Successful Surgical Treatment of Severe Perforating Diametric Craniocerebral Gunshot Wound Sustained during Combat: A Case Report

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ABSTRACT Many researchers classify perforating diametric craniocerebral gunshot wounds as fatal because mortality exceeds 96% and the majority of patients with such injuries die before hospitalization. A 23-year-old Ukrainian male soldier was admitted to a regional hospital with a severe perforating craniocerebral wound in a comatose state (Glasgow Coma Scale score, 5). Following brain helical computed tomography, the patient underwent primary treatment of the cerebral wound with primary duraplasty and inflow/outflow drainage. After 18 days of treatment in the intensive care unit, he was transferred to a military hospital for further rehabilitation. This report details our unusual case of successful treatment of a perforating diametric craniocerebral gunshot wound.

INTRODUCTION

Gunshot wounds with injury-causing projectile penetration through the dura mater into the brain parenchyma are classified into two groups: perforating wounds and penetrating wounds.[1,2] Perforating and penetrating wounds are gunshot injuries of the skull and dura mater with and without the presence of exit wounds, respectively. Kinetic/wounding energy in such wounds is represented as $E = 1/2\ m\ (Vi2 - Vr2)$, where m is projectile mass, Vi is impact velocity, and Vr is residual velocity.[3,4] Many researchers classify these types of injury as fatal because mortality exceeds 96% and the majority of patients with such injuries die before hospitalization.[5] Here we present an unusual case of successful treatment of a diametric craniocerebral gunshot wound sustained during combat.

CASE

A 23-year-old male soldier was transported to a mobile military hospital with a gunshot wound of the head inflicted by a sniper shot during local armed conflict in eastern Ukraine. The patient's condition on admission was extremely severe, with a Glasgow Coma Scale (GCS) score (based on three responses: eye, voice, and motor) of 3–15. In our case, GCS score was 5 (eye response 1, voice response 1, motor response 3).[6,7]

The patient presented with pale skin, occasional respiratory movements, and involuntary urination. His arterial pressure was 165/80 mmHg and heart rate was 87 bpm. Local findings were as follows: an inlet up to 0.5 cm in diameter in the right temporal region with no signs of active bleeding (Fig. 1A); soft tissues bulging out in the left parietal region (up to 8 cm in diameter) with a 2 × 2.5 cm outlet in the middle of the bulge from which brain detritus was released (Fig. 1B); and bleeding from the left external auditory canal. In an antishock ward, the patient was intubated and fitted with a urinary catheter and a catheter into the right subclavian vein. Arterial oxygen saturation (SaO2) was 97%–99%. The patient was administered the following drugs: 2 g IV ceftriaxone (a third generation cephalosporin), 1 ml (20 units) IM tetanus toxoid, 200 ml IV 15% mannitol, and 5 ml (250 mg) IV hemotran (tranexamic acid). An infusion therapy with colloidal and crystalloid solutions was initiated. The gunshot wounds were bandaged, and when the patient's vital functions stabilized, he was evacuated by air to a regional multidisciplinary clinical hospital. The time from injury to admission to the regional hospital was 3 h 40 min.

On admission to the regional hospital, the patient underwent brain helical computed tomography (HCT) using an Optima CT660 helical computed tomography system (GE Healthcare, USA) using bone and brain modes for data analysis; data from HCT was used to construct a three-dimensional skull model. The field-of-view included all sections of the patient's head and upper cervical vertebrae (C1–C3).

Severe perforating diametric gunshot wounds were diagnosed, with an inlet in the right temporal region (Figs 2A and 3A) and outlet in the left parietal region of the head. The channel of the wound was located diametrically through the skull, passing through the right and left hemispheres and the ventricular system of the brain. The channel of the wound crossed the projection of the right middle cerebral artery branches and midline structures at the level of the

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third ventricle and septum pellucidum. The entire wound channel was filled with blood and multiple small bone fragments were detected in the first section of the right frontal lobe (Fig. 2B). Clinical observations included hemorrhages in the lateral (Fig. 2C), 3rd and 4th brain ventricles, acute subdural hematoma over the right hemisphere, and massive subarachnoid hemorrhage. The injury was accompanied by brain swelling, with a 9 mm midline shift to the left. On the left side of the skull, an explosive fracture of the cranial vault bones was observed (Fig. 2D), with a transition to the base of the skull (Fig. 3B); epidural clots and air bubbles were detected below the fracture. Bone fragments were detected in the soft tissues of the left parietal region.

