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Lead toxicity due to retained intracranial bullet fragments: illustrative case

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BACKGROUND Lead toxicity (plumbism) secondary to retained lead bullet fragments is a rare complication in patients with gunshot wounds. To the authors' knowledge, there has been no definitive case reported of lead toxicity due to retained intracranial bullet fragments.

OBSERVATIONS The authors reported the case of a 23-year-old man who presented after being found down. Computed tomography scanning of the head revealed bullet fragments within the calvaria adjacent to the left transverse sinus. During follow-up, he developed symptoms of plumbism with paresthesias in his bilateral hands and thighs, abdominal cramping, labile mood, and intermittent psychosis. Plumbism was confirmed with sequentially elevated blood lead levels (BLLs). The patient opted for surgical removal of the bullet fragments, which led to reduction in BLLs and resolution of his symptoms.

LESSONS Although rare, lead toxicity from retained intracranial bullet fragments should be considered in patients who have suffered a gunshot wound to the head and have symptoms of lead toxicity with elevated BLLs. For safe and accessible intracranial bullet fragments in patients with plumbism, surgical intervention may be indicated.

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KEYWORDS plumbism; craniotomy; retained bullet fragments; lead toxicity

Firearm injury remains a significant public health disease burden in the United States,[1] with almost 40,000 fatal injuries reported in 2019 according the Centers For Disease Control and Prevention,[2] with an even higher number of individuals suffering nonfatal firearm-related injuries.[3] Many of these nonfatal firearm injuries result in retained bullet fragments (RBFs), for which removal may be indicated for various reasons, including retained bullets in joints and the globe, fragments causing nerve impingement, and cosmetic deformity.[4] Rarely, RBFs have been shown to be associated with lead levels sufficient to cause symptoms, a condition known as plumbism.[5]

Intracranial penetrating injuries can result in significant morbidity and mortality. Among persons who have survivable injuries, routine extraction of penetrating bone or missile fragments is not recommended[6] because less aggressive surgical management has been shown to have equivalent outcomes.[6–9] We present the first definitive documented case of lead toxicity due to intracranial bullet fragments and discuss the operative management and relevant literature.

Illustrative Case

A 23-year-old man was brought to our level 1 trauma center after being found down by police, who were alerted to the vicinity by the sound of gunshots. The patient recalled walking in the area yet claimed no recollection of the events thereafter. On primary survey, the patient was noted to have a small laceration to the left forehead and what appeared to be a gunshot wound to the left occiput. He had a Glasgow Coma Scale score of 15, with the only pertinent neurological finding being a right homonymous hemianopia. Noncontrasted computed tomography (CT) scanning of the head and cervical spine revealed an impacted comminuted left occipital skull fracture close to midline at the level of the transverse sinus with adjacent dispersed bullet fragments, a small associated left occipital intraparenchymal contusion, and a small focus of pneumocephalus. The patient's wound was superficially washed out and closed primarily. He was admitted to the neuroscience intensive care unit for close monitoring. He remained in a stable neurological condition and was discharged home after 5 days.

On follow-up in clinic 2 weeks after the incident, the patient's visual field cut had improved. He denied headaches but did report paresthesias in bilateral hands and thighs. His cervical spine CT was negative for pathology. Because of his symptoms and the nature of his injury, a venous lead level was obtained, which was

ABBREVIATION BLL = blood lead level; CSF = cerebrospinal fluid; CT = computed tomography; GSW = gunshot wound; RBF = retained bullet fragment.
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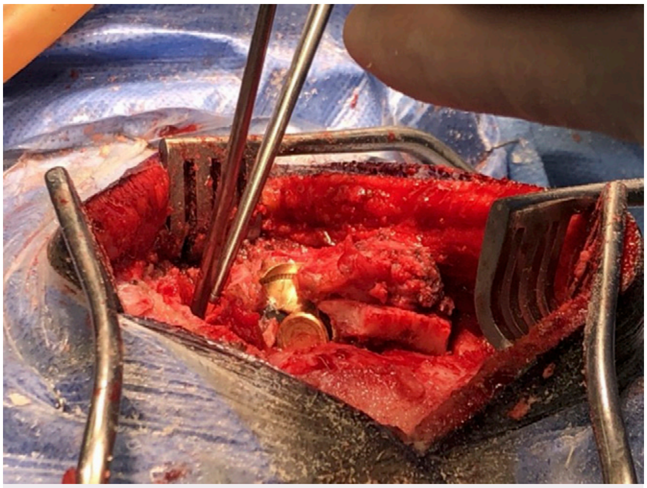

FIG. 1. Intraoperative photograph showing the contorted bullet casing impacted into bone.

FIG. 3. Extracted bullet mass with adjacent impacted bony fragments measuring approximately 4 × 3 × 3 cm.

mildly elevated to 5.1 μg/dL (normal 0.0–5.0 μg/dL) in the absence of any known home or occupational exposure. Over the proceeding weeks, his symptoms worsened with development of new abdominal cramping, diarrhea, and mood swings. His venous lead levels continued to trend upward to 8 μg/dL and then 10.2 μg/dL. Given the worsening symptoms in conjunction with rising lead levels, the patient was offered operative intervention to remove the bullet. Because of a streak artifact on CT and contraindication to magnetic resonance imaging due to the bullet fragments, catheter angiography was performed to investigate the patency of the left transverse sinus, which was found to be patent but with significantly reduced flow. The patient underwent left occipital craniectomy with removal of the bone fragments and retained bullet components. The occipital bone surrounding the bullet was fractured into multiple pieces (Figs. 1 and 2), and the dura was significantly thickened in the region. Dissection around the bullet and bone fragments was performed until the mass was extracted en bloc (Fig. 3). There was no intradural involvement from the bullet or any egress of CSF, and the transverse sinus was patent following bullet removal. This procedure was followed by an overlay of synthetic dural material to buttress the sinus (Fig. 4) and a cranioplasty with titanium mesh and methylmethacrylate cement. Repeat lead levels postoperatively showed an immediate reduction in value to 7.5 μg/dL. He was seen for follow-up approximately 2 weeks postoperatively, when he reported near complete resolution of his peripheral neuropathy, abdominal discomfort, and mood swings, which was sustained at his 2-month follow-up visit. A delayed lead level was planned 6 months postoperatively; however, by that time the patient was lost to follow-up.

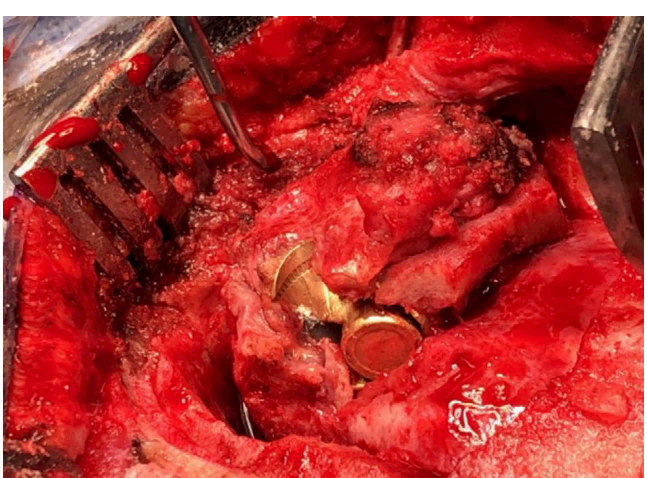

FIG. 2. Intraoperative photograph showing the bullet casing with surrounding comminuted fractures of the occipital bone and thickened dura.

Discussion

Observations

Firearm injury in the United States continues to be a significant source of trauma, with more than 350,000 deaths and more than 980,000 nonfatal injuries since 2010.[2] As projectile missiles encounter bony surfaces, they can fragment,[10] which can have multiple clinical implications. Fragmentation can cause increased local tissue destruction,[11] decrease the likelihood of complete extraction of the

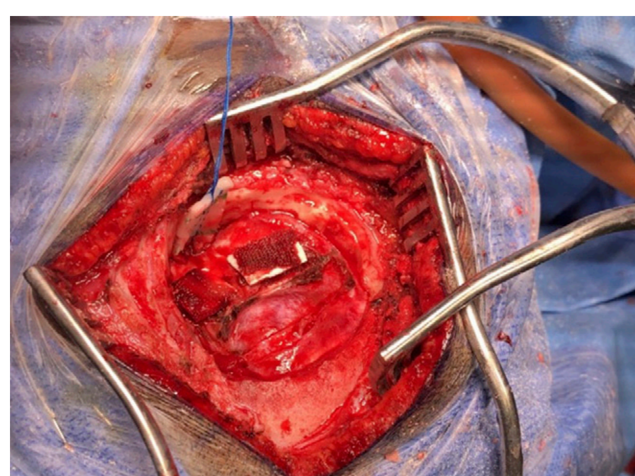

FIG. 4. Overlay of synthetic dural material to buttress the transverse sinus.

foreign bodies, and increase the total surface area exposed to internal tissue.[12,13] Firearm injuries to the brain are some of the most lethal, with approximately 90% of patients dying prior to arrival and up to 50% of surviving patients succumbing to their injuries in the emergency department.[14–16] More recent evidence suggests that with modern trauma recommendations, such as prioritizing blood products over crystalloid, survival rates for cranial gunshot wounds (GSW) are increasing. With increased survival rates, we therefore expect a concomitant rise in the number of patients with intracranial retained bullet fragments.

