Short Reports

Hypoglycemic Convulsions Cause Serious Musculoskeletal Injuries in Patients With IDDM

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Six diabetic patients are described who sustained
serious musculoskeletal injuries during insulin-induced
hypoglycemia. The convulsions were associated with
nocturnal hypoglycemia, superoptimal glycemic control,
pregnancy, hypoglycemic unawareness, or errors in self-
management. *Diabetes Care* 12:32–34, 1989

Hypoglycemia, and its ability to cause diminished consciousness, is a common side effect of insulin therapy and is associated with significant mortality and morbidity rates in diabetic patients (1). The morbidity related to hypoglycemia is poorly defined, with most reports emphasizing the effects of neuroglycopenia on the central nervous system or associated vascular events, such as myocardial infarction, stroke, and cardiac arrhythmias (2). Physical injuries related to hypoglycemia are seldom reported. We describe six patients (1 who has been described previously; 3) who sustained significant trauma as a result of the coma and convulsions caused by hypoglycemia. None of the patients were involved in high-risk activities nor had any clinical, radiological, or biochemical evidence of preexisting metabolic bone disease.

CASE REPORTS

Case 1. A 24-yr-old woman with insulin-dependent diabetes mellitus (IDDM) of 15 yr duration was controlled with twice daily injections of soluble and isophane insulins (Velosulin and Insulatard, Nordisk, Gentofte, Denmark). The prepregnancy insulin dose was 1.2 U $\cdot$ kg^{-1} $\cdot$ day^{-1}. At the time of the study, the subject was 7 wk pregnant and had a glycosylated hemoglobin level of 9.9% (nondiabetic range 5–8%). During the first trimester the subject's total insulin requirement was 1.6 U $\cdot$ kg^{-1} $\cdot$ day^{-1}, and her glycosylated hemoglobin level fell to 5.8%. The subject began to experience frequent severe episodes of hypoglycemia without warning symptoms; during one of these episodes, the subject became comatose, convulsed, and fell onto a carpeted bedroom floor. As a result, the subject sustained a comminuted spiral fracture of the left tibia and fibula. Conservative management was unsuccessful, and the fracture of the tibia required internal fixation that was complicated by infection. A healthy baby was delivered by cesarean section during the 38th wk of pregnancy, but the fracture and incision did not heal satisfactorily until 8 mo postpartum.

Case 2. A 50-yr-old art teacher with IDDM for 6 yr required 0.7 U/kg of insulin twice daily of soluble and lente insulins (Actrapid and Monotard, Novo, Copenhagen). The subject led a self-confessed chaotic lifestyle with erratic mealtimes and had a history of recurrent hypoglycemia and symptomatic unawareness. One evening the subject incorrectly estimated his dose of insulin while using a noninsulin syringe and as a result developed hypoglycemia while asleep. After regaining consciousness on the floor, the subject complained of a painful back. Radiographic examination demonstrated

compression fractures of vertebrae T12 and L1, which responded to conservative management. The subject's glycosylated hemoglobin level 1 mo previously was 11%. The subject had previously suffered hypoglyce-mia-induced convulsions, which had been witnessed by others. Therefore, the skeletal injury was attributed to a convulsion induced by hypoglycemia.

Case 3. A 32-yr-old university lecturer with IDDM for 21 yr required 0.8 U/kg of insulin twice daily of soluble and lente insulins (Actrapid and Monotard). One month after conversion to U-100 insulin, the subject had a hypoglycemic convulsion during the night and required intravenous dextrose to restore consciousness. After recovery, the subject complained of a painful back. Radiographic examination demonstrated crush fractures of vertebrae T8 and T9, which responded to conservative management. The subject's concurrent glycosylated hemoglobin level was 11.1%.

Case 4. A 31-yr-old engineer with IDDM for 12 yr controlled by 1.2 U/kg of insulin twice daily of soluble and lente insulins (Actrapid and Monotard) developed severe nocturnal hypoglycemia. On awakening one morning, the subject complained of severe tenderness and restricted mobility of both shoulders. Radiographic examination demonstrated fractures of both humeral necks. In the absence of a history of trauma, it was suspected that the subject had experienced a convulsion, although this episode was not witnessed. The subject had a concurrent glycosylated hemoglobin value of 6.4%. Two months later, the subject suffered a nocturnal convulsion, and coexisting hypoglycemic coma was demonstrated (blood glucose 1.0 mM), requiring intramuscular injection of glucagon to facilitate recovery.

Case 5. A 17-yr-old schoolboy with IDDM for 2 mo had been stabilized on an insulin dose of 0.6 U/kg twice daily of human soluble and crystalline ultralente insulins (Humulin S and Humulin Zn, Lilly, Indianapolis, IN). While on vacation, the subject exercised excessively after an insulin dose and evening meal without consuming additional calories. The subject developed severe nocturnal hypoglycemia that induced a generalized convulsion. Consciousness was restored with an intramuscular injection of glucagon. The subject complained of a painful left shoulder, and radiographic examination demonstrated dislocation of the shoulder joint that required reduction under general anesthesia. The subject's concurrent glycosylated hemoglobin level was 6.7%.