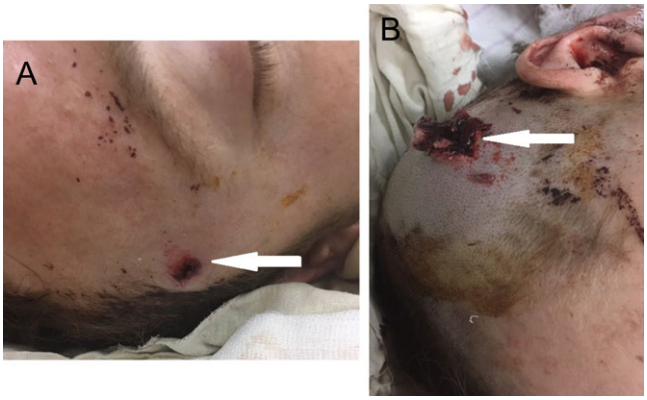

FIGURE 1. Patient's photograph on admission showing (A) inlet (up to 0.5 cm in diameter) in the right temporal area (white arrow) and (B) outlet (2 × 2.5 cm) in the left parietal area, with traces of coagulated blood (white arrow) and bleeding from the ear.

To counter intracranial hypertension and prevent internal occlusive hydrocephalus in the acute period and subsequent non-obstructive (communicating) hydrocephalus and purulent septic complications, the patient underwent urgent surgery. Surgical intervention was performed using a microsurgical technique with an OPMI VARIO 700 microscope (Carl Zeiss, Oberkochen, Germany). The patient's preoperative GCS score of 5 was stable; anisocoria D > S was determined, and pupil reaction to light was reduced.

The first stage of surgery comprised primary surgery of the head injury near the inlet in the right temporal region. The following steps were performed: skull resection via trepanation; removal of intracerebral and subdural hematoma, brain detritus, and bone fragments from the wound channel; hemostasis in the small branches of the right middle cerebral artery; and removal of hematomas from the 3rd ventricle and

