Archives of Anesthesiology and Critical Care (In Press); x(x): xx-xx.

Available online at http://aacc.tums.ac.ir

Case Report

Soldier Suicide with a Firearm: A Case Report with an Approach to Hemorrhagic Shock Control in the Emergency Department

Hojat Rastegari Najafabadi[1], Aliakbar Keykha[2,3]*

[1]Department of Emergency Medicine, Khatami Al-Anbia Hospital, Zahedan University of Medical Sciences, Zahedan, Iran.

[2]Community Nursing Research Center, Zahedan University of Medical Sciences, Zahedan, Iran.

[3]Department of Medical-Surgical Nursing, School of Nursing and Midwifery, Zahedan University of Medical Sciences, Zahedan, Iran.

ARTICLE INFO

Article history:
Received 16 October 2025
Revised 06 November 2025
Accepted 20 November 2025

Keywords:
Suicide;
Firearm;
Hemorrhagic shock;
Case report

ABSTRACT

Military personnel face a heightened risk of suicide due to firearm accessibility. Bullet impacts can cause significant tissue damage and bleeding, which varies depending on the affected area. This report details a case of a 20-year-old Iranian Baluch soldier who was admitted to the emergency room with hemorrhagic shock after suicide with a firearm. The bullet damaged his mandible, tongue, and maxilla, exited from his forehead, and damaged his nose. In hemorrhagic shock, prioritize blood products over isotonic fluids; consider norepinephrine if hemodynamics remain unstable.

Introduction

Suicide in soldiers is a complex issue influenced by factors such as pre-existing mental illness, a history of self-harm or suicide attempts, and family problems. Additionally, psychological conflicts with colleagues, separation from family, and access to firearms significantly contribute to suicide risk within the military environment [1-2]. In Iran, the compulsory military service for all males complicates this issue, as even highly educated individuals must serve for a minimal salary. Inappropriate and disrespectful behavior by commanders and officers, heavy and unprofessional workload, and waste of time have been the main reasons for soldiers' dissatisfaction with compulsory military service. These conditions, when combined with low levels of education, mental illness in the soldier or family, a history of suicide in the soldier or family, service in frontier zones, and a history of drug abuse, increase the risk of suicide by firearm [3].

Case Report

A 20-year-old soldier was transported by EMS to Khatam Al-Anbia Hospital in Zahedan (Sistan and Baluchestan Province, Iran) at 4:15 a.m. for a suicide attempt with a firearm.

Military officers reported the patient shot himself while on guard duty at an inner-city checkpoint. Clinical examination revealed a submental bullet entry and forehead exit wound, with extensive damage to the oral cavity, tongue, maxilla, and nose. Severe hemorrhage was present at the bullet entry and exit sites, as well as from the mouth and nose. The patient's vital signs and consciousness were as follows:

GCS 7, BP 70/40 mmHg, HR 137 bpm, RR 34, T 37°C, SpO_2 70%.

The authors declare no conflicts of interest.
*Corresponding author.
E-mail address: aliakbar.keykha@gmail.com
DOI:

Copyright © 2026 Tehran University of Medical Sciences. Published by Tehran University of Medical Sciences.
This work is licensed under a Creative Commons Attribution-NonCommercial 4.0 International license (https://creativecommons.org/licenses/by-nc/4.0/). Noncommercial uses of the work are permitted, provided the original work is properly cited.

The patient was promptly intubated for airway protection and mechanically ventilated with AC/VC+ (PRCV), TV = 450 mL, RR = 14, Ti = 1.2, PEEP = 5, and FiO_2 = 100-40%. To control bleeding, the patient's mouth and nose were packed with adrenaline-soaked gauze. A size 14 venous line was inserted in the upper limb, and a Shaldon catheter was inserted into the subclavian vein. Blood was drawn for testing, and fluid therapy was initiated with 2 liters of normal saline. Emergency ICU, ENT, and maxillofacial surgery consultations were performed, and bleeding was controlled as much as possible by suturing the patient's wounds. (Figure 1) shows the patient's condition after initial bleeding control.

The patient received emergency transfusions of 2 units of PRBCs, 4 units of FFP, and 4 units of platelets. The patient's blood pressure remained low at 80/50 mmHg. We started administering another liter of normal saline; however, bleeding recurred from the facial injuries. We reduced the serum infusion rate and initiated norepinephrine infusion at 5-20 μg/min. Hemodynamic stability was achieved by titrating the norepinephrine infusion rate. Following stabilization, a CT scan of the patient's chest, neck, maxillofacial area, and brain was performed to assess bullet-related injuries. Maxillofacial CT revealed multiple fractures of the mandible, zygomatic, nasal, orbital, and frontal bones, accompanied by severe soft tissue damage and hematoma. Neck and chest CTs were normal. The brain CT scan showed no brain parenchyma damage, as the bullet's trajectory resulted only in facial bone fractures and soft tissue damage (Figure 2). Facial bone fractures were more clearly visible in the CT reconstruction (Figure 3).

Figure 1- The patient's image reveals severe facial injuries with significant bleeding, which was controlled with packing.

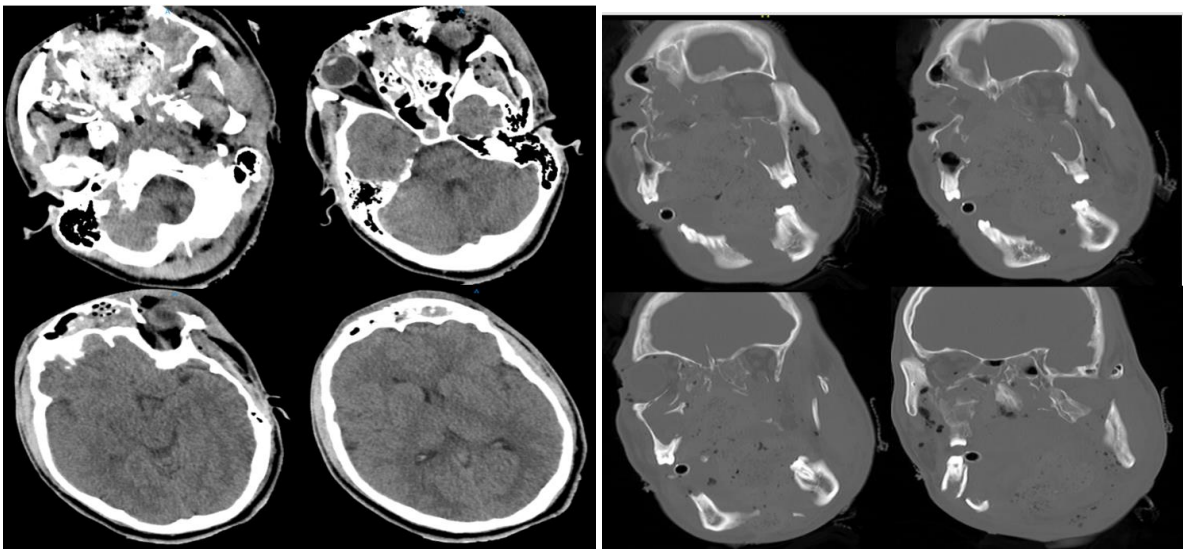

Figure 2- The brain and maxillofacial CT scan showed that the brain parenchyma is normal. Bilateral bone fractures of the frontal and maxillary sinuses with displacement into the sinuses and hematomas are present. Also, the nasal septum and bone, hard palate, pterygoid, maxilla, alveolar process, mandible, and palatine bone had suffered a comminuted fracture. Soft tissue swelling with hematoma and intratissue emphysema is evident.

