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Transient Global Amnesia in a Collegiate Baseball Player with Type I Diabetes Mellitus: A Case Report

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Objective: To present the case of a collegiate pitcher with type I diabetes mellitus who developed transient global amnesia and to characterize the acute onset of symptoms and clinical diagnosis of this rarely reported neurologic condition in the student–athlete population.

Background: A 21-year-old collegiate pitcher with type I diabetes mellitus was found by his roommate to have acute-onset memory loss. The athletic trainer identified normal blood glucose levels and normal vital signs but profound amnesia. The patient was evaluated by his team physician and referred to the local emergency department for acute-onset memory disturbance.

Differential Diagnosis: Hypoglycemia, ketoacidosis, adverse drug reaction, infectious disease, transient epileptic amnesia, transient ischemic attack, acute confusional state, complex partial seizure, psychogenic amnesia, migraine, intracerebral hemorrhage, traumatic brain injury, tumor, and transient global amnesia.

Treatment: Diagnostic studies included computed tomography of the head, urine and serum toxicology, urinalysis, blood glucose level, electrolytes, blood urea nitrogen level, creatinine level, complete blood count, and electroencephalography. The patient was admitted overnight to the neurology service. The next morning, electroencephalography was repeated, and magnetic resonance imaging of the head with contrast was performed. The patient was discharged with the diagnosis of transient global amnesia.

Uniqueness: Transient global amnesia is considered a benign condition characterized by an acute episode of memory disturbance involving the inability to form new memories and recall recent events. It is rare in young people, with only 3 case reports involving young athletes published in the literature.

Conclusions: Transient global amnesia is a rarely diagnosed neurologic disturbance that may present acutely in student–athletes, although most reported cases affect older adults. Unfamiliarity with the symptoms may cause anxiety for the athlete and bystanders. Transient global amnesia does not result in long-term neurologic deficit, and neurologic function will return to baseline.

Key Words: memory loss, neurologic conditions, athletes

Features of transient memory loss were first described independently by Bender[1] and Guyotat and Courjon[2] in 1956 and formally named *transient global amnesia* (TGA) by Fisher and Adams[3] in 1958. Few cases of TGA have been documented in children and young adults.[4–7] We present the case of a collegiate baseball player with diabetes who experienced transient global amnesia.

CASE REPORT

A 21-year-old collegiate baseball player with type I diabetes mellitus diagnosed at age 9 years was brought to the athletic training room by his roommate on a Sunday morning at approximately 10 AM during the fall practice season because of memory loss. The roommate reported that the patient kept asking questions such as "When is practice?" and "What is my blood sugar?" Upon questioning by the athletic trainer, the patient was unable to recall any events of the previous day and whether he had eaten, checked his blood sugar, or given himself any insulin. His roommate reported that they had spent the prior evening watching football and baseball on television. The roommate and the patient both denied alcohol or drug use, and the roommate reported that the patient was his usual self before going to bed at about 1 AM. According to the log on his insulin pump, the patient had given himself a bolus of 5 units of Novolog (Novo Nordisk, Inc, Princeton, NJ) at about midnight and inferred that he must have had a light snack; he stated that he never gave himself insulin boluses unless he was about to eat. He was unable to recall what he ate, but his roommate confirmed that he had eaten a snack. The insulin pump showed no record of any other insulin boluses after midnight. The patient denied headache, visual changes, nausea or vomiting, numbness, tingling or weakness in the extremities, or slurred speech. He had no history of migraines, recent illness, or head trauma. Current medications included Novolog at a basal rate of 1.1 to 1.5 units per hour in addition to 1 unit per 12 grams of carbohydrates before each meal.

Upon examination in the athletic training room by the team physician and athletic trainer, the patient was alert but somewhat anxious. He asked the athletic trainer several times whether he had checked his blood sugar. His vital signs were as follows: temperature, 98.2ºF (36.7ºC); blood pressure, 143/90 mm Hg; heart rate, 82 beats/min; respiratory rate, 18 breaths/min. His speech was fluid. He was oriented to person, place, and time, except that he reported the date as 1 day earlier. He performed serial 7s correctly and missed 1 month when stating the calendar months backward. He was able to recall 3/3 words immediately and 0/3 after distraction. He was unable to

recall the words with prompting. Abstract thinking was intact. He was able to give only 7 words that started with the letter "A" or "F." There was no agnosia or apraxia. He was able to recall the names and ages of his family members, which high school he attended, and the classes he was enrolled in. Cranial nerves II through XII were intact. Muscle strength was 5/5 in all major muscle groups of the upper and lower extremities bilaterally. Cerebellar functioning was intact; reflexes were 1+ in the Achilles tendon bilaterally and could not be elicited in the biceps or triceps muscle or patella bilaterally. Gait was normal. Cardiac examination revealed a regular rate and rhythm, and his lungs were clear to auscultation bilaterally. Fingerstick blood glucose level was 5.33 mmol/L (96 mg/dL). Dipstick urinalysis was negative for ketones.

