Low velocity gunshot injuries to the temporal bone

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Abstract

Objective: To review the presentation, symptoms and management associated with low velocity gunshot injuries to the temporal bone.

Methods: A retrospective analysis of 26 patients treated for low velocity gunshot injuries to the temporal bone.

Results: Initial presentation included otorrhoea (69 per cent), facial nerve injury (27 per cent), hearing loss (65 per cent), intracranial injuries (50 per cent), and cranial neuropathies (58 per cent). Nine patients (35 per cent) underwent angiography, which showed vascular injury in five of them. Four patients died. **Conclusions:** Low velocity gunshot injuries can be devastating and may result in functional sequelae. Low velocity missiles crush and lacerate surrounding structures, while high velocity missiles cause extensive wound cavity formation. Early aggressive management for intracranial, vascular and facial nerve injury can improve outcome.

Key words: Temporal bone; Wounds, gunshot

Introduction

Injury is the leading cause of death in persons aged one to 44 years in the United States. It is predicted that by the year 2003 firearm-related deaths will become the leading cause of injury-related death (CDC, 1994). Among deaths in persons 15–24 years of age, one out of every four is related to firearms (Fingerhot *et al.*, 1991). Gunshot wounds to the head and neck are the most lethal of firearm injuries, with a mortality rate estimated to be in the range of 75–80 per cent (Kaufman, 1993; Stone *et al.*, 1995).

Gunshot wounds of the temporal bone can result in a wide variety of immediate life-threatening injuries as well as long-term morbidity (Shindo *et al.*, 1995). The temporal bone is densely packed with structures of functional importance (Goodwin, 1983). Mechanisms of hearing, balance and facial movement can be permanently destroyed. The internal carotid artery with its proximity to the petrous portion of the temporal bone is at high risk of injury. The temporal bone has an intimate relationship with the dura mater where injury can cause serious and lasting disability (Goodwin, 1983). The accompanying intracranial damage with contaminated bony fragments can lead to permanent sequelae.

A bullet that travels less than 1000 feet/second (ft/ sec) is classified as low velocity. Low velocity firearm injuries differ from high velocity ones. Most hand gun injuries are classified as the low velocity range and are most commonly seen at our urban centre. Structures in immediate contact with the low velocity missile are crushed or lacerated (Tainmont, 1983). Some low velocity bullets are designed to expand on impact. The expansion causes an increased surface area with a greater release of energy resulting in a large primary wound cavity. Most low velocity missiles do not have the kinetic energy to pass completely through tissue and bone.

In contrast, high velocity bullets travel faster than 2000 ft/sec. Direct crush and lacerating injuries occur, and shock waves are produced (Tainmont, 1983). This leads to secondary missile formation from the surrounding bone. A cavity is created which can be 40 times the diameter of the bullet fragments. It reaches its maximal size in a few milliseconds. By this mechanism, missiles cause injury to surrounding structures outside its direct path. Extensive tissue damage occurs as the cavity collapses which may lead to extensive intracranial injury and possible death.

A basic ballistic principle is that the 'wounding power' of a missile is a function of its kinetic energy. The kinetic energy of a moving object is expressed as $E = mv^2/2$ where m indicates the mass and v represents the velocity. Although the mass is directly proportional to the energy dissipated to the surrounding tissue, it is the velocity that plays a much more significant role. This determines if the bullet penetrates through tissue and bone.

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Gunshot wounds to the temporal bone result in fractures. Although much has been written about blunt injuries to the temporal bone resulting in longitudinal or transverse fractures, very few investigators have studied low velocity penetrating trauma. By understanding wound ballistics one can realize why gunshot injuries to the temporal bone have a much less predictable clinical course and outcome.

Materials and methods

We conducted a retrospective analysis of patients with the diagnosis of skull base fractures with firearm injury at Kings County Hospital Center, Brooklyn, New York from 1991 to February 1996. Thirty-one patients were identified with low velocity gunshot injuries to the temporal bone with 26 charts available for review. The patients' records were reviewed with special emphasis on initial management, computerized tomography (CT) results, angiographic findings, facial nerve injury and intracranial damage.

Results

A total of 26 patients, 23 men and three women, were included in the study. Their ages ranged from six to 56 with a median of 29 years. The injuries were divided by anatomical location as shown in Table I. The mastoid portion was the most frequently injured followed by the external auditory canal (EAC). Mixed fractures, which involve multiple anatomical segments, occurred in nine patients. Six out of nine mixed fractures involved the mastoid and/or EAC.

Four patients suffered multiple gunshot injuries with wounds to the extremities (four), abdomen (two), chest (one), neck (one) and gluteal region (one). These associated injuries were treated by the trauma service and did not have an acute impact on the temporal bone injury.

Nine patients (35 per cent) underwent carotid angiography. Vascular injury was detected in five patients (20 per cent). Injuries were sustained to the facial artery (two), lingual artery (one), internal maxillary artery (one), superficial temporal artery (one) and vertebral artery (one). Arterial embolization was performed for a lingual artery injury in one patient, while the internal maxillary and facial arteries were embolized in another. Three other injuries were observed without further sequelae. Two out of seven patients with entrance wounds to the face underwent angiography compared to seven out of 19 with entrance wounds to the head.

Peripheral facial nerve injury was seen in seven patients (27 per cent). All nerve branches were involved in six patients and an isolated peripheral temporal branch in one. In six of these patients the injury was documented in the mastoid and/or EAC. Of these patients, five underwent exploration and partial recovery was reported in two of them. One patient with complete paralysis on presentation improved to grade 5 in the House-Brackman

TABLE I LOCATION AND TYPE OF INJURY

Location	Number of patients	Facial nerve injury	Hearing loss	Intracranial injury
Mastoid	7	2	4	5
EAC	5	0	3	0
Squamous	3	0	1	3
Petrous	2	0	1	1
Mixed	9	5	8	4
TOTAL	26	7	17	13

classification without initial exploration. This patient underwent a mastoidectomy one year post-injury for chronic otorrhoea.

Seventeen patients (65 per cent) complained of hearing loss. Audiograms were obtained in 14 of them. Sensorineural or mixed hearing loss occurred in 12 out of 14 patients (86 per cent). Eighteen patients (69 per cent) presented with bloody otorrhoea on initial examination. However, tympanic membrane injuries were documented in only seven patients. Four patients complained of vertigo. A total of 12 patients did not have audiometric studies: four patients died, and eight patients were not evaluated by the Otolaryngology service.

Intracranial injuries were seen in 13 patients (50 per cent). Seven patients underwent craniectomy, with six of them surviving. An additional two patients required neurosurgical intervention within one month post-injury. One patient developed a temporal lobe abscess while another required a shunt procedure for hydrocephalus. Four patients with intracranial injuries died within 48 hours post-injury.

Cranial nerve involvement was documented in 15 patients (58 per cent). Twelve patients had involvement of the auditory nerve, seven patients had facial nerve injury, two patients had optic nerve injury, one patient had an abducen nerve injury and one patient had oculomotor nerve damage. Only one patient had documented trigeminal nerve injury. We suspect that this injury may have been underdiagnosed.

Discussion

A multidisciplinary approach is necessary for the majority of gunshot wounds to the temporal bone. The trauma/emergency physician is often the first to encounter the patient. At our institution advanced trauma life support (ATLS) protocol is used (Ramenofsky *et al.*, 1989). Establishing an airway and resuscitation are the first priorities (Stiernberg *et al.*, 1992). Multiple injuries are often present and will require a close co-operation between the trauma surgeon, neurosurgeon, interventional radiologist and otolaryngologist.

Once the patient is stabilized, a more thorough diagnostic workup is initiated (Stiernberg *et al.*, 1992). CT scanning is essential in evaluating intracranial as well as temporal bone damage. CT scans are very effective in evaluating the extent of bony

LOW VELOCITY GUNSHOT INJURIES TO THE TEMPORAL BONE



FIG. 1 CT scan depicting bullet fragments in a mastoid cavity.

damage (Figure 1). Often the extent of inner ear injury can be determined by CT alone (Haberkamp, 1995).

A thorough otological evaluation is important. A complete initial examination may be delayed until the patient is discharged from the intensive care unit. However, an initial bedside evaluation can be performed in all patients. The canal should be thoroughly cleaned under the otomicroscope. Injuries to the external auditory canal, middle ear cleft and facial nerve should be carefully described.

Audiometry is recommended for all patients sustaining temporal bone trauma. Sixty-five per cent of the patients in our series complained of hearing loss. Audiometry is often not performed until several weeks post-injury due to the patient's condition. However, tuning fork testing can be performed at the bedside to help determine if there is a sensorineural component.

Vertiginous symptoms may not be present until the patient is walking. Four patients in our series reported vertigo. Unusual clumsiness or falls may imply vestibular damage. Differentiating a labyrinthine concussion from a perilymph fistula can be difficult. Vertigo and hearing loss can occur with both. If the cochlea sustained a concussion, the hearing loss is often temporary. Hearing loss may be at low or high frequencies or a combination of both (Gros, 1967).

