ORIGINAL RESEARCH

Risk factors for postoperative infection and its impact on overall outcome after combat related head injuries

Goran Pavlićević, MD, PhD[1]

1. Clinic for Neurosurgery, Military Medical Academy, Belgrade, Serbia

Abstract

Introduction: The prevalence of penetrating head injuries (PBI) has increased during the latest wars, comprising up to 37,4% of all injuries. The microbiology of modern war wounds depends on the climatic and geographical features of the theater of combat.

Material and methods: A total of 286 patients were operated on after penetrating cranial combat injury in our institution between 1991-1999, of which 202 were included in this study based on inclusion criteria of combat-related cranial injury, absence of severe abdominal or chest combat injuries, and ability to report for a follow-up exam. Initial surgical treatment included removal of devitalized soft tissue and bone fragments with craniectomy and removal of devitalized brain tissue, easily accessible intracerebral bone and metal fragments, and intracranial hematoma. All patients received standardized postoperative care with triple antibiotics.

Results: Infection occurred in 36 patients (17,82%), most commonly in the form of brain abscess (31, 86.11%), in addition to meningitis (4, 11.1%) and osteomyelitis and epidural infection (1, 2.78%). Retained metal and bone fragments and postoperative CSF leak had significant influences on the occurrence of postoperative infection.

Conclusion: Postoperative infection considerably worsens long-term functional outcome, and it was favored in patients with retained metal and bone fragments and postoperative CSF leak. Autograft appears as preferable option for dural reconstruction in penetrating combat-related cranial injuries, although our study failed to find statistically significant correlation between the postoperative infection and the material used for the reconstruction.

Keywords: combat injury; head; brain; traumatic brain injury; infection; outcome

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Introduction

The prevalence of penetrating head injuries (PBI) has increased in the latest wars in south-east of Ukraine, Iraq, Afghanistan, and Syrian Arab Republic, comprising up to 37,4% of all injuries [1,2].

The microbiology of modern war wounds is unique to each military conflict depending on the climatic and geographical features of the theater of combat [3,4]. The mechanism of high velocity weapons injury predisposes to development of intracranial infection, mostly due to the cavitation effect, causing initial expansion in brain tissue (often 10 to 20 times the size of the projectile), which collapses under negative pressure that may draw in external debris [27]. Patients with severe head injuries are also prone to infection as they have prolonged intubation, decreased respiratory function, prolonged immobilization, and posttraumatic immunosuppression.

On the other hand, different types of invasive monitoring, including intracranial pressure monitoring, increase the risk for wound infection.Cranial infection after war injury can develop in the form of epicranial infection, osteomyelitis, epidural abscess, subdural empyema, meningitis, brain abscess, or ventriculitis. All forms of posttraumatic infection considerably increase mortality and morbidity.

The current concept of surgical treatment is controversial given uncertainty regarding the infective potential of retained intracranial foreign object and the different surgical strategies are used [5,6,7,8,10,12,13,14,15,19,20,21,25,26]. We present our experience of postoperative infection in patients with combat-related penetrating cranial injuries and the long-term functional outcome of these events.

Materials and methods

Patient selection

A total of 286 patients were operatively treated after penetrating cranial combat injury in our institution between 1991-1999. For this study, the data of 202 patients were retrospectively reviewed, and Glasgow outcome score (GOS) was determined on examination. The inclusion criteria were as follows: combat-related cranial injury, absence of severe abdominal or chest combat injuries, and ability to report follow-up examination. Forty-one patients died during initial treatment because of the severity of injury (92% of them had GCS≤8), and they were excluded from the study. Forty-three patients were also excluded from the study as they were lost to follow-up (due to the address change, death etc.).

Initial surgical treatment

Initial surgical treatment after craniocerebral injury included removal of devitalized soft tissue and bone fragments with craniectomy and removal of devitalized brain tissue, easily accessible intracerebral bone and metal fragments, and intracranial hematoma. The dura was closed and rendered watertight, which in almost all cases required a dural autograft (periosteum, temporalis fascia, fascia lata) or allograft. In cases with opened air cavities, obliteration of these cavities was performed with fat graft followed by suturing of the graft (periosteum, fascia, muscle) to dura and the epicranial aponeurosis (***Figure 1***). Soft tissues were closed without suture tension.

