



Case report

A case report of exertional heat stroke with multi-organ failure following GLP-1 receptor agonist while on the Kokoda Track

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Abstract

An Australian male in his early fifties developed severe exertional heat stroke [EHS] trekking on the Kokoda Track in Papua New Guinea. The patient had been taking semaglutide (Ozempic), a glucagon-like peptide-1 receptor agonist for weight management. Initial presentation included generalised tonic-clonic seizures, altered consciousness, tachycardia, and tachypnoea in the field. Immediate creek immersion cooling was performed, as a presumptive diagnosis of EHS was made in the absence of rectal temperature measurement. The efficacy and practicality of cooling is irrespective of location and best-available attempts are a priority in EHS. Subsequent domestic then international medical evacuation was initiated due to multi-organ dysfunction after field cooling. The patient achieved complete neurological recovery without long-term sequelae. This case highlights GLP-1 agonist-induced dehydration as a modifiable risk factor for heat illness in austere trekking environments and underscores the critical importance of immediate field-based cooling and early recognition of heat stress.

Keywords GLP-1 agonist, Kokoda track, cooling, cold water immersion, exertion heat stroke.

For referencing Solano T. A case report of exertional heat stroke with multi-organ failure following GLP-1 receptor agonist while on the Kokoda Track. *JHTAM*. 2026;8(1):32-36.

DOI <https://doi.org/10.33235/JHTAM.8.1.32-36>

Submitted 21 April 2026, Accepted 28 April 2026

Background

Exertional heat stroke (EHS) is defined by core body temperature exceeding 40°C combined with central nervous system dysfunction arising during physical activity which proceeds into a noncompensable phase.^{1,2,3} EHS represents a medical emergency with outcomes strongly dependent on the speed and quality of cooling in the initial minutes after collapse.^{1,2,3} The Kokoda Track in Papua New Guinea traverses 96kms of dense tropical jungle, with ambient temperatures frequently exceeding 35°C and relative humidity often above 80%.^{4,5} This unforgiving terrain has claimed the lives of numerous trekkers; since 2001, at least six Australian trekkers have died on the track from suspected heat-related causes, with numerous trekkers per year requiring helicopter medical evacuation.^{6,7,8} Emergency evacuation involves complex satellite/radio communications, helicopter transport and onward international medical repatriation—a complex logistical chain.^{9,10}

GLP-1 receptor agonists—including semaglutide (Ozempic, Wegovy)—are prescribed with increasing frequency for obesity and weight management.^{11,12} These medications suppress appetite and attenuate thirst drive, creating susceptibility to dehydration under heat stress.^{11,12,13} Gastrointestinal side effects including nausea, vomiting and diarrhoea further compound

fluid losses and often detract from the caloric intake required for trekking.^{11,12} Semaglutide has a prolonged half-life of approximately 168 hours (7 days), meaning biologically active drug concentrations persist for substantial periods following administration.^{14,15} Despite widespread use, GLP-1 receptor agonists constitute an underrecognised and highly modifiable risk factor for EHS in austere trekking environments.

Case presentation

The patient, an Australian male in his early fifties, was trekking the Kokoda Track in a group setting. Weather conditions on the day of collapse included sunny skies with a midday temperature of approximately 33°C and humidity exceeding 85%. His past medical history included obstructive sleep apnoea managed with continuous positive airway pressure and hypercholesterolaemia. Regular medications comprised semaglutide (weekly, last dose one day prior to collapse) rosuvastatin 20mg oral daily, aspirin 100mg daily for cardiovascular primary prevention, and doxycycline for antimalarial prophylaxis.

On the second day of trekking, the patient's companions reported a prodrome of several hours: muscle cramps, altered mentation, vomiting and progressive delirium, culminating in syncope and

generalised tonic-clonic seizures at approximately 12:30. Initial medical care was provided by another medical practitioner who placed an intravenous cannula, administered metoclopramide for vomiting, moved the patient into the shade, and doused him with water. The reporting clinician arrived approximately 45 minutes after initial collapse and found the patient unresponsive (Glasgow Coma Scale E1V1M2) on a hillside. In this field-based emergency, a presumptive diagnosis of EHS was made on clinical grounds, based on the constellation of altered mental status, seizure activity, recent strenuous exertion in extreme heat and humidity and collapse.

Initial field observations included heart rate approximately 160 beats per minute, respiratory rate 60 breaths per minute, and a thready pulse. Immediate management comprised of rapid transport approximately 100m to a nearby creek via stretcher where the patient was submerged neck down in a cool creek with clothing removed for over 60 minutes. Concurrently, a retrieval request was initiated via satellite phone communication to ground support, and local porters constructed a makeshift helicopter landing zone. Intravenous midazolam 5mg bolus was administered terminating the seizure activity. Due to weak thready pulse on carotid palpation, intravenous adrenaline 20micrograms was administered, after which the patient demonstrated clinical improvement with Glasgow Coma Scale improving to E4V4M4. Legs were elevated to assist venous return. The helicopter landed at 15:15; during transfer the patient sustained a further generalised tonic-clonic seizure, likely precipitated by the warm aircraft environment and cessation of active cooling. The intravenous cannula was dislodged mid-flight. Intramuscular midazolam 5mg was administered successfully, terminating the recurrent seizure.

