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Absence of Adverse Neurological Outcomes in a Non-Neurologically Injured Polytrauma Patient Despite Extreme and Prolonged Treatment-Resistant Hypotension: A Case Report

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Temporary hypotension after severe trauma might help achieve hemostasis and increase the chances of survival. However, excessive hypotension can lead to adverse neurological sequelae or be fatal. The relationship between the degree of hypotension and neurological prognosis after trauma is not fully understood. Our report describes a patient with severe trauma who survived with a favorable neurological outcome despite extreme and prolonged treatment-resistant hypotension. (A&A Practice. 2019;13:358–61.)

GLOSSARY

BP = blood pressure; **FFP** = fresh frozen plasma; **MAP** = mean arterial pressure; **pRBCs** = packed red blood cells.

Temporary hypotension after trauma can potentially decrease bleeding and help in achieving hemostasis. For instance, hypotensive resuscitation, which is an established therapeutic method before definitive hemostasis in patients with trauma, might increase survival rates, decrease bleeding, and help achieve hemostasis.[1–5] However, the lowest tolerable degree of hypotension, the maximal allowable duration of such hypotension for survival, and the appropriate blood pressure (BP) for preservation of central neurological function are unclear. A few reports have described neurological function after resuscitation from severe trauma.

We describe a patient with severe trauma who survived and achieved a favorable neurological prognosis despite extreme and prolonged treatment-resistant hypotension.

Written consent was obtained from the patient for publishing this case report.

CASE DESCRIPTION

A 25-year-old man (body mass index, 20 kg/m^2) with no previous medical illness was involved in a motorcycle accident, in which his motorcycle, traveling at 60 km/h, crashed into a sedan car traveling at 100 km/h. At the time of the accident, he was wearing a helmet but no protective spinal gear. Before his hospitalization, he was diagnosed with hemorrhagic shock without any neurological symptoms by the emergency response team. On admission, his vital signs were as follows: BP 80/60 mm Hg; pulse rate 120 beats/min; oxygen saturation 99% while breathing 100% oxygen at a flow rate of 10 L/min with a nonrebreather mask, and respiratory rate 30 breaths/min. His cold and wet hands, prolonged capillary refill time >5 seconds, and serum lactate level of 2.3 mmol/L indicated a severe shock state. His Glasgow Coma Scale score was 15 points, and he was neurologically normal. Computed tomography images indicated a ruptured spleen, deep and complex damage to the right kidney, damage to the right adrenal gland and liver, a very small subarachnoid hemorrhage, right pneumothorax, pulmonary contusion, unstable fracture of the pelvis, fractured second cervical and first thoracic vertebrae, multiple rib fractures, and complex fractures of both lower limbs. We immediately inserted large bore peripheral and central venous lines and a resuscitative endovascular balloon occlusion catheter of the aorta via the right femoral artery. Four units of packed red blood cells (pRBCs) and fresh frozen plasma (FFP) were transfused, and the balloon of the occlusion catheter was correctly positioned slightly above the level of the celiac artery to prevent bleeding from the ruptured spleen. Thereafter, his BP stabilized and he was given an additional 6 units of pRBC and FFP before the first surgery. He underwent splenectomy, right nephrectomy, right adrenalectomy, and external fixation of the pelvis and left lower limb. The first operation was performed 133 minutes after his hospital arrival. He was conscious and had no evidence of neurological symptoms before the first operation. The balloon of the aortic occlusion catheter was deflated immediately after splenectomy (occlusion time, 144 minutes). During these procedures, his mean arterial

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The Ethics Committee at Kitami Red Cross Hospital approved this study.

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pressure (MAP) remained below 60 mm Hg because of uncontrollable hemorrhage.

Despite 3 surgeries within the first 3 days after his accident, we were unable to achieve hemostasis because of extensive retroperitoneal crush injuries. Administration of vasopressors could not increase his BP, and his MAP remained at 40–45 mm Hg. During this period, his vital signs and laboratory data were as follows: MAP 40–45 mm Hg; pulse rate 140–150 beats/min; urine output 0 mL/h; hemoglobin 6.6–9.0 g/dL; platelet count 31–68 × 10^9/L; serum lactate level 6.0–8.3 mmol/L; and serum fibrinogen 167–281 mg/dL.