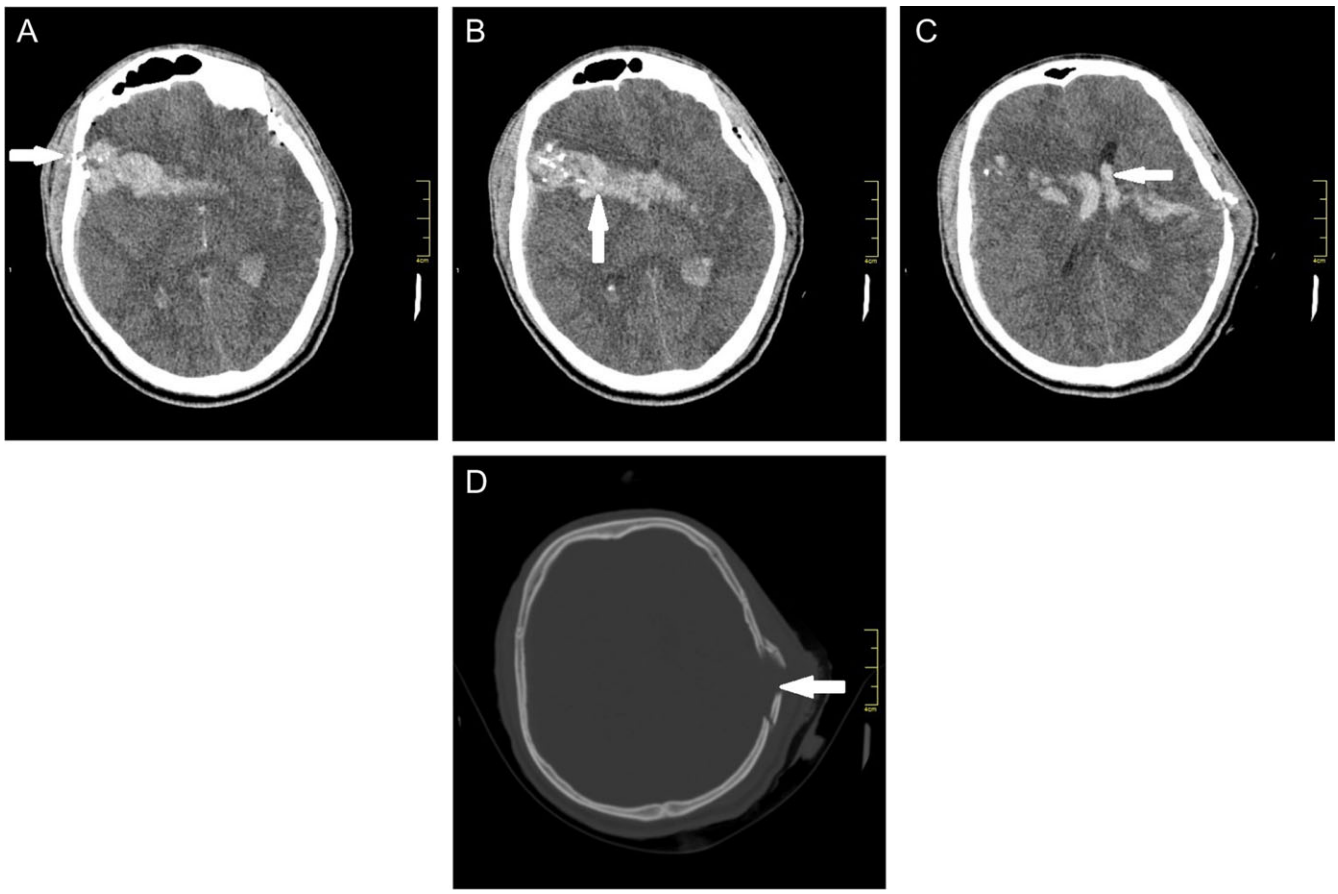

FIGURE 2. Brain computed tomography (CT) showing (A) inlet (white arrow) in the right temporal bone, (B) wound channel filled with blood and bone fragments (white arrow), (C) intraventricular hemorrhage (white arrow), and (D) explosive fracture of skull base near the outlet.

right lateral ventricle. The cerebral wound was subjected to inflow/outflow drainage. This first stage lasted 2 h 10 min. The second stage comprised primary surgery of the craniocerebral wound near the wound outlet. The following steps were performed: skull resection via trepanation, followed by removal of bone fragments, brain detritus, and foci of crush injury from the left parietal and temporal lobes. Inflow/outflow drainage of the brain wound was performed on the left side using silicone drains with an internal diameter of 3.5 mm, with a blunt, closed end, and contained multiple lateral holes over 5 cm from the drain tip. A sterile isotonic 0.9% NaCl solution, 1,200–2,000 ml per day, was used for washing. A fluid flowed through a drainage pipe to a closed sterile system. During both the first and second stages of surgery, duraplasty was performed using autografts composed of periosteum and superficial temporal fascia. Silicone drains were introduced through specially created tunnels in the dura mater and subaponeurotic space and exited through counteropenings at considerable distances (8 and 10 cm) from the primary wounds. This second stage lasted 2 h.

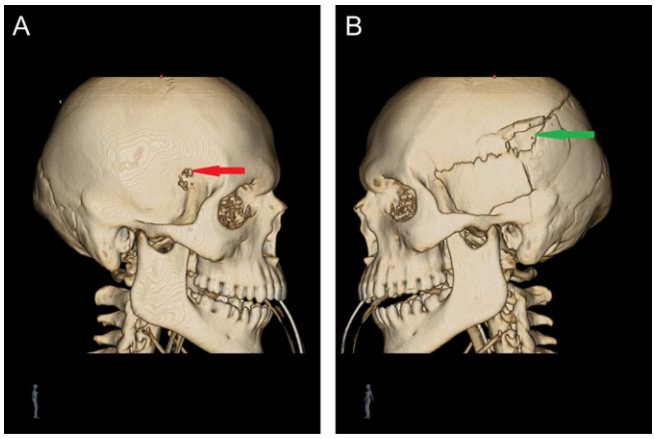

FIGURE 3. 3D model of the patient's skull showing (A) inlet in the right temporal bone with small bone fragments (red arrow) and (B) concentric swollen fracture of the left parietal bone (green arrow) with fracture lines spreading across the skull vault and base.