On arrival to a Port Moresby hospital at approximately 16:00, the patient had Glasgow Coma Scale of 3, non-invasive blood pressure approximately 73/40mmHg, heart rate 168 beats per minute, and oxygen saturation 92% on 10 litres per minute supplemental oxygen. Rapid sequence intubation was performed with subsequent dental trauma by the local clinician. The patient was then cooled with intravenous Hartmann's solution and iced towels. The patient was transferred to intensive care at approximately 19:30 with dark concentrated urine, blood pressure supported on noradrenaline infusion (non-invasive blood pressure approximately 120/50mmHg), oxygen saturation 98%, minimal urine output, mechanical ventilation, and midazolam/fentanyl infusion via right internal jugular central line.

By approximately 22:30 (seven hours post-collapse), the patient was haemodynamically stable with noradrenaline weaned off. By 06:00 the following morning, approximately 14 hours post-event, the patient was E4VTM6 and following commands, with approximately 4 litres of crystalloid administered overnight.

Indwelling catheter output was approximately 120mL of concentrated urine. The ventilator had been transitioned to spontaneous mode. International medical repatriation to a Brisbane-based intensive care unit in Australia was completed. Upon arrival to the Australian intensive care unit, the patient was successfully extubated with no ongoing haemodynamic support required and no need for renal replacement therapy. The patient was discharged to the ward the following day and achieved complete neurological recovery without long-term sequelae.

Investigations

Laboratory investigations obtained on arrival to the Port Moresby hospital confirmed multi-organ dysfunction consistent with EHS. Liver function tests demonstrated elevated aspartate aminotransferase (152.3IU/L; reference 8–37) and mildly elevated alanine aminotransferase (50.8IU/L; reference 20–40). Renal function revealed markedly elevated blood urea nitrogen (13.5 mmol/L; reference 2.21–7.7) and creatinine (251.9µmol/L; reference 53–123), confirming acute kidney injury. Inflammatory markers included markedly elevated procalcitonin (2.68ng/mL; reference 0–0.3) with normal C-reactive protein (3.44mg/L; reference 0–6.0). Creatine kinase was critically elevated (3946.8IU/L; reference 25–200), confirming exertional rhabdomyolysis. Lactate dehydrogenase was elevated (953.8U/L; reference 140–280). D-dimer was elevated (9.250µg/mL; reference 0.0–0.5), raising concern for early disseminated intravascular coagulation, an independent predictor of mortality in EHS.^{1,2,16} A computed tomography brain image demonstrated a normal study with no cerebral oedema identified. These laboratory findings are consistent with the multi-organ manifestations of severe EHS.

Discussion

Field-based emergency cooling in austere environments

This case exemplifies the critical importance of immediate field-based cooling in EHS, particularly in austere environments where sophisticated medical equipment or even ice is unavailable. Cold water immersion is the gold-standard cooling modality for EHS, achieving cooling rates of 0.15–0.35°C per minute with near-100% survival when initiated within 30 minutes of collapse under controlled conditions.^{17,19,21} In this case, an adjacent creek served as the improvised cooling medium—a pragmatic application of the cold water immersion principle in an environment where ice baths or dedicated cooling equipment was unavailable. The efficacy and practicality of cooling is irrespective of location and environment. The “cool first, transport second” model endorsed by the Wilderness Medical Society and international consensus statements was implemented in this case, with evident clinical benefit demonstrated by Glasgow Coma Scale improvement from E1V1M2 to E4V4M4 within approximately 60 minutes of cooling.^{17,18,19,20,23,24} Wilderness Medical Society guidelines explicitly state that in the field-based emergency setting, cooling should be initiated immediately at time of collapse and should be

based on feasible field measures. The cold water immersion was also reflected as a recommendation update for EHS from the Australian and New Zealand Committee of Resuscitation [ANZCOR] in September 2020.²³ Specifically ANZCOR recommend a immersion [from neck down / whole body] in cold water for 15 minutes while awaiting transport as the most effective method for core cooling.

