Dear Elizabeth,

Interesting question.

It is difficult to make any conclusion on the three documents supplied by Peter Corris. You intimate in your email that the fragments are from a bullet, but the documents say:

- “No point of entrance is to be determined.”
- “three foreign bodies of high specific gravity and presumably metal.” (and presumably lead) “Immediately below these are finer pieces of little significance”.
- These foreign bodies are located:
  1. “lying in the outer table of bone immediately laterally to the superior mastoic air cells”;
  2. “lying anterosuperiorly to the auditory meatus and close to the skull wall but extracranially”; and
  3. “lying deeply placed in the bone and apparently close to the internal acoustic meatus”.

While this anatomical exactness is commendable, the most important comment is the last one: “There is no evidence to suggest that any part of the foreign body is intracranial”, indicating these particles did not pass through the skull and penetrate the brain.

- The size of these foreign bodies are:
  1. “maximum measurement of 4 mm”;
  2. “maximum transverse measurement is 6 mm”; and
  3. “its measurements are 8 mm by 3.5 mm and situated 25 mm from the skin surface”.

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Regarding: Request for Information from Peter Corris on Toxicity of lead from bullets
These relatively large particles could contribute lead to the body, although lead dissolves slowly in body fluids, and if there were enough of them, the contribution of the “finer pieces of little significance” might make a larger contribution by virtue of their relative surface area to mass ratio. However, no information is present on how long they were present, and for long term toxicity from a source, I would suggest years, rather than anything shorter. It is probable that any person who was to show lead related effects from such an exposure would show at least some conventional signs of lead poisoning. These could include: gastrointestinal symptoms (such as colic, constipation, abdominal problems), neurological (such as depression, poor concentration, loss of short term memory, numbness/tingling, sleep problems), blood forming tissues (anaemia) and general problems (headaches, fatigue).

My take on this is:

1. Lead toxicity from bullets is rare, mainly because lead dissolves slowly in body fluids.
2. If lead fragments from bullets can get to a place in the body where they are in contact with body fluids, and they break down into smaller fragments (or are already in small fragments), then lead can leach out of the fragments and, if released in large enough amounts, can produce toxicity.

I attach some abstracts from articles on this matter.

Hope this helps.

Regards

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3 August, 2010
Dear Chris,

I wonder if you can help me with the following inquiry which comes from one of Australia's most famous crime writers, Peter Corris who was advised to contact me by another client, Evan Whitton, also a writer.

I sent this to Carol Bodle and she thought you might be a good person to comment on Peter’s question in relation to the two attached reports (in both jpg and doc formats).

I think the key question really is: is it likely that either the bullet fragments OR lead poisoning from the bullet fragments (or from shooting if he practised a lot) could have affected the executed man's behaviour?

If you can comment or locate another expert who can comment, could you please email your or his/her comments to me so that I can forward them to Peter, and also to let us know whether your or his/her name can be stated and comments can be quoted, or just the comments quoted by Peter in the book he is writing.

Thanks very much for any help you can give.

Much appreciated

Elizabeth
Yours Sincerely
Elizabeth O’Brien, Manager,
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www.lead.org.au
Dr. Paul Tillett.

Hengrove Hall,
193 Macquarie St,
Sydney
28th May, 1932.

Patient. William Cyril Moxley.

Examination made of head At The Coast Hospital on 25th May, 1932.

In the petrous area of the right temporal bone, there are three foreign bodies of high specific gravity and presumably metal. Immediately below these are finer pieces of little significance.

The piece lying most posteriorly is situated in the upper part of mastoid bone with a maximum measurement of 4 mm. Anteriorly and slightly inferiorly the second piece is lying deep in the osseus portion of auditory canal or in the middle ear. Its maximum transverse measurement is 6 mm.

The third piece is lying 17 mm. above and anteriorly about 60 degrees from the horizontal. Its measurements are 8 mm by 3.5 mm and situated 26 mm from the skin surface. This places the fragment immediately above the anterior superior border of the petrous portion of the temporal bone and abutting on the inferior temporal gyrus of the brain.

