Reversible decerebrate posturing after profound and prolonged hypoglycemia

MALCOLM M. KIRK, MD; BYRON J. HOOGWERF, MD; JAMES K. STOLLER, MD

Decerebrate rigidity is one of several reversible neurological abnormalities which have been observed in the setting of metabolic coma. We present the case of a patient who recovered fully from prolonged decerebrate rigidity associated with hypoglycemic coma. This case emphasizes the possibility of recovery from severe, prolonged hypoglycemia.

HYPOGLYCEMIA may cause a number of changes in brain function, some of which are reversible. We describe the case of a patient who recovered after exhibiting decerebrate posturing for six hours during hypoglycemia from a massive insulin overdose.

From the Departments of Pulmonary Disease (M.M.K., J.K.S.), and Endocrinology (B.J.H.), The Cleveland Clinic Foundation.

Address reprint requests to M.M.K., Department of Medicine, The University Hospitals of Cleveland, 2074 Abington Road, Cleveland, Ohio 44106-5000.
hypoglycemia. During the first 6 hours, decerebrate posturing
and a total of 800 mL of 50% dextrose solution.

When the serum glucose level rose to 238 mg/L and
was maintained at roughly this level, decerebrate pos-
turing disappeared, and the patient's level of conscious-
ness progressively improved. Over the next 3 days,
serum glucose levels ranged from 130 mg/dL to 250
mg/dL, and his mental status returned to a reported
baseline of verbal coherence, alertness, answering yes
or no questions, and following simple commands.
Decerebrate posturing did not return once the
hypoglycemia was corrected.

This patient's prior episodes of sustained
hypoglycemia, and the severe degree and long duration
of the current hypoglycemic episode were consistent
with an insulin overdose. This was confirmed by a
serum insulin concentration of greater than 11,000
μU/mL (normal fasting concentration 4 to 24 μU/mL)
in a sample obtained 12 hours after his initial presen-
tation to the local hospital. At 96 hours after presenta-
tion, the level was 79.7 μU/mL, and at 108 hours, it
was 65.0 μU/mL. Assays for plasma protein binding of
insulin ("insulin antibodies") showed <5% binding,
which is considered to be a normal value and of no
clinical significance in patients with diabetes mellitus.

DISCUSSION

The current report demonstrates two uncommon
but noteworthy clinical points: (1) decerebrate postur-
ing in hypoglycemia may be transient and reversible
with correction of hypoglycemia, and (2) neurologic
recovery is possible despite severe insulin overdose
with prolonged hypoglycemia.

Abnormal posturing was first observed in exper-
imental animals following intercollicular section of
the brain stem (decerebrate)1,2 and removal of all cor-
tex and white matter above the level of the basal
ganglia (decorticate). These two postures have been
consistently produced by specific anatomic lesions in
experimental animals, and it has been inferred that
analogous anatomic lesions are responsible for these
postures in humans. Indeed, clinical decortication and
decerebration most often result from a structural lesion
(usually trauma). However, a variety of metabolic and
other causes have been reported, including anoxia,3
infections,4 hepatic coma,5,6 the syndrome of inap-
propriate secretion of antidiuretic hormone (SIADH),7
and hypoglycemia.8,9 A few of these non-structural
causes have been associated with reversible posturing.

In our patient, decerebrate rigidity coincided with
hypoglycemia. During the first 6 hours, decerebration
rapidly resolved when hypoglycemia was corrected and
recurred when hypoglycemia returned.

Two previous cases of reversible decerebrate postur-
ing secondary to hypoglycemia have been reported.
Seibert8 described the remission of decerebrate postur-
ing after administration of intravenous glucose in two
patients, one of whom was a man with adult-onset
diabetes who presented in coma with decerebrate pos-
turing in response to painful stimuli and a serum
glucone of 35 mg/dL. He became alert and conversant
following 50 mL of 50% dextrose. (The other patient
was a 59-year-old male alcoholic with neither diabetes
nor insulinoma and without documented hypogly-
ecemia, who presented with hypothermia, tachycar-
dia, diaphoresis, and coma with decerebrate posturing,
as well as anisocoria and bilateral extensor plantar
reflexes. He awoke and became neurologically normal
immediately after receiving 25 g of dextrose.)

The second case, described by Ogunyemi and
Olowoyeye,9 concerned a 65-year-old man with
hypoglycemia caused by religious fasting and diarrhea.
He became comatose with decorticate posturing on the
right side in response to painful stimuli on either side of
the body. The serum glucose was 8 mg/dL. An in-
travenous bolus of 100 mL of 50% dextrose solution
resulted in resolution of the decorticate posture and
return of consciousness within 90 minutes.

Our patient's course differs from the previously
described experience, principally in the duration of the
decerebrate posturing. In each previous case, the com-
atose patient demonstrated decerebrate posturing in
response to painful stimuli, but decerebration was
rapidly reversed and consciousness restored by a single
dose of dextrose. In the current case, posturing oc-
curred intermittently for 6 hours after admission, then
constantly for 6 hours, and the presence of decerebrate
rigidity coincided with hypoglycemia.

It is known that hypoglycemia can cause seizures,
and Seibert8 postulated hypoglycemia-induced tonic
seizure activity as a mechanism for the decerebrate
rigidity seen in his patients. Our patient, however,
clearly demonstrated adduction, extension, and pron-
atation of the arms, a posture very different from the
abduction and flexion of the arms seen in global tonic
seizures.10 Furthermore, this patient's posture was made
more prominent by noxious stimuli, which is typical of
decerebrate rigidity11 but not of seizures. Haines12
describes five cases of decerebrate posturing which
were misinterpreted as seizures, and gives an excellent
account of the manner and importance of differentiat-
ing between the two.
Our patient's return to baseline neurologic status after a total of more than 6 hours of decerebration was unexpected. Arem and Zoghbi\textsuperscript{13} indicate that full neurologic recovery after prolonged hypoglycemic coma without posturing (up to 6 days) is not unusual, although there is no account of recovery from such prolonged hypoglycemic decerebration. Using a pooled analysis of eight of their own patients and 38 cases from the literature, Arem and Zoghbi found that neither the magnitude of the insulin dose nor the severity of the resulting hypoglycemia had any effect on the clinical outcome in intentional insulin overdose. The most important factor was delay in initiation of treatment.\textsuperscript{13} That the human brain can withstand long periods of metabolic insult severe enough to cause decerebration is shown by the reports of Conomy and Swash\textsuperscript{5} in which two patients with hepatic coma demonstrated decerebrate rigidity that lasted about 2 days and resolved as the coma cleared, without neurological sequelae.

The total amount of insulin given to this patient is unknown. That the insulin dose was especially large can be inferred from the serum insulin level: 12 hours after admission it exceeded 11,000 µU/mL, which is among the highest values ever reported. However, confounding factors make it difficult to estimate the size of the dose. When injected intravenously, insulin has a plasma half-life of about 10 minutes,\textsuperscript{14} but the principal determinant of serum levels is the rate of release from the injection site which, in turn, is influenced by the type of insulin used. An added factor in this case was the presence of complete renal failure, a variable not discussed in most of the earlier reports of insulin overdose. Insulin is degraded mainly in the liver and kidneys, and renal failure causes a significant lengthening of the half-life of insulin (the liver normally operates close to its capacity to metabolize insulin and cannot compensate for loss of kidney function).\textsuperscript{14}

The current case demonstrates that decerebrate posturing in hypoglycemia may be transient and reversible with correction of hypoglycemia, and that neurologic recovery may be possible despite severe insulin overdose with resulting prolonged hypoglycemia.

\textbf{REFERENCES}