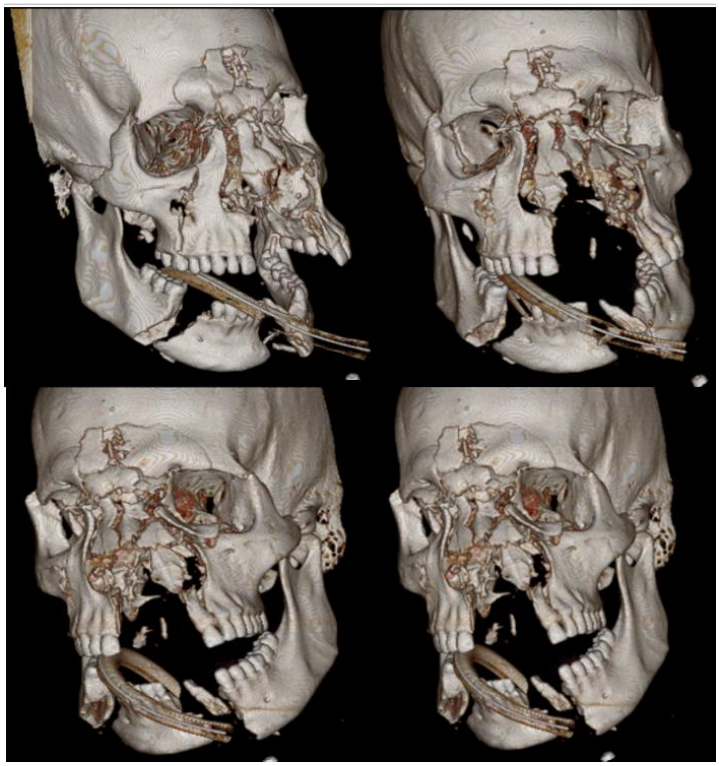

Figure 3- Facial CT revealed bilateral comminuted bone fractures of the mandible, maxilla, nasal, and orbits.

The patient's initial laboratory findings were as follows:

WBC= 17.5×10^3/µl, RBC= 4.4 mil/µl, HB= 13.4 g/dL, HCT= 42.2%, PLT= 255×1000, PT= 12/sec, PTT= 30/sec, INR= 1, BS= 205 mg/dl, BUN= 17 mg/dl, CR= 1.2 mg/dl, K= 4.7 meq/l, Na= 141 meq/l, ABG (PH= 7.18, $PaCO_2$= 47.3, HCO_3= 17.1, BE= -10.7, PaO_2= 48.8, O_2Sat= 78.7)

Following serum and blood transfusions, a CBC revealed significantly decreased hemoglobin and hematocrit levels compared to initial values.

WBC= 14.6×10^3/µl, RBC= 3.2 mil/µl, HB= 8.7 g/dL, HCT= 30.2 %

After transfusion of 2 units of PRBCs, the patient was transferred to the intensive care unit with the following medication orders.

Ceftriaxone 2 g BD and vancomycin 1 g BD were initiated to target sinus organisms and prevent meningitis. Also, to reduce the risk of seizures, increased intracranial pressure, and rebleeding, the patient received the following treatments: 20% mannitol (300 cc stat, then 100 cc TDS), phenytoin (750 mg stat, then 125 mg TDS), and tranexamic acid (1 g stat, then 1 g over 8 hours). We performed an early tracheostomy three days after ICU admission due to facial injuries and anticipated need for long-term airway management.

Two days after the tracheostomy, the patient underwent jaw reconstruction surgery by an oral and maxillofacial surgeon (Figure 4).

Figure 4- A, B and C: Before surgery, D: After surgery

An orogastric tube was placed during jaw surgery to facilitate early feeding. A postoperative CT scan showed well-fixed fractures of the maxilla and mandible (Figure 5).

Two weeks post-surgery, the patient regained consciousness and was removed from the ventilator. However, the tracheostomy could not be removed due to oral injuries.

The patient reported transient blurred vision in the right eye after extubation, which was resolved after two days. The ophthalmologist discharged the patient and scheduled a follow-up examination at the ophthalmology hospital.

The ENT surgeon postponed nasal bone reconstruction until the patient had fully recovered after discharge from the hospital. Due to mouth ulcers, pain, and poor swallowing, a gastroenterologist placed a percutaneous gastrostomy. After four days of family education on nutrition and patient care, the patient was discharged.

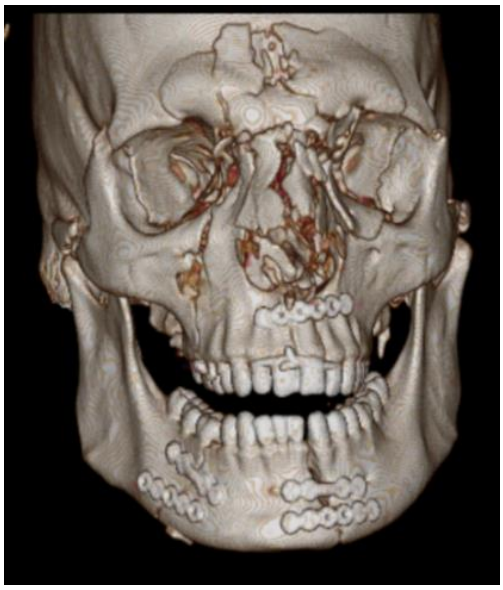

Figure 5- A postoperative CT showed proper fixation of the fractured maxilla and mandible.

Discussion

This patient was in hemorrhagic shock upon admission due to blood loss from a gunshot wound. Despite administration of isotonic saline, PRBCs, FFP, and platelet transfusions, the patient's hemodynamic status remained unstable. Continued saline infusion caused rebleeding from the gunshot wound. Norepinephrine infusion stabilized the patient's hemodynamics.

