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Exceptional survival of an airplane stowaway, treated successfully with hyperbaric oxygen

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ABSTRACT

Survival of airplane stowaways is rare. Here we report an exceptional case of successful treatment and full recovery. After a transcontinental flight an unconscious stowaway was discovered in a wheel well of a Boeing 747-400F. Airport paramedics confirmed regular respiration and achieved 100% oxygen saturation (pulse oximetry) by high-flow oxygen. Rectal body temperature was 35.5 °C. On arrival at the emergency department, the patient's vital signs were stable. He did not respond to verbal stimuli. He localized to painful stimuli with both arms, however, there was no reaction to stimuli to both legs. We suspected his neurological deficits were caused by posthypoxic encephalopathy or altitude decompression sickness (DCS), the latter amenable to hyperbaric oxygen therapy (HBOT). HBOT was performed for 5 h (US Navy Treatment Table 6) and afterwards, full neurological recovery was documented. About 24 h after admission a new proximal paresis of the left leg was noted. Assuming recurrence of DCS, daily HBOT was scheduled for three days, after which motor function had again returned to normal.

Stowaways travelling in airplane wheel wells experience extreme environmental circumstances. The presented patient survived an eight-hour exposure to calculated barometric pressures as low as 190 mmHg and ambient PO_2 of 40 mmHg. Apart from creating awareness of this rare patient category, we want to stress the risk of altitude DCS in unpressurized flights. When DCS is suspected, immediate high-flow oxygen therapy should be initiated, followed by HBOT at the earliest opportunity.

1. Introduction

Survival of airplane stowaways hiding outside the pressurized compartment is rare, because of the extremely low atmospheric pressure, partial oxygen tension and temperature during flight [1,2]. Here we report an exceptional case, with successful treatment and full recovery.

2. Case report

About 45 min after arrival at the airport of Amsterdam (The Netherlands) a stowaway was discovered in a wheel well of a Boeing 747-400F, coming from Nairobi (Kenia). The apparently lifeless victim, later confirmed to be a 22 year old man, was found on a small latch within the wheel bay (Fig. 1). Paramedics of the Airport Medical Services detected signs of life and dispatched a physician-staffed emergency medical service.

The patient presented with an open airway, regular breathing pattern with normal bilateral breath sounds, 100% oxygen saturation (pulse oximetry) on high-flow oxygen through a non-rebreathing mask, a blood pressure of 145/115 mmHg and a heart rate of 115/min.

Both eyes were open spontaneously, however, not fixating nor following objects. He demonstrated localization to painful stimuli to both arms and trunk, without verbal response. No external signs of trauma, nor skin abnormalities (e.g. frost bite, hematoma or mottled skin), nor subcutaneous emphysema were identified. Rectal body temperature was 35.5 °C. He was heated passively, and actively with 500 mL warmed Ringer's lactate. The patient was transported to a Level-1 trauma center.

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Fig. 1. Photograph of the stowaway as found in the left body gear wheel well of a Boeing 747-400F. Visible are yellow ropes that the patient had secured himself with to prevent him from falling. He was wearing thin, but layered clothing and a knit cap. Also depicted: Part of the paramedic (right margin) who climbed into the wheel-well to attach monitoring, such as the blood pressure spiral-hose and blood pressure cuff to the victim's right arm.

On arrival at the emergency department, the patient's vital signs were stable. Pupils were isocoric and responsive to light. He did not respond to verbal stimuli. He localized to painful stimuli with both arms, however, there was no reaction to painful stimuli to both legs. Plantar stimulation elicited no response on the left and a Babinski-sign on the right side.

Height (175 cm) and body weight (55 kg) yielded a low body mass index (BMI) of 18 ('underweight').

Initial arterial blood gas analysis showed a PO_2 of 418 mmHg with high-flow oxygen through a non-rebreathing mask, a normal pH of 7.36 with hypocapnia (PCO_2 29 mmHg) and an acidotic metabolic component (base excess −8.1 mmol/L; HCO_3^- 15.8 mmol/L; lactate 2.7 mmol/L). Screening lab results were without major abnormalities.

CT-imaging (without contrast) of the head and thorax showed no abnormalities. With no radiologic signs supporting high altitude cerebral edema, we suspected his neurological deficits were either caused by posthypoxic encephalopathy or by altitude decompression sickness (DCS), the latter of which would be amenable to hyperbaric oxygen therapy (HBOT). HBOT was performed for 5 h, according to US Navy Treatment Table 6, the accepted standard treatment for DCS [3]. Afterwards, full neurological recovery was documented and he was discharged to the regular ward.

About 24 h after admission a new proximal paresis of the left leg was noted. Assuming recurrence of DCS, daily HBOT (at 2.4 atm for 90 min) was scheduled for three days, after which motor function was normal. The patient was discharged from the hospital three days after admission.

3. Discussion

Stowaways travelling in airplanes are a global challenge, likely to be aggravated by political or economic pressure. Survival in airplane wheel wells is exceptional due to the extreme environmental circumstances [2]. The presented patient survived an eight-hour exposure to calculated barometric pressures as low as 190 mmHg, based on the recorded maximum altitude of 34,000 ft. (10.363 m), with a corresponding ambient PO_2 of 40 mmHg.

While outside air temperature at this altitude is approximately −50 °C, we conclude from the initial body temperature of 35.5 °C that temperature within the wheel well was markedly higher. Indeed, the specific Boeing 747-400F wheel well is adjacent to hot air ducts between engines and air conditioning systems. This, together with residual heat from, e.g., the retracted landing gear, hydraulics and electrical equipment reportedly contributes to a survivable environment in wheel wells [4].

Possible contributors to the patient's neurologic presentation were posthypoxic encephalopathy, high altitude cerebral edema [5], and altitude DCS. The loss of consciousness shortly after take-off, as reported by the victim, was probably caused by hypobaric hypoxia. High altitude cerebral edema seemed unlikely since no cerebral edema was found on CT and neurological symptoms persisted at ground level.

The rapid decrease in ambient pressure most likely caused altitude DCS. DCS is initiated by formation of nitrogen gas bubbles during decompression of tissues, saturated with dissolved nitrogen, a phenomenon similar to the formation of gas bubbles after opening a soda bottle [3]. Nitrogen gas bubbles may travel within the circulation, causing vascular obstruction, and trigger coagulant and inflammatory pathways [3]. These secondary effects explain persisting symptoms even after recompression, e.g., return to ground level. HBOT acts both on primary (e.g., shrinking of nitrogen bubbles, enhanced elimination of nitrogen) and secondary aspects (e.g., anti-ischemic) of DCS.

Several risk factors have been suggested to modify the individual risk of DCS, e.g., a larger fraction of adipose tissue increases DCS risk, since the relative low perfusion of adipose tissue prevents the rapid wash-out of tissue nitrogen during decompression and thus supports local bubble formation [6].

According to US Air Force resources [7], the DCS risk for this patient's exposure exceeded 80%. However, this model does not account for a departure at 1800 m above sea level (Nairobi-airport), as well as his physical fitness and low body mass (BMI of 18), all of which likely lowered his DCS risk [6,8].

The course of his neurological symptoms, i.e., absent responses to painful stimuli in both legs and the Babinski sign on initial presentation, the resolution of these symptoms after initial HBOT, as well as the re-occurrence of neurological symptoms after several hours and again their resolution after additional HBOT, are compatible with the diagnosis of altitude DCS.

While hypothermia, given the initial body temperature of 35.5 °C, was not a considerable threat in this particular case, his slight degree of hypothermia may have provided some (neuro)protection against hypoxic damage [8-10].

Aside creation of awareness for this rare patient category, we want to specifically stress the risk of altitude DCS in unpressurized flights. When DCS is suspected, immediate high-flow oxygen therapy should be initiated, followed by HBOT at the earliest opportunity.

Author statement

All authors contributed substantially to pre- or intra-hospital patient treatment and the preparation of the initial (LAS, HJvdW, RPW) or final (MLR, JF, DvE) manuscript.

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Consent

Photo usage with informed, written permission of the patient.

Credit authorship contribution statement

Lothar A. Schwarte: Writing – original draft, Resources, Conceptualization. **Huub J. van der Werf:** Writing – original draft, Resources, Conceptualization. **Milan L. Ridderikhof:** Writing – review & editing, Resources. **Jan Fräβdorf:** Writing – review & editing, Resources. **Daphne van Embden:** Writing – review & editing, Resources. **Robert P. Weenink:** Writing – original draft, Resources, Conceptualization.

Declaration of Competing Interest

None of the authors has any conflict of interest.

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