The patient was referred to the local emergency department for further evaluation of his persistent amnesia. He still showed deficits in short-term memory recall; otherwise, his neurologic examination remained normal. Reflexes in the emergency department were 2+ at the brachial and patellar tendons. While in the emergency department, he developed a dull headache; computed tomography scan of the head was normal. Toxicology screens were negative for benzodiazepines, barbiturates, cocaine, opiates, cannabinoids, and amphetamines (urine screen) and acetaminophen, salicylate, and ethanol (blood screen). Urinalysis was negative. Blood glucose was 7.5 mmol/L (135 mg/dL); electrolyte, blood urea nitrogen, and creatinine levels and complete blood count were all normal. The patient was admitted overnight to the neurology service. On admission, reflexes were documented as 2/4 at all sites. His ability to form new memories started to improve about 6 hours after his initial presentation in the athletic training room, but he remained unable to recall any events from the night before or the morning of presentation. Additional diagnostic studies included electroencephalography, which initially showed mild bitemporal lobe slowing. By the next morning, the patient's headache had resolved and he had complete return of anterograde memory. He still lacked memory for events that occurred the night before and the morning of presentation, but his memory for events after hospital admission returned before discharge. Repeat electroencephalography was normal. Magnetic resonance imaging of the head with contrast was also normal. The patient was discharged with the diagnosis of transient global amnesia. The neurologist was puzzled by the patient's lack of ability to name at least 10 words per minute starting with the letters "A" or "F." Therefore, the patient was asked to follow up in 3 weeks for further evaluation.

At his 3-week and 6-month follow-ups, the patient was still able to give only 7 words beginning with the letters "A" or "F." This deficit remains unexplainable. We have no baseline for comparison and so are unable to interpret these results. His neurologic examination otherwise remained normal except for an amnestic gap for the duration of the attack. He performed well in school and on the field the remainder of the year.

Consideration was given to the possibility that the transient global amnesia might have resulted from a hypoglycemic event leading to hypoxia, but hypoglycemia was never found, and he never showed any altered level of consciousness throughout the episode. We believe that the initial absence of reflexes was not pathologic but examiner dependent. The team physician still has difficulty obtaining the patient's reflexes bilaterally, and the patient later reported that many physicians have had difficulty eliciting his reflexes in the past. The fact that the neurologist could elicit reflexes on admission lessens the likelihood that the initial absence of reflexes was a pathologic finding.

DISCUSSION

Transient global amnesia is considered a benign condition involving the inability to form new memories (anterograde amnesia) for several hours, along with the inability to recall recent events (retrograde amnesia). Remote memory is not disrupted. The incidence is reported to be 0.005% to 0.010% per year.[4,8] The condition is seen more commonly in those 50 to 80 years of age; most patients are in their 60s.[9]

Diagnostic criteria[10] (Table 1) include the acute onset of a witnessed anterograde amnesia that is usually discovered after the patient is found to ask the same questions repeatedly despite just having been given an answer. Cognition and consciousness are not affected, and no focal neurologic deficits exist. Attacks typically last from 1 to 8 hours[8,9] but not more than 24 hours.[10] There is no history of head trauma or seizures. Occasionally headache, nausea, or vomiting accompanies TGA. Patients return to baseline but have a gap in their memories for the duration of the attacks.[10]

The differential diagnosis of transient global amnesia is listed in Table 2. Temporal lobe epilepsy has been dismissed as a cause because epileptic symptoms usually last less than 1 hour, and the condition has a high recurrence rate.[4,8] Transient ischemic attacks are usually associated with motor and sensory deficits and not with anterograde amnesia by itself. Also, transient ischemic attacks can recur. An acute confusional state commonly occurs over a longer period of time (hours to days) and includes disorientation, impaired cognition, and hallucinations.[11] Inattention is a key difference between TGA and acute confusional state.[8] Complex partial seizures most often begin with an aura or tunnel vision, followed by impaired consciousness and automatisms.[11] Psychogenic amnesia involves loss of autobiographical memories and self-identity, usually triggered by stress. Retrograde amnesia is evident, but new learning is not disrupted; therefore, repetitive questioning by the patient does not occur.

