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A Rare Case of Intravenous Mercury Poisoning in a 35 Year Old Male

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Abstract

The only metal that is liquid at ambient temperature is mercury. It is usually employed in the production of sphygmomanometers, thermometers, and switches. Mercury poisoning, often called mercurialism or hydrargyria, can result from exposure to mercury. Exposure may occur through injection, ingestion, or inhalation. In this report, we describe effects of mercury poisoning in a 35 year old male, nurse by occupation, who presented with an alleged history of mercury poisoning following injecting large amount of mercury intravenously after breaking through a sphygmomanometer in a rural primary health care centre. Investigations revealed diffuse metallic depositions in both of his lung fields. The patient was treated with chelation therapy and other supportive management for which he responded well and was eventually treated and discharged. While hydrargyrum has been extensively documented, reports of hydrargyrum following an intravenous dose are very rare, like in our case. Furthermore, it underscores the challenges associated with managing mercury poisoning, including the limited efficacy and availability of chelation therapy in rural set ups with cases of severe toxicity. Further research is needed to elucidate optimal treatment strategies and improve outcomes in patients with mercury poisoning.

Keywords: Hydrargyria, Intravenous, Mercurialism, Metallic Deposits, Mercury Poisoning

1. Introduction

Mercury poisoning is a rare but potentially life-threatening condition that can result from various forms of exposure, including inhalation, ingestion, and in exceptional cases, intravenous administration. Mercury, a ubiquitous heavy metal, exists in multiple forms, each with its own toxicological profile. While industrial and environmental sources remain the primary routes of exposure, cases of intentional or accidental mercury injection have been rarely documented, posing unique diagnostic and management challenges to healthcare providers[1].

The prevalence and incidence of mercury poisoning vary depending on factors such as geographic location, occupational exposure, dietary habits, and regulatory measures in place[2]. In general, acute cases of mercury poisoning are relatively rare but can occur in industrial settings where mercury is used or in incidents of chemical spills or contamination[2].

Through this case study, we highlight the importance of prompt recognition and intervention in cases of mercury toxicity to mitigate potential complications and improve patient outcomes. Additionally, we discuss the limitations of current treatment modalities and the need

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for further research to optimise management strategies for mercury poisoning.

By elucidating the clinical course of this rare presentation of mercury poisoning, we hope to contribute to the body of literature on heavy metal toxicity and enhance awareness among healthcare providers regarding the diverse manifestations of mercury exposure.

2. Case Presentation

A 35 year old male, nurse by occupation at a rural primary health care, was brought by his family to the emergency department with alleged history of mercury poisoning after injecting a large amount of mercury after breaking through a sphygmomanometer at his place of work, at a primary health care hospital, 1 day ago around 11 am in the morning. He was a known case of depressive disorder and had history of suicidal tendencies in the past. After injecting the mercury, he did not reveal the act to anyone and remained silent. Six hours after ingestion he started to have a severe headache and palpitations. By night he started to experience *dyspnea* and a non productive cough. Breathlessness increased in intensity which was followed by *orthopnea* and by morning head grade IV dyspnea and came to this hospital.

On general examination, patient's condition was poor with severe restlessness and a pulse rate of 130 beats per minute, respiratory rate of 35 cycles per minute associated with use of accessory muscles of respiration, 100/60 mm of hg blood pressure in his right arm in supine position and an oxygen saturation of 86% on room air.

On respiratory system examination, coarse crepitations were heard over both of his lung fields involving more than 2/3rd areas with a decreased air entry bilaterally over basal areas. Bronchial breath sounds were heard over bilateral lung fields with severe bronchospasm. While other systemic examinations were unremarkable.

2.1 Results

An emergency chest x-ray in sitting position was performed bedside which revealed diffuse homogeneous opacities irregularly appearing in bilateral lung fields suggesting diffuse metallic deposition, as shown in Figure 1.