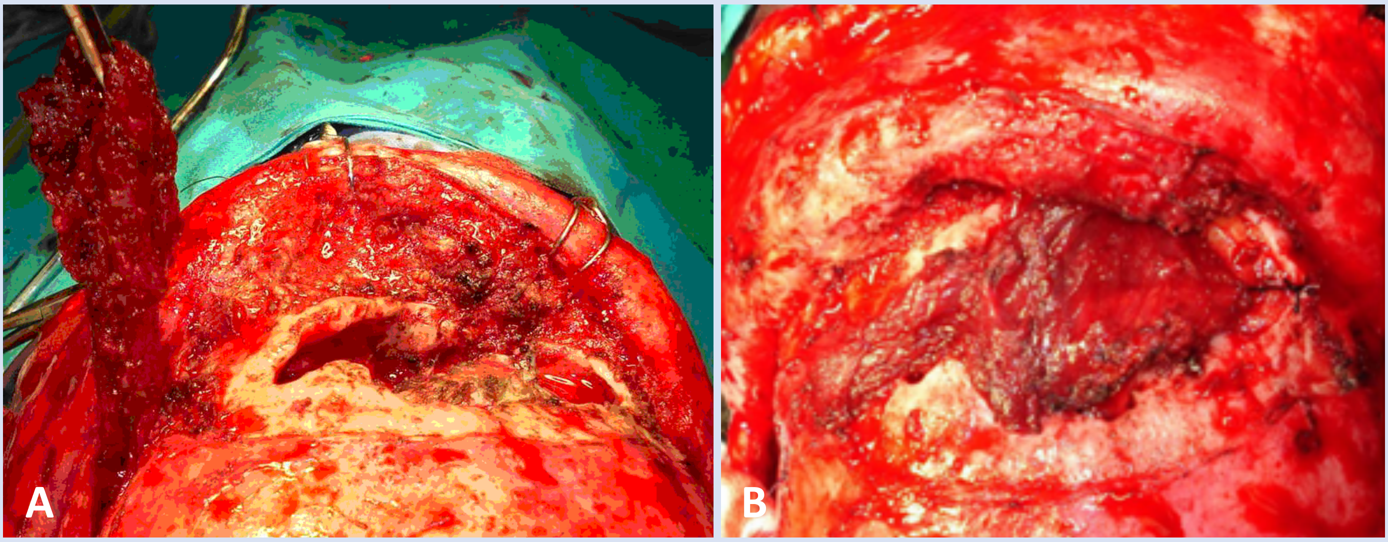

Figure 1. Surgical air cavities obliteration. A. Preparation of the graft. B. Graft in place secured with sutures.

Postoperative care

Drains were removed when the daily drainage volume was less then 50 ml, at least 24-48 hours after the operation. Subsequent epicranial collections were removed by puncture under sterile conditions and treated with compressive dressings until coalescence between the skin flap and the graft occurred. Third generation cephalosporine, aminoglycoside and metronidazole were used for prophylaxis over a ten-day period. Postoperative cerebrospinal fluid (CSF) leak was treated with lumbar drainage for 7 days. In the cases with persistent CSF leak, wound revision was performed.

Assessment of outcome

The mean postoperative follow-up period was 12.3 years (10-15 years). A follow-up CT was done to locate any retained metal or bone fragments and to assess the dominant localization of brain damage before control examination. GOS was determined on control examination.

Statistical analysis and Factors Influencing the Outcome

We evaluated the influence of the following 7 factors: age, mechanism of injury (bullet or explosive injury), localization of injury, dura (autograft or allograft), communication with paranasal cavities, retained metal or bone fragments (larger than 10mm) and postoperative CSF leak, on the development of postoperative intracranial infection. We have also analyzed the influence of postoperative infection on the Glasgow outcome score.