In our patient's case, hypoglycemia was also entertained as the underlying problem because of his history of type I diabetes mellitus. However, the patient had lived with diabetes for 12 years and reported that his symptoms were not similar to those of previous hypoglycemic episodes. In addition, he was never found to be hypoglycemic. His blood sugar at initial presentation was 5.33 mmol/L (96 mg/dL). It is possible that the patient suffered a hypoglycemic event while sleeping but recovered by morning. This could have happened if low glucose led to the release of stress hormones such as epinephrine, cortisol, and growth hormone, which raise glucose levels. Two case reports[12,13] of prolonged amnesia after hypoglycemia have been published. However, both cases involved a severe hypoglycemic coma, changes on magnetic resonance imaging, and amnesia (including working and short-term memory) that resolved within months rather than hours of presentation.

Table 1. Diagnostic Criteria for Transient Global Amnesia[10]

Acute onset of anterograde amnesia
Witnessed attack
No change in cognition or level of consciousness (except for amnesia)
No loss of personal identity
No focal neurologic deficits on examination
Symptoms clear within 24 hours
No history of head trauma
No epileptic features or history of seizures

Table 2. Differential Diagnosis of Transient Global Amnesia

	Impaired Cognition	Impaired Consciousness	Focal Neurologic Deficits	Duration of Symptoms	Rate of Recurrence	Anterograde Amnesia	Inattention	Automatisms
Transient global amnesia	No	No	No	<24 h	Low	Yes	No	No
Transient epileptic amnesia	No	No	No	<1 h	High	Yes	No	Yes
Transient ischemic attack	No	No	Yes	<24 h	High	No	No	No
Acute confusional state	Yes	Yes	No	Hours to days	High[a]	No	Yes	No
Complex partial seizure	Yes	Yes	No	Minutes	High	No	Yes	Yes
Psychogenic amnesia	No	No	No	Hours to days to years	Low to high	No	No	No

[a] If underlying cause is not found.

In their review of the literature, Quinette et al[9] found that the 3 most common precipitators of an attack were emotional stress, physical effort, and extreme temperature change, such as immersion in cold water. Our patient lacked any of these precipitators. He had served as umpire for a baseball scrimmage the day before the onset of amnesia but otherwise had not engaged in any intense physical activity. No emotional stressors were identified, and he had no recent history of experiencing extreme temperature changes.

The areas of the brain involved in TGA are known: the mediobasal temporal region, hippocampus, and parahippocampus.[8] Yet the mechanism for TGA remains controversial. Various hypotheses have been put forth to explain the cause of TGA, including spreading depression of cortical electrical activity (from migraine headache) and venous congestion with ischemia in areas involving memory. Based on their literature review and study of 142 patients with TGA, Quinette et al[9] postulated that TGA may have at least 3 different causes: a neurotoxic effect on hippocampal function occurring after emotional or physical stress, venous congestion due to insufficient jugular vein valves precipitated by a Valsalva maneuver, and spreading depression of cortical activity in younger patients with a history of migraine. Cortical depression involves a wave of cellular depolarization that causes a brief period of cortical excitation followed by prolonged nerve depression and can be seen during a migraine attack. Quinette et al[9] questioned this last hypothesis because the risk of experiencing a migraine is higher in younger than in older patients. Therefore, coexistence of migraine and TGA cannot be ruled out. They found that patients with TGA were no more likely than a control group to have vascular risk factors or a history of migraines.

Very few cases of TGA have been reported in athletes. Tosi and Righetti[4] documented 2 cases of TGA, one in a 13-year-old female volleyball player and another in a 16-year-old male soccer player. Both occurred during athletic participation and were associated with migraine headache. The only other published case report[5] of TGA in an athlete involves a 13-year-old male soccer player who developed symptoms consistent with TGA after an evening practice. Although he did not have a history of migraines, he complained of a headache on admission.

CONCLUSIONS

Although TGA can bring much anxiety to the patient and witnesses of the attack, it is considered a benign condition and is not thought to be a precursor for stroke. Transient global amnesia rarely recurs, and the only lasting deficit is an amnestic gap for the duration of the attack. Health care providers should be aware of the signs and symptoms to address this condition promptly and appropriately.

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