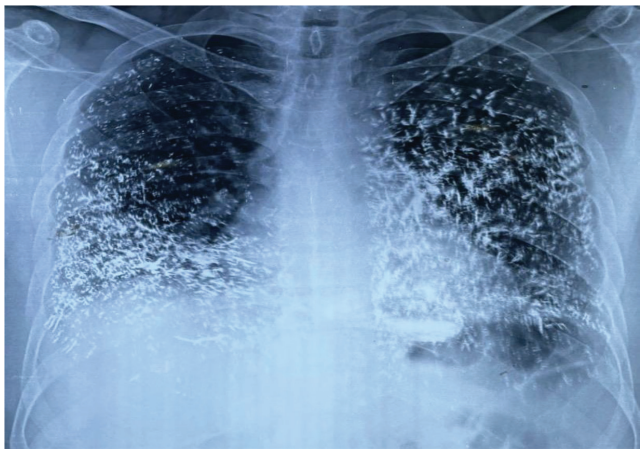


Figure 1. Chest x ray in anterior-posterior view suggestive of diffuse metallic depositions.

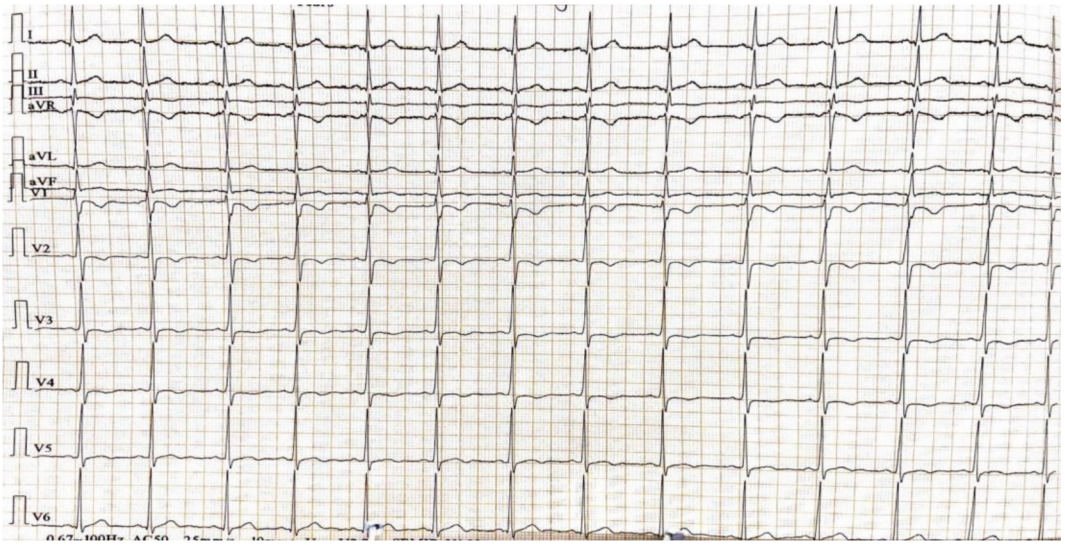


Figure 2. Electrocardiograph suggestive of normal sinus rhythm with non specific T wave inversions in leads in V1-V4.

Electrocardiogram of the patient was done which was suggestive of sinus rhythm with non specific T wave inversions in leads V1-V4 as shown in Figure 2.

All routine blood investigations were done which were suggestive of sepsis with coagulopathy and deranged kidney and liver function tests, raised troponin I levels done on the second day of admission along with significant presence of serum and urine mercury levels, as shown in Table 1.

In view of tachycardia, tachypnea, hypoxia and severe respiratory acidosis with metabolic acidosis on Arterial Blood Gas (ABG), patient was immediately started in non invasive positive pressure ventilation and shifted to ICU Serum mercury levels and urine mercury levels were raised which was suggestive of acute mercury toxicity. High Resolution Computed Tomography (HRCT) of thorax was done which showed scattered high attenuation foci in bilateral lung fields with tiny ground glass opacities, as shown in Figure 3.

A bedside target 2 Dimensional (2D) echocardiography was done for the patient to reveal infiltration of right ventricular myocardium with metallic deposits with an ejection fraction of 50% as explained in Figure 4.

On further investigating, x-ray erect anterior-posterior view of abdomen revealed high attenuated foci suggestive of metallic deposits in bilateral iliac fossa, suggestive of metallic deposits in both renal systems with deposits in pubic fossa, as shown in Figure 5.

2.2 Management

The patient was immediately started on chelation therapy considering severe mercury toxicity with multisystem involvement. Chelation therapy with injection dimercaprol 5mg/kg (350mg in our case) intramuscular stat was given on the first day followed by 150 mg intramuscular twice daily for next 11 days along with tablet selenite sodium (500mcg per day) and zinc supplements. During the hospital stay, patient was also started on higher antibiotics like injection piperacillin-tazobactam 4.45gm intravenous stat followed by 2.25gm intravenous thrice a day along with injection levofloxacin and injection clindamycin on renal in renal doses. The patient was also started on injection vitamin K 10mg intravenous once a day for 3 days with injection n-acetylcysteine

Table 1. All routine blood investigation results of the patient

Blood Investigation	Value/Result	Normal Values
Complete Blood Count (CBC)	WBC: 18,000 cells/µL; RBC: 4.5 million cells/µL; Hb: 10 g/dL; Hct: 30%; Platelets: 80,000 cells/µL	WBC: 4,000-11,000 cells/µL; RBC: 4.2-5.4 million cells/µL; Hb: 12-16 g/dL; Hct: 37-47%; Platelets: 150,000-400,000 cells/µL
Blood Culture	Positive for Staphylococcus aureus	No growth expected
Basic Metabolic Panel (BMP)	Sodium: 138 mmol/L; Potassium: 3.0 mmol/L; Chloride: 105 mmol/L; Creatinine: 2.0 mg/dL; BUN: 40 mg/dL	Sodium: 135-145 mmol/L; Potassium: 3.5-5.0 mmol/L; Chloride: 98-106 mmol/L; Creatinine: 0.6-1.2 mg/dL; BUN: 6-20 mg/dL
Liver Function Tests (LFTs)	AST: 120 U/L; ALT: 100 U/L; ALP: 80 U/L; Bilirubin: 2.5 mg/dL	AST: 10-40 U/L; ALT: 7-56 U/L; ALP: 44-147 U/L; Bilirubin: 0.1-1.2 mg/dL
Coagulation Profile	PT: 18 seconds; INR: 1.5; PTT: 40 seconds	PT: 11-13 seconds; INR: 0.8-1.2; PTT: 25-35 seconds
Arterial Blood Gas (ABG)	pH: 7.25; PaO2: 60 mmHg; PaCO2: 50 mmHg; HCO3-: 20 mmol/L	pH: 7.35-7.45; PaO2: 75-100 mmHg; PaCO2: 35-45 mmHg; HCO3-: 22-26 mmol/L
Serum Lactate	4.0 mmol/L	0.5-2.2 mmol/L
Urinalysis	Proteinuria: 2+; Hematuria: Negative; Specific Gravity: 1.025	Proteinuria: Absent; Hematuria: Absent; Specific Gravity: 1.005-1.030
Serum Mercury Levels	>250 µg/L	N/A (Depends on exposure history)
Urine Mercury Levels	>250 µg/L	N/A (Depends on exposure history)
Troponin I	180.2 ng/mL	0-0.04ng/mL