GLP-1 Receptor agonists as modifiable risk factor for exertional heat stroke

The patient had taken his last dose of semaglutide (Ozempic) one day before the EHS event. Semaglutide's pharmacological half-life of approximately 168 hours (7 days) means biologically active drug concentrations were present at the time of collapse.²⁶ Semaglutide suppresses appetite through GLP-1 receptor agonism in hypothalamic nuclei, and this appetite suppression concurrently attenuates thirst drive—a mechanism that predisposes to underhydration.^{11,12,13,27,28} Combined with heat stress and a reduced appetite and thirst drive this attenuates caloric and fluid input and increased predisposition to EHS.^{11,12,27} GLP-1 receptor agonists delay gastric emptying, which represents the primary mechanism underlying their glucose control and weight loss benefits, but also increases susceptibility to dehydration and reduces the physiological drive to consume fluids.^{11,12,27,28}

Pre-expedition medical assessment should identify GLP-1 receptor agonist use as a significant modifiable risk factor requiring careful consideration and trip planning. Current perioperative clinical guidelines by the Australian and New Zealand College of Anaesthetists [ANZCA] no longer recommend cessation of GLP-1 medications for elective procedures.²⁹ Given the role these medicines have for diabetic indications the role of GLP-1 receptor agonists may be viewed differently in the light of weight loss in austere environments. Cessation of weekly-dosed GLP-1 receptor agonists 14–21 days prior to commencing high-exertion trekking in hot, humid austere environments would theoretically allow for substantial clearance of biologically active drug (approximately 2–3 half-lives).^{14,15,27,28,30,31,32,33}

However, this recommendation must be balanced against the potential for deteriorating glycaemic control in patients with diabetes mellitus who require GLP-1 agonist therapy for glucose management. An individualised approach involving consultation with endocrinology specialists, structured pre-trek acclimatisation with close monitoring, aggressive hydration protocols with consideration of alternative shorter-acting glucose control strategies during the peri-trek period would represent optimal risk mitigation. For patients using GLP-1 agonists primarily for weight management rather than diabetes control, discontinuation 14–21 days pre-trek represents a modifiable intervention with minimal metabolic consequence and substantial potential benefit in reducing EHS risk.^{11,12,27,28,30,31,32,33} In particular to recreational trekking in austere

locations these medicines can be safely stopped while in an austere trekking location for a short duration with minimal to no clinical implications. Given the documented side effects of GLP-1 agonists, pre-departure trek medical professionals should strongly consider cessation of GLP-1 agonists prescribed for weight loss.

Clinical implications and future directions

This case underscores several critical clinical implications for pre-expedition medical screening and risk stratification. First, GLP-1 receptor agonist use must be identified during pre-trek medical assessments, with specific counselling regarding dehydration risk and consideration of medication cessation 14–21 days prior to high-exertion trekking in hot environments.^{11,12,27,28,30,31,32,33} Second, expedition medical teams and trek leaders must be trained in the recognition of early warning signs of heat illness—muscle cramps, altered mentation, vomiting, progressive delirium—and empowered to halt exertion and initiate immediate cooling at the first sign of deterioration.^{1,2,17,18,19,20}

Third, the efficacy and practicality of therapeutic cooling is irrespective of location and needs to occur early in EHS; natural water sources (creeks, rivers) represent readily available and effective improvised cooling resources in jungle and wilderness settings.^{17,18,19,20,23,24,34} Fourth, the presumptive diagnosis of EHS in field-based emergency settings should be based on clinical criteria—recent exertion in heat, central nervous system dysfunction, cardiovascular compromise—rather than delayed pending rectal temperature measurement, which may be unavailable or impractical in austere environments.^{31, 32,33,35}

The complete neurological recovery and resolution of multi-organ dysfunction in this case demonstrates that even severe EHS with multi-organ failure can have excellent outcomes when immediate field-based cooling is implemented, followed by coordinated international medical repatriation and intensive care management.^{1,2,17,22} Future research should investigate the specific pharmacokinetic and physiological interactions between GLP-1 receptor agonists and thermoregulation during exertional heat stress, develop evidence-based guidelines for GLP-1 agonist cessation timing prior to high-exertion activities in hot environments, and evaluate structured pre-trek acclimatisation protocols for patients using these increasingly common medications.^{11,12,27,28,30,31,32,33}

Learning points

- GLP-1 receptor agonists constitute a highly modifiable risk factor for EHS through suppression of thirst drive and gastrointestinal side effects; pre-expedition medical screening must systematically identify GLP-1 agonist use, with consideration of medication cessation 14–21 days prior to high-exertion trekking in hot, humid austere environments, particularly for patients using these agents for weight management rather than diabetes control.

- The efficacy and practicality of cooling is irrespective of location in EHS: improvised cold water immersion using natural water sources (creek immersion in this case) represents a life-saving field-based emergency intervention in austere environments and should follow the “cool first, transport second” model endorsed by Wilderness Medical Society guidelines, even when sophisticated equipment is unavailable.
- Complete neurological recovery and resolution of multi-organ dysfunction (rhabdomyolysis with creatine kinase 3946.8 IU/L, acute kidney injury with creatinine 251.9µmol/L, coagulopathy with D-dimer 9.250µg/mL) can be achieved in severe EHS when immediate field-based cooling is implemented within 60 minutes of collapse, highlighting that outcomes are strongly dependent on the speed and quality of initial cooling.

Competing interests

None declared.

Funding

No external funding was received for this case report.

Patient consent for publication

Informed consent was obtained from the patient for publication of this case report and any accompanying data. Patient anonymisation has been implemented.

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