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CLINICAL CASE

# Inflammatory low back pain with radiculopathy revealing epidural disintegration of a bullet complicated by lead poisoning

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## ABSTRACT

Inflammatory low back pain with radiculopathy is suggestive of cancer, infection or inflammatory diseases. We report a unique case of a 42-year-old patient with an acute inflammatory low back pain with bilateral radiculopathy associated with weight loss and abdominal pain, revealing the disintegration of a lead bullet along the epidural space and the S1 nerve root complicated by lead poisoning. Because of the high blood lead level of intoxication (>10 times over the usual lead levels) and the failure of repeated lead chelator cycles, a surgical treatment to remove bullet fragments was performed. It resulted in a significant decrease of pain and lead intoxication.

### WHAT IS ALREADY KNOWN ON THIS TOPIC

⇒ Gunshot with retained bullets or fragments (RBF) can cause systemic lead poisoning.
⇒ The clinical presentation of systemic poisoning is rich and non-specific including asthenia, anorexia and abdominal pain.
⇒ When lead intoxication is suspected, blood lead level (BLL) have to be monitored. With BLL>100 µg/dL, risks of severe encephalopathy, cytolytic liver disease and even death are significant. Chelation is formally indicated if BLL>100 µg/dL.

### WHAT THIS STUDY ADDS

⇒ Bullets can disintegrate.
⇒ Depending on the localisation of the RBF, local complication also exist,such as radiculopathy because of an epidural localisation in this case.
⇒ Bullet removal can decrease the blood lead level.

### HOW THIS STUDY MIGHT AFFECT RESEARCH, PRACTICE OR POLICY

⇒ To assess local and systemic lead poisoning in the event of a gunshot
⇒ In failure of chelation, surgical treatment with bullet removal have to be discussed.

## REPORT

A 42-year-old man was seen in the rheumatology department for an inflammatory low back pain with radiculopathy.

Two weeks earlier, this patient suffered from an acute inflammatory low back pain with abdominal pain. He has a history of a mild spondyloarthritis, confirmed by the presence of a sacroillitis on a CT scan, diagnosed 7 years ago, never requiring analgesics or non-steroidal anti-inflammatory drugs (NSAIDs), and a 20-year history of abdominal trauma by gunshot, with perforation of the inferior vena cava. He was treated by venoplasty and the bullet had never been removed. His motivation for visiting the hospital was a feverish feeling with a generalised weakness and the loss of 4 kg in 1 month. Temperature was 36.7°C, abdomen was tender and painful. Low back pain was not induced by lumbar palpation and neurological examination was normal. Laboratory results were notable for an inflammatory syndrome with hyperleucocytosis at 11 980 per µL including 8750 per µL absolute neutrophil count and C reactive protein (CRP) level at 8.5 mg/dL. Lumbar CT scan was not in favour of any spine or abdominal infection, but showed a hyperdense foreign body material in the L4/L5 disk space (figure 1A). The patient went home with analgesic treatments.

A few days later, a radicular pain with paresthesia appeared in both legs along with an increased abdominal and low back pain. Analgesic treatments were ineffective. He consulted again at the emergency department and was hospitalised in the rheumatology department. On examination, there was still no fever. The blood pressure was 110/70 mm Hg, the heart rate was 57 bpm and the oxygen saturation was 100%. The