On the first day after surgery, follow-up CT imaging of the brain revealed postoperative cranial vault defects on the right- and left-hand side, presence of drainage tubes along the length of the wound channel, absence of bone fragments in the wound channel, significant decrease in the volume of intracerebral and intraventricular hematomas, regression in lateral dislocation with midline structures in the expected location (Fig. 4A). On the same day, inflow/outflow drainage tubes were removed on the left (Fig. 4B) and on the right on the following day, with counteropenings subsequently being stitched. Thus, the drainage system was in place for less than 3 days. Further follow-up CT imaging of the brain was performed on day 5 after surgery (Fig. 4C).

The patient was comatose for 9 postoperative days (GCS score of 7). On day 2 after surgery, a lower tracheotomy was performed. On day 14, with a GCS score of 10, the patient was disconnected from the oxygen breathing apparatus.

General cerebrospinal fluid (CSF) test on the first day after removal of the inflow-outflow system revealed the following: cytosis 6×10^6 cells/l; protein 1.5 g/l; hemorrhagic color of fluid before centrifugation and saturated xanthochromic color after centrifugation. Maximum CSF cytosis (341.3×10^6/l) was detected on day 5 after surgery. Table I contains more detailed information on the patient's vital functions dynamics and test results.

Microbiological tests on blood, cerebrospinal fluid (CSF), urine, and sputum were performed using a VITEK-2 bacteriological analyzer (bioMerieux., Inc., France). Repeated cerebrospinal fluid analysis showed no growth of bacteria. In fluid from bronchoalveolar lavage were isolated *Pseudomonas aeruginosa* (104 colony forming unit, CFU), *Proteus mirabilis* (104 CFU), *Corynebacterium xerosis* (104 CFU), and *Klebsiella pneumonia* (104 CFU).

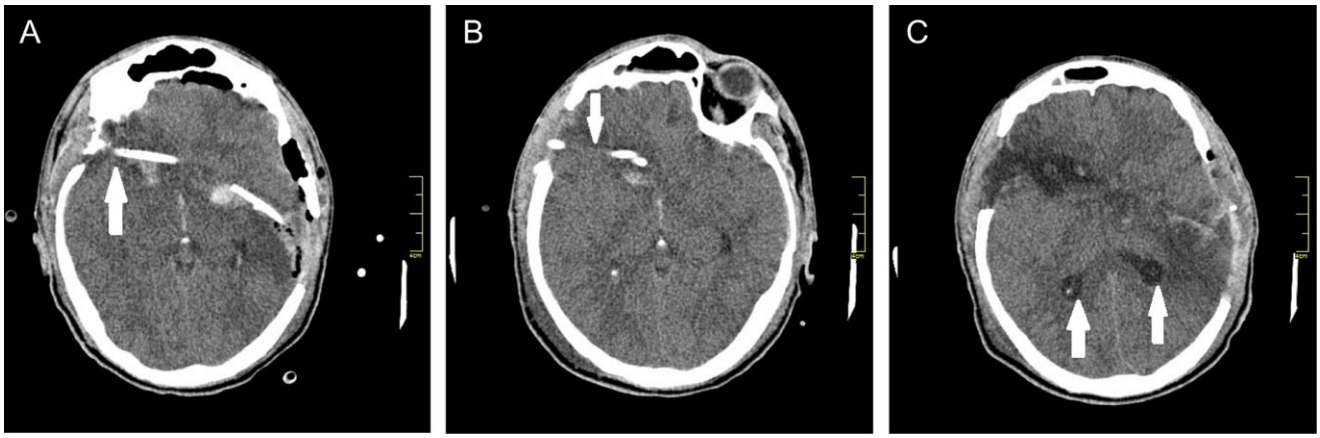

FIGURE 4. Brain computed tomography (CT) images showing (A) follow-up CT on day 1 following surgery and drainage, the absence of bone fragments in the wound channel (white arrow), (B) follow-up CT on day 2 after removal of drains, reduction of blood accumulation along the wound channel (white arrow) and (C) follow-up CT on day 5, lack of blood in the posterior horns of the lateral ventricles (white arrows).

TABLE I. Patient's vital functions dynamics and test results

Parameter	On admission	1st day after surgery	2nd	3rd	5th	7th	11th	At discharge 18th	Norm
GCS (points)	5	7	7	7	7	7	9	15	15
BP (mm Hg)	165/80	130/80	115/70	120/70	110/60	130/70	120/75	120/80	120/80–140/90
HR (bpm)	87	103	78	87	79	94	84	80	60–90
t °C	36.5	37.1	37.6	36.7	36.4	37.8	37.3	37.1	36.6
Ht (%)	34	30	27	27	29	29	25	30	35–50
Er ($\times 10^{12}$ cells/l)	4.2	3.5	3.1	3.04	3.2	3.26	2.82	3.28	3.8–5.8
Hb (g/l)	122	105	95	92	96	99	87	100	100–165
WBC ($\times 10^9$ cells/l)	23.5	14.6	9.4	6.5	5.3	8.2	13.2	11.1	4–9
Pl ($\times 10^9$ cells/l)	160	111	110	102	135	161	259	324	180–320
K^+ (mmol/l)	2.5	2.4	3.7	4.7	4.6	4.5	3.2	4.5	3.5–5.5
Na^+ (mmol/l)	153	152	151	152	150	145	161	140	130–155
Cl^- (mmol/l)	108	111	113	113	111	107	111	103	95–110
Glucose (mmol/l)	7.1	7.63	5.28	4.93	5.17	7.46	7	6	3.5–5.6
Total protein (g/l)	62	60.2	57.8	56.2	53.8	56	56.5	64.5	65–85
Creatinine (μmol/l)	106	122	105	129	113	100	85	53	44–115
Urea (mmol/l)	6.8	4.5	5.8	8.2	9.1	10.7	11.1	11	2.5–8.3
Procal-citonin (ng/ml)	–	–	–	–	0.245	0.163	0.106	0.081	up to 0.5
CSF cytosis ($\times 10^6$ cells/l)	–	–	–	6	341.3	106.6	121.6	–	0–4
Protein in CSF (g/l)	–	–	–	1.5	3.5	1.8	1.32	–	0.22–0.33

According to the standards of antibiotic prophylaxis and treatment of gunshot wounds, from the first day of the patient's stay in the ICU, he took empiric antimicrobial therapy (8 g IV cefazolin per day – during 5 days after surgery).

Then, due to the increased risk of nosocomial infection, was prescribed 4 g IV cefoperazone-sulbactam per day from 6 to 10 day after surgery.

Then, starting from the tenth day after surgery, due to clinical and laboratory signs of post-traumatic meningoencephalitis were prescribed 1500 mg IV meropenem per day and 2000 mg IV vancomycin per day.

Level of serum procalcitonin in this patient was as follows: 0.163 ng/ml on day 7 after surgery; 0.106 ng/ml on day 11; and 0.081 ng/ml on day 15 (normal value is up to 0.5 ng/ml).

The postoperative wounds healed through primary intention with no signs of wound liquorrhea. The stitches were removed on day 12 after surgery.

Upon being transferred to a military hospital rehabilitation center on day 18 after injury, the patient was conscious, with a GCS score of 15 and adequate breathing, stable hemodynamics and ingestion, and no evidence of fever. The patient was able to understand speech directed at him and follow instructions; moderate motor speech disorders and tetraparesis (minor in the left extremities and severe in the right extremities) were still present. At the time of compilation of this article, the patient's follow-up period had reached 4 months, with a Glasgow Outcome Scale result corresponding to severe disability (upper level). Patient's current condition (6 months after the injury), along with continuing improvement of Glasgow Outcome Scale score, corresponds to moderate disability (lower level).[8,9] Cranioplasty for bone defects of the cranial vault has been scheduled for 6 months post-injury.

Ethical approval from an institutional review board, informed consent of the patient and his parents was obtained.

