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Thallium Poisoning: Case Report and Scoping Review on Diagnostic Delay and Therapeutic Outcome

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Featured Application: Early treatment of suspected cases of thallium intoxication with Prussian Blue is recommended as an "ex juvantibus" strategy, because it offers immediate clinical benefit in positive cases and has negligible side effects in non-intoxicated cases.

Abstract: Thallium poisoning, which may be accidental or criminal, presents with a non-specific clinical picture but is rapidly progressive. A delay in diagnosis may cause the appearance of serious, often irreversible, and sometimes fatal lesions. Prompt treatment with Prussian Blue before toxicological confirmation results in immediate improvement in cases of intoxication, without appreciable side effects, and is, therefore, recommended as an "ex juvantibus" strategy in cases of suspected thallium poisoning. A successfully treated case of poisoning is presented as an example of this strategy. An analysis of the contaminated well water the patient had unknowingly drunk subsequently showed pollution over 75 times higher than the potable limit, and plasma levels revealed values 267 times higher than the normal range. All the test results were received when the patient undergoing treatment had improved so much that she had been discharged from hospital. To complete the study, we conducted a scoping review to understand the extent and type of evidence in relation to the latency in the diagnosis of intoxication and health effects. The review of 30 articles covering 115 cases of thallotoxicosis confirmed that early treatment with Prussian Blue offers the best chance of achieving complete recovery.

Keywords: metal toxicity; neurotoxicity; toxicokinetic; therapy; environmental pollution; emerging contaminants; low-dose effects; human health

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1. Introduction

Tasteless, colorless, odorless, and soluble in water, thallium is a chemical element that has been known to cause accidental intoxication as well as intentional poisoning. In Western countries, thallium zinc sulfate was used in the 1950s as rat and ant poison [1] but was banned, firstly, in American homes in 1965 and then, commercially in 1975. However, thallium is still utilized in rodenticides and insecticides in several countries where, on account of its odorless and tasteless properties, it has led to both unintentional poisoning and, in some cases, illegal use. In medicine, thallium salts were previously employed in the treatment of dermatophytosis [2]. Prior to the adoption of technetium-99m in nuclear medicine, thallium-201, a radioactive isotope, was the primary agent [3] and

continues to be utilized in neuroradiology [4], radiotherapy [5], and scintigraphy [6], and for stress tests assessing coronary artery disease [7]. This kind of thallium is around 4000 times less powerful than zinc sulfate.

In industry, thallium is a vital element commonly used in high-technology industries where there is currently a growing demand for semiconductors. The occupational exposure limit for thallium is 0.1 milligrams (mg) per cubic meter for no longer than eight hours a day. Acutely hazardous levels are those reaching 15 mg/m^3 and over. Thallium can easily enter the body via skin absorption and inhalation. Cases of occupational thallium intoxication have been reported, mainly in Asian countries [8–10].

Wastewater discharges from manufacturing plants or metal-mining activities may result in elevated levels of thallium in the receiving water. For this reason, it is a priority pollutant regulated by the American Environmental Protection Agency (US EPA) [11–14]. Thallium has been utilized as a pesticide in Africa, where it has led to food contamination. Chinese herbal medicines have also been compromised. Intoxication results from cumulative absorption via dermal, respiratory, and gastrointestinal pathways. Instances of inadvertent snorting by cocaine users [15], unintentional injection by heroin users [16–18], and dermal absorption through protective gloves have been documented [9].

Thallium contamination has cumulative effects. The chronic exposure of populations to this pollutant has been associated with many pathologies. Thallium urinary levels have been associated with a reduction in cognitive performance in older US adults [19], with Parkinson's disease [20], and also with a risk of gestational diabetes [21]. Plasma thallium levels have been associated with decreased renal function [22], central obesity [23,24], ischemic stroke [25] and increased all-cause and cardiovascular mortality in the general population in China [26]. Studies with DNA methylation probes in exposed populations indicated alterations that could be implicated in cancer progression and respiratory diseases [27]. A longitudinal study showed that thallium exposure at low concentration leads to the early damage of multiple organs in children [28].

Thallium (Tl^+), one of the most toxic heavy metals, shares an ionic ratio similar to potassium (K^+), allowing it to replace K^+ in enzymatic processes. Its primary target within cells is the mitochondria, where it disrupts intrinsic pathways, affects antiapoptotic and proapoptotic proteins, and activates oxidative stress mechanisms. Tl^+ exposure leads to increased reactive oxygen species (ROS) and lipid peroxidation, causing cellular damage and triggering antioxidant responses. In humans, Tl^+ is absorbed through skin and mucous membranes, distributing across organs like bones, kidneys, liver, and the central nervous system, with neurotoxic effects that are now recognized as a significant global health concern due to rising reports of Tl^+ pollution [29].

The establishment of surveillance protocols to monitor toxic substances like thallium is essential, particularly in high-risk environments and workplaces. According to the Environmental Protection Agency (EPA) and the Occupational Safety and Health Administration (OSHA), thallium poses significant health risks through occupational and environmental exposure and has been identified at multiple hazardous waste sites nationwide. OSHA has set workplace exposure limits (PEL-TWA) at 0.1 mg of soluble thallium compounds per cubic meter of air for an 8 h workday, underscoring the need for strict control measures to limit skin and respiratory exposure [30]. The National Institute for Occupational Safety and Health (NIOSH) has indicated the same limit value (REL-TWA = 0.1 mg/m^3) for work, up to 10 h. The Association of American Industrial Hygienists (ACGIH) set a much lower limit in 2009 (TLV-TWA = 0.02 mg/m^3), confirming the notations "skin" (skin passage), "stel" (short time exposure limit, limit valid even for a short period), and "ceiling" (limit not to be exceeded).