Data processing was performed using SPSS 11.5 (SPSS Inc., Chicago, Illinois, United States of America) for Windows. Average values were presented as mean ± standard deviation. P values less than 0.05 (two-tailed) were considered to be statistically significant. Groups were compared using an unpaired Student t test for parametric data and the Mann-Whitney U test for nonparametric data. Data for 3 or more groups were compared using 1-way analysis of variance, the Kruskal-Wallis H test.

Results

Characteristics of the series

All patients were males, aged from 18 to 61 years of age (average of 30,61±9,5years). Only five patients were older than 50 years.

Considering the mechanism of injury, 136 (67.4%) patients had explosive injuries, and 66 (32.6%) had injuries caused by bullets. The most severe brain damage was localized most often in frontal region 89 (44,1%), then parietal (84, 41,1%), occipital (16, 7,9%) and the temporal 13 (13, 6,4%) regions. Opened air cavities were noted during operation in 11 patients (5,4%), and 17 (8,4%) patients had retained bone or metal fragments larger then 10mm in brain tissue. For dural repair, autograft (fascia lata or periosteum) was used in 47 (23,3%), and allograft in 155 patients (76,7%).

CSF leak was noted in 10 patients (4,9%). According to the Glasgow Outcome Score, 116 patients (57.4%) had a good recovery, 55 (27.2%) were left with moderate disability, and 31 (15.3%) of the patients were severely disabled.

Risk factors for infection and influence on functional outcome

Infection occurred in 36 patients (17,82%), most commonly in the form of brain abscess (31, 86.11%), in addition to meningitis (4, 11.1%) and osteomyelitis and epidural infection (1, 2.78%). Infection developed from 3 to 18 days after debridement and was caused by gram positive bacteria in 60% of the cases.

Postoperative infection occurred almost equally among the patients of different age (29,2 vs 31,1 years) and among patients with different mechanisms of injury (17,65% after explosive and 18,18% after bullet injury).

Infection most commonly occurred in in the dominant occipital region of brain injury (37,5%), then in parietal region (19,04%), temporal region (15,38%) and most rarely frontal localization (10,5%) The distribution of infection was the same across different localizations (Kruskal Wallis test, p>0,05).

Infection occurred in 1/11 (9.1%) patients with open cavities and in 35/191 (18.3%) without open paranasal cavities (Mann-Whitney U test p>0,05).

Type of the dural graft was not associated with developing an infection (10,6% in autograft group, 20% in allograft group, Mann Whitney U test p>0,05).

Postoperative infection was twice as frequent in patients with retained fragments in brain tissue (6/17, 35.3%) as it was in those without retained fragments (30/185, 16.2%), (Mann Whitney U test p<0,05).

Postoperative infection occurred in 40% of patients with CSF leak and in 16,7% without CSF leak (Mann Whitney U test, p<0,05. There were a small number of patients (10) with CSF leak in study.

According to GOS, good recovery was noted in only 16.7% of patients with infection and in 66,27% of the patients without infection. Moderate disability occurred 50% of cases with infection and 22,29% without infection, and severe disability occurred in 33,4 % of patients with infection and 11,4% without infection (Kruskal Wallis Test p<0,001).

Discussion

In patients with combat-related penetrating brain injury, the incidence of wound contamination ranges from 39-80% [3,4,26]. The high incidence of wound contamination results from contaminated foreign objects, skin, hair, and bone fragments driven into the brain tissue along the projectile tract [12]. High velocity projectiles, dominantly used in wars, create temporary cavitation during brain penetration as a result of the transmission of the kinetic energy of the projectile into surrounding tissue. Formation of a temporary cavitation produces an enormous increase in intracranial pressure. The temporary cavitation then collapses, resulting in negative intracranial pressure. Due to brain elasticity, the cavitation forms and then collapses several times. When negative intracranial pressure occurs during the temporary cavitation collapse, aspiration of the foreign contents into the wound occurs, causing additional contamination of the intracranial space [11]. Bone fragments have higher potential for infection than metal fragments as high kinetic energy and temperature from metal fragments during brain penetration sterilizes surrounding brain tissue. Necrotic and devitalized brain tissue around the projectile trajectory also increases risk for the development of infection.

The main goals of surgical treatment of casualties with penetrating brain injuries are to normalize intracranial pressure and to prevent infection. Operative techniques have been changed to reduce risk factors for postoperative infection over time.

