REVIEW ARTICLE

The Chronicle of Arsenic Poisoning in the 19th Century

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INTRODUCTION

Arsenic (As) exposure is a worldwide health problem, although overdose with arsenic, in its classic term, has almost disappeared in the 21st century in high income countries. The magnitude of this problem has been shifted towards environmental long-term exposure rather than acute overdose (1-3).

Old literature related to arsenic-induced medical and public health problems are certainly enlightening from a historical point of view. In addition, in cases of vanished toxicological diseases, they may have some implications even today such as their clinical manifestations.

In this study, medical literature related to arsenic poisoning in the 19th century was searched and finally 89 articles were included. Obsolete clinical applications of arsenic, mechanisms of toxicity, accidental (food, iatrogenic, others), intentional and homicidal overdoses, and development of forensic information, occupational and environmental toxicology, public health and legislation, laboratory detection methodologies, clinical findings and treatment of arsenic poisoning in the 19th century are discussed in this article.

Arsenic was in use for clinical purposes, and accidental and homicidal human overdoses were particularly common in the early to mid-19th century. Arsenic poisoning received major attention following the highly publicized and controversial conviction of Marie-Fortunée Lafarge (1816 - 1852) for murdering her husband in 1840, which was confirmed based on forensic toxicological evidence (4-6).

It seems that arsenic poisoning in 19th century was predominantly accidental and food-related. Homicides were also frequently reported. Occupational and environmental toxicities related to arsenic were born in the middle and late 19th century. Public policies and forensic medicine were developed in this century and treatment of arsenic poisoning was expanded to include chemically developed antidotes.

Clinical application of arsenic

Applying arsenic for medical purposes was common in the 19th century, including reports for *malignancy* by Barton(1806), Littlep (1808), Crane (1839), and others, *leprosy* and *headache* by Johns (1832) and *cholera by* Martin (1813) (7-15). It has also reported that arsenic has been successfully employed in 200 chorea cases with no side effect by Reese (1840) and Black and Sarjeant (1857) (16-18)

Arsenic was also reported to be useful in treating *ulcer of the tongue* (19). Jones (1832) claimed that he had controlled neuralgic-related *angina pectoris* by using arsenic (14). Arsenic was also used for *removal of skin complications of psoriasis* by Jones (1843) and treating *furunculus* (1848), *scabies* by Williams (1853), *asthma* by Julius (1861) and Wahltuch 1877, *gastric pain* by Leared (1867), *menorrhagia* by Aveling (1872), *cardiac tone* by Lockie (1878), *skin diseases by* Farquharson (1880), *gastric ulcer* by Strahan (1884), *malaria* by Drummond (1884) and *anemia* by Wiles (1885) and Barton (1891) (20-32).

Mechanisms of toxicity

Potential mechanisms related to the toxicity of Arsenic were not clear until the 19th century. Wright (1829) and later Orfila (1831) attributed the cause of death in overdosed subjects to intestinal inflammation (33-35). Later Greening (1835) questioned intestinal inflammation as the sole cause of death and suggested direct effects on the blood, heart and the brain (36).

The cause of death was also attributed to mechanical obstruction of the passage of blood through the heart [and obstructing vital energies] following autopsy (37). Emily A. Nunn (1878) described the histologic characteristics of arsenic poisoning in an animal model (Figure 1) (38).

Laboratory (detecting arsenic)

Marcet (1812) used nitrate of silver to detect arsenic and Berzelius (Ann. de Physik, 1828) improved the detection method using animal fluids by mixing the sulphuret of arsenic with an excess of carbonate of soda and water (39,40)

Orfila (1831) performed an interesting animal experiment by giving a portion of the responsible liquid in a case of human toxicity as a method to diagnose arsenic toxicity. He also discussed the potential reasons for discrepancies in clinical findings in these animals (34).