Case 6. A 31-yr-old teacher with IDDM for 12 yr requiring 0.9 U/kg of insulin twice daily of soluble and isophane human insulins (Velosulin and Insulatard) suffered a severe nocturnal episode of hypoglycemia after strenuous exercise without consuming additional calories. This hypoglycemic episode provoked a generalized convulsion, and an intramuscular injection of glucagon was administered to restore consciousness. After recovery, the subject complained of a painful left shoulder, and radiographic examination demonstrated a dislocation that required reduction under Entonox analgesia. The subject's concurrent glycosylated hemoglobin level was 9.0%.

DISCUSSION

Although severe hypoglycemia is of obvious danger to the diabetic patient, few reports have been published citing serious injury arising de novo from acute hypoglycemic coma. In this study, six patients sustained serious musculoskeletal injuries during hypoglycemic coma, which included long-bone and vertebral fractures and major joint dislocations. None of the patients were known to have idiopathic epilepsy; therefore, it was likely that hypoglycemia-induced convulsions caused the musculoskeletal injuries. Convulsions were witnessed in three of the patients and strongly suspected in the remaining three based on patients' histories. Injuries from hypoglycemic convulsions were sustained during sleep in five patients, three of whom committed errors in the management of their diabetes that may have contributed to the development of severe hypoglycemia. Nocturnal hypoglycemia may be protracted because the warning symptoms are often unrecognized, and the risk of inducing convulsions may increase (4). The sixth patient suffered from symptomatic hypoglycemic unawareness associated with pregnancy, which may also predispose to profound and prolonged hypoglycemia.

Compression fractures of spinal dorsal vertebrae after hypoglycemia-induced convulsions have been described in four diabetic patients (5). Bony fractures during convulsions are related to muscular spasm, and dorsal vertebral compression fractures have been attributed to relatively unopposed spinal flexion (6). In epileptic patients, such fractures have been associated with nocturnal convulsions. The incidence of injuries related to convulsions of any cause has rarely been reported, although vertebral compression fractures are considered to be the most common skeletal injury. Musculoskeletal trauma during convulsions was noted in 1.1% of 3000 patients undergoing convulsive therapy during psychiatric treatment in 1939 (7). The mechanical injuries included joint dislocations and bony fractures and involved the shoulder joint, scapula, femur, and humerus; vertebral compression fractures were not reported. One percent of epileptic patients were reported to have sustained symptomatic vertebral compression fractures during convulsions, but the overall incidence in unselected cases was estimated at 15% (8). A recent survey of our diabetic clinic population (unpublished observations) indicated that 10% of the patients have sustained a musculoskeletal or head injury related to hypoglycemia, and 7.9% have had one or more hypoglycemic convulsions during their diabetes.

Hypoglycemic convulsions can cause severe injuries in IDDM patients. In this study, hypoglycemic convulsions were associated with nocturnal hypoglycemia, superoptimal glycemic control, pregnancy, hypoglycemic unawareness, or errors in self-management. The frequency of injuries related to hypoglycemic convulsions may increase as a result of the current intensification of insulin regimens.

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Effect of Life-Style Activity of Varying Duration on Glycemic Control in Type II Diabetic Women

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Life-style activities such as walking are often recommended for patients with type II (non-insulin-dependent) diabetes. Because many of these patients are overweight and sedentary, such low-intensity activity would appear most appropriate, especially during initial intervention. However, there has been little research on the effects of low-intensity life-style activity on glycemic control. This study examined the effects of varying the duration (0, 20, or 40 min) of low-intensity exercise (50–55% of age-predicted max heart rate) on glycemic responses during exercise and a subsequent meal in type II diabetic patients. Glycemic response to exercise was significantly related to the duration of activity; 20 min of activity decreased blood glucose (BG) by 6 mg/dl, whereas 40 min decreased BG by 16 mg/dl. The effect of exercise on glucose was maintained over a 30-min rest period but disappeared after a meal was consumed. Insulin and the insulin-to-glucose ratio were not affected by the length of activity. These data suggest that life-style activity of long duration (20–40 min) produces a significant, but modest, decrease in glucose levels in type II diabetic women. Diabetes Care 12:34–37, 1989

Exercise is recommended for patients with type II (non-insulin-dependent) diabetes to improve weight loss and insulin sensitivity and decrease coronary heart disease risk factors (1–4). Because most of these patients are middle aged, overweight, and sedentary, the type of exercise that would appear most appropriate is low-intensity life-style activity, such as walking. However, there has been little research on the acute effects of such life-style activity on glycemic control. Moreover, although the glycemic response to exercise has been related to the amount of energy expended (5), there has been no research on the effect of increasing energy expenditure by increasing the duration of low-intensity activity (rather than increasing the intensity of exercise). Therefore, this study examined whether the duration of low-intensity preprandial activity would affect glucose and insulin responses during exercise and/or a subsequent meal in type II diabetic females.

MATERIALS AND METHODS

Subjects. Ten women with type II diabetes (aged 48.9 ± 17 yr, mean ± SD, 147 ± 31% of ideal body wt) were studied. Subjects had diabetes for an average of 4.5 yr and controlled their blood glucose (BG) by diet ($n = 7$) only or with oral medication (2 used chlorpropamide, 1 used tolazamide).
Procedures. Target heart rate for the exercise sessions was set at 50–55% of age-predicted maximum heart rate. This heart rate range was chosen because it is a level of activity that patients can safely and comfortably perform in an unsupervised setting and maintain for 20- to 40-min sessions and because it is comparable with that observed in life-style activities such as walking. The power output equivalent to this heart rate range was determined by submaximal cycle tests conducted on a Monarch 868 ergometer.
  Each subject completed three experimental sessions