

REVIEW ARTICLE

Clinical and Forensic Aspects of Aluminum Phosphide Poisoning: An Update and Review of Suicidal, Homicidal, and Accidental Poisoning Cases

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Abstract

Background: Aluminum phosphide (AIP) is a cheap and broad-spectrum fumigant that remains one of the leading agents of fatal poisoning. It is also associated with forensically important homicidal and accidental/occupational poisoning. This narrative review synthesizes recent clinical, forensic toxicological, and medico-legal data on AIP poisoning, with specific attention to the cautious differentiation of suicidal, homicidal, and accidental circumstances when reconstructing the circumstances of death.

Methods: A literature search was conducted in PubMed/MEDLINE, Scopus, ScienceDirect, Google Scholar, and the Directory of Open Access Journals (DOAJ) for English-language publications from 1st January 2010 to 1st August 2026, using the combinations of the terms "aluminum/aluminium phosphide", "phosphine", "rice tablet", "celphos", "forensic toxicology", "autopsy", "suicide", "homicide" and "accidental poisoning". Original research, case reports, systematic reviews, and meta-analyses published in indexed journals were prioritized. Non-peer-reviewed sources and pre-2010 publications were excluded.

Results: Sixty-four sources meeting the eligibility criteria were retained. The reviewed literature confirms AIP homicidal and accidental poisoning are less common but forensically significant. These types of AIP poisoning are arising from covert administration, occupational fumigation, warehouse storage, and pediatric exposure. Autopsy findings are frequently nonspecific. Confirmatory diagnosis based on headspace gas-chromatographic with nitrogen-phosphorus or mass-spectrometric detection.

Conclusion: AIP poisoning remains a major clinical, forensic and public-health problem in developing countries. Systematic autopsy protocols, prompt toxicological confirmation, and careful reconstruction of scene evidence are essential to correctly AIP fatal poisoning cases. Cause of death and manner of death should be determined separately and through integration of the totality of available evidences.

Keywords: Aluminum phosphide; Phosphine; Suicide; Homicide; Accidental poisoning

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INTRODUCTION

Aluminum phosphide (AIP) is a cheap, solid and highly effective fumigant pesticide extensively used for grain preservation in many developing countries in South and Southeast Asia, the Middle East, the Balkans, and parts of Africa particularly in India, Iran, Egypt and Türkiye [1, 2]. It sold under different trade names such as Phostoxin® and street names as "rice tablet" or "wheat tablet" in Iran [2, 3]. When ingested or exposed to water/moisture in atmospheric humidity or gastric hydrochloric acid in stomach, AIP

liberates phosphine (PH₃) gas, a potent cellular toxin that inhibits cytochrome C oxidase and disrupts mitochondrial oxidative phosphorylation, resulting in multi-organ failure and death [4-7].

Ready availability, rapid onset of toxicity, and the absence of a specific antidote have made AIP one of the most common tool of fatal AIP self-poisoning in developing countries [6, 8-10].

From a forensic standpoint, AIP poisoning occupies a distinctive position among toxic agents. Unlike many poisons that produce specific, easily recognized

histopathological signatures at autopsy, phosphine toxicity is largely a story of nonspecific multi-organ congestion, and cardiovascular collapse, so that the forensic pathologist must rely heavily on scene investigation, circumstantial evidence, and toxicological confirmation rather than gross pathology alone [1, 11]. Furthermore, because AIP is cheap and unregulated in many jurisdictions, it is encountered not only in suicidal ingestion but also in accidental exposure, particularly among agricultural workers, warehouse staff, and family members inadvertently exposed to fumigated grain and, less frequently, in cases with genuine or alleged homicidal intent [2-4, 12-14]. The majority of reported cases are suicidal, in low- and middle-income countries. For example, Kumar et al. in a systematic review and meta-analysis showed that among 3449 Indian patients of AIP poisoning were included in the study, 94% of the cases were suicide, while accidental cases were observed only in 5% of cases [15]. In another study from Nepal, the researchers showed in a hospital record-based study includes 25 AIP poisoning patients presenting to the emergency department during 2017- 2020, most of them were female (60%) and married (56%). The case fatality rate was 84% and all poisoning cases were suicidal in intent [16].

The AIP accidental poisoning, mainly occupational-related is frequently reported due to improper storage, inhalation of phosphine during fumigation, or contamination of food and household environments [17-20]. For example, Hájek et al. described an accidental AIP poisoning case due to the fumigation of stored grain in an agricultural building near to the temporarily housed building of a patient, while both buildings were connected by an underground corridor. The released PH_3 gas was penetrated to the building through this way [18].

Forensic interpretation requires a clear distinction between the cause of death and the manner of death. AIP/phosphine poisoning may constitute the cause of death when adequately supported by the clinical, autopsy, circumstantial, and toxicological evidence. However, identification of the toxic agent does not, by itself, establish whether the death was suicidal, homicidal, accidental, or undetermined. Manner of death determination requires an integrated medico-legal assessment of the totality of the evidence.