No point of entrance is to be determined.

This examination was made stereoscopically and the localisation made by a double tube shift on flat projection.

The antero-posterior and postero-anterior projections reveal no further abnormality in the skull and confirm the position of the third piece of foreign body.

Sgd. Paul Tillett.

This is the annexure marked "K" referred to in the affidavit of Charles Edmund Godwin sworn before me this eighth day of July in the year one thousand, nine hundred and thirty-two.

"M"

Dr. Paul Tillett.

Hengrove Hall,
193 Macquarie St,
Sydney
7th July, 1932.

Amended Report William Cyril Moxley.

X-Ray examination made at The Coast Hospital on 7th July, 1932.

In the right petrous area of the temporal bone are three foreign bodies of high specific gravity and evidently metal.

Finer particles are immediately below but of little significance lying extra-cranially.

Of the three larger pieces one is lying in the outer table of bone immediately laterally to the superior mastoid air cells.

The second piece is lying anterosuperiorly to the auditory meatus and close to the skull wall but extra-cranially. The third piece is lying deeply placed in the bone and apparently close to the internal acoustic meatus. There is no evidence to suggest that any part of the foreign body is intra-cranial.

Sgd. Paul Tillett.

This is the annexure marked "M" referred to in the affidavit of Charles Edmund Godwin sworn before me this eighth day of July in the year one thousand, nine hundred and thirty-two.


A man was hospitalized on three occasions for symptoms of lead intoxication 20 to 25 years after a gunshot wound that resulted in retention of a lead bullet in his hip joint. The potential for lead toxicity as a complication of a lead missile injury appears to be related to (1) the surface area of lead exposed for dissolution, (2) the location of the lead projectile, and (3) the length of time during which body tissues are exposed to absorbable lead. Cases of lead poisoning of immediate onset resulting from lead shot have been reported in Europe, but all documented cases of ammunition-related plumbism reported in the United States have involved synovial fluid dissolution of a single lead bullet over many years. The solvent characteristics of synovial fluid and associated local arthritis are apparently important factors in the dissolution and absorption of lead from projectiles located in joints. Awareness that lead intoxication can be a complication of retained lead projectiles should allow rapid institution of appropriate diagnostic and therapeutic modalities when such a clinical situation arises.


A 26-year-old man developed a movement disorder characterised by bradyphrenia, bradykinesia, rigidity, tremor and dystonia, several years after having been shot by a gun in the hip. Laboratory investigations revealed anaemia and porphyria. The authors demonstrate that his neurological condition was a delayed manifestation of lead toxicity, caused by slow absorption of lead from persisting bullet fragments in the hip joint. Treatment with excision of the femoral head and debridement of the hip followed by a total hip, in combination with chelating therapy, led to a remarkable remission.


In the past 3 decades, lead levels in North American children have been declining. Despite the decline in lead exposure, lead toxicity remains a significant childhood environmental health hazard. The usual route of lead exposure is through ingestion, but lead toxicity secondary to retained bullet fragments has been well documented in the adult literature. The diagnosis of lead toxicity is often difficult and delayed secondary to vague and transient symptoms. Recognizing high-risk characteristics of bullet fragments can improve clinician awareness to the possibility of lead toxicity. The primary management of patients with continued lead exposure is to remove the source of exposure. However, in the case of retained bullet fragments, initiation of chelation therapy before surgical removal may be essential in preventing systemic toxicity. We present the case of a 14-year-old female with lead toxicity who presented with an 18-month course of chronic abdominal pain, vomiting, and anorexia 2 years after sustaining a gunshot wound to the right leg. The patient was treated with oral succimer and operative removal of bullet fragments.