This brief review of the effects of thallium indicates how important it is for the clinician to consider this form of intoxication among the many possible etiological diagnoses

of cases with neurological symptoms. In fact, even in Western countries, a person can be intoxicated by inadvertent exposure through alternative drugs, uncontrolled food products, and contaminated water. Even intentional ingestion for suicidal purposes must be considered on account of the possibility of purchasing it online [31]. The rarity of thallium poisoning and the non-specific clinical signs of presentation may cause a delay in treatment and admission. Because intoxication often appears to be analogous to other diseases, ill patients may go unnoticed. If left untreated, thallium poisoning can cause irreversible harm to the digestive and neurological systems, or, in extreme situations, result in paralysis, coma, and death.

In this article, we report on a case of unintentional thallium poisoning which had not been correctly identified in two previous hospital centers and had presented to our Center with symptoms compatible with thallium poisoning. In thallium poisoning, the therapeutic goal is to lower the levels of circulating metal as quickly as possible, before it is deposited in the tissues, by means of Prussian Blue (PB). We asked ourselves whether it was correct to start treatment with PB before having toxicological confirmation of the poisoning. For this reason, we undertook a scoping review to assess the relationship between the onset of treatment and the outcome.

2. Methods

Considering that there were no reviews in the literature that allowed us to study the relationship between delay in diagnosis, the treatment of intoxication, and residual health problems, we conducted a rapid scoping review on this topic, following the Preferred Reporting Items for Systematic Reviews and Meta-Analyses extension for Scoping Reviews (PRISMA-Scr) checklist [32]. We opted for a rapid review to have immediate reassurance for our idea of administering the drug before receiving toxicological confirmation of the diagnosis. We used the PCC framework, according to the JBI Scoping Review Methodology Group [33].

A scoping review approach was selected to provide a broad overview of available evidence on Prussian Blue (PB) therapy for thallium intoxication. Given the rarity of documented cases and the fragmented nature of available data, a full systematic review was deemed impractical. A scoping review allows for the identification and mapping of key findings across diverse studies since it highlights treatment outcomes and gaps in literature without requiring comprehensive quality assessments. This approach is particularly suited to emerging clinical topics where formal guidelines are absent and rapid evidence synthesis can inform clinical decision-making.

Given the objectives of the review, the inclusion criteria (PCC framework) were as follows: cases of thallium intoxication or poisoning were included as “participants”; the “concept” was the relationship between the latency between exposure and treatment and the outcome; the “context” included case reports/series of intoxication, whatever their origin (accidental, voluntary, and unknown). The overview of the existing evidence was conducted without a formal assessment of the methodological quality of the included studies. The rapid scoping search was conducted on a single database (PubMed) and included publications in English, German, and Italian.

Cases or case series where treatment had included Prussian Blue were included; cases where PB was not used were excluded. Experimental, in vitro, and animal studies and toxicological investigations were not included; reviews were not included but were consulted for additional reports. The selection of the articles was carried out by two authors (N.M. and F.C.); a third author (P.M.S.) was called upon for doubtful cases. The search for the phrase “thallium (intoxication or poisoning) and (case-report or case series)” returned 188 results, while adding the term “Prussian Blue” caused the search to return 32 articles. Examination of titles and abstracts identified 41 articles that were read. Articles that did

not report the time of starting treatment or the outcomes were excluded. The remaining 30 articles were included in the review. We extracted clinical data, such as onset of symptoms, treatment initiation, and outcome, listing the treatments that were associated with PB.

We limited the search to one database to respect the urgency of a clinical decision on a patient in emergency conditions. Considering that the results were positive and potentially beneficial for other patients, we opted for a prompt publication rather than broadening the search to include other databases.

This article is a literature review with a clinical case. Literature reviews do not require an ethical opinion. Likewise, the publication of a clinical case requires the consent of the interested party and respect for confidentiality, avoiding any element that could lead to the identification of the patient [34–37]. The patient gave preliminary permission to publish, and read the final manuscript text, making sure that her privacy was respected.

3. Detailed Case Description

A 40-year-old female patient, working as a pharmacist in central Italy, came to our emergency room after a week's progressive onset of paresthesia of the lower arms, with severe muscle pain, reduced strength, generalized asthenia, visual disturbances with conjunctival hyperemia, chest pain, abdominal pain, nausea, and diarrhea. During the week, the extent and intensity of her symptoms had gradually worsened. The patient had taken ibuprofen, without gaining relief. Finally, being unable to walk unaided because of severe pain in the trunk and limbs, and subsequently in the arms, she was confined to a wheelchair. Previously, she had been to two different peripheral hospital emergency rooms but had not been hospitalized. Eye drops had been prescribed for eye symptoms.

At the clinical examination in the emergency department, the patient was alert and lucid and eupneic, with rhythmic cardiac action and normal pulmonary findings. The abdomen was treatable; Murphy's and Blumberg's signs were negative. Severe arthralgias prevented walking and movement. There was no fever, but pharyngeal hyperemia, lymphadenopathy, and skin rashes were observed. The patient complained of paresthesia of the tingling type at the tips of the fingers and the tips of the feet, with a slight superficial hypoesthesia of the anterior surface of the legs. Discriminative tactile, thermal, and painful sensitivity was normal. The patient did not present any deficits of the cranial nerves or global strength (although this was difficult to evaluate due to the pain limitation); the index–nose test was well performed symmetrically, and she had normally evoked osteotendinous reflexes.