Homicidal poisoning using AIP is rare in the published literature and the evidence base is substantially smaller than that for suicidal and accidental poisoning. The unpleasant odor often associated with commercial phosphine-releasing products and the rapid clinical course may further limit covert administration, but these features are neither sensitive nor specific [21]. Accordingly, any inference regarding homicidal intent must be made cautiously and should not be based on toxicological detection or autopsy findings alone. In all three circumstances, the cause of death and the manner of death should be considered as separate medico-legal determinations, integrating circumstantial, clinical, autopsy, and analytical toxicological findings.

The purpose of this review is to bring together in a single clinical and forensic-oriented synthesis, the current literatures on the chemistry and mechanism of AIP/phosphine toxicity, its global epidemiology, clinical course, the autopsy and histopathological features, the analytical methods used to confirm the diagnosis in living and deceased individuals, and the specific forensic considerations that arise in suicidal, homicidal, and accidental poisoning scenarios. Clinical management is summarized briefly in so far as it clarifies the pathophysiological basis of death and the postmortem findings; the emphasis throughout remains on the forensic and medico-legal dimensions of AIP poisoning.

Several narrative and systematic reviews have previously addressed the clinical toxicology or the treatment of AIP poisoning in isolation. The present review differs from that body of work in four respects. First, it is organized explicitly around the medico-legal question that most concerns the forensic practitioner (the differentiation of suicidal, homicidal, and accidental manners of death) rather than around organ-system toxicity alone. Second, it consolidates, for the first time in a single synthesis, the scene-investigation, autopsy/histopathological, and clinical and analytical-toxicology evidence bases that jointly support manner of death classification, including recent gas-chromatographic and machine-learning-assisted prognostic approaches [22-26]. Third, it provides an explicit comparative contrasting the epidemiological, circumstantial, and analytical features of suicidal, homicidal, and accidental AIP poisoning, intended as a practical reference for forensic practice. Fourth, the literature base was deliberately restricted to sources published from 2010 onward, ensuring that the synthesis reflects contemporary analytical methods (headspace GC-MS/MS, multi-kernel machine-learning mortality models) and updated epidemiological patterns rather than superseded data.

METHODS

Design

This article was prepared as a narrative (non-systematic) review, appropriate for synthesizing a heterogeneous body of forensic, clinical, and analytical toxicology literature that cannot readily be reduced to a single quantitative outcome measure. Where the underlying primary studies were themselves systematic reviews or meta-analyses, this is noted explicitly in the text.

Information sources and search strategy

A comprehensive literature search was performed in PubMed/MEDLINE, Scopus, ScienceDirect, Google Scholar, and the Directory of Open Access Journals (DOAJ), supplemented by hand-searching of reference lists of retrieved reviews. The search performed from 1st January 2010 to 1st August 2026 using combined the following keyword groups using Boolean operators: (i) substance terms including "aluminum phosphide", "aluminium phosphide," "phosphine," "metal phosphide", "zinc

phosphide", "rice tablet", "celphos"; (ii) forensic/clinical terms including "forensic toxicology", "autopsy", "postmortem", "histopathology", "medico-legal", "cause of death", "manner of death"; and (iii) circumstance terms including "suicide", "suicidal poisoning", "homicide", "homicidal poisoning", "accidental poisoning", "occupational exposure".

English-language, peer-reviewed original research articles, case reports and case series, narrative and systematic reviews, and meta-analyses addressing AIP or phosphine toxicity relevant to forensic, clinical, or analytical toxicology were eligible. Priority was given to indexed publications with verifiable bibliographic information and direct relevance to the claim being supported. Titles and abstracts were screened for relevance; full texts were reviewed when potentially eligible. Publications were excluded when they were non-peer-reviewed, lacked verifiable bibliographic information, or did not materially address the review question. Publications before 2010 were excluded during the final revision when an equivalent contemporary source was available; where an older source was retained because it documented a specific historical or forensic phenomenon not adequately represented in the post-2010 literature, this was treated as an exception rather than part of the general eligibility set.

Study selection and data synthesis

Titles and abstracts were screened for relevance to AIP/phosphine toxicity and forensic or clinical toxicology. Full texts of potentially relevant articles were reviewed, and data on mechanism of toxicity, epidemiology, clinical features, diagnostic methods, autopsy and histopathological findings, and manner of death classification were extracted narratively.

RESULTS

A total of sixty-four sources satisfying the eligibility criteria were incorporated into the final synthesis and are listed in the reference section.

Chemistry, Toxicokinetics, and Mechanism of Toxicity

AIP is a dark gray or dark yellow solid crystalline powder which remains stable up to 1000°C but is highly sensitive to moisture [1, 27]. AIP has been extensively utilized since 1940s in India as a solid fumigant to protect stored agricultural crops and buildings from pests and as a rodenticide [1]. It is typically formulated as tablets, pellets, granules or powder sachets. In India, AIP is marketed as a tablet under trade names such as Celphos®, Alphos®, Quickphos® and Phostoxin®. Also, in Iran it is known "rice tablet" as a common name [2-4]. This formulation is marked as in 3-gram tablets containing 56% AIP and 44% of ammonium carbamate as a stabilizer to control PH₃ gas release and prevent flammability [1,2]. This preparation can release 1g of PH₃ gas as a fumigation agent along with ammonia and carbon dioxide when reacting with water or moisture [1-3, 27]. PH₃ is a colorless, highly toxic, and

flammable gas with a garlic or rotting fish odor in commercial products but the pure PH₃ is odorless [1-3].