Our objective is to present a case of symptomatic lead toxicity (plumbism) with abdominal colic and hemolytic anemia following a gunshot wound. It is a retrospective case report and the setting is in a teaching hospital in south central Los Angeles. The case report is that of a patient who presented with abdominal pain, generalized weakness, and hypertension following multiple gunshot wounds, 15 years previously. Other causes of abdominal pain and weakness--such as diabetes mellitus, alcohol abuse, pancreatitis, and substance abuse--were ruled out. Interventions included treatment with the newer oral chelating agent, Succimer (2, 3-dimercaptosuccinic acid), and subsequent surgery. The main outcome was the initial reduction in blood lead levels with improvement of symptoms. Because of a recurrent rise in the blood lead levels, the patient was again treated with Succimer and underwent surgery to remove two bullet fragments from the face. We conclude that lead toxicity should be ruled out in patients presenting with abdominal cramps and a history of a gunshot wound. Prompt therapy--including environmental intervention and chelation therapy--is mandatory, and surgical intervention may be necessary.


Gunshot wounds to the spine are becoming more prevalent, especially in urban areas. Neurologic injury can occur even if the projectile does not penetrate the spinal canal. Initial treatment should consist of stabilizing the patient's medical condition followed by a careful neurologic examination. Spinal stability can be determined from plain radiographs and CT scan, with the overwhelming majority of injuries being stable. Retained bullets rarely cause problems of delayed infection, late neurologic decline, or lead toxicity, eliminating the need for prophylactic bullet removal; however, in the thoracolumbar spine, removal of bullet fragments lodged within the spinal canal has been shown to significantly improve neurologic recovery.


Lead poisoning is an unusual complication of gunshot wounds that occurs when retained lead bullet fragments are in contact with body fluids capable of solubilizing lead. The epidemic of violence by gunfire may result in increasing numbers of lead poisoning cases from this exposure. The use of oral chelation for toxicity resulting from this mode of exposure has not been previously discussed. Cases of lead poisoning arising from bullet lead in the synovial cavity of the hip, synovial cavity of the chest, and pleural space are reported. A combination of surgical debridement and chelation therapy with oral succimer produced a satisfactory outcome in all three cases. Oral succimer may be a safe and effective chelation agent for treating lead toxicity in adults with high lead levels secondary to gun shot wounds.

Lead poisoning is a common disease that, if not detected, can lead to developmental delay and other serious sequelae. We report the case of a child with retained intracranial lead pellets from a gunshot injury, in whom elevated blood lead levels were detected approximately 1 year after the injury. No environmental source of lead was found, and a twin sister living in the same dwelling had considerably lower lead levels. The patient's lead levels diminished after each of four courses of chelation, but rebounded each time to potentially toxic levels after termination of therapy. Physicians should be particularly alert in screening for elevated lead levels in children with retained bullet fragments. In patients in whom removal of the bullet fragments is impractical, the potential risks of long-term chelation therapy must be weighed against the risks of lead toxicity.


Bullet wounds causing lead synovitis in the wrist and knee are reported in two patients, one of whom also developed clinical plumbism. Very high lead levels in the synovial fluid are believed to be responsible for toxicity changes that occurred in the synovium and bone. Ultrastructurally, these alterations included the formation of nuclear lead inclusions, dilation, and degranulation of the rough endoplasmic reticulum and deposition of crystalline precipitates in the matrix of the mitochondria in macrophages, osteoclasts, and synovocytes, as well as the development of cytoplasmic lead inclusions in osteoclasts. Energy-dispersive x-ray elemental analysis (EDXEA) indicated that the nuclear inclusions contained only lead, whereas precipitates within the mitochondria and elsewhere in the cytoplasm were composed of complexes containing lead, calcium, and phosphorus. Similarly constituted extracellular complexes were incorporated into newly formed trabecular bone laid down as a physiologic response to the bullet lodged within the wrist bones. This bone subsequently exhibited defects in bone resorption, which were characterized by depressed osteoclastic function and a unique lesion termed incomplete osteocytic osteolysis. The genesis of this latter lesion is uncertain. The sequestration of the partially degraded bone fragments containing lead complexes into the marrow and eventually into the joint spaces and synovium permitted the recycling of bone lead, and this may have played an important role in inducing clinical plumbism in one of the patients in this study.