The patient was admitted to the Department of Emergency, Anesthesiology, and Resuscitation Sciences of the Gemelli General Hospital. At anamnesis, she failed to report any chronic pathology, drug usage, or food allergies. She denied taking any food supplements, alternative medicines, cosmetics, or exotic food products. She lived in a country house and had not travelled outside Italy. She owned a cat and had a flower garden and a vegetable garden and no history of tick bites.

The patient's background was further explored to assess potential chronic exposure sources, including any prior use of contaminated water, dietary habits that could increase thallium ingestion (such as home-grown produce), and possible occupational or environmental contacts. This investigation aimed to determine whether the acute presentation was compounded by underlying, low-level exposure over time that might have contributed to the severity of symptoms and a delayed diagnosis.

Her vital parameters were within limits: blood pressure 121/67 mmHg, heart rate 79 bpm, SpO2 98%, and temperature 36.5 °C. The venous blood gas analysis showed pH 7.39, Lactates 0.7 mmol/L, potassium 3.7, and no electrolyte alterations. The chest X-ray, electrocardiogram, urine tests, and basic blood tests were negative. The blood tests for Leptospira, Borrelia, Rickettsia, Bartonella, human immunodeficiency virus (HIV), cytomegalovirus (CMV), Epstein–Barr virus (EBV), toxoplasma, syphilis, hepatitis C virus (HCV), hepatitis B virus (HBV), enterovirus, adenovirus, and Chikungunya virus were all negative.

Considering that the patient lived in the countryside, the doctors investigated the source of the water supply. The patient confirmed that the house was supplied with drinkable water from the public water supply, but also had well water that was not drinkable and used only for cleaning.

Having learned of the existence of a well with non-potable water, the doctors investigated further by asking if the water line of the well was separate from that of the municipal water supply with potable water. To their surprise, they learned that the line was the same and that the type of water introduced into the domestic pipes could be potable or not, depending on the positioning of a valve. The patient added that the partition valve was always operated by her husband and that he had been away on business for a week. She did not know how to position the valve correctly.

The information gathered prompted the doctors to request a water analysis from the relevant Regional Agency (ARPA), which, due to the urgency of the case, provided the report within a few days.

Toxicological analyses were carried out by the national reference laboratory, situated in another Region. Plasma and urinary samples for barium and thallium were obtained on the second day after admission, but the results naturally arrived a few days later when the patient had already been discharged. However, based on the patient's clinical history, the doctors decided to start treatment with Prussian Blue (Radiogardase, 3 g × 3/die) before receiving the results of the tests. Prussian Blue can attach to other oral medications (specifically, it may prevent tetracycline absorption) and vital nutrients while being taken orally. The gastrointestinal tract's electrolytes, such as potassium, can be bound by Prussian Blue, lowering serum potassium levels and causing asymptomatic hypokalaemia. As a result, throughout treatment, the serum electrolyte levels should be frequently checked. Patients with pre-existing cardiac arrhythmias and electrolyte abnormalities should exercise extra caution. Prussian Blue consumption may result in constipation. Fiber-based laxatives or a high-fiber diet may be used to treat this. Since Prussian Blue is essentially not absorbed, does not cross the placental barrier, and does not enter breast milk, there are no issues with using it during pregnancy or lactation. There are currently no known consequences to one's capacity to operate machinery and drive. The low toxicity of Prussian Blue made the clinically guided "ex juvantibus" treatment advisable.

The Prussian Blue treatment, that was started on the first day of hospitalization, immediately resulted in pain relief and a general improvement in symptoms. Twelve hours after the start of Prussian Blue therapy, the pain was noticeably reduced. In the following days, there was a significant improvement in the patient's clinical conditions: paresthesia, myalgias, and joint pains disappeared, and tenderness in her knee joints diminished. Table 1 reports the results of toxicological analyses conducted on the patient during hospitalization and over a period of several months after discharge.

Table 1. Barium and thallium values measured at different time intervals after admission.

Determination	Day 2	Day 10	Day 30	Day 50	Day 75	Day 120	Normality Range *
Urinary Barium	2.76 μg/L						ND-6.96 μg/L
Plasma Barium	1 μg/L						0.2–1.2 μg/L
Urinary Thallium	24 μg/L	26 μg/L	48 μg/L	7.4 μg/L	2.7 μg/L	0.65 μg/L	0.06–0.759 μg/L
Plasma Thallium	19 μg/L	8 μg/L	4 μg/L	0.4 μg/L	0.22 μg/L	0.12 μg/L	0.012–0.071 μg/L

* Reference values for the general population; data from the Italian Society for Reference Values, SIVR 2023 [38].

On the fifth day, the patient was discharged with instructions to remain in telematic contact with the Poison Control Center of the Gemelli General Hospital and to repeat the urine and plasma tests on the dates indicated by the center.

The water quality was analyzed using inductively coupled plasma mass spectrometry (ICP-MS), according to the ISO 7294-2:2023 method [39]. When the water analyses became available, they indicated evident thallium contamination in the well water, with values off the scale. The laboratory provided the highest measurable value, 150 μg/L, which is 75 times higher than the maximum United States Environmental Protection Agency (US EPA) safety value of 2 μg/L for drinkable water. The levels of other elements (barium, chromium, copper, vanadium, aluminum, cadmium, arsenic, lead, mercury, boron, nickel, selenium, manganese, and antimony) in the well water were within the safety levels.