Aluminum phosphide reacts with moisture in air, water, or gastric hydrochloric acid to liberate phosphine gas together with aluminum hydroxide [6, 8, 27]. Under trace environmental moisture, phosphine can undergo further disproportionation to diphosphine (P₂H₄), a spontaneously flammable compound that accounts for occasional reports of spontaneous ignition of gastric contents during resuscitation, gastric decontamination or autopsy of AIP poisoned patients

[28-30]. Phosphine is a small molecule, lipophilic and colorless gas classically described as having a garlic- or decaying-fish like odor, which is itself an important, though not entirely reliable, clue at the scene of death and at autopsy [8, 24]. Once absorbed by ingestion of the tablet with subsequent intragastric gas release, or by direct inhalation of PH₃, the phosphine gas is rapidly distributed throughout the body and metabolized to phosphite and hypophosphite, which can themselves serve as analytical markers of exposure [1, 4]. The weight of contemporary mechanistic evidence points to non-competitive inhibition of mitochondrial cytochrome-c oxidase (Complex IV) of the electron transport chain, with consequent impairment of oxidative phosphorylation, depletion of adenosine triphosphate, and generation of reactive oxygen species that produce lipid peroxidation and depletion of cellular glutathione [1, 5, 31-33]. This combination of energy failure and oxidative stress preferentially injures tissues with high metabolic demand including myocardium, vascular endothelium, liver, kidney, and brain and explaining the characteristic clinical picture of refractory cardiogenic shock, multi-organ failure, and severe metabolic (lactic) acidosis that dominates the terminal course of severe poisoning [1, 32, 34-38].

Contemporary translational work has further framed AIP poisoning as a useful acute model of mitochondrial disease, given its reproducible inhibition of Complex IV, rapid bioenergetic collapse, and measurable oxidative-stress biomarkers, and has explored antioxidant and mitochondria-targeted strategies (N-acetylcysteine, myricetin, glutathione, sodium selenite, vitamin C and E, triiodothyronine, liothyronine, vasopressin, milrinone, 6-aminonicotinamide, boric acid, acetyl-L-carnitine and, melatonin, and nanomicelle-curcumin) intended to counter oxidative injury, although none has yet emerged as a validated antidote in clinical practice [36, 39-41].

Epidemiology

AIP poisoning is reported from agricultural, middle- and lower-income countries where the compound is inexpensive, minimally regulated, and stored in domestic and agricultural settings. Iran, India, Pakistan, Nepal, Ethiopia, Egypt, Saudi Arabia, and Albania have all reported substantial case series [2, 9, 12, 15, 42-44]. Broader narrative reviews of acute adult poisoning admissions in

Iran confirm that AIP consistently ranks among the leading causes of fatal poisoning nationally, alongside other pesticides and pharmaceuticals [2, 45]. Retrospective forensic autopsy series from Tehran documented a rising incidence of fatal AIP poisoning between 2006 and 2013, with self-poisoning accounting for approximately 85% of manner of death classifications [9], and a parallel forensic toxicology analysis of self-poisoning deaths in Tehran between 2011 and 2015 corroborated this trend and identified a predominance of young decedents [46]. Comparable Iranian cohorts from Kermanshah province likewise found that essentially all cases were intentional AIP self-poisonings, with blood pressure, arterial pH, and the interval between ingestion and treatment emerging as principal predictors of mortality [10].

Systematic review and meta-analytic data from Iran estimate a pooled mortality of approximately 40% among treated patients, with non-survivors skewing male and slightly older than survivors [47]. A five-year clinical series from Tirana, Albania, documented that fatalities more than doubled between 2009 and 2013, attributing the trend to unrestricted market availability, low cost, and a cluster effect following media coverage of individual cases [42]. A nine-year Saudi Arabian series and Pakistani retrospective cohorts confirm a broadly similar demographic pattern of young-adult, intentional self-poisoning across culturally and geographically diverse settings, indicating that the phenomenon is not confined to a single region [43, 48-51]. Toxicoepidemiological surveys from southern India, a regional demographic profile from Gandhinagar, Gujarat, and broader systematic reviews of pesticide-related lethal poisoning in India and Pakistan confirm that metal phosphides rank among the leading causes of fatal self-poisoning nationally, alongside organophosphates, and that regulatory restriction of pesticide sale has measurably reduced suicide mortality in some jurisdictions [43, 49, 52, 53]. A World Health Organization Eastern Mediterranean regional analysis similarly identifies pesticide ingestion, including AIP, as among the most common suicide methods in the region [54].