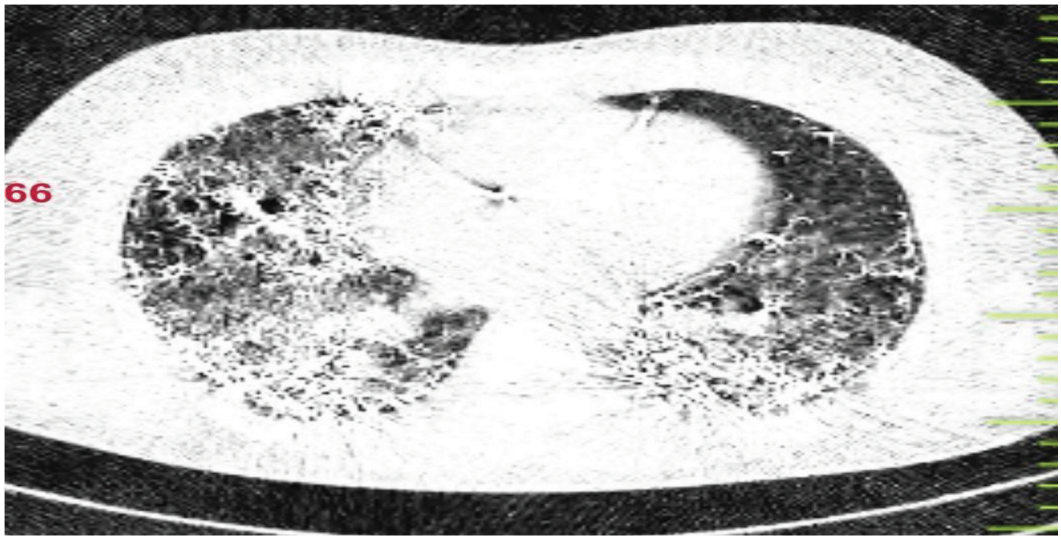


Figure 3. High Resolution Computed Tomography (HRCT) thorax showing scattered high attenuation foci in bilateral lung parenchyma predominantly in peripheral distribution with tiny areas of ground glass opacities bilaterally.

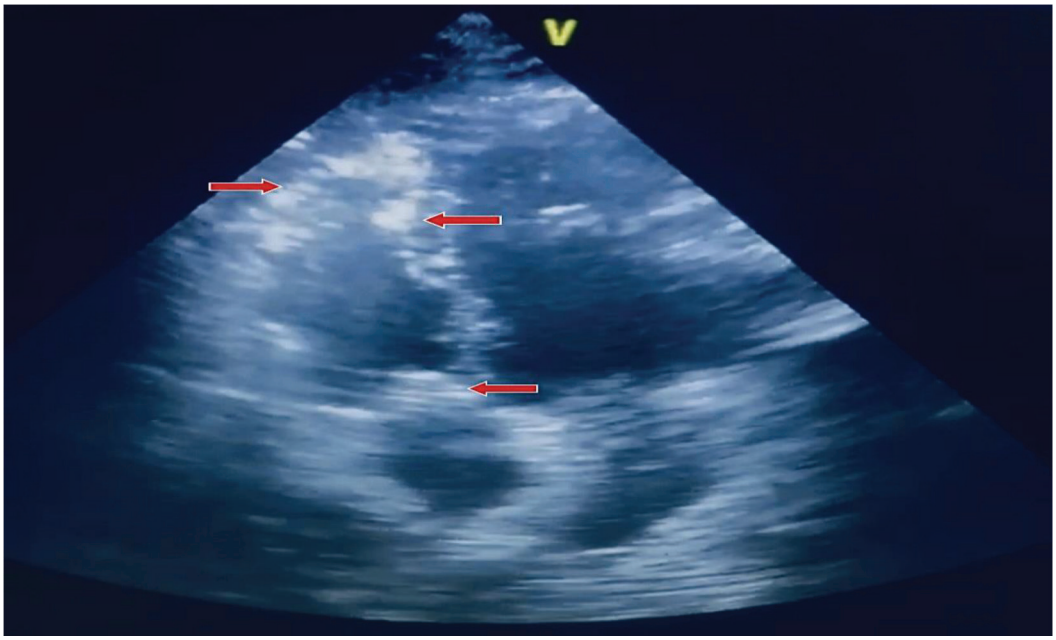


Figure 4. Transthoracic 2 Dimensional (2D) echocardiography four chamber view showing hyperechoic infiltrations with red arrows on distal interventricular septum, right ventricular apex and right ventricular free wall including infiltration in the tricuspid valve.

600mg intravenous thrice a day in view of deranged kidney function tests, deranged coagulation profile and raised liver enzymes as shown in Table 1. Patient was also started on unfractionated heparin injection 5000 IU intravenous thrice a day for 5 days with a close watch on thrombocytopenia and bleeding tendencies, in view of raised troponin I levels along with single antiplatelet and statins. Other supportive management along with rigorous intravenous fluid administration around 3000-3500 ml per day was maintained with close monitoring of input and output status along with all routine blood investigations.