After 28 hours of extreme hypotension, his MAP increased and the amount of blood from the drainage tubes decreased. Finally, we confirmed hemostasis during the fourth operation. After the fourth operation, platelet count and serum fibrinogen were 93 × 10^9/L and 242 mg/dL, respectively. His pupillary response was maintained during hypotension, but his Glasgow Coma Scale score was 3 points (eye 1, verbal not testable, motor 1) without any sedation. The total amount of blood and blood products transfused over 4 days included 130 units of pRBC, 130 units of FFP, 110 units of platelet concentrates, and 8 L of irradiated fresh whole blood. Transfusion of each component was performed with frequent evaluations of hemoglobin, platelet count, and serum fibrinogen levels. The Figure shows the course of the patient's MAP, hemoglobin, platelet count, and serum fibrinogen over the first 4 days of hospitalization. The Table shows the patient's clinical course after admission.

Subsequently, he regained consciousness, function of all major organs, and motor function except for his right lower limb. Magnetic resonance imaging showed a surprisingly intact brain and spinal cord. However, he had no motor nerve conduction of his right lower limb and could not sense or move the limb, which was deemed to be because of irreversible ischemic peripheral nerve injury secondary to prolonged resuscitative endovascular balloon occlusion of the aorta on the first day (Figure), rather than the lower extremity and pelvic fractures.

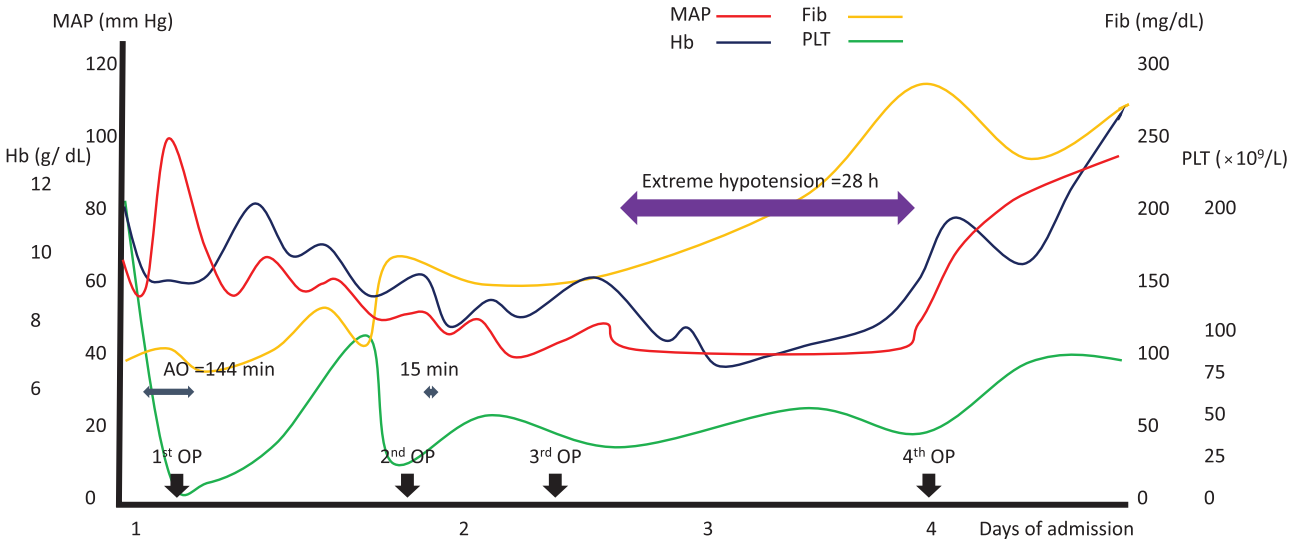

Figure. The patient's clinical course in the hospital. AO indicates aortic occlusion; Fib, serum fibrinogen; Hb, hemoglobin; MAP, mean arterial pressure; OP, operation; PLT, platelet count.