Occupational and accidental exposures, while numerically smaller, are recurrent in the literature. Cases include warehouse or transport-company workers exposed to phosphine released from improperly stored fumigant tablets, agricultural fumigators, and bystanders inadvertently exposed to treated grain or storage containers [12, 13, 55]. Pediatric poisoning, whether from accidental ingestion of tablets left within reach or from environmental exposure to fumigated dwellings, has also been documented and carries an especially poor prognosis given lower physiological reserve [56].

Clinical Manifestations

The clinical presentations of AIP poisoning is characteristically rapid and dominated by gastrointestinal and cardiovascular features. Early symptoms, appearing within minutes to a few hours of ingestion, include nausea,

vomiting, epigastric pain, and a sensation of chest tightness, often accompanied by the characteristic garlicky breath odor [6, 57]. Severe cases progress rapidly to profound, catecholamine-refractory hypotension, cardiogenic shock, and life-threatening arrhythmias, reflecting the marked cardiotoxicity of phosphine described in contemporary mechanistic and electrocardiographic studies [1, 5, 23, 58].

Metabolic disturbances are prominent and prognostically important. Severe anion-gap metabolic (lactic) acidosis, hypomagnesemia, and adrenocortical insufficiency have all been associated with a fatal outcome, and arterial blood gas and serum cortisol values have been validated as mortality predictors in prospective cohorts [34, 38, 58]. Composite prognostic tools, including shock indices and dedicated poisoning mortality scores, and machine-learning models trained on admission variables, have more recently been developed to stratify risk at presentation [22, 23, 35, 59]. Acute kidney injury, hepatocellular injury, and adult respiratory distress syndrome frequently complicate the clinical course in patients who survive the initial cardiovascular insult, and neurological manifestations, ranging from agitation and reduced consciousness to frank coma, are common in critically ill patients [5, 34]. Because these findings are systemic and nonspecific, and because rapid deterioration frequently precludes extensive investigation, a high index of clinical suspicion prompted principally by history of exposure or the characteristic odor remains central to timely diagnosis [6, 34].

Diagnosis and Forensic Toxicological Analysis

In living patients, diagnosis is based principally on a history of exposure, corroborating scene evidence (such as a tablet container), compatible clinical findings, and, where available, presumptive testing such as the silver-nitrate test on gastric aspirate or exhaled breath. Silver-nitrate-impregnated paper darkens on contact with PH_3 . This presumptive test is rapid but non-quantitative and may produce false-negative results, particularly when the analyte has dissipated or the specimen is diluted [26, 34].

Confirmatory analytical methods

Definitive forensic confirmation requires detection and quantification of phosphine or its metabolites (phosphite, hypophosphite) in gastric content, blood, or tissue. Headspace gas chromatography coupled with a nitrogen-phosphorus detector (HS-GC-NPD) or with tandem mass spectrometry (HS-GC-MS/MS) is the most widely validated approach, offering sensitivity to sub-microgram-per-milliliter concentrations and applicability to postmortem specimens, including putrefied or exhumed material [24-26]. In a forensic case series of seven fatal pediatric poisonings, total PH_3 concentrations in postmortem fluids and tissues ranged from approximately 0.2 to 4.7 micrograms per milliliter, illustrating both the analytical feasibility and the wide biological variability of measured concentrations [24]. A separate two-case analysis quantified phosphine at 264 ng/mL in gastric contents and 30 ng/g in liver in one decedent, and 21, 5, and 268 ng/mL in blood,

urine, and gastric lavage respectively in a second, non-fatal case, using GC-NPD with a headspace zinc/sulfuric-acid derivatization step [26]. Validated tandem mass-spectrometric methods with multiple-reaction monitoring have extended this capability to veterinary and other unusual matrices, which is of particular value in delayed-discovery homicide or accidental-death investigations [25].

Because phosphine is volatile and prone to loss from decomposing tissue, forensic protocols should emphasize prompt, airtight headspace sampling at autopsy and collection of multiple appropriate specimen types (including gastric content, blood, liver, and lung) to maximize the likelihood of detection [24, 26]. The concentration measured postmortem may decrease with delay, inadequate sealing, inappropriate storage or transport, decomposition, and other pre-analytical factors. Therefore, a negative or low phosphine concentration should not be interpreted in isolation as excluding AIP exposure. Interpretation should consider the sampling interval, specimen type, preservation conditions, analytical method, and the totality of scene, autopsy, clinical, and investigative evidence. Toxicological confirmation can strongly support the cause of death when positive, but determination of manner of death remains a separate integrated medico-legal judgment.

Autopsy and histopathological findings

Gross autopsy findings in fatal AIP poisoning are frequently nonspecific but, taken together with scene and historical information, are strongly supportive of the diagnosis. A recent systematic review and meta-analysis of observational autopsy series comprising more than two thousand postmortem examinations found that congestion of the respiratory system was present in nearly all reported cases, alongside a distinctive garlicky or fish-like odor on opening the stomach and, in a minority of cases, an unusual greenish visceral discoloration whose mechanism remains incompletely elucidated [11]. Gastric mucosal congestion, hemorrhage, and partial loss of shrinkage are common, explicit mucosal necrosis is reported with variable frequency across series [1, 11, 60].