The patient was eventually weaned off non invasive positive pressure ventilation after thorough arterial blood

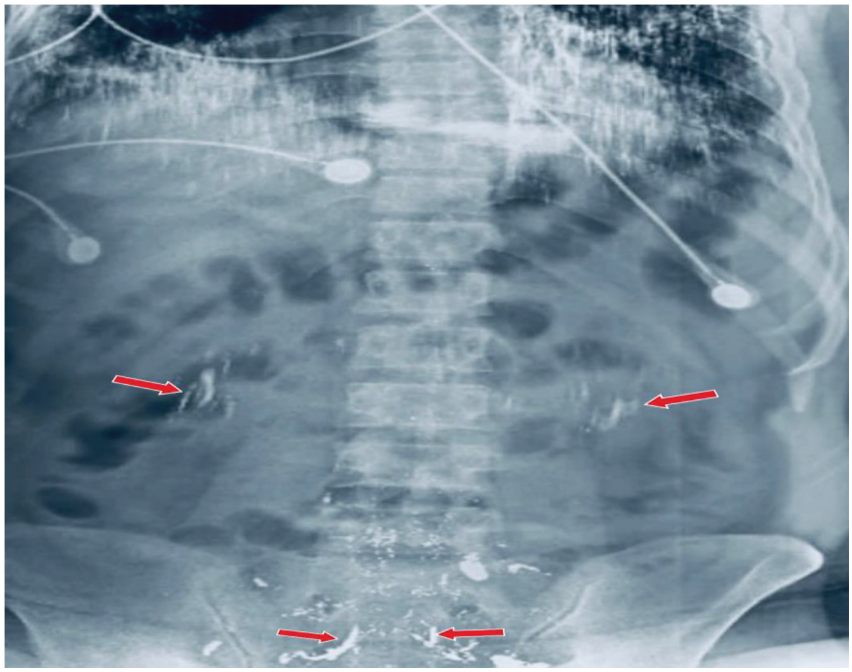


Figure 5. X ray erect anterior-posterior view of abdomen revealed high attenuated foci suggestive of metallic deposits in bilateral iliac fossa, suggestive of metallic deposits in both renal systems with deposits in pubic fossa.

gas monitoring and treating respiratory acidosis. All routine blood parameters of the patient like the kidney and liver function test along with coagulation profile recovered and sepsis was treated eventually. During the 15 day ICU stay, the patient did not manifest any neurological complaints and the patient's condition got better and was shifted to general ward for observation. He was also consulted by the psychiatry department after stabilisation and was diagnosed with major depressive disorder without psychotic symptoms with borderline personality traits. He was further transferred to psychiatry department for evaluation and management under close monitoring of the medicine department. The patient was then discharged on tablet escitalopram 10 mg one tablet at night and advised dialectical behavioural therapy along with multivitamin supplements after keeping in observation for 5 more days and was asked to follow up after 1 month. On close follow up of the patient, there were no complaints and no signs of any chronic toxicity.

3. Discussion

Mercury poisoning is a significant health concern worldwide, characterised by elevated levels of mercury in the body, leading to a range of acute and chronic health effects. Mercury is a naturally occurring heavy metal that exists in various forms, including elemental mercury (Hg^0), inorganic mercury compounds, and organic mercury compounds like methylmercury. Each form of mercury has distinct toxicological properties and routes of exposure, contributing to the complexity of mercury poisoning[3].

Mercury exists in various forms like elemental, inorganic and organic forms of which elemental form is associated with respiratory symptoms, neurological effects, and kidney damage and its chronic exposure may result in tremors, cognitive impairment, and emotional disturbances[4]. The most common organic form of mercury, also known as methylmercury, bioaccumulates in the food chain, particularly in predatory fish. Chronic exposure to methylmercury through consumption of contaminated fish can lead to neurological deficits, developmental delays, and cardiovascular effects, especially in vulnerable populations such as pregnant women and young children. Inorganic form may be associated with corrosive effects on the gastrointestinal tract and systemic toxicity, affecting the kidneys, gastrointestinal system, and nervous system[4].

Various routes of exposure to mercury include inhalation, ingestion, dermal and intravenous, out of which intravenous mercury poisoning is relatively rare compared to other routes of exposure and very few cases have been reported with intravenous mercury abuse.

Incidents of intravenous mercury poisoning often occur accidentally or through illicit drug use, occupational exposure, or intentional self-harm[5]. The incidence and prevalence of intravenous mercury poisoning may vary depending on factors such as geographic location, healthcare practices, and regulatory measures in place to prevent mercury exposure[5].