Table. Surgical Procedures, Neurological Findings, and Clinical Course After Admission

Day After Admission	Surgical Procedures	Neurological Findings	Development
1	Hemostasis, splenectomy, right nephrectomy, right adrenalectomy, external skeletal fixation of pelvis, and left lower limb	All neurological findings clear on admission	REBOA inserted
	Hemostasis, decompression	…	ACS
2	Hemostasis, decompression	…	ACS
4	Hemostasis	…	Complete hemostasis REBOA removed
7	Abdominal closure	…	HD started for AKI
8	…	Awakening Right arm movement	…
9	Complex fixation of lower limbs	…	…
12	Pelvic fixation	…	…
13	…	…	Extubation
14	…	Left arm movement	…
22	…	Left leg movement	…
60	…	…	HD withdrawn
70	…	…	Discharged from ICU

Abbreviations: ACS, abdominal compartment syndrome; AKI, acute kidney injury; HD, hemodialysis; ICU, intensive care unit; REBOA, resuscitative endovascular balloon occlusion of the aorta.

DISCUSSION

Our patient completely recovered consciousness and motor function, except of his right lower extremity, despite extreme and prolonged treatment-resistant hypotension. His injury severity score was 75, and his Predicted Risk of Mortality was 76.9%.

The minimum BP level required for survival after trauma is unclear, although research on hypotensive resuscitation might help our understanding. The importance of hypotensive resuscitation for trauma has been previously described, although its efficacy is unclear.[1–5] One randomized controlled trial found that early mortality rates after blunt or penetrating trauma were lower for patients with a MAP of 50 mm Hg as compared to those with a MAP of 65 mm Hg.[5] A previous animal study showed that permissive hypotension for >90 minutes was associated with worse survival rates and organ function.[6] However, no information exists on extreme and prolonged hypotension as in this case. Although our patient with blunt trauma had a far lower MAP and a longer duration of hypotension than patients in published studies, he survived.

The association between hypotension after trauma and neurological prognosis is not clearly known. In this case, neurological findings and magnetic resonance imaging showed that our patient had an intact brain and spinal cord. Guidelines of the Brain Trauma Foundation indicate that the systolic BP of patients 15–49 years old with traumatic brain injury should be controlled at or above 110 mm Hg, to decrease mortality and improve neurological outcomes.[7] One report recommended maintenance of MAP between 85 and 90 mm Hg for the first 7 days after acute spinal cord injury.[8] A recent review showed that there is no ideal vasopressor for neuroprotection.[9] Our patient did not have any neurological symptoms suggestive of central nervous system injury on admission, and we could not maintain his BP at the level recommended in previous reports with commonly used vasopressors. However, his central nervous system remained well preserved. This suggests the possibility that trauma patients with no central nervous system injury can achieve favorable neurological outcomes despite maintenance of MAP as low as 40–45 mm Hg.

Apart from BP regulation, there are other strategies to protect the brain and spinal cord.[10,11] However, we did not apply any special protective strategies for the brain and spinal cord in this patient because computed tomography images of the brain and spinal cord on admission revealed no abnormalities. Reportedly, resuscitative endovascular balloon occlusion of the aorta can induce remote ischemic preconditioning by causing transient ischemia of the ipsilateral lower limb.[12] Remote ischemic preconditioning by balloon occlusion of the aorta might have contributed to protecting our patient's central nervous system.[13,14]

A limitation of this report is that we cannot conclude that this degree of extreme and prolonged hypotension is safe and effective in preserving central neurological function in all patients with trauma because our patient was young and healthy and did not have severe traumatic brain or spinal cord injury. Hypotensive resuscitation strategies are contraindicated in patients with brain or spinal injuries because maintaining adequate perfusion pressure is crucial to ensuring tissue oxygenation of the injured central nervous system.[1–5] Patients with central nervous system injury and older patients were excluded in previous articles about hypotensive resuscitation.[1,5]

CONCLUSIONS

Our patient with severe trauma survived and achieved good central neurological outcomes despite extreme and prolonged treatment-resistant hypotension, to a level that was lower and for a longer duration than that previously reported as being permissible. Future reports elucidating details about the hypotension might help clarify the mechanisms affecting neurological outcomes in trauma patients.

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DISCLOSURES

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