Histopathological examination consistently demonstrates centrilobular hepatic congestion and, in more severe cases, hepatocellular necrosis, sinusoidal dilatation, and mononuclear infiltration; pulmonary findings include alveolar wall thickening, intra-alveolar hemorrhage, and pulmonary edema; renal specimens show glomerular and interstitial congestion with acute tubular necrosis. In the myocardium may show focal necrosis consistent with the severe cardiotoxic component of the syndrome [1, 11]. Adrenal cortical involvement, ranging from mild lipid depletion to severe hemorrhagic necrosis, correlates with impaired cortisol response and a worse prognosis in several series [11, 58]. Cerebral changes are generally limited to congestion and nonspecific neuronal degeneration attributable to hypoxic-ischemic injury rather than a direct neurotoxic action of phosphine [1, 11].

From a forensic pathology standpoint, the principal value of these findings lies not in specificity; since congestion and hypoxic organ injury are common to many causes of rapid cardiovascular collapse, but in their consistency with a diagnosis of phosphide poisoning when combined with a compatible history, scene evidence, and positive toxicological confirmation. Case reports of spontaneous ignition of gastric aspirate during suction, attributable to release of pyrophoric diphosphine, represent a distinctive and hazardous phenomenon of direct relevance to autopsy and emergency department safety protocols [28, 29].

DISCUSSION

Suicidal Poisoning

Intentional self-poisoning constitutes by far the largest share of fatal AIP exposures reported in the forensic and clinical literature. Iranian, Indian, Pakistani, Nepali, Ethiopian, Albanian, and Egyptian series consistently report that 85% to essentially 100% of AIP fatalities are attributable to deliberate self-poisoning, most often precipitated by interpersonal or family conflict, and disproportionately affecting adolescents and young adults [9, 10, 12, 13, 43, 44, 61, 62]. The availability of AIP tablet as a household or agricultural product, its low cost, and the popular (frequently mistaken) perception that death will be rapid and free of the disfigurement associated with some other means of suicide, are recurrently cited as contributing to its selection as a method [6, 42, 63]. Cross-sectional studies of pesticide self-poisoning more broadly, including large cohorts from China, confirm that the ready accessibility of a lethal agent is a stronger predictor of method choice than any specific psychiatric diagnosis, a finding directly relevant to prevention strategy [64].

From the forensic investigator's perspective, suicidal AIP poisoning is usually established through a convergent pattern of evidences including a scene consistent with self-administration (tablet containers near the body, absence of signs of restraint or struggle), a plausible psychosocial precipitant, a compatible clinical or postmortem course, and positive toxicological confirmation of phosphine or its metabolites [8, 26]. Regulatory bans or restrictions on AIP sale, introduced in Iran and studied in Pakistan following pesticide regulation reform, have in some analyses been associated with only a partial and sometimes temporary reduction in incidence, underscoring that legislative restriction alone, without concurrent public education and mental-health/suicide-prevention measures, may be insufficient to substantially reduce suicide by this method [9, 44].

Homicidal Poisoning

True homicidal AIP poisoning is considerably rarer than suicidal or accidental exposure in the published literature, and the evidence concerning homicide is substantially limited, consisting predominantly of isolated case reports or small case-based observations. This limited evidence base

should be explicitly recognized when interpreting alleged homicidal poisoning, and conclusions should not be generalized beyond the documented cases. Reported scenarios include covert administration of AIP in food or drink and initially unexplained deaths in which toxicological analysis subsequently supported phosphide exposure [26].

Because AIP produces a clinical and autopsy picture that overlaps substantially with several other causes of rapid cardiovascular collapse, and because the characteristic odor may dissipate or be masked in outdoor or ventilated settings, homicidal poisoning can present a genuine diagnostic challenge, particularly when the poison is administered without the victim's knowledge and no tablet residue is recovered at the scene [24,26].

Forensic pathologists and toxicologists must therefore maintain a high index of suspicion in unexplained sudden deaths, particularly where scene investigation reveals third-party access to AIP, and must ensure that gastric content, blood, and visceral tissue are systematically collected and analyzed even when the initial impression favors a natural or accidental cause [13,26]. Cases in which multiple individuals sharing a meal or environment are simultaneously affected warrant particular scrutiny to differentiate mass accidental exposure (for example, from fumigant off-gassing) from deliberate poisoning of a group [13].

Accidental and Occupational Poisoning

Accidental exposure to phosphine arising from AIP is a well-documented, if less frequent, category of forensic case. It typically involves occupational or environmental release of gas from improperly stored or handled fumigant rather than deliberate ingestion. A forensic case series described the deaths of two warehouse workers who slept in a room where AIP tablets had been stored in cartons; a running cooler intensified air circulation and drew phosphine into the sleeping area, and meticulous crime-scene investigation was required to exclude homicide before an accidental mechanism was established [13]. Comparable reports describe fatal pediatric exposure related to residential proximity to fumigated grain stores, occupational exposure among fumigation workers and agricultural laborers, and accidental poisoning with the related compound zinc phosphide, which shares the same phosphine-release mechanism and toxicological profile [14, 24, 65, 66].

Pediatric accidental poisoning, whether from ingestion of tablets inadvertently left accessible in the household or from inhalation of phosphine in poorly ventilated storage areas, is repeatedly documented and carries a particularly guarded prognosis given children's smaller physiological reserve and the difficulty of early recognition [14, 67, 68].

The forensic distinction between accidental and homicidal exposure in these scenarios depends heavily on reconstruction of the physical environment, including ventilation, proximity to stored fumigant, presence or absence of intact packaging, and the possibility of environmental phosphine release, together with

toxicological confirmation and, where relevant, environmental air or residue sampling. These features are supportive rather than definitive; the final manner-of-death determination requires integration of all available evidence. Occupational exposure cases additionally raise questions of regulatory compliance and workplace safety that extend beyond immediate cause-of-death determination into broader public-health and occupational-medicine considerations [13].

Comparative overview of suicidal, homicidal, and accidental AIP poisoning

Table 1 summarizes the epidemiological, circumstantial, and forensic analytical features that may help differentiate suicidal, homicidal, and accidental AIP poisoning. These characteristics are supportive findings rather than definitive diagnostic criteria for manner of death. Individual cases may present with overlapping or incomplete features, and classification ultimately depends on the totality of scene, historical, autopsy, toxicological, and investigative evidence.

Figure 1 demonstrates a comprehensive and integrated approach on AIP poisoning from molecular mechanisms, clinical and forensic toxicology pathway and manner of death classifications.

Figure 2 demonstrates a practical forensic algorithm for suspected AIP poisoning from scene of death to classification of manner of death.

Clinical Management (Forensic Relevance)

Although a detailed treatment protocol lies outside the primary forensic scope of this review, an understanding of the therapeutic course is relevant because it shapes both the clinical trajectory preceding death and, in survivors, the residual organ injury that may itself become the subject of medico-legal disability assessment. No specific antidote exists for AIP/phosphine poisoning and management remains fundamentally supportive including gastric decontamination, aggressive correction of metabolic acidosis, vasopressor and inotropic support for refractory shock, and intensive cardiac monitoring for life-threatening arrhythmia [37, 69].

Intravenous magnesium sulfate remains the most extensively studied adjunctive therapy, valued for its membrane-stabilizing, antiarrhythmic, and antioxidant properties, although a formal survival benefit remains debated across published protocols [37, 69]. Additional measures reported in recent case series and experimental studies include N-acetylcysteine and other antioxidants (myricetin, nanomicelle-curcumin), vasopressin and milrinone for cardioprotection, and, in refractory cardiogenic shock, mechanical circulatory and respiratory support such as extracorporeal membrane oxygenation, for which a recent systematic review and meta-analysis suggests a possible mortality benefit in selected severe cases [31, 36, 39, 40, 49]. Despite these interventions, mortality in severe poisoning remains high due to the overall clinical

Table 1. Comparative features of suicidal, homicidal, and accidental/occupational aluminum phosphide poisoning

Feature	Suicidal Poisoning	Homicidal Poisoning	Accidental / Occupational Poisoning
Relative frequency	Overwhelming majority of fatal cases (~85–100% in most regional series) [8, 9, 10, 13, 20]	Rare; isolated case reports rather than case series [40]	Uncommon but recurrent, especially occupational/warehouse exposure [12, 40–44]
Typical victim profile	Adolescents/young adults, often female-predominant in some series, history of psychosocial conflict [9, 36, 62, 64]	Variable; determined by relationship to perpetrator rather than a demographic pattern [13]	Agricultural/warehouse workers, fumigators, and children in fumigated dwellings [12, 14]
Typical route	Oral ingestion of tablets/pellets [6, 9]	Covert oral administration in food/drink, or confined-space gas exposure [13]	Inhalation of ambient phosphine from stored/fumigated grain; occasional ingestion by children [12, 14]
Scene evidence	Tablet containers near the body; absence of struggle [9, 44]	Absent or removed tablet residue; possible signs of restraint/struggle; access to AIP by a third party [13]	AIP tablets/cartons in storage areas; poor ventilation; shared exposure of several victims [13, 14]
Key forensic challenge	Distinguishing intent from accidental exposure when scene evidence is ambiguous [9, 44]	Excluding natural/accidental causes in an unexplained death; odor may dissipate before discovery [13, 24]	Differentiating mass accidental exposure from deliberate poisoning of a group; reconstructing ventilation/exposure pathway [13]
Essential confirmatory step	Toxicological confirmation (silver-nitrate test, HS-GC-NPD/MS) plus psychosocial history [36, 38]	Systematic toxicology of gastric content, blood and viscera even when odor/scene clues are absent [36, 38]	Environmental/air sampling and reconstruction of storage/ventilation, plus toxicology [37, 44]

HS-GC-NPD/MS: Headspace gas chromatography coupled with a nitrogen-phosphorus detector (HS-GC-NPD) or mass spectrometry