Systemic manifestations of mercury poisoning are widespread involving various neurological, gastrointestinal, renal, cardiovascular, endocrine and respiratory symptoms out of which patient presenting with respiratory symptoms are not very common[6]. Pathophysiology of intravenous mercury poisoning leading to severe metallic pneumonitis, like in our case, involves the following mechanisms[7].

3.1 Direct Pulmonary Injury

Elemental mercury (Hg^0) vaporises readily at room temperature and can be absorbed into the bloodstream through the lungs when injected intravenously. Once in the bloodstream, elemental mercury can bind to haemoglobin and be transported to the pulmonary capillaries. Elemental mercury vapour can also directly come into contact with the alveolar epithelium, leading to local tissue damage and inflammation in the lungs[7].

3.2 Inflammatory Response

Exposure to elemental mercury vapour in the alveoli triggers an inflammatory response characterised by the recruitment of inflammatory cells such as neutrophils and macrophages to the site of injury. Activated neutrophils release pro-inflammatory cytokines, chemokines, and Reactive Oxygen Species (ROS), contributing to tissue damage and inflammation in the lungs. Macrophages phagocytose mercury particles and release inflammatory mediators, further amplifying the inflammatory cascade[7].

3.3 Oxidative Stress

Mercury-induced oxidative stress plays a significant role in the pathogenesis of pneumonitis.

Elemental mercury and its vapour can induce the generation of Reactive Oxygen Species (ROS) in lung tissues, leading to oxidative damage to cellular components such as lipids, proteins, and DNA. Oxidative stress disrupts cellular integrity, impairs cellular function, and exacerbates inflammation and tissue injury in the lungs[7].

3.4 Alveolar Damage and Edema

Mercury exposure disrupts the integrity of the alveolar-capillary barrier, leading to increased permeability and leakage of plasma proteins and fluid into the alveolar spaces. Alveolar damage and increased permeability result in the accumulation of protein-rich fluid in the alveoli, leading to pulmonary edema and impaired gas exchange[7].

3.5 Chemical Pneumonitis

The combination of direct tissue injury, inflammation, oxidative stress, and pulmonary edema leads to the development of chemical pneumonitis. Chemical pneumonitis is characterised by diffuse alveolar damage, interstitial inflammation, edema, and hyaline membrane formation, resembling the histological features of Acute Respiratory Distress Syndrome (ARDS)[7].

3.6 Clinical Manifestations

Patients with severe metallic pneumonitis may present with acute onset of respiratory distress, cough, chest pain, and hemoptysis. Severe cases may progress to respiratory failure, requiring mechanical ventilation and intensive care support. Chest X-ray or CT imaging may reveal diffuse bilateral infiltrates consistent with acute lung injury or ARDS[7].

In summary, intravenous mercury poisoning can lead to severe metallic pneumonitis through a combination of direct pulmonary injury, inflammation, oxidative stress, and disruption of the alveolar-capillary barrier. Early recognition and aggressive management are essential to minimise morbidity and mortality in patients with severe pneumonitis secondary to intravenous mercury exposure. While, management of mercury toxicity involves only chelation therapy, supportive management to treat multiorgan dysfunction syndrome is of utmost importance with close monitoring of vital signs and systemic examinations to early identify involvement of any further system to start treatment promptly[8].

Also, prevention is key to reducing the risk of mercury poisoning. This involves minimising exposure to mercury by avoiding contaminated food and water, using protective equipment in occupational settings, and properly disposing of mercury-containing products. It's important to consult with healthcare professionals for personalised management and treatment plans based on the individual's level of exposure and symptoms[9].

4. Conclusion

Mercury poisoning poses significant risks to human health and the environment. Prevention strategies focus on reducing exposure to mercury, including regulatory measures to limit environmental contamination, public health interventions to educate communities about safe consumption practices, and occupational health and safety measures to protect workers from mercury exposure. Additionally, counselling services should be made available to individuals at risk, providing them with guidance on monitoring and reducing exposure. Early recognition, prompt treatment, and preventive measures are essential for mitigating the impact of mercury toxicity on human health and the environment. Multidisciplinary collaboration among healthcare professionals, regulatory agencies, environmental experts, and community stakeholders is crucial for addressing the complex challenges associated with mercury poisoning. Continued research and monitoring are necessary to better understand the full spectrum of mercury's impact on health and to develop more effective strategies for prevention and treatment.

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