Blood and urine were analyzed by dynamic cell reaction inductively coupled plasma mass spectrometry (DRC-ICP-MS). Toxicology tests indicated elevated urinary thallium levels from the second day of hospitalization, with increased urinary excretion during therapy (between the tenth and thirtieth day) and a subsequent reduction, which, nevertheless, reached normality only after 120 days. The plasma thallium levels were very high on admission (267 times the upper reference limit) and showed a gradual reduction but failed to return to normal after 30 days of treatment. For this reason and given the absence of side effects from the treatment, the doctors decided to continue treatment with Prussian Blue, reducing the dose (1.5 g three times a day) on day 45. The plasma thallium levels were not normal even on the 120th day after the start of treatment.

The first telematic clinical check-up enabled doctors to detect the appearance of a rash on one hand (Figure 1). The hand appeared hyperemic, with signs of parakeratosis of the skin and nails. Moreover, there was the onset of alopecia. The patient reported the disappearance of pain and recovery of mobility, even if marked asthenia persisted. The alopecia had worsened during the 30-day check-up, while the general clinical picture showed a slow but gradual improvement. By day 120, the hair loss had been resolved, and the patient had returned to the pre-intoxication clinical and functional state.

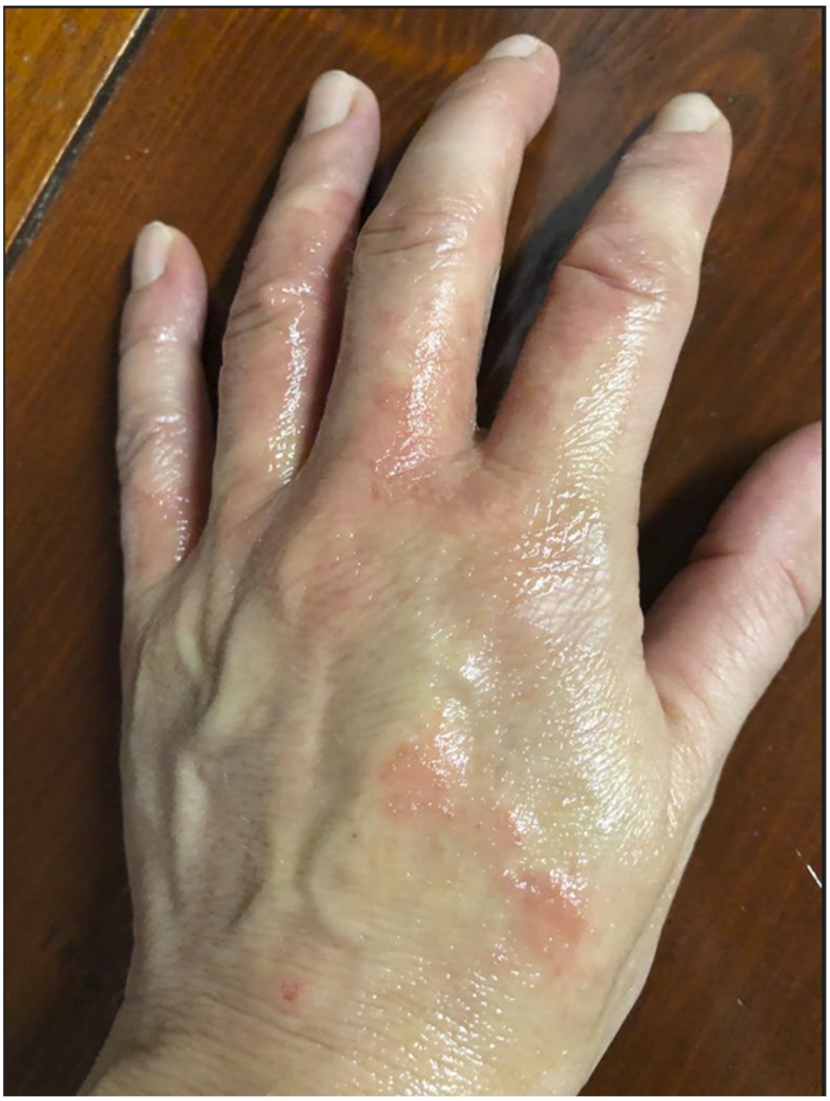

Figure 1. Left hand at 30th day after admission.

4. Results of the Review

From each of the selected articles, the age and sex of the victim, the cause of poisoning, the symptoms, the latency between the onset of symptoms and treatment with Prussian Blue, any additional treatments, and the patients conditions at the end of the follow-up were extracted.

Table 2 reports the result of the review.

Table 2. Cases of thallium intoxication.

Author, Year	Patient Age, Sex	Cause	Symptoms	Latency	Other Therapy	Outcome
Pedersen et al., 1978 [40]	56-year-old woman	Unknown	Gastrointestinal, painful neuropathy, alopecia	>20 days	Hemodialysis	Residual neurological problems
Stevens, 1978 [41]	52-year-old woman	Homeopathic drug	Neurological	Day 1		Complete recovery
Heath et al., 1983 [42]	58-year-old woman 59-year-old man 28-year-old man	Criminal	Neurological and cardiological Neurological, alopecia Neurological, alopecia	Day 4	Hemoperfusions Hemoperfusions Hemoperfusions	Death Residual neurological problems Residual neurological problems
Wainwright et al., 1988 [43]	33-year-old man	Unknown	Neurological and cardiological	>1 year	Forced diuresis and hemodialysis	Neurological sequelae
Meggs et al., 1994 [44]	4 young men	Criminal	Gastrointestinal, and painful paresthesia; hypertension and tachycardia	3 days		Complete recovery
Niehues et al., 1995 [45]	15-year-old woman	Voluntary (twice)	Colic-like abdominal pain, vomiting, paresthesias	<1 h		Complete recovery
Malbrain et al., 1997 [46]	38-year-old man	Unknown	Neurological	Day 2	Forced diuresis, hemodialysis	Complete recovery
Atsmon et al., 2000 [47]	40-year-old man	Criminal	Gastrointestinal, neurological, alopecia	>30 days		Residual neurological problems
Pau, 2000 [48]	67-year-old woman	Unknown	Pain in the chest, abdomen, and lower limbs, alopecia	>30 days		Residual sensory neuropathy after 1-year
Lu et al., 2007 [49]	52-year-old man 48-year-old woman	Accidental	Gastrointestinal, neurological and dermatological	21 days	Forced diuresis, hemoperfusion	Residual neurologicalproblems
CDC, 2008 [50]	10 family members	Accidental	Gastrointestinal, neurological, alopecia, coma	>11 days		4 deaths, 6 residual neuropathy
Pelclova et al., 2009 [51]	44-year-old woman 22-year-old woman	Criminal	Pain in the chest, abdomen, and lower limbs. Alopecia, visual impairment Pain, neurological symptoms	>15 months >3 months		Neuro-ophtalmological symptoms at 18 months from treatment
Sun et al., 2012 [52]	14 patients (mean age: 36 years)	Accidental, poisoned food	Gastrointestinal, painful polyneuropathy, hair loss	9–19 days	Chelant, hemodialisis	1 death 13 residual neuropathy after 7 months
Riyaz et al., 2013 [53]	36-year-old man	Voluntary	Neurological	18 h		Death
Zhang et al., 2014 [54]	9 patients, 2 to 60 years old, 4 M, 5 W	Accidental	Gastrointestinal, neurological	12 days (mean)	Hemodialysis, hemoperfusion, veno-venous hemofiltration	Mild neurological problems

Huang et al., 2014 [55]	40-year-old man	Criminal	Numbness and intense stabbing pains	Day 5	hemoperfusion (HP) and continuous veno-venous hemofiltration (CVVH)	Recovery on day 33
Li et al., 2014 [56]	13 patients	Various	Various	24 days (mean)		2 deaths, 11 neurological problems
Sojáková et al., 2015 [57]	24-year-old man	Voluntary	Gastrointestinal complaints, painful polyneuropathy; alopecia	A few hours	Stomach irrigation, active charcoal	Complete recovery
Li et al., 2015 [58]	2 patients	Criminal	Toxic encephalopathy, alopecia	>150 days		Death
Yumoto et al., 2017 [59]	23-year-old woman	Criminal	Gastrointestinal, neurological symptoms and alopecia.	Day 11		Partial recovery of polyneuropathy
Almassri et al., 2018 [60]	3 patients (out of 23 poisoned)	Criminal	Abdominal pain, neuropathy, alopecia,	45 days		Neuropathy
Yang et al., 2018 [61]	53-year-old man	Unknown	Stabbing pain in the abdomen and lower extremities; alopecia	>20 days	Hemoperfusion, veno-venous hemofiltration	Residual neurological problems at 6-month follow-up
Ash and He, 2018 [62]	18-year-old woman	Criminal	Encephalopathy	>5 months		Death
Lin et al., 2019 [63]	31 patients	Various	Various	13 days (mean)		Neurological problems
Linet al., 2019 [64]	42-year-old woman	Unknown	Toxic encephalopathy, coma	Day 44	Plasma exchange	Recovery of consciousness on the 50th day of admission. Neurological symptoms at 37-month follow-up
Liu et al., 2021 [65]	43-year-old man	Criminal	Gastrointestinal complaints, painful polyneuropathy	21 months		Neurological symptoms at 6-year follow-up
Pragst and Hartwig, 2021 [66]	44-year-old man	Criminal (repeated)	Paraparesis of the legs, and hypersensitivity and strong pains in whole body	40 days	Forced diuresis	Neurological
Wang et al., 2021 [67]	5 patients 33 to 49 years old men	Accidental, poisoned food	Hyperalgesia of the limbs and abdominalgia	9–12 days		Complete recovery in 4 cases, blindness and paralysis at 20 months in 1 case
Graham, 2023 [68]	20-year-old man	Unknown	Neurological, alopecia	Day15		Neurological at >10 years
Spadaro et al., 2024 [31]	18-year-old man	Voluntary	Asymptomatic	Day 1	Surgical remotion of a metal bar of thallium	Asymptomatic at day 86

Generally, patients with delayed treatment exhibited severe symptoms upon admission. Treatment with Prussian Blue significantly reduced thallium levels in plasma and urine; however, in cases with a longer treatment latency, permanent neurological sequelae were invariably present. Complete recovery was observed only in cases where Prussian Blue was administered within the first few hours of exposure. In some cases, doctors started treatment before having toxicological confirmation [44].

In Table 3, we have summarized the symptoms and signs of intoxication obtained from the literature and their evolution after the treatment. We have compared the data with the observations made in our case.

Table 3. Reported symptoms and progression of intoxication as a function of treatment, according to literature data and in our observation.

Symptoms and Signs	Literature Data	Our Observation
Paresthesia of the limbs	Reported in all cases	Relief after 2–3 days of treatment with PB.
Muscle pain	Reported in all cases	No improvement with ibuprofen. Pain relief 12 h after the start of therapy with PB.
Chest pain, cardiological signs	Dominant symptoms in cases with late diagnosis [43,48,50] or high dose, criminal intent [42]	No improvement with ibuprofen. Disappearance after the therapy with PB.
Abdominal pain, nausea, diarrhea	Frequently reported [40,45,47–50,54,57,59–61,65,67]	No improvement with ibuprofen. Disappearance after the therapy with PB
Reduced strength, functional impotence	Frequently reported	Marked relief in 4 days of treatment with PB (discharge). Resumption of household activities after 15 days of treatment.
Generalized asthenia	Frequently reported	Gradual and very slow improvement.
Peripheral neuropathy	Very common [40–44,46–48,50,52–59,61,63,65–68].	Rapid relief of neurological symptoms after treatment.
Toxic encephalopathy	In the most serious cases [58,62,64]	No.
Conjunctival hyperemia, visual disturbances	After high-dose exposure [50,67]	No improvement with topical preparations. Complete relief after treatment with PB.
Alopecia	Late onset [34,42,47,48,52,56–61,63,68]	Evident 15 days after onset, worsened after 30 days, resolved after 120 days of treatment.
Skin changes	Late onset [49]	Hyperemia and parakeratosis 15 days after the onset, regressed after 30 days.
Persistent neurological changes	Reported in all cases treated with PB more than 10 days after intoxication [40,43,47–50,52,54,59–61,63–68] or after exposure to high doses for criminal intent [42,56]	None.
Complete recovery	Reported in all cases of early treatment with PB [41,44–46,55]	Complete recovery after 120 days of treatment with PB.
Death	Reported in criminal and voluntary cases [42,53,56,58,62] and in delayed treatment [50,52]	No.

PB = Prussian Blue.

5. Discussion

5.1. Adverse Effects of Thallium

Thallium poisoning has a long history and, as we have seen, the occurrence of cases of poisoning cannot be ruled out even in the most advanced countries. The United States ceased the domestic mining of thallium in 1981, but, since then, the growing demand in the semiconductor industry (more than 2 metric tons in 1987) has been met by imports from Belgium (54%), the Netherlands (16%), the Federal Republic of Germany (14%), the United Kingdom (6%), and other sources (10%) [69]. Production was estimated at 14.06 metric tons in the rest of the world [70], and this has caused increasing ecological problems globally. Furthermore, thallium is widely distributed in the earth's crust, and this multiplies the possibility that waters are polluted by this element, as was evident in the case reported above.

In 2019, the American Association of Poison Control Centers reported 49 single exposures in the US, resulting in one major outcome but no fatalities, which are a rare but not impossible occurrence. Thallium poisoning is prevalent in developing countries; however, few data are available. In this rapid review, we found 115 patients treated with Prussian Blue, 11 of whom had fatal outcomes (Table 2). Mortality rates for acute thallium toxicity have been reported to range from six to fifteen percent. A lethal dose for human beings ranges from 10 to 15 mg/kg, but mortality can still occur at lower dosages [71–73].

The major hazard for the general population is exposure to polluted water. Water pollution is generally attributed to the interaction between groundwater and thallium-bearing pyrite ores. In China, a water source contaminated with thallium (with values up to 10 μg/L) caused significant public health problems [74]. In Italy, high levels of pollution (up to 9000 μg/L) have been observed in wastewater from abandoned mining plants [75–79] and resulting in the contamination of the water drunk by the surrounding population [80]. Exposure to low doses of thallium, far below the US EPA maximum allowable level in drinking water (2 μg/L) [81], is a threat to human health [82]. Over 10 years ago, the US EPA aimed to lower the maximum contaminant level of thallium in drinking water to 0.5 μg/L, but this measure has not yet been adopted in America or any European country.

Studies conducted on general population subjects with moderate thallium absorption (urinary levels less than 0.2 μg/L) reported a number of health problems, including impaired thyroid function [83], metabolic changes (increased waist circumference and body mass index) [84], hypertension [85], impaired glomerular filtration [86], and autism spectrum disorders [87,88], as well as osteoarthritis [89]. The placenta transports about 50% of the metal from mother to fetus [90] and maternal exposure is associated with a low birth weight [91] and preterm birth [92]. Thallium is also associated with genetic and epigenetic changes [93]. Although we cannot state that there is consolidated evidence of harm, the complex set of reports suggests that careful monitoring of the possible effects on populations drinking water with thallium levels between 0.5 and 2 μg/L is necessary.

Thallium is similar in structure to potassium and is, consequently, processed in a comparable manner at the cellular level [94]. Thallium toxicity is associated with some primary toxicologic effects. Tissues exhibiting elevated potassium concentrations accumulate significant levels of thallium. This results in initial stimulation, subsequently leading to the inhibition of potassium-dependent processes. The inhibition of pyruvate kinase and succinate dehydrogenase disrupts the Krebs cycle and glucose metabolism, resulting in decreased ATP production, swelling, and vacuolization due to the impairment of the sodium-potassium ATPase. Thallium's strong capacity to form disulfide bonds interferes with the cross-linking of cysteine residues, leading to a decrease in keratin synthesis. Riboflavin sequestration caused by thallium and the inhibition of flavin adenine dinucleotide disrupts the electron transport chain, resulting in decreased ATP production [95].