Later, Dodgson (1831) applied oxalic acid to improve the detection of oxide of arsenic (41). However, the validity of the current methods and potential errors in toxicological testing in legal cases was raised by Venables (1834) (42). In 1836, James Marsh (chemist) developed an accurate test for

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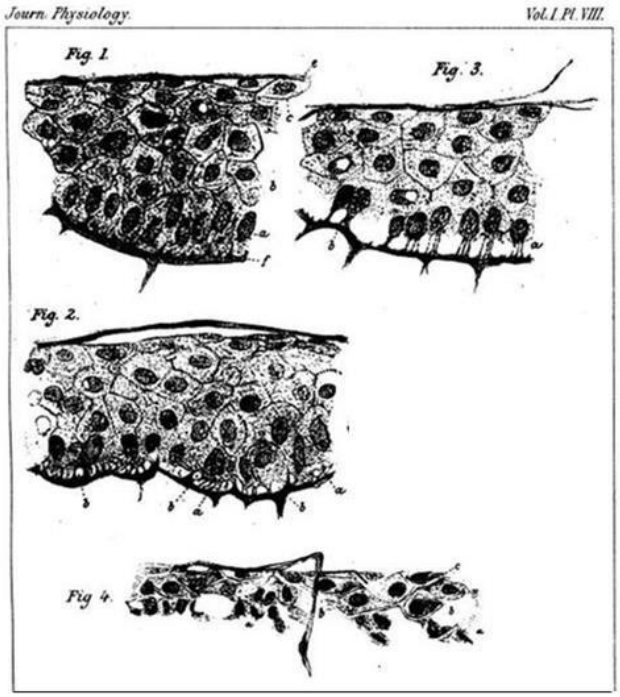

Figure 1. Epidermis of the frog exposed to arsenic (1. Healthy case, 2. Minor poisoning, 3. Severe poisoning and 4. Three days later)(adopted from J Physiol, 1878).

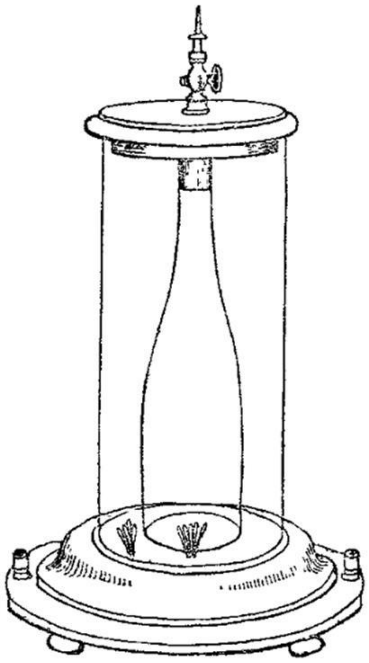

Figure 2. Morton's Apparatus for detection of arsenic (adopted from The Lancet, 1841).

detecting arsenic in forensic toxicology (43). The Academy of Sciences, Paris (1840) evaluated the measurement of arsenic, and recommended a proper application of Marsh's apparatus, which is capable of easily detecting the 1×10^{-6} part of arsenious acid in a fluid, to be used in legal cases (44).

Morton's apparatus (Figure 2) and the Galvanic test for detecting arsenic were introduced in 1841(45,46). A self-detective arsenic test usable by a wide range of professionals was introduced by Cattell (1847) in the light of increasing rates of homicide by arsenic (47). In a case of food-related arsenic poisoning, Skevington (1852) had scientifically quantified the amount of adulterated tea and arsenic that was taken (48).

Accidental overdose

It seems that the most dominant mode of arsenic poisoning was accidental and food consumption -related in the 19th century. The majority of homicidal cases were also food-mediated.

Food-related accidents

Accidental poisonings via consumption of puddings were frequently reported, including an arsenic-contaminated pudding leading to the poisoning of a family of nine in 1805 [although no deaths were reported] (49). Ewen (1842) reported four cases of food-related acute poisoning (50). Barnes (1847) also reported six fatal cases among nine members of a family that consumed arsenic-contaminated pudding. Fatal cases included five children who were below 9 years of age and their father (51).

Other accidental overdoses

May (1845) reported a fatal case of child death as a result of accidental ingestion of a paste kept for the purpose of exterminating mice (52).

Iatrogenic overdoses

Tomkins (1843) reported an Iatrogenic fatal poisoning by a run-about doctor, where despite the rage of the medical community, the perpetrator was not prosecuted for manslaugther as he was not officailly a physician (53). Jones (1843) reported using local application of "the liquor arsenicalis" [sulphure] for removal of skin complications related to *psoriasis guttata*, which led to chronic arsenic toxicity (20).

Intentional overdoses

Murray (1838) and Foster (1841) reported cases of suicide with arsenic (54,55). Allison (1845) also reported deliberate arsenic ingestion by a 13-year-old girl following an assault by a man (56).

Homicidal overdoses

Rayner (1838) reported the murder of a 17-year-old girