Tidsskriftet
DEN NORSKE LEGEFORENING

A man in his forties with impaired cognitive and circulatory function after a fall

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Falls can be dangerous, but sometimes whatever caused the fall can be even more dangerous. Here we present the case of a man who was hospitalised after a fall, but for whom identifying the cause and appropriate treatment took some time.

A man in his forties was brought to the trauma unit of a university hospital. According to the information received by the Emergency Medical Communications Centre, he had fallen down a 3–4 m slope, possibly as a result of intoxication. The ambulance team reported both cognitive and circulatory impairment. There was no information to suggest previous illness or a history of substance abuse.

Prior to admission, the man was initially awake and alert. However, his level of consciousness changed rapidly, and he became aggressive and was considered to be psychotic. A police escort was required, and handcuffs had to be used. On the way to the hospital, assistance was requested from the air ambulance doctor, as the patient was showing increasing signs of circulatory failure with tachycardia of up to 120 beats/min and his skin was cold and clammy. He lost consciousness and his respiratory rate slowed to the extent that he required Bag-Valve-Mask ventilation. The final readings in the ambulance showed a systolic blood pressure of 90 mm Hg, pulse of 120 beats/min and SpO_2 of 94 %. His pupils were 4 mm, equal on both sides, and unresponsive to light. The patient arrived at the hospital about one hour after his fall.

The trauma team discussed the available information prior to the patient's arrival at the hospital. Based on the mechanism of injury and the clinical picture, the primary suspicions were haemorrhagic shock and possibly a head injury. The person who had contacted the emergency services had also reported the possibility of intoxication: the patient could have taken cocaine, benzodiazepines and alcohol. This was reported by the air ambulance doctor upon handover at the trauma unit.

On admission, the patient underwent a trauma assessment in line with the Advanced Trauma Life Support (ATLS) principles (1).

When the patient arrived at the trauma unit, his circulation was extremely poor. He was pale and sweaty with cold, clammy skin, and was receiving Bag-Valve-Mask ventilation with an oropharyngeal airway. Pulmonary auscultation revealed faint wheezing sounds bilaterally. The thorax was stable upon palpation. Peripheral oxygen saturation could not be measured. A chest X-ray suggested slight mediastinal widening, but no other visible pathology such as pneumothorax or haemothorax.

Initial cardiac rhythm was sinus tachycardia with a frequency of 140 beats/min, with palpable peripheral pulses. Blood pressure was initially measured at 160/140 mm Hg, but a subsequent measurement showed 75/36 mm Hg. X-rays of the pelvis and long tubular bones provided no evidence of fractures. The man had a large abdominal girth, but a preliminary ultrasound scan (focused assessment with sonography for trauma, FAST) revealed neither free abdominal fluid nor signs of pericardial fluid. The patient was deeply unconscious, with a Glasgow Coma Scale (GCS) score of 4. His pupils were 3 mm in diameter.

Arterial blood gases showed pronounced lactic and respiratory acidosis as well as hyperkalaemia: pH 6.83 (reference range 7.36–7.44), pCO_2 10.0 kPa (4.5–6.1 kPa), pO_2 13.5 kPa (11.0–13.0 kPa), HCO_3 12 mmol/L (22–26 mmol/L), base excess –19.9 mmol/L (0 ± 3.0 mmol/L), Hb 16.0 g/dL (13.4–17 g/dL), sodium 145 mmol/L (137–145 mmol/L), potassium 7.3 mmol/L (3.5–5 mmol/L), lactate 20.0 mmol/L (0.4–1.3 mmol/L), anion gap 30.3 mmol/L, glucose 20.0 mmol/L (4.0–6.0 mmol/L). Tympanic temperature was 37.5 °C.

The patient was clearly critically ill. Airway obstruction and poor spontaneous respiration were initially managed with an oropharyngeal airway and mask ventilation. Clinical findings, vital parameters and arterial blood gases showed marked circulatory failure. The elevated glucose level was interpreted as part of a stress response rather than a sign of ketoacidosis.

The patient's condition was considered to reflect traumatic hypovolaemic shock, with no evidence of pneumothorax or cardiac tamponade. Haemorrhagic shock was considered the most likely explanation, although no clear focus of bleeding could be found.

Transfusion of two units of blood was started in the Emergency Department, but with no improvement in vital parameters.
A chest X-ray showed only a possible mediastinal widening, and the chest was considered a less likely site of bleeding than the abdomen. The pelvis and long tubular bones appeared unscathed. However, the patient was too unstable to undergo trauma CT, and as haemorrhagic shock was suspected, it was decided to transfer him directly to the operating theatre for surgery in accordance with the ATLS principles of trauma management (1).
The patient received ketamine, fentanyl and rocuronium for intubation in the operating theatre. Surgery began 35 minutes after admission. The patient underwent laparotomy, but no bleeding was detected in the abdomen. The surgery took 15 minutes. A bilateral intercostal drain was inserted to aid diagnosis, but revealed no bleeding in the pleural cavities. Supportive care to help stabilise the patient was provided at the same time, including medications and fluids to raise blood pressure, normalise acidosis and lower potassium levels. Fifty-five minutes after admission, a run of ventricular tachycardia occurred along with circulatory collapse requiring advanced cardiopulmonary resuscitation. The acute Extracorporeal Membrane Oxygenation (ECMO) team was summoned to assess the patient's suitability for venoarterial extracorporeal mechanical circulatory support. Return of spontaneous circulation (ROSC) was achieved after approximately 15 minutes, and ECMO was considered not to be indicated.
Echocardiography revealed no sign of cardiac tamponade, and suggested a hyperdynamic circulation without regional cardiac dysfunction. A neurosurgeon considered the clinical picture inconsistent with a traumatic brain injury.
The patient's circulation remained extremely poor, and he was given a high-dose noradrenaline infusion plus bolus doses of adrenaline. He had been medicated for intubation, but not for the maintenance of anaesthesia. Transfusion of blood products yielded little improvement. A total of five units of red cells, four units of plasma and two units of platelet concentrate were administered in the course of the first 90 minutes. The patient's lactate level fell briefly from 20 to 10 mmol/L, but quickly rose again. The respiratory acidosis worsened, with pCO_2(a) rising to 15 kPa despite an increase in minute ventilation from the ventilator.
Additional interventions were discussed while the patient was still in the operating theatre. About 80 minutes after admission, it was noted that the patient's skin had become remarkably hot. His temperature had not been continuously monitored up to this point, but his nasal temperature was now found to have risen to 41.3 °C, from 37.5 °C on admission. External cooling with ice packs was started immediately. A central venous cooling catheter was inserted, and temperature control was achieved after the initiation of internal cooling.

The patient's clinical picture was dominated by circulatory failure. No evidence was found for ongoing bleeding, cardiac tamponade or tension pneumothorax; conditions that should always be suspected after trauma. The patient's body temperature had risen significantly. We realised that his circulatory failure was not the result of bleeding, and that his clinical status was probably due more to drug intoxication than to trauma. Treatment options were discussed in the operating theatre immediately after surgery while advanced cardiovascular life support was being provided. The discussions were interdisciplinary, involving anaesthesiologists and intensive care doctors, surgical gastroenterologists, a neurosurgeon, cardiac surgeons and a cardiologist.

Upon transfer from the operating theatre to the intensive care unit three hours after admission, the patient was deeply comatose without sedation. Oxygenation proved extremely challenging and saturation remained below 90 % despite treatment with 100 % oxygen ($pO_2(a)/FO_2(I)$ ratio approximately 7 kPa). In addition to vasodilation, the patient developed increasing heart failure with low cardiac output as revealed by thermodilution (pulse index continuous cardiac output, PiCCO) and an echocardiogram, and required very high doses of noradrenaline and adrenaline. Levosimendan was also tried but with no improvement. It was difficult to keep the mean arterial pressure (MAP) above 60 mm Hg, and there was no improvement in the lactic acidosis.
Another set of blood samples taken 3.5 hours after admission showed extensive rhabdomyolysis with a myoglobin concentration of 163 000 µg/L (< 70 µg/L) and CK of 21 800 U/L (50–400 U/L). The creatinine concentration was 286 µmol/L (60–105 µmol/L), urea 7.9 mmol/L (3.2–8.1 mmol/L) and potassium 4.4 mmol/L (3.5–5.0 mmol/L). His platelet count was $73 \cdot 10^9$/L ($145–348 \cdot 10^9$/L), INR 1.4 (0.8–1.2), activated partial thromboplastin time (APTT) > 180 s (30–44 s) and fibrinogen < 0.4 g/L (2.0–4.0 g/L), consistent with consumption coagulopathy. Diffuse bleeding began to occur from the surgical wounds and injection sites, as well as from the mouth, nose and respiratory tract, consistent with disseminated intravascular coagulopathy (DIC). Thromboelastography (TEG) was highly abnormal and revealed an almost complete absence of coagulation.
The patient died while receiving intensive care less than seven hours after arrival at the hospital, with clinical and biochemical status consistent with multi-organ failure.

A forensic autopsy was performed. Apart from subcutaneous haematomas and some insignificant skin abrasions, no signs of traumatic injury were found. The heart was clearly enlarged as in dilated cardiomyopathy. The liver was greatly enlarged with cirrhosis and findings consistent with both acute and chronic inflammation. There was no sign of brain damage. Toxicology revealed cocaine and its major metabolite benzoylecgonine as well as amphetamine and mianserin. Fentanyl, ketamine and paracetamol were also detected, all of which had been administered in hospital.

The cause of death was assumed to be drug intoxication. It was unclear whether this was a direct consequence of a high dose of cocaine or whether the patient had developed serotonin syndrome (2, 3).

The patient's general practitioner (GP) was contacted post mortem. There was no history of drug abuse mentioned in the patient's records, nor any use of drugs that alter serotonin metabolism (typical antidepressants). The GP was aware only of a minor prescription of a low-dose benzodiazepine. We therefore assume that the main toxic agent in this case was cocaine.

Discussion

Cocaine poisoning could account for our patient's clinical picture. Acute cocaine poisoning can give rise to a wide range of signs and symptoms, the severity of which varies greatly between individuals and is partly dose-dependent (4–7). Most symptoms involve the cardiovascular system and central nervous system, but almost all organ systems are vulnerable, primarily via haemodynamic effects. Cocaine inhibits the reuptake of monoamine neurotransmitters (dopamine, noradrenaline and serotonin) and has a local anaesthetic action by blocking Na+ channels. Effects on consciousness can range from mild agitation to coma.

Cocaine can affect the cardiovascular system directly by acting on the heart and blood vessels, or indirectly via effects on the central nervous system. Tachycardia, vasoconstriction and hypertension are typically seen, as well as increased myocardial oxygen consumption. Cocaine can give rise to arrhythmia by inhibiting impulse conduction in the myocardium via a local anaesthetic action, and a direct negative inotropic effect on the myocardium may be seen. Most deaths are the result of cardiac arrhythmias, circulatory failure, seizures, cerebral haemorrhage or hyperthermia (7).

Lactic acidosis occurs secondary to tissue hypoxia. Skeletal muscle damage gives rise to rhabdomyolysis and kidney failure, and may result in hyperkalaemia. Disseminated intravascular coagulopathy also occurs.

The treatment of cocaine toxicity is primarily symptomatic and depends on the severity of the symptoms. Cardiovascular symptoms may be reduced by sedation.

Circulatory support may be required, either with vasoactive or inotropic drugs, or with mechanical circulatory support (ECMO) in the most severe cases of circulatory failure. Acidosis, kidney failure and electrolyte disturbances may necessitate renal replacement therapy. Hyperthermia should be treated aggressively, with an emphasis on external cooling.

Serotonin syndrome, also known as serotonin toxicity, results from serotonergic overstimulation of the central nervous system (8, 9). The syndrome most often arises when two or more substances with serotonergic activity are used in combination, but can also be seen with use of a single substance. The diagnosis is clinical and can reflect varying severities of autonomic, neuromuscular and cognitive symptoms. Multiple organ dysfunction such as coma, circulatory failure, disseminated intravascular coagulopathy, hyperthermia and rhabdomyolysis can occur and the condition can be fatal. Both mianserin and fentanyl have serotonergic activity and can trigger and exacerbate this condition.

Our patient's death was probably secondary to drug intoxication. It was known at an early stage that the patient had been using drugs prior to his fall, and in retrospect it is clear that this should have been given greater emphasis. We placed too much weight on the trauma history. We assumed that the drug use was responsible for his loss of consciousness. The air ambulance doctor did state on handover that he considered drug intoxication to be important for the patient's condition, but the dramatic circulatory collapse led us to assume that there must be 'something more'.

In the absence of clinical findings or test results pointing to any particular haemorrhagic focus, we should perhaps have questioned sooner whether trauma could in fact explain the patient's condition. It is possible that considering differential diagnoses related to intoxication or other possible causes might have led to an earlier focus on organ support therapy in intensive care, as opposed to surgery. A CT scan could also potentially have been performed rather than proceeding directly to laparotomy. CT scanning of unstable patients is, of course, risky and is contrary to the ATLS principles for the initial assessment of trauma patients. In our case, however, a scan might have resulted in an earlier shift in focus away from trauma management towards the treatment of a drug poisoning, thereby avoiding an unnecessary laparotomy that did not benefit the patient. We found it difficult to escape the mindset that trauma was the root cause of the patient's condition, and therefore continued to search for injuries that could explain his dramatic deterioration.

Although hypovolaemia is the most common cause of circulatory failure in trauma patients, it is important to always keep in mind that there may be underlying factors that explain why the patient sustained the trauma in the first place. These factors can themselves be life-threatening, and may require a completely different approach to that of surgical trauma management.

EPILOGUE

Many patients are assigned to a predefined treatment pathway on the basis of their symptoms or reason for admission, such as individuals with suspected stroke, myocardial infarction or trauma. For most patients, this means that they receive the correct treatment without loss of time. However, our recent experience shows that this approach can lead to a narrow focus on a particular treatment algorithm when the patient is critically ill and decisions must be made quickly. This case report illustrates why other causes should also be considered when the presumed cause appears inconsistent with the clinical picture, or when there is more than one possible explanation.

We believe that our story will be relevant to others who encounter patients early in the patient pathway. We offer a reminder that drug intoxication can also give rise to dramatic symptoms and clinical findings for which an interdisciplinary approach is required. We have used this case as an example during internal discussions and teaching to help foster greater focus on differential diagnosis when receiving trauma patients and others who are critically ill.

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Published: 1 February 2021. Tidsskr Nor Legeforen. DOI: 10.4045/tidsskr.20.0546
Received 22.6.2020, first revision submitted 6.10.2020, accepted 20.11.2020.