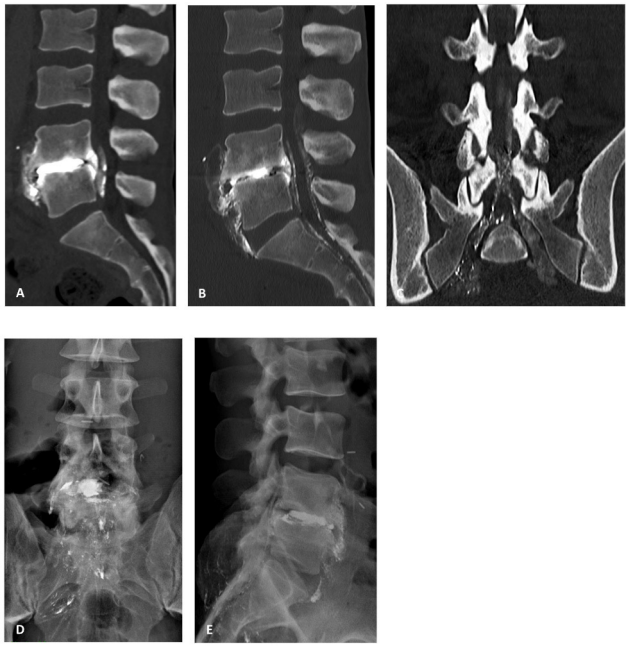


**Figure 1** Lumbar CT scan and lumbar X-ray of the patient revealing a disintegration of the hyperdense material along the epidural space and right nerve S1. (A) First lumbar CT scan, sagittal section with hyperdense material in L4/L5 intervertebral disc with minimal posterior flow. (B) Second lumbar CT scan, 2 weeks after the first CT scan, sagittal section with hyperdense material with flow in the anterior and posterior epidural space. (C) Second lumbar CT scan, 2 weeks after the first CT scan, coronal section with hyperdense material with in the epidural space and among nerve roots. (D) Face X-ray, 2 weeks after the first CT scan, hyperdense material with in the epidural space and among nerve roots. (E) Profile X-ray, 2 weeks after the first CT scan, posterior migration of the hyperdense materiel through the sacral foramina into the paraspinal soft tissue, and into the anterior paravertebral space.

rheumatological examination revealed no deformation but a loss of anterior and lateral flexion to the mobilisation with pain at the lumbar spine level. Neurological examination did not reveal any motor deficit or sphincter disorders. Osteotendinous reflexes were symmetrical and non-pathological. The abdominal area was tender as a whole, without any defence or contracture. The cardiovascular examination was normal. The lymph node areas were clear. Laboratory results showed a persistence of the inflammatory syndrome with hyperleukocytosis at 11 600 per µL and CRP level at 11.1 mg/dL. Blood and urinary cultures were sterile. Since radiculopathy appeared and CRP level increased, a second CT scan and a lumbar X-ray were performed and revealed a disintegration of the metallic material along the epidural space and right nerve S1 (figure 1B–E). There was no sign of spondylodiscitis or enthesitis. MRI was contraindicated because of the bullet history. Thoracic-abdominal-pelvic CT scan did not reveal infectious or neoplastic lesions.

Subsequent recovered operative reports and a 7-year-old CT scan confirmed that no metallic foreign material has been placed, including no cement, in the

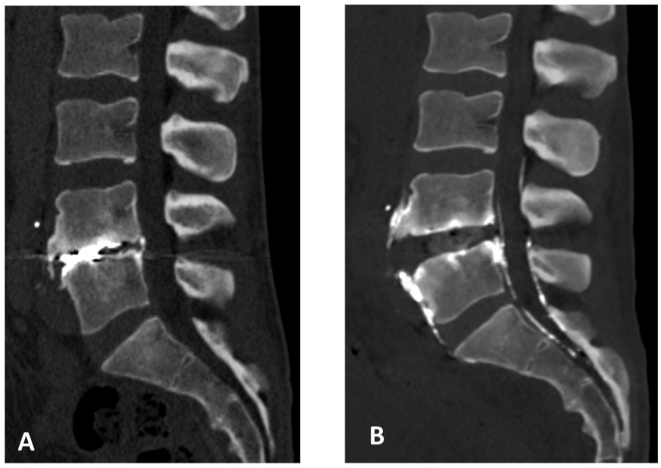


**Figure 2** Lumbar CT scan before material disintegration and after surgery. (A) 7 years before lumbar CT scan: sagittal section with well limited hyperdense material corresponding to the bullet. (B) Lumbar CT scan after surgery, sagittal section with removal of hyperdense materiel and sequelae of disintegration in the epidural space.

L4/L5 disk space, and that the radiological findings described above were in fact consistent with the disintegration of the lead projectile that had been left in place, 20 years before (figure 2A). Because lead intoxication was suspected, blood lead level (BLL) was measured from a blood sample with an inductively coupled plasma mass spectrometry technique. BLL was at 119.4 µg/dL (95th percentile in the French adult population: 5 µg/dL) with zinc protoprophyrin (ZPP) at 82 µg/g haemoglobin (Hb) (N<3), indicating significant lead poisoning, from at least 2–4 months.

The patient was transferred to a poison control centre to assess complications of lead poisoning. Blood analysis revealed BLL at 119.7 µg/dL, ZPP at 74 µg/g Hb, 876 µg/5 hours (<600), 6.06 µg/5 hours/EDTA $Na_2Ca$ (<0.6), Hb at 101 g/L, mean corpuscular volume at 83 $\mu m^3$, clearance at 78.4 mL/mn, alanine and aspartate transaminases, respectively, at 53 U/L (N<35) and 34 U/L (N<34), gamma-glutamyltransferase at 34 U/L (N<64), bilirubin at 12 µmol/L, ferritin at 92.7 µg/L and uric acid at 386 µmol/L (N 202–417). Psychometric tests and somatosensory evoked potentials did not show any impairment attributable to lead poisoning. Electromyogram of the lower limbs showed L5–S1 root damage.

The patient received a chelating treatment which consisted in a daily 1-hour infusion of 1 g of calcicodisodium EDTA (EDTA $Na_2Ca$) for five consecutive days. The decrease in BLL was significant but insufficient (from 117.9 to 97.1 µg/dL), forcing the repetition of lead chelator cycles every month (figure 3). Abdominal, lumbar and radicular pain persisted but CRP normalised. It was decided to surgically remove the lead bullet fragments in order to stop the lead systemic diffusion and to decrease the pain. A discectomy with an L4/L5 arthrodesis and bone-grafting was performed by an anterior approach.

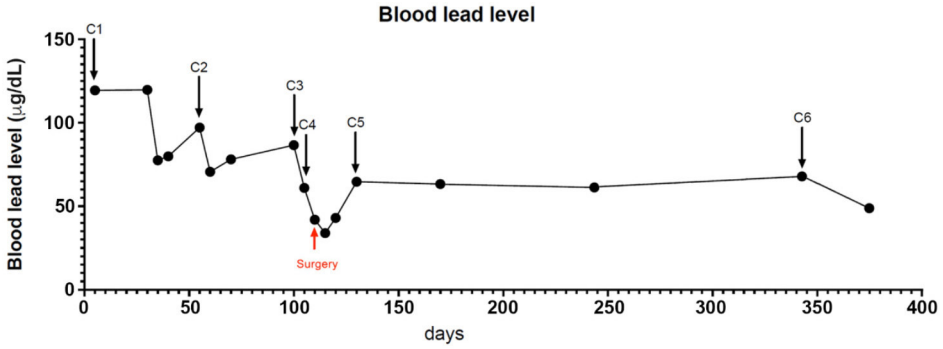


**Figure 3** Blood lead level among days. C1, first cycle; C2, second cycle; C3, third cycle; C4, fourth cycle; C5, fifth cycle; C6, sixth cycle.

Postsurgical lumbar CT scan confirmed that the majority of the disintegrated lead bullet was removed with some remaining fragments in the epidural space (figure 2B). Low back pain and radiculopathy improved along with the abdominal pain. Postoperative lead levels after surgery significantly decreased but remained above the nontoxic standard (figure 3). Postoperative follow-up was complicated by a lymphoedema caused by an intraoperative lymph wound, which was surgically closed, with a persistent lymphoedema of the left leg.

## DISCUSSION

We describe here an unusual case of local and systemic lead poisoning due to retained bullets or fragments (RBF). The proportion of patients with RBF having elevated blood levels is unknown but RBF accounts for approximately 10% of patients with BLL>250 µg/L.[1] Data regarding RBF duration and BLL elevation are conflicting, with most studies describing an increase within the first 3 months associated with a progressive decrease after 6 months or 1 year.[2 3] This early elevation of BLL after gunshot wound is explained, for some authors, by the initial resorption of the RBF leading to inflammation and the formation of a fibrous shell.[4 5]

Variation of BLL depends on location. Most cases of lead poisoning due to gunshot wounds result from particular locations of the projectiles in contact with body fluids: mainly synovial fluid, but sometimes bursa, cerebrospinal fluid and eye or fracture lesions.[1 6]

Lead arthropathy, caused by RBF lodged inside a joint, is suspected to result from biomechanical forces and permanent contact with synovial fluid (rich in hyaluronic acid) expose to greater risk of lead dissolution, inflammation and diffusion to adjacent tissues and the bloodstream.[7–10]

The diagnosis of systemic lead poisoning is complex since the clinical presentation can be very rich, nonspecific, given many possible systemic effects. General signs may include asthenia, malaise, neurological, gastrointestinal, rheumatological or renal signs.[4] No linear correlation has been found between the severity of symptoms and BLL.[1]

Although lead intoxication caused by an intra-articular bullet is well described, only 12 cases of bullet remaining in an intervertebral disc are described in overview literature.[11] Moreover, only four cases of intradiscal bullet responsible for a lead poisoning have been described to this date.[11–14] Here, we report the first case with associated radiculoneuritis.

The distinctive feature of this patient is that he presented symptoms related to a systemic lead intoxication (asthenia, anorexia, abdominal pain, nausea and anaemia) but also inflammatory sciatica related to the local disintegration of the bullet, in the epidural space and along the S1 nerve root. The delay of lead poisoning is also singular and revealing of the supposed underlying pathophysiology because the patient was asymptomatic for 20 years as long as the bullet was located in the intervertebral disc, therefore an avascular and fibrocartilaginous space.[13] Even if we cannot formally rule out a role played by the spondyloarthritis diagnosed 7 years earlier, the fact that it never required analgesic or anti-inflammatory medication, and that the nature of the symptoms was very different from those described in this pathology, makes us highly doubtful of a possible role played by this disease in this case.

The intoxication began when the bullet spontaneously disintegrated in the epidural space, as shown by the successive scans confirming its progressive disintegration. The extension to the epidural space being highly vascularised could explain blood intoxication and regional radiculoneuritis.

How the bullet lodged in the intervertebral disc spontaneously disintegrated is unclear. No history of uncommon mechanical or metabolic stress was found, and this could simply result from repetitive motion and frictional forces leading to its dissolution.

With BLL>100 µg/dL, risks of severe encephalopathy, cytolytic liver disease and even death are significant. Chelation is formally indicated if BLL>100 µg/dL.[15] Chelating agents mostly used for lead poisoning are calcicodisodium EDTA (EDTA $Na_2Ca$) or dimercaptosuccinique acid (DMSA). In cases where medical treatment does not allow a persisting decrease of BLL, removal

of the bullet or bullet fragments may be warranted.[14 16] As lead intoxication depends on the localisation of the bullet and as postoperative intoxication and complications are not rare, it is still debated whether fragments should be removed, followed or ignored.[17 18] In 2012, European guidelines were published for trauma surgeons on the indication of bullet removal. They proposed indications for bullet removal, according to the localisation of the bullet (joints, cerebrospinal fluid, eye), to its impact on roots, nerve, vessel and finally to lead poisoning.[19]

The surgical intervention for its extraction may result in a transient but often large increase in lead release that could lead to serious neurological complications, when the preoperative BLLs are too high.[7] BLLs have to be carefully monitored before and after surgery, since rebound may appear.

If concomitant removal of our patient's RBF should have been performed in the past, local and general complication may have been avoided. Lead intoxication symptoms and lead levels improved after surgery but remained above the nontoxic standard. That could be explained by a redistribution from the lead flow along the anterior longitudinal ligament that could not be removed. Its persistence for several weeks is also explained by redistribution of lead from bone which represents more than 90% of lead body burden. Indeed, preoperative chelation sharply decreased lead levels in blood and soft tissues but only moderately decreased bone lead pool.

In conclusion, we described the first case of a spontaneous disintegration of a lead bullet from the intervertebral disc to the epidural space, responsible of local and systemic lead toxicity.

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