The principles of penetrating head trauma management, radical debridement of the scalp and skull and aggressive irrigation of the projectile trajectory to remove foreign bodies with water-tight closure. were established by Harvey Cushing during the World War One. T Using this approach Cushing significantly decreased infection rates, which reportedly mitigated the major cause of mortality due to penetrating head injuries [7].

Experiences gained during the Korean War and Vietnam War have changed surgical approaches such that that only easily accessible fragments should be removed, as evacuating the foreign bodies that are distant from the projectile trajectory may result in additional neurologic deficit or a lesser degree of recovery of functions due to brain damage [7,15]. Fragments distant from the projectile trajectory are left in the brain tissue [7,15]. This strategy was used in the majority of operative treatments during recent wars, including wars in the former Yugoslavia in the 1990s [21].

Many studies conducted in last decades of the 20th century suggested that retained metal and bone fragments in the brain did not increase the incidence of immediate or late complications such as infection or epilepsy [20,21,19]. As a result, conservative management of retained shrapnel is recommended in view of low long-term infection rates and worsened neurological outcome with shrapnel retrieval [12,13,14,15,5].

Surgical strategy has shifted further toward the conservative approach based on studies in which minimal debridement or even simple wound closure was successfully used as the only surgical treatment in war victims [8,20]. Some authors have even found out that conservative approach promoted superior outcomes to standard surgery, as no mortality in was noted in conservatively treated patients in comparison to 48,5% of mortality rate in surgically treated cases [10].

With the adoption of a less aggressive approach of treatment, it is likely that large percent of the patients will have retained intracranial fragments.

in contrast, other studies have suggested that retained bone and metal fragments are the risk factor for development of postoperative infection [22,24] and that the presence of intracranial retained foreign bodies promotes worse outcome in comparison with their absence [25]. Some studies found out that foreign bodies have the potential to initiate infection decades after the injury: brain abscess has been noted in patients 30 and 52 years after the injury around the retained shrapnel [6,23]. Long-term follow-up also revealed potential complications such as migration of the foreign bodies or development of the hydrocephalus [23]. Foreign bodies in the eloquent cortex may also increase the risk of posttraumatic epilepsy [16,24]. Those findings rendered it acceptable to remove all bony and metallic fragments that are accessible without additional trauma to non-damaged brain regions [24].

Careful preoperative planning for secondary removal of retained missile fragments, the use of neuronavigation system, and choosing a less invasive approach for the exact intraoperative localization of the fragment can result in extraction without additional neurologic deficit, infections, or seizures [24].

In our series, bone and metal fragments that were distant from the projectile trajectory were left intact. Postoperative infection was twice as frequent in patients with retained fragments in brain tissue than those without retained fragments. These finding support the view that additional effort should be used to remove retained brain fragments using contemporary devices such as neuronavigation, open multi-slice computed tomography, and minimal invasive surgical approaches.

CSF leaks after penetrating brain injury are highly predictive of infectious complications [17]. Infection rates in patients with CSF leak are 49,5-68% compared to 1,5-4,6% in those without leak (17,18,19,). In our study, the infection rate was 40% in patients with leak relative to 16,7% in without leak. Our results suggest that early revision surgery is better option than lumbar drainage as CSF leak carries a high risk for postoperative infection.

Anterior cranial fossa injury is an important subgroup of craniocerebral missile injuries, as the projectile trajectory traverses the facio-orbital plane before penetrating the cranium. CSF spaces communicate with air-filled mucosa-lined spaces, and patients may later develop CSF rhinorrhea or orbitorrhea [9]. Such patients are prone to infection. The infection rate in some studies is higher in patients with anterior cranial fossa injury [11].

In our study, the presence of open air cavities was not a risk factor for postoperative infection, as the incidence of CSF leak in this location was similar to other locations. We can conclude that appropriate surgical technique, including using fat graft for obliterating paranasal cavities and pericrania-fascia lata flap for covering anterior base, can prevent CSF leak and diminish the risk for infection in this location.