Aluminum Phosphide (AIP) Poisoning: Mechanism, Forensic Pathway, and Manner-of-Death Classification

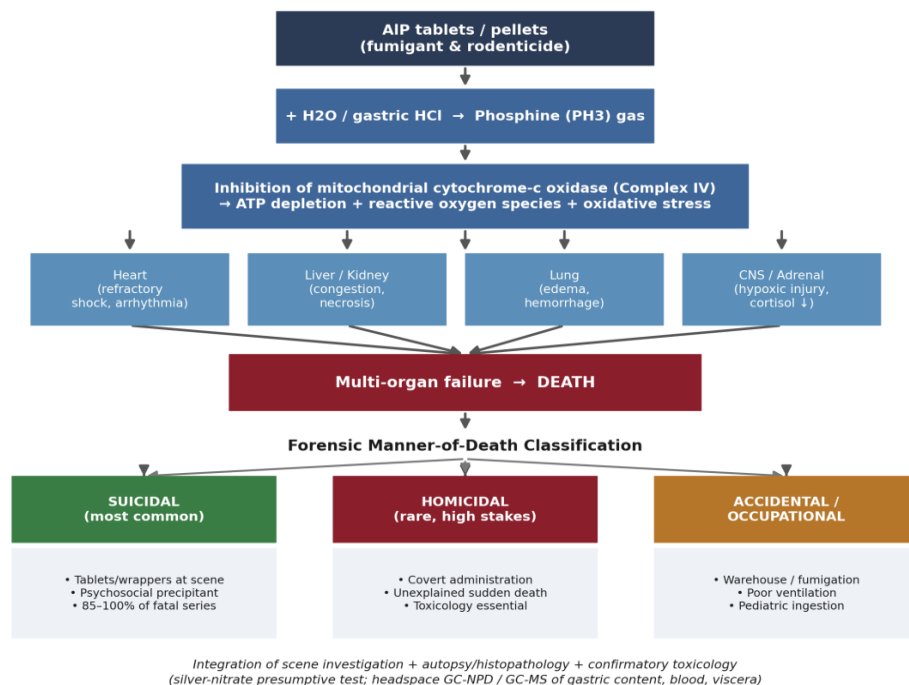


Figure 1. Schematic overview of aluminum phosphide toxicokinetics, the mitochondrial mechanism of injury, the resulting multi-organ failure, and the three forensic manners of death (suicidal, homicidal, accidental/occupational), together with the evidence streams used to confirm each. Generated by Artificial Intelligence [Claude (Anthropic, San Francisco, CA, USA)].