Neurosurgical intervention is important. Fifty per cent of our patients sustained intracranial damage. The most common entrance wound was to the head (73 per cent). Early debridement of bone and necrotic brain tissue can improve outcome significantly (Levy *et al.*, 1993; Stone *et al.*, 1995). Emergent craniectomy had a significant impact on survival. Six of seven patients who underwent emergent craniectomy survived. All four deaths were from intracranial damage.

Missile injuries to the temporal bone can cause major vascular injury. The trajectory of the bullet may be a clue to accompanying vascular injury especially if there is penetration into the neck. Although none of the patients in our series sustained a carotid artery injury, the internal carotid artery with its proximity to the petrous portion of the temporal bone is at high risk of injury. Missile injuries can cause local bullet and bone fragmentation which can lead to injury unrelated to the trajectory of the bullet. Controversy exists in the literature as to the necessity of angiography in penetrating neck trauma in stable asymptomatic patients (Scalfani, 1991). We advocate that angiography should be performed in all stable patients sustaining gunshot wounds to the temporal bone. Intimal injuries can occur from surrounding local shock waves. Undiagnosed and untreated carotid injuries can lead to the subsequent formation of pseudoaneurysms and arteriovenous malformations resulting in blindness or neurological sequelae (Scalfani, 1991).

Scalfani and Scalfani (1996) published that patients sustaining trauma to zone III of the neck are more likely to have multiple vessel injuries than those in zone II or below. They found that approximately 21 per cent of patients with entry or exit wounds in zone III had multiple asymptomatic vascular injuries. This is similar to our series with 20 per cent of patients demonstrating vascular injury. Scalfani believes that the kinetic energy of the bullet is distributed within a limited area leading to increased tissue destruction. Embolization is a useful modality as an alternative to surgery for vascular injuries detected during angiography. Transcatheter arterial embolization is an ideal treatment option for small multiple vessel injuries in zone III because this area is difficult to access surgically. Two patients in our series were treated by this modality.

None of our patients required base of skull exploration for excessive bleeding or sigmoid sinus injury. A plausible explanation is that the injury was caused by low velocity handguns which cause direct lacerating, crushing effects and local bone destruction. This is in contrast to high velocity guns with secondary missile and cavitary formation causing extensive destruction. These injuries, as seen with military weapons, would likely cause immediate death.

Twenty-seven per cent of our patients sustained facial nerve injury. This is lower than the previously reported incidence of 45–75 per cent (Gros, 1967; Hooper *et al.*, 1972; Hagan *et al.*, 1979; Goodwin, 1983; Duncan *et al.*, 1986). The difference may be related to the low velocity weapons used. A thorough evaluation and documentation of facial nerve function should be conducted in the emergency department. Painful stimuli may be necessary to elicit facial grimacing in severely injured patients. Differentiating central from peripheral facial nerve palsy is essential. Although peripheral nerve injury is expected, central causes should not be overlooked. No patients in our series sustained central facial nerve palsy.

Gunshot wounds are always contaminated by infectious agents (Thorsby and Darlow, 1967; Dahlgram *et al.*, 1979; Tian *et al.*, 1988). Patients may present several months after the injury with otorrhea. One patient in our series presented one



FIG. 2 Bullet fragments in a mastoid cavity (see arrow).

year post-injury with chronic otorrhea. Treatment with an ototopical preparation is not likely to adequately control the drainage because retained bullet fragments will obstruct mastoid air cells and often cause a persistent foreign body reaction. Removal of the bullet is recommended to prevent infectious complications (Figure 2).

The extent of surgical exploration and reconstruction depends on the magnitude of injury which is often unknown. Damage may be limited to the tympanic membrane and middle ear cleft requiring tympanoplasty with ossicular reconstruction. Reconstructive efforts for middle-ear damage are withheld until appropriate debridement has been performed to prevent future infectious complications. Wide surgical exposure is necessary for full exploration and to relieve post-traumatic obstruction. This may necessitate radical mastoidectomy or cavity obliteration when extensive injury is present with functional loss.

Conclusion

Low velocity gunshot injuries to the temporal bone can be devastating and may result in functional sequelae. A multidisciplinary approach is necessary to optimize outcome. Once the patient is stabilized, CT scan evaluates intracranial as well as temporal bone damage. The mastoid and/or EAC portion of the temporal bone were the most commonly injured segments in our series. A thorough otological examination with audiometry is essential for diagnosis and treatment. Peripheral facial nerve injury occurred in 27 per cent and is best managed by early exploration. Carotid angiography is recommended for all patients since missed vascular injuries can lead to future sequelae. Removal of the bullet and bony fragments may prevent future infectious complications.

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LOW VELOCITY GUNSHOT INJURIES TO THE TEMPORAL BONE

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