Ribosomes are adversely affected by thallium, particularly impacting protein synthesis through damage to the 60S ribosome. The reduction in ribosome synthesis and its biogenesis results in the impairment of protein synthesis, blockage of the cell cycle progression, and apoptosis [96]. Thallium at high doses induces the degeneration of myelin in both the central and peripheral nervous systems [29]. Low-dose exposure is associated with mitochondrial dysfunction, neurite shortening, a loss of substrate adhesion, and an increase in cytoplasmic calcium [97,98].

The toxicokinetic phases of thallium are divided into three distinct stages: (1) the Intravascular Distribution Phase, during which, in the first 4 h following exposure, thallium is distributed to organs through the bloodstream; (2) the CNS Distribution Phase, in which, over a period of 4 to 48 h, thallium is distributed within the central nervous system; and (3) the Elimination Phase that commences approximately 24 h after exposure and is primarily facilitated through renal excretion and fecal elimination. This phase is gradual and may require up to 30 days for completion [95]. The analysis of thallium accumulated in hair by laser ablation inductively coupled plasma mass spectrometry (LA-ICP-MS) allows for the reconstruction of the exposure for forensic purposes [62].

Acute exposure symptoms include gastrointestinal manifestations, which may arise within 3–4 h. These include abdominal pain, nausea, vomiting, diarrhea, or constipation, with rare instances of blood in the vomitus or stools. Neurological symptoms typically present 2–5 days post-exposure and may include ascending peripheral neuropathies, distal motor weakness, ataxia, tremor, cranial nerve palsies, headache, seizures, insomnia, and coma, potentially leading to death. Ocular symptoms may involve diplopia, ptosis, seventh cranial nerve palsy, nystagmus, optic neuropathy, and lens opacities. Dermatological symptoms can present early as non-specific scaling and acneiform or pustular eruptions, followed by alopecia due to the disruption of cysteine disulfide bonds, and also as late manifestations such as Mees lines on nails, hypohidrosis, anhidrosis, and painful glossitis. Chronic exposure leads to the continuation of the aforementioned effects. In chronic poisoning, neurological symptoms may persist despite a decrease in blood thallium levels [95].

5.2. Our Observations

Our clinical case matches those of other cases that have been documented in the literature, where the presence of a triad has been demonstrated, raising the possibility of thallium intoxication. This triad includes alopecia, signs of motor or sensory neurological impairment, and abdominal pain [98]. However, alopecia appears, as we have seen, several days after the onset of the clinical picture, which is initially characterized by paresthesia and sharp pain. Following thallium exposure, the presentation pattern typically includes vomiting, nausea, and abdominal pain in the first few hours after exposure, peripheral sensorimotor neuropathy after four or more days, and generalized alopecia weeks later.

Cases of accidental poisoning have been reported recently [99], as well as cases of attempted suicide [31] and criminal intoxication in different countries, including Italy [59,100–102]. A case of acute, nonintentional thallium poisoning was due to thallium-contaminated alternative medicine [103]. However, reports of poisoning were more frequent in the past. Thallium poisoning during pregnancy may have fetal effects ranging from severe toxicity with residual sequelae to outwardly normal development [104]. A recent case of criminal intoxication with long-term misdiagnosis and neurologic outcomes has also been observed [65]. Another case of criminal intent was studied for three years in six different hospitals without reaching a diagnosis [66]. Chronic intoxication is sometimes suspected with the onset of alopecia [61]. However, since thallium is tasteless, odorless, and water-soluble, and can be absorbed through the skin, inhaled, or ingested, thallium

intoxication is frequently misdiagnosed, or the diagnosis is delayed. A retrospective analysis of cases with delayed admission showed significant symptoms associated with central nervous system damage and changes in magnetic resonance images and electroencephalograms [63]. Cases of severe neurological impairment with coma have been shown to respond positively to treatment; but the rarity of these occurrences prevents the generalizability of these observations [63].

Recently, an interesting report of a case of accidental thallium poisoning in a chemist who survived 10 years after intoxication has been published [68]. The first symptom was numbness in the feet; this rose up the legs, causing terrific pain. The paralysis included the digestive system, thus making the body unable to absorb food and causing cachexia. Severe nerve damage was followed by alopecia. Initially, a neurologist diagnosed Guillain–Barré syndrome and prescribed immunoglobulin treatment and plasmapheresis. This incorrect treatment had no effect, whereas the correct diagnosis and treatment with Prussian Blue resulted in a very slow improvement. Thallium was used in the laboratory, but there had been no occupational exposure; the metal had probably been ingested through beverages.

The early recognition of the diagnosis is generally the key to successful treatment, as was seen recently in reported cases of contaminated meat poisoning [19]. Cases of delayed diagnosis have an unfavorable outcome, as in the one recently reported by Zou [105]. A 41-year-old male was admitted with acute polyneuropathy and abdominal pain. The patient received treatment for suspected Guillain–Barré syndrome and subsequently for autoimmune encephalopathy. Over the following 42 days, he exhibited progressive muscle weakness, delirium, and alopecia, ultimately leading to a diagnosis of thallium toxicity. After treatment with a combination of Prussian Blue, activated charcoal, and continuous venous hemofiltration, the patient showed improvement; however, neuropsychiatric and neuromuscular sequelae persisted. This case demonstrates that delaying a diagnosis until symptoms have evolved can compromise the possibility of recovery and, probably, the life of the patient. The decision to proceed with the administration of Prussian Blue was taken before obtaining confirmation of water pollution and poisoning. This ex juvantibus criterion should be followed in all cases in which the clinical picture is compatible with thallium poisoning because Prussian Blue is not very toxic and induces an improvement that can be the best guide for the clinician.