Hemorrhagic shock, a subtype of hypovolemic shock, occurs when critical blood loss overwhelms the body's compensatory mechanisms, leading to impaired tissue oxygenation and anaerobic metabolism. Continued tissue ischemia and toxic substance production lead to cell death [4]. Hemorrhagic shock, a common and frequently fatal consequence of traumatic injuries, often leads to poor outcomes even with optimal treatment [5]. The 'lethal triad of trauma' (hypoperfusion, acidosis, and coagulopathy) plays a crucial role in the pathophysiology of hemorrhagic shock [6-7]. Blood loss decreases intravascular volume, hindering oxygen delivery and causing cells to produce lactate via anaerobic metabolism. Initial compensation involves increased heart rate and vasoconstriction to maintain blood pressure, but systolic pressure eventually drops with declining cardiac output.

Blood shunting to vital organs exacerbates acidosis in peripheral tissues. Ultimately, uncorrected acidosis and hypoxemia result in loss of vasoconstriction, hemodynamic collapse, and death [8]. Preventing further blood loss is the first step in controlling hemorrhagic shock and the progression of acidosis. Therefore, hemorrhaging should be controlled by applying pressure dressings, applying tourniquets to the extremities, using hemostatic agents, or using surgical techniques [9-10].

Trauma-induced coagulopathy, characterized by coagulation factor loss, hemodilution, acidosis, and hypothermia, significantly contributes to mortality [11]. This acute coagulopathy can begin before resuscitation, with acidosis and hypothermia further impairing coagulation and exacerbating the condition [6,12]. Given the pathophysiology of hemorrhagic shock, management should not rely solely on fluid infusion and blood transfusion. Therefore, damage control resuscitation should be considered. In hemorrhagic shock without head trauma, target a systolic blood pressure of 90 mmHg to balance the risks of rebleeding and inadequate tissue perfusion [7].

Preventing trauma-induced coagulopathy requires maintaining patient warmth or actively rewarming, limiting isotonic fluids (these fluids, colder than body temperature, worsen trauma-induced hypothermia), and early blood product transfusion [9].

Normal saline and lactated Ringer's are the most common crystalloid resuscitation fluids. The chloride content of normal saline is significantly higher than that of blood, and by altering the strong ion difference of the blood, it causes non-anion gap hyperchloremic metabolic acidosis, which intensifies the patient's underlying metabolic acidosis caused by trauma. Conversely, Ringer lactate may induce metabolic alkalosis as lactate metabolizes into bicarbonate.

Crystalloid fluids are inefficient at expanding intravascular volume, with only 25-30% remaining in the vasculature; the majority shifts into interstitial and intracellular spaces. Overinfusion can also cause hemodilution and impair tissue perfusion [13]. Patients receiving over 2 liters of isotonic fluid have a greater than 40% increased risk of coagulopathy and hemodilution [9,14].

Compared to crystalloid fluids, administering blood products in ratios of 1:1:1 or 1:1:2 (plasma: platelets: packed RBCs) can improve hemodynamic stability and reduce metabolic acidosis and mortality [6-7]. Colloids,

particularly hydroxyethyl starch, may impair renal function and coagulation. Albumin use remains controversial, with no proven benefit and higher mortality in traumatic brain injury [6].

Also, vasoconstrictive drugs effectively improve hemodynamics and maintain tissue perfusion when intravenous fluid intake is restricted. Norepinephrine (NE), the preferred vasopressor for septic and hemorrhagic shock, is a sympathomimetic agent that causes vasoconstriction via α-adrenergic stimulation. NE mobilizes venous blood via arterial and venous constriction, while β_2 ‑ receptor activation reduces venous resistance and improves venous return [8,15].

Tranexamic acid (1 g loading dose over 10 min, followed by 1 g infusion over 8 hr) in hemorrhagic shock reduces mortality without increasing thromboembolic events. Therefore, tranexamic acid should be a standard component of traumatic hemorrhagic shock management [8].

Conclusion

For the treatment of hemorrhagic shock, immediately control bleeding. Limit isotonic fluid resuscitation to 2-3 liters due to poor volume expansion and risk of worsening metabolic acidosis. Administer blood products in a 1:1:1 or 1:1:2 ratio of FFP, platelets, and PRBCs to treat the shock and prevent acidosis. If instability continues, cautiously use norepinephrine to increase blood pressure, avoiding excessive systolic pressure to minimize rebleeding risk. Consider tranexamic acid as a standard adjunct to reduce bleeding.

