified and human insulins, but their management started with conventional insulins. Accordingly, all new cases, especially those in whom interrupted insulin treatment can be expected or a history of allergic reactions is present, should be treated with such insulins to avoid a possible booster effect.

The authors thank Dr. J. Smedegaard Christensen from the Novo Research Institute for the test solutions and IgE determinations, as well as for the most interesting discussions on this topic.

M. GRANIĆ, M.D., D.Sc.
I. PAVLIĆ RENAR, M.D.
Ž. METELKO, M.D., M.Sc.
Z. ŠKRABALO, M.D., D.Sc.

From the Vuk Vrhovac Institute for Diabetes, Endocrinology, and Metabolic Diseases, Faculty of Medicine, University of Zagreb, Krijesnice bb, YU-41000 Zagreb, Yugoslavia.

Address reprint requests to Dr. I. Pavlić Renar at the above address.

REFERENCES

[1] Galloway, J. A.: When the patient is resistant or allergic to insulin. Med. Times 1980; 108:91–101.

[2] Goldstein, H. H.: Allergy to insulin. *In* Clinical Diabetes Mellitus. Kozak, G. P., Ed. Philadelphia, W. B. Saunders Co., 1982:146–53.

[3] Falholt, K.: Determination of insulin specific IgE in serum of diabetic patients by solid phase radioimmunoassay. Diabetologia 1982; 22:245–57.

[4] Grüneklee, D.: Die Insulin Allergie. Internistische Welt 1980; 12:442–50.

Rhabdomyolysis Associated with HNKC

We would like to make some observations regarding the case report in the March-April 1985 issue of DIABETES CARE.[1] The proposal that hyperosmolality or hyperglycemia "per se" was the primary cause for rhabdomyolysis (RM) cannot be clearly established from the presented data. Known causes for RM, such as fever and tissue hypoperfusion,[2,3] were present; hypokalemia or hypophosphatemia[2,4,5] if present would be obscured by the RM itself. In order to suggest the hyperosmolality as the inducer, other known causes must be absent. To document this, blood chemistry before RM ensues is needed.

Over the last 2 yr we treated two women (aged 67 and 75) with "unexplained high creatine kinase (CK)" in the setting of hyperosmolar nonketotic coma (HNKC). The first patient presented on admission with a CK value of 653 IU/L (normal <135) and a maximal concentration of 12,400 IU/L 5 days later. For the second patient, CK levels on admission were 126 IU/L with a maximal peak of 629 IU/L on the third day. Aspartate aminotransferase (AST) was also elevated. The most common causes of elevated CK were excluded. Although we suspected RM, a firm diagnosis could not be established (neither myoglobinuria nor a urine heme test was performed).

RM may not be so rare in HNKC. We believe that establishing this diagnosis can benefit the patient by alerting the physician of the possibility of acute failure, the more dangerous complication.

ROSA CORCOY, M.D.
R. WALTER DE LA TORRE, M.D.
JOSE Mª POU, M.D., Ph.D.
ALBERTO DE LEIVA, M.D., Ph.D.

From the Service of Endocrinology and Nutrition, Hospital de Sant Pau, Autonomous University, Barcelona, Spain.

Address reprint requests to Alberto de Leiva, M.D., Ph.D., at the above address.

REFERENCES

[1] Schlepphorst, E., and Levin, M. E.: Rhabdomyolysis associated with hyperosmolar nonketotic coma. Diabetes Care 1985; 8:198–200.

[2] Gabow, P. D., Kaehny, W. D., and Kelleher, S. P.: The spectrum of rhabdomyolysis. Medicine 1982; 61:141–52.

[3] Berg, P., and Frenkel, E. P.: Myoglobinuria after spontaneous and induced fever: report of a case. Ann. Intern. Med. 1958; 48:380–88.

[4] Woolridge, T. D., Bower, J. D., and Nelson, N. C.: Rhabdomiolysis. A complication of parathyrodectomy and calcium supplementation. JAMA 1978; 239:643.

[5] Knochel, J. P., Barcenas, C., Cotton, J. R., Fuller, T. J., Haller, R., and Carter, N. W.: Hypophosphatemia and rhabdomyolysis. J. Clin. Invest. 1978; 1240–46.

Alternating Choreoathetosis Associated with Uncontrolled Diabetes Mellitus and Basal Ganglia Calcification

Choreoathetotic movements have been described in patients with several metabolic diseases, including three patients with non-insulin-dependent diabetes mellitus.[1] We report the case of an elderly patient who presented with choreoathetosis as the initial manifestation of diabetes mellitus and subsequently developed a contralateral recurrence of these symptoms during another episode of marked hyperglycemia. CT scanning performed twice, 1 yr apart, revealed increasing calcification of the basal ganglia in the region of the caudate nuclei.