In combat penetrating craniocerebral injury, dura is usually lost, necessitating the use of dural substitutes. Some authors suggest that autologous tissues are preferred because a synthetic dural substitute, as a foreign body, may become a potential source of infection, particularly in grossly contaminated wounds (9,11). Using of auto or allograft in our study was not a statistical risk factor for postoperative infection, but patients receiving allograft had postoperative infection twice as frequently as patients with autograft.

Increasing age is correlated with a worse prognosis in penetrating brain injury, especially in patients older than 50 years (11,12). In our study, infection was almost equal in different age groups, but we have only five patients older than 50 years, and the soldiers were in good physical condition and without comorbidities.

Comparing groups with different GOS in our study revealed that postoperative intracranial infection had strong influence on functional recovery, as the chance for good recovery based on GOS was four times higher in patients without infection.

Conclusion

Postoperative infection considerably worsens long-term functional outcome. Retained metal and bone fragments and postoperative CSF leak have significant influences on occurrence of postoperative infection. Although our study failed to find statistically significant correlation between the postoperative infection and the material used for dural reconstruction, autograft appears as preferable option in penetrating combat-related cranial injuries

Disclosures

Conflict of Interest: The author certify that he have no affiliations with or involvement in any organization or entity with any financial interest (such as honoraria; educational grants; participation in speakers' bureaus; membership, employment, consultancies, stock ownership, or other equity interest; and expert testimony or patent-licensing arrangements), or non-financial interest (such as personal or professional relationships, affiliations, knowledge or beliefs) in the subject matter or materials discussed in this manuscript.

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Correspondence

Goran Pavlićević

Clinic for Neurosurgery, Military Medical Academy
Crnotravska 17, 11000, Belgrade, Serbia