Abbreviations

PRBCs= Packed Red Blood Cells, FFP= Fresh Frozen Plasma, CBC= Complete Blood Count, BD= Twice in a day, TDS= 3 times in a day, PEEP= Positive End-expiratory Pressure, ICU= Intensive Care Unit, AC/VC+ (PRCV)= Assist Control/ Volume Control+ (Pressure Regulated Volume Control), RR= Respiratory Rate, TV= Tidal Volume, Ti= Time Inspiration

Acknowledgment

The researchers thank the otolaryngology, maxillofacial surgery, ophthalmology, radiology, and intensive care teams for their invaluable clinical support. They also acknowledge the Vice President for Research and Technology, the research experts, and the ethics committee of Zahedan University of Medical Sciences for their contributions to ethical approval and oversight.

Authors' contributions

AK: Conceptualization, Patient Care, Data Collection, writing – Original Draft, Writing – Review & Editing, Final Approval.

HRN: Patient Follow‑up, Data Collection, Writing – Review & Editing.

Availability of data and materials

All patient data, including medical records, radiographs, and computed tomography scans, are available from the corresponding author upon reasonable request.

Ethics approval and consent to participate

The study protocol was approved by the ethics committee of Zahedan University of Medical Sciences (ethical code: IR.ZAUMS.REC.1404.271).

Consent for publication

Informed written consent was obtained from the patient authorizing publication of this case report and related images, and the consent document may be reviewed by the journal's Editor‑in‑Chief upon request.

References

[1] Nouri R, Fathi-Ashtiani A, Salimi SH, Soltani Nejad A. Effective factors of suicide in soldiers of a military force. J Mil Med. 2012; 14(2):99-103.
[2] Hoyt T, Duffy V. Implementing firearms restriction for preventing US Army suicide. Mil Psychol. 2015; 27(6):384-90.
[3] Anisi J, Majdian M, Mirzamani SM. The factors associated with suicide ideation in Iranian soldiers. Iran J Psychiatry. 2010; 5(3):97-101.
[4] Cannon JW. Hemorrhagic Shock. N Engl J Med. 2018; 378(4):370-9.
[5] Eastridge BJ, Holcomb JB, Shackelford S. Outcomes of traumatic hemorrhagic shock and the epidemiology of preventable death from injury. Transfusion. 2019; 59 Suppl 2:1423-8.
[6] Markl-Le Levé A, Haller I, Bachler M. Fluid management in hemorrhagic shock. Curr Opin Anaesthesiol. 2025; 38(3):316-22.
[7] Vishwanathan K, Chhajwani S, Gupta A, Vaishya R. Evaluation and management of haemorrhagic shock in polytrauma: Clinical practice guidelines. J Clin Orthop Trauma. 2020; 13:106-15.
[8] Bouglé A, Harrois A, Duranteau J. Resuscitative strategies in traumatic hemorrhagic shock. Ann Intensive Care. 2013; 3(1):1.
[9] Jacob M, Kumar P. The challenge in management of

hemorrhagic shock in trauma. Med J Armed Forces India. 2014; 70(2):163-9.
[10] David JS, Spann C, Marcotte G, Reynaud B, Fontaine O, Lefèvre M, et al. Haemorrhagic shock, therapeutic management. Ann Fr Anesth Reanim. 2013; 32(7-8):497-503.
[11] Petros S. Trauma-Induced Coagulopathy. Hamostaseologie. 2019; 39(1):20-7.
[12] Moore EE, Moore HB, Kornblith LZ, Neal MD, Hoffman M, Mutch NJ, et al. Trauma-induced coagulopathy. Nat Rev Dis Primers. 2022; 8(1):25.
[13] Rudloff E, Hopper K. Crystalloid and Colloid Compositions and Their Impact. Front Vet Sci. 2022; 9:878702.
[14] Gillissen A, van den Akker T, Caram-Deelder C, Henriquez DDCA, Bloemenkamp KWM, van Roosmalen JJM, et al. Association between fluid management and dilutional coagulopathy in severe postpartum haemorrhage: a nationwide retrospective cohort study. BMC Pregnancy Childbirth. 2018; 18(1):398.
[15] Fage N, Asfar P, Radermacher P, Demiselle J. Norepinephrine and Vasopressin in Hemorrhagic Shock: A Focus on Renal Hemodynamics. Int J Mol Sci. 2023; 24(4):4103.