CASE REPORT

A 76-yr-old Black woman presented with a 1-day history of involuntary movements of the right arm and face and a 3-

wk history of polyuria and polydipsia. Past medical history was positive for hypertension and negative for diabetes mellitus, neurologic, thyroid, or liver disease. Medications included a thiazide diuretic and alpha-methyldopa. Physical examination revealed an alert, oriented woman in no acute distress. Pulse was 72 beats/min and blood pressure was 136/88 mm Hg. The patient exhibited choreoathetotic movements of the right face, tongue, arm, and leg, and increased tone in the right upper and lower extremities. The serum glucose concentration was 584 mg/dl. Serum electrolytes, blood urea nitrogen, creatinine, calcium, phosphate, thyroid, and liver function tests were within normal limits. A CT scan of the brain revealed bilateral, 1-mm calcification of the caudate nuclei. The EEG was within normal limits. The choreoathetotic movements abated with return of the serum glucose to the 200–300-mg/dl range with insulin and hydration. The patient was discharged on haloperidol, intermediate-acting insulin, and the same antihypertensive regimen. Haloperidol was subsequently discontinued due to excessive sedation.

The patient did well for ~1 yr without any abnormal movements and serum glucose concentrations between 150 and 275 mg/dl when she presented with a 2-wk history of polyuria and a 3-day history of involuntary movements of the left face, tongue, arm, and leg, but no abnormal movements of the right side. The remainder of the examination was unchanged. The serum glucose level was 622 mg/dl. Serum electrolytes, calcium, phosphate, thyroid, and parathyroid hormone levels were within normal limits. CT scan of the brain revealed increasing calcification of the posterior head of the caudate nuclei bilaterally. EEG was again within normal limits. The choreoathetotic movements abated after the serum glucose level was normalized with insulin. The patient was discharged on intermediate-acting insulin and has had no further abnormal movements with continued good control of hyperglycemia.

DISCUSSION

Acquired paroxysmal choreoathetotic movement disorders arise from hypoparathyroidism, hyperthyroidism, and several neurologic diseases.[2,3] A review of the etiologies of acquired paroxysmal choreoathetosis revealed that 60% of the cases are unilateral, 30% are bilateral, and the remaining 10% are alternately unilateral and bilateral.[2] Our case demonstrates that choreoathetotic movements associated with diabetes mellitus may be alternately unilateral during periods of hyperglycemia and be successfully managed by restoring normoglycemia with insulin. Insulin therapy and hydration have been shown to be effective therapies for other neurologic manifestations of hyperglycemia and hyperosmolarity, including focal seizures and transient ischemic attacks,[4] by reestablishing previous osmotic gradients.

The choreoathetotic movements and basal ganglia calcification reported in this case are also seen in idiopathic hypoparathyroidism, a rare disorder in which neurons in the basal ganglia damaged by anoxia or vascular insufficiency are predisposed to reversible dysfunction during hypocalcemia. The calcification of the basal ganglia is felt to represent an irreversible process of pericapillary deposition of acid mucopolysaccharides, iron, and calcium that follows an anoxic, vascular, or encephalitic injury.[5] In several autopsy studies in diabetic individuals, areas of calcification correspond to severe sclerosis of basal ganglia vasculature with resultant encephalomalacia and loss of neuronal parenchyma.[6] These histopathologic findings have not been correlated clinically with the presence of a movement disorder since only 25% of patients with basal ganglia calcification will develop extrapyramidal deficits.[5] The infrequent reporting of choreoathetosis with diabetes mellitus would indicate that both preexisting neuronal degeneration of the basal ganglia and the acute metabolic effects of hyperglycemia are required to produce a clinical picture of alternating choreoathetosis.

JEFFREY A. SANFIELD, M.D.
JEROME FINKEL, M.D.
SCOTT LEWIS, M.D.
STEPHEN G. ROSEN, M.D.

From the Department of Medicine, Sinai Hospital of Detroit, Detroit, Michigan (J.A.S., J.F., S.L.); and the Division of Endocrinology and Metabolism, Department of Internal Medicine, University of Michigan, Ann Arbor, Michigan 48109 (J.A.S., S.G.R.).

Address reprint requests to Jeffrey A. Sanfield, M.D., at the above address.

REFERENCES

[1] Rector, W. G., Franklin, H. H., and Moses, H.: Nonketotic hyperglycemia appearing as choreoathetosis or ballism. Arch. Intern. Med. 1982; 142:154–55.

[2] Goodenough, D. J., Farrello, R. G., Annis, B. L., and Chun, R. W.: Familial and acquired paroxysmal dyskinesias. Arch. Neurol. 1978; 35:827–31.

[3] Fischbeck, K. H., and Layzer, R. B.: Paroxysmal choreoathetosis associated with thyrotoxicosis. Ann. Neurol. 1979; 6:453–54.

[4] Guisado, R., and Arieff, A. I.: Neurological manifestations of diabetic comas: correlation with biochemical alterations in the brain. Metabolism 1975; 24:665–79.

[5] Muenter, M. D., and Whisnant, J. P.: Basal ganglia calcification, hypoparathyroidism, and extrapyramidal motor manifestations. Neurology 1968; 18:1075–83.

[6] Vinken, P. J., and Bruyn, O. W.: Neurological manifestations of diabetes mellitus. *In* Handbook of Clinical Neurology. Vol. 27. New York, North Holland Publishing Co., 1976:99–142.

Remission of NIDDM After Irradiation of Metastatic Cervical Lymph Nodes

Remission of overt diabetes mellitus (DM) after removal of invasive epidermoid carcinoma of the tongue was reported by Rex and Duckworth[1] in 1984. Diabetes in the 54-yr-old man was detected when he was admitted for sore throat, dysphagia, and weight loss of 4 mo duration. Examination revealed a 2-cm exophytic lesion at the base of the tongue