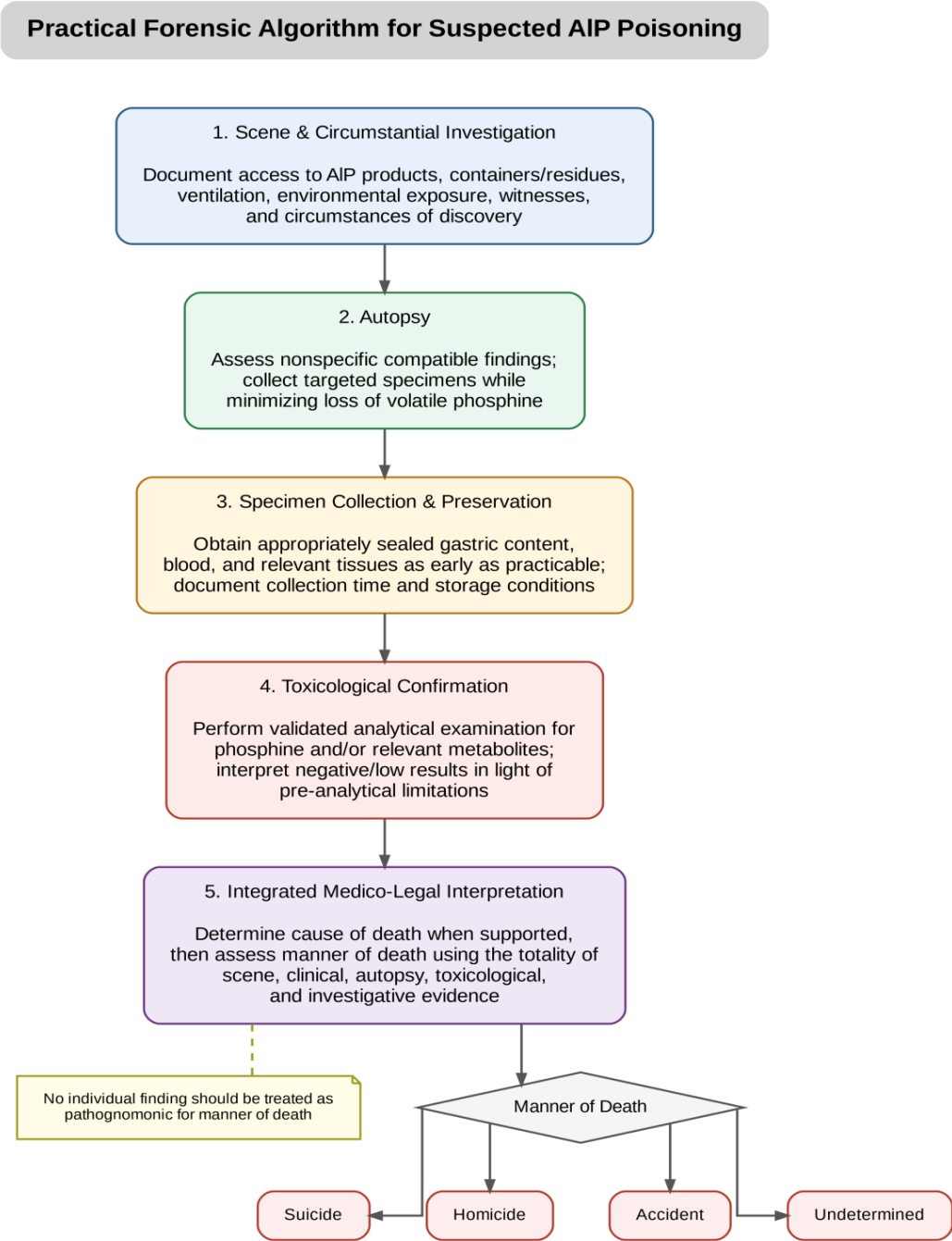


Figure 2. Practical forensic algorithm for suspected aluminum phosphide (AIP) poisoning from scene of death to classification of manner of death. Generated by Artificial Intelligence [Claude (Anthropic, San Francisco, CA, USA)]

course (rapid, refractory and multi-system) [5, 47].

Prevention and regulatory aspects

Because no reliable antidote exists and mortality remains high even with aggressive supportive care, prevention is widely regarded as the most effective strategy for reducing

the burden of fatal AIP poisoning. Measures proposed and, in some jurisdictions, implemented include restricting over-the-counter sale of AIP tablets to licensed pest-control operators, mandating tamper-resistant and smaller-quantity packaging, requiring secure storage separate from living

quarters, and public education on the hazards of fumigant off-gassing in enclosed or poorly ventilated spaces [1, 11, 42]. Analyses from Iran and Pakistan suggest that legislative restriction alone produced only partial and sometimes temporary reductions in poisoning incidence, and that sustained impact requires integration with broader suicide-prevention strategies, given that most fatalities are intentional [10, 43].

From an occupational safety perspective, enforcement of fumigation protocols such as adequate ventilation, exclusion periods before re-entry, and use of personal protective equipment together with training of agricultural and warehouse personnel, is essential to reduce accidental and occupational exposure of the kind documented in warehouse-related fatalities [1, 13]. Forensic and public-health authorities have an important complementary role in systematically documenting AIP-related deaths through medico-legal autopsy registries, which not only inform manner-of-death determinations in individual cases but also generate the epidemiological data necessary to evaluate the effectiveness of regulatory interventions over time [9, 11].

This review has several limitations that should be considered when interpreting its conclusions. First, as a narrative rather than systematic review, study selection was not subject to dual independent screening or formal risk-of-bias assessment, and the synthesis may therefore be susceptible to selection emphasis on more accessible or more frequently cited sources. Second, most of the underlying epidemiological literature originates from a relatively small number of high-incidence countries (chiefly Iran, India, Pakistan, and Egypt), which may limit generalizability. Third, the deliberate emphasis on post-2010 literature may omit some historically important descriptive and mechanistic studies. Finally, the evidence concerning homicidal AIP poisoning is particularly limited and is dominated by case reports, which restricts generalization. In addition, postmortem phosphine analysis has important pre-analytical and analytical limitations because phosphine is volatile and may be lost or altered after sampling. Consequently, a negative or low postmortem concentration cannot necessarily exclude exposure, and toxicological results should be interpreted together with specimen type, collection interval, preservation and storage conditions, analytical method, and the overall forensic evidence.

CONCLUSION

AIP poisoning remains a leading cause of fatal self-poisoning in several agricultural and rural regions and a recurrent, if less common, cause of accidental and occupational death. Homicidal AIP poisoning is an infrequent but forensically consequential possibility for which the published evidence is limited. The pathophysiology of phosphine, including mitochondrial disturbances leading to refractory cardiogenic shock,

metabolic acidosis, and multi-organ failure, produces a clinical and autopsy picture that is systemic and often nonspecific. Determination of the cause of death requires careful scene investigation, thorough autopsy with targeted sampling, and appropriately collected specimens for confirmatory chemical analysis when feasible. Determination of the manner of death is a separate medico-legal judgment and requires integration of circumstantial, clinical, histopathological, toxicological, and investigative evidence; no single finding is pathognomonic. Continued epidemiological surveillance, harmonized autopsy and analytical protocols, and appropriate regulatory measures represent important strategies for reducing mortality attributable to this highly lethal compound.

Declarations

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Ethical approval

Not applicable; this article is a literature review and did not involve primary data collection from human or animal subjects.

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