Lead, commonly found in bullets, is a chemical element (atomic number 82, symbol Pb) classified as a heavy metal that appears to have no innate function in the human body.[17] Lead can cause damage through ionic mimicry, disruption of intracellular calcium homeostasis, inhibition of nitric oxide synthases, production of oxidative stress, and alteration of gene transcription.[18] Lead toxicity, poisoning, or plumbism is the clinical manifestation of these pathological mechanisms and can lead to renal dysfunction, hypertension, gastrointestinal distress, neuropsychological effects, reduction in IQ, speech and hearing impairment, and infertility.[19] Initial symptoms can be vague and vary significantly, which can make diagnosis difficult. Clinical suspicion can be verified by blood lead levels (BLLs), with values >5 μg/dL considered to be above acceptable levels, although many health professionals advocate that there is no safe lead level.[20]

Systemic lead poisoning from retained bullet fragments has been described as far back at 1867;[21] since then, there have been more than 100 case reports citing an association between elevated BLL and RBF.[22] A recent meta-analysis by Apte et al.[5] showed a statistically significant elevation in BLL in patients with RBF across 12 studies, with independent predictors of elevated BLL being a higher number of RBFs and the presence of a bony fracture. Increased fragmentation, as previously mentioned, increases the surface area of exposure,[13] and bony fracture involvement is thought to involve osteoclast-mediated absorption of lead into the systemic circulation.[5,13,23,24] This meta-analysis did not show a relationship between RBF location and BLL, although prior studies[13] had. Conventional belief that fluid contact leads to increased systemic absorption continues to influence guidelines regarding removal of RBFs if they are in contact with joint spaces or the globe of the eye or in communication with cerebrospinal fluid (CSF).[4]

Lead poisoning from RBFs is not a novel concept in neurosurgery. GSWs to the spine account for 13% to 17% of all firearm injuries,[16,25] and RBFs in various locations within the spinal column have been linked to plumbism. RBFs lodged in intervertebral disc spaces have been reported to cause elevated BLL,[26–30] as the disc space is thought to contain a bursa-like fluid that can contribute to dissolution and absorption of metal particles.[29,30] Additionally, fragments that violate the spinal dura mater and are in contact with CSF have also been implicated.[31] It would therefore follow logically that intracranial RBFs in contact with fractured bony surfaces or CSF may cause lead toxicity. The authors of this report, however, have found only one such case described,[32] in which the exact location of the bullet fragment, whether intradural or extradural, is unclear. Additionally, the patient in that reported case had elevated BLL both prior to and following fragment extraction; thus, the causal relationship may be questioned.

Intracranial RBFs can cause a multitude of complications. Outside of the acute penetrating injury from the initial GSW, feared complications include abscess formation, both subacute[33,34] and delayed,[35,36] and migration of fragments[37–40] that can lead to new deficits. Additionally, patients with GSWs to the head almost ubiquitously suffer neurological sequelae from their initial injury, which may mask symptoms of lead toxicity. Parsing out neurological symptoms specifically due to lead toxicity or identifying extracranial symptoms such as abdominal pain and discomfort in a patient population with a high rate of cognitive deficits or aphasia may be difficult.

A definitive case of lead poisoning as a complication of an intracranial RBF has not been described in the literature until this report. In our patient, the RBFs were exclusively extradural; thus, a proposed mechanism involving CSF degradation and absorption of the foreign lead material would be excluded. Rather, in this scenario, we believe the mechanism of lead absorption was related to the position of the bullet fragments in relation to the transverse sinus. Blood flow through the confluence of sinuses has known patterns of anatomical variance, with an estimated 300 to 400 mL of venous blood passing through the transverse sinus per minute.[41,42] The large volume of blood passing directly over the site of lead fragment lodging, coupled with the increased surface area from multiple bullet fragments, and the bony fragmentation inducing osteoclast-mediated resorption, all likely played a role in the increased systemic lead absorption in our patient. Because of worsening symptoms and rising BLL, surgical intervention was offered and pursued by our patient, which led to resolution of his symptoms and an objective drop in his BLL.

Limitations to this case report include the fact that although most bullets are composed of a metal jacket with a lead core, some specialty bullets are not composed of lead. The core of the bullet in this case was not analyzed to confirm the presence of lead. Given the rarity of nonlead bullets in common ammunition, this analysis was not pursued further.

Lessons

Intracranial GSWs remain a significant etiology of presentation in trauma centers within the United States. As prehospital and emergency department interventions continue to improve, leading to increased survival in patients with GSWs to the head, prevalence of patients with retained intracranial bullet fragments will continue to increase. Neurological sequelae are common in this patient population, and symptoms of lead toxicity, which can be vague and difficult to identify, may be easily overlooked. Patients with retained intracranial bullet fragments are at risk of lead toxicity, and it is reasonable to consider monitoring BLLs in select patients who may be symptomatic. In cases of elevated BLLs and retained intracranial bullet fragments that are easily accessible, neurosurgical intervention may be warranted.

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Disclosures

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

Author Contributions

Conception and design: Hedayat. Acquisition of data: Hedayat. Analysis and interpretation of data: Hedayat. Drafting the article: Aaronson, Awad. Critically revising the article: all authors. Reviewed submitted version of manuscript: all authors. Approved the final version of the manuscript on behalf of all authors: Aaronson. Administrative/technical/material support: Hedayat. Study supervision: Hedayat. Primary surgeon: Hedayat.

Supplemental Information

Previous Presentations

Portions of this paper were presented virtually as an abstract at the International Brain Injury Associated Virtual World Congress, July 28, 2021.

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