Even in a medically advanced country like Italy, and in a hospital that, for decades, has had a Poison Control Center, of which the first author of this article is the Director, it is not easy to obtain a water dosage and a toxicological analysis within a period of time compatible with the survival of a thallium-poisoned patient. This is the main reason that leads us to stress the need to start immediate treatment for cases that could be caused by thallium intoxication, before the diagnosis of thallotoxicosis is confirmed. The rapid relief of painful symptoms and a pause in the progression of neurological damage are important indicators that enable us to calmly await the results of toxicological tests confirming the diagnosis. Conversely, in cases where the symptoms are due to a different cause, a few days of administration of a drug of low toxicity prevents the patient from running excessive risks.

Lowering blood levels as soon as possible is the main objective when treating acute thallium poisoning [55]. Prussian Blue functions as a univalent cation exchanger that improves fecal clearance, blocks the enterohepatic circulation of thallium, and preferentially binds unabsorbed thallium in the gut. Activated charcoal, which is often administered alongside Prussian Blue, improves fecal removal while simultaneously adsorbing thallium. Activated charcoal and Prussian Blue are both recognized therapies; however, Prussian Blue is the standard of care because of its greater safety and effectiveness [106]. Nevertheless, these therapies cannot eliminate absorbed thallium that has reached the

bloodstream. Prussian Blue cannot sequester thallium outside of the digestive tract, and thallium has a lengthy physiological half-life. Consequently, additional research has suggested combining Prussian Blue with blood purification procedures, including continuous veno venous hemofiltration, hemoperfusion, and hemodialysate, that use extracorporeal filtration devices to eliminate thallium from the bloodstream [107,108]. Animal studies suggest that the effect of PB may be increased by the concomitant administration of an ion exchange resin (calcium polystyrene sulfonate) [109].

The recommendation to use Prussian Blue for the emergency treatment of patients with symptoms such as those described above can encounter two main obstacles: firstly, the emergency physician must be aware of thallium poisoning and its treatment and suspect the disease, and, secondly, it is essential that the drug be available. Drug availability can be a problem since the limited number of cases is an obvious impediment to the hospital supply of the drug. The lack of the drug can be a serious problem, especially in countries where the probability of thallotoxicosis is higher [50,60]. We therefore recommend that doctors working in peripheral hospitals contact larger hospitals with clinical toxicology services. The lack of knowledge about the effects of thallium and the clinical picture of intoxication can be effectively countered by referring to facilities, such as the Poison Control Center of the Gemelli General Hospital, that are able to provide advice in doubtful cases.

The case presented also raises the problem of hygiene in homes. Italian law prohibits the introduction of non-potable water into the domestic water supply, and a dwelling with the characteristics described in the case reported is deemed unfit for habitation. It is surprising that the person who was poisoned had a degree in a subject that requires knowledge of the toxic effects of thallium, and it is difficult to understand how one could live for years in such a high-risk situation. The slow rate at which urinary and plasma thallium levels decreased (the latter failed to reach normal limits even after 120 days of treatment) suggests that, besides the acute exposure episode during which the patient drank contaminated water for the entire week of her husband's absence, there could have been previous chronic low-level exposure due to contaminated water used for washing or watering vegetables.

5.3. Implications for Physicians and Policymakers

Given the severe outcomes associated with the delayed diagnosis of thallium poisoning, it is essential that we standardize the protocols for the emergency management of suspected cases. The rapid access to Prussian Blue and hemoperfusion should be prioritized in emergency settings, particularly in regions with higher thallium contamination risks. Training and awareness programs for emergency physicians could enhance the recognition of early symptoms, especially priority indicators like peripheral neuropathy and abdominal pain; alopecia may have a delayed onset. Furthermore, initiating treatment with Prussian Blue as an "ex juvantibus" approach, even before toxicological confirmation, is advisable due to its low toxicity and proven efficacy in reducing thallium levels. For policymakers, the implementation of surveillance and control measures as well as guidelines for clinical practice could significantly improve patient outcomes and reduce the incidence of long-term complications.

5.4. Study Limitations

This study has several limitations that should be considered when interpreting the findings. Firstly, the rarity of documented thallium poisoning cases limits the generalizability of the results, as the cases reviewed may not fully represent the variability in clinical presentation and treatment response across different populations. Additionally, the fragmented nature of the available data restricts the depth of analysis and prevents the

application of a fully systematic review approach, imposing the use of a scoping review. Although this methodology is suitable for mapping the available evidence, it does not allow for a rigorous quality assessment of each study, and, therefore, potentially introduces bias. Lastly, the absence of long-term follow-up data in some cases limits insights into the chronic effects of thallium poisoning and the durability of Prussian Blue® efficacy in preventing sequelae. Future research with larger sample sizes and a more comprehensive follow-up would be valuable for confirming these preliminary findings and enhancing clinical guidance.

6. Conclusions

In conclusion, thallium poisoning, although rare, should be considered in cases presenting with paresthesia, progressive neurological deficits, and severe osteomuscular pain in the abdomen and thorax. Prompt treatment with Prussian Blue not only offers the potential for rapid symptom relief but also serves as a therapeutic confirmation of the diagnosis. In the literature, early treatment with Prussian Blue is associated with a better patient outcome and lower incidence of permanent neurological deficit. Given the challenges in obtaining a prompt diagnosis, heightened awareness and standardized protocols are essential in order to enhance the clinical response and minimize the risk of long-term complications.

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