

From fatal to functional: a trauma case with extreme acidosis and full recovery

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DOI: XXX

Background: Arterial pH below 6.8 has traditionally been regarded as incompatible with life because of profound cellular dysfunction and high mortality. When accompanied by severe hyperkalemia and critical anemia following traumatic hemorrhagic shock, survival is exceedingly rare. This case highlights successful reversal of extreme physiological derangement through rapid, protocol-driven, multidisciplinary management.

Methods: A multidisciplinary trauma, surgical, and critical care team managed the patient using institutional massive transfusion and resuscitation protocols. Serial clinical, hemodynamic, and metabolic assessments guided fluid resuscitation, blood component therapy, correction of acidosis and hyperkalemia, vasopressor support, and definitive surgical hemorrhage control.

Case Presentation: A 42-year-old previously healthy male presented 15 minutes after a high-speed motor vehicle collision with a Glasgow Coma Scale score of 3, a pulse rate of 148 beats/minute, unrecordable blood pressure, a respiratory rate of 32/minute, oxygen saturation of 88% on high-flow oxygen, and delayed capillary refill. Focused Assessment with Sonography for Trauma demonstrated intra-abdominal free fluid. Arterial blood gas revealed pH 6.75, bicarbonate 14.1 mmol/l, lactate 7.2 mmol/l, potassium 7.1 mmol/l, and hemoglobin 4.8 g/dl. Electrocardiography showed peaked T waves and widened QRS complexes. Massive transfusion, correction of hyperkalemia and acidosis, vasopressor support, and emergency laparotomy were initiated promptly.

Results: Despite minimal early improvement in arterial pH, exploratory laparotomy evacuated approximately 3 L of hemoperitoneum resulting from a grade IV liver laceration and mesenteric tears, followed by damage-control packing. Following hemorrhage control and continued resuscitation, blood pressure improved to 100/60 mmHg, metabolic acidosis and hyperkalemia resolved, electrocardiographic abnormalities normalized, and hemoglobin increased to 10.2 g/dl. The patient was extubated on day 4, discharged on day 14, and demonstrated complete neurological and hepatic recovery at the 6-week follow-up.

Conclusion: Extreme acidemia (pH 6.75) should not be considered futile in trauma resuscitation. Early recognition, aggressive massive transfusion, targeted correction of metabolic abnormalities, and prompt surgical hemorrhage control can achieve complete recovery even in patients with physiological parameters traditionally considered incompatible with survival.

Keywords: Severe acidosis, trauma, resuscitation, hemorrhagic shock.

Acknowledgments

The authors thank the nursing staff and allied healthcare professionals of the Emergency Medicine at Max Super Speciality Hospital, New Delhi, for their contribution to patient care.

Funding

No external funding was received.