☏ +381641189

@ drpavlicevic@gmail.com

References

1. MacGregor AJ, Dougherty AL, Galarneau MR. Injury-specific correlates of combat-related traumatic brain injury in Operation Iraqi Freedom. J Head Trauma Rehabil. 2011;26(4):312-318. doi: 10.1097/HTR.0b013e3181e94404
2. Gizatullin SK, Stanishevskiy AV, Svistov DV. Combat gunshot skull and brain injuries. Zh Vopr Neirokhir Im N N Burdenko. 2021;85(5):124-131. doi: 10.17116/neiro202185051124
3. Valentine KP, Viacheslav KM. Bacterial flora of combat wounds from eastern Ukraine and time-specified changes of bacterial recovery during treatment in Ukrainian military hospital. BMC Res Notes. 2017;10(1):152. Published 2017 Apr 7. doi: 10.1186/s13104-017-2481-4
4. Murray CK, Roop SA, Hospenthal DR, et al. Bacteriology of war wounds at the time of injury. Mil Med. 2006;171(9):826-829. doi: 10.7205/milmed.171.9.826
5. Pringle C, Bailey M, Bukhari S, et al. Manchester Arena Attack: management of paediatric penetrating brain injuries. Br J Neurosurg. 2021;35(1):103-111. doi: 10.1080/02688697.2020.1787339
6. Marquardt G, Schick U, Möller-Hartmann W. Brain abscess decades after a penetrating shrapnel injury. Br J Neurosurg. 2000;14(3):246-248. doi:10.1080/026886900408450
7. Milton J, Rugino A, Narayan K, Karas C, Awuor V. A Case-Based Review of the Management of Penetrating Brain Trauma. Cureus. 2017;9(6):e1342. Published 2017 Jun 12. doi: 10.7759/cureus.1342
8. Amirjamshidi A, Abbassioun K, Rahmat H. Minimal debridement or simple wound closure as the only surgical treatment in war victims with low-velocity penetrating head injuries. Indications and management protocol based upon more than 8 years follow-up of 99 cases from Iran-Iraq conflict. Surg Neurol. 2003;60(2):105-111. doi: 10.1016/s0090-3019(03)00358-6
9. Bhatoe HS. Missile injuries of the anterior skull base. Skull Base. 2004;14(1):1-8. doi: 10.1055/s-2004-821351
10. Darwazeh R, Darwazeh M, Sbeih I, Yan Y, Wang J, Sun X. Traumatic Brain Injury Caused by Missile Wounds in the North of Palestine: A Single Institution's Experience with 520 Consecutive Civilian Patients. World Neurosurg. 2018;116:e329-e339. doi: 10.1016/j.wneu.2018.04.202
11. Kazim SF, Shamim MS, Tahir MZ, Enam SA, Waheed S. Management of penetrating brain injury. J Emerg Trauma Shock. 2011;4(3):395-402. doi: 10.4103/0974-2700.83871
12. Esposito, D P. Walker, J B. Contemporary Management of Penetrating Brain Injury, Neurosurgery Q. 2009;19(4): 249-254
doi: 10.1097/WNQ.0b013e3181bd1d53
13. Chaudhri KA, Choudhury AR, al Moutaery KR, Cybulski GR. Penetrating craniocerebral shrapnel injuries during "Operation Desert Storm": early results of a conservative surgical treatment. Acta Neurochir (Wien). 1994;126(2-4):120-123. doi: 10.1007/BF01476420
14. Surgical management of penetrating brain injury. J Trauma. 2001;51(2 Suppl):S16-S25.
15. Hammon WM. Analysis of 2187 consecutive penetrating wounds of the brain from Vietnam. J Neurosurg. 1971;34(2 Pt 1):127-131. doi: 10.3171/jns.1971.34.2part1.0127
16. Salazar AM, Jabbari B, Vance SC, Grafman J, Amin D, Dillon JD. Epilepsy after penetrating head injury. I. Clinical correlates: a report of the Vietnam Head Injury Study. Neurology. 1985;35(10):1406-1414. doi: 10.1212/wnl.35.10.1406
17. Management of cerebrospinal fluid leaks. J Trauma. 2001;51:S29–33.
18. Meirowsky AM, Caveness WF, Dillon JD, et al. Cerebrospinal fluid fistulas complicating missile wounds of the brain. J Neurosurg. 1981;54(1):44-48. doi: 10.3171/jns.1981.54.1.0044
19. Gönül E, Baysefer A, Kahraman S, et al. Causes of infections and management results in penetrating craniocerebral injuries. Neurosurg Rev. 1997;20(3):177-181. doi: 10.1007/BF01105561
20. Chaudhri KA, Choudhury AR, al Moutaery KR, Cybulski GR. Penetrating craniocerebral shrapnel injuries during "Operation Desert Storm": early results of a conservative surgical treatment. Acta Neurochir (Wien). 1994;126(2-4):120-123. doi: 10.1007/BF01476420
21. Levi L, Borovich B, Guilburd JN, et al. Wartime neurosurgical experience in Lebanon, 1982-85. I: Penetrating craniocerebral injuries. Isr J Med Sci. 1990;26(10):548-554.
22. Aarabi B, Taghipour M, Alibaii E, Kamgarpour A. Central nervous system infections after military missile head wounds. Neurosurgery. 1998;42(3):500-509. doi: 10.1097/00006123-199803000-00014
23. Swain SK, Arora RK, Mittal RS. Migrating Souvenir Bullet: The Management Dilemma. Ann Neurosci. 2021;28(1-2):79-81. doi: 10.1177/0972753120966495
24. Schulz C, Woerner U, Luelsdorf P. Image-guided neurosurgery for secondary operative removal of projectiles after missile injury of the brain. Surg Neurol. 2008;69(4):364-368. doi: 10.1016/j.surneu.2006.11.058
25. Splavski B, Vranković D, Saftić R, Muzević D, Kosuta M, Gmajnić R. Clinical predictors correlated to outcome of war missile penetrating brain injury. Acta Med Croatica. 2006;60(4):369-373.
26. Aarabi B. Comparative study of bacteriological contamination between primary and secondary exploration of missile head wounds. Neurosurgery. 1987;20(4):610-616. doi:10.1227/00006123-198704000-00018
27. Neal CJ, Ling GS, Ecklund JM. (2005) Management of Ballistic Trauma to the Head. In: Mahoney PF, Ryan JM, Brooks AJ, William Schwab C. (eds) Ballistic Trauma. Springer, London. 2005:325-347. doi: 10.1007/1-84628-060-5_