DISCUSSION

Aggressive surgical intervention in hemodynamically stable patients with low GCS scores in the absence of intracranial hematoma with mass effect is an extremely difficult and debatable decision. Most researchers agree that patients with GCS scores of 3–5 have poor outcomes regardless of intervention, recommending conservative treatment unless large intracranial hematomas exist, which must be removed.[10,11] However, as in our case, these decisions must be made on a case-by-case basis, particularly for young, hemodynamically stable patients. Considering the relatively favorable trajectory of the wound channel observed from HCT data and the presence of pupillary reaction to light, surgery was decided upon despite the patient's low GCS score.

Postresuscitation GCS score is the most significant clinical predictor of patient outcome.[10,12,13] Other prognostic factors include hypotension, hypoxia, coagulopathy, dilated and nonresponsive pupils, advanced age, suicidal attempts, and perforating wounds.[14–16] Upper bifrontal perforating wounds are associated with reduced functional damage compared with lower penetrating wounds that pass through the vital structures of the brain.[17] Bihemispheric, multilobar, and ventricular injuries, which were diagnosed in the present case, are generally associated with poor patient outcome.[10,13,17,18] Bihemispheric and ventricular injuries are associated with poor outcome due to pathological processes involving bilateral structures, upper brain stem, basal ganglia and critical vascular elements. Furthermore, tram-track signs in CT images correlate with fatal outcomes ($p = 0.005$). Four potential mechanisms explaining

increased severity of such injuries have been described: 1) diencephalic and mesencephalic destruction; 2) diffuse edema causing intracranial hypertension; 3) localized lesion associated with diffuse edema causing brain stem herniation; and 4) great vessel injuries with profuse hemorrhage.[19] Vector analysis (missile trajectory analysis) of non-survivors has indicated an area of the brain approximately 4 cm above the dorsum sella, which, when penetrated through the midline, leads to brain death ($p = 0.0006$). This zone is known as the "zona fatalis".[20] The presence of intracranial hematomas and wounds of the structures of the posterior cranial fossa are also associated with poor outcome.[11,12]

In such a wound, successful surgery is subject to the availability of full data on severity, nature, and location of intracranial injuries. Therefore, the soonest possible post-injury brain CT can be decisive. In our case, due to non-availability of CT in the zone of military operation, the patient needed to be transported to the nearest medical center. In the absence of CT, decompressive craniotomy can be considered as a method of reducing intracranial pressure and gaining time for transportation to the center where CT and trained neurosurgeon team are available.

Decompressive craniectomy during primary surgical treatment of craniocerebral wounds is only required in specific cases in which cerebral edema is expressed.[21] Most researchers insist on thorough hermetic closure of the dura mater and skin to prevent post-surgical leakage of CSF, which causes purulent septic complications and poor outcomes.[17,22] In our case, decompressive craniectomy was not deemed necessary because removal of the intracerebral hematoma along the wound channel reduced the intracranial pressure (ICP) during surgery. Installation of an inflow/outflow system for 3 days following injury allowed control of ICP by removing CSF from the ventricular system into a sterile, closed system. This drainage system also permitted blood to be quickly released from the brain's ventricular system, reducing the risk of occlusive and non-obstructive (communicating) hydrocephalus. Finally, duraplasty and creation of specific tunnels for drainage tubes helped prevent wound liquorrhea.

In the case of orbital or pterional injuries, the proximity of the wound trajectory to major cerebral arteries, presence of intracerebral hematomas and massive subarachnoid hemorrhage (SAH), possible MCA branch injuries, and formation of further pseudoaneurysms should be considered.[12,16,23]

In addition to adequate craniocerebral wound surgery, intensive postoperative therapy along with maintenance of adequate brain perfusion and oxygenation and adequate antibiotic therapy are of high importance. Antibiotic schemes, monitoring of treatment efficacy, and indications for the discontinuation of treatment have been detailed previously.[24,25]

Radical surgical TBI treatment with a removal of all brain compression factors, hydrocephalus development monitoring, neurophysiological monitoring-based intensive therapy, and balanced antibiotic therapy allow achieving good functional outcomes, even in cases of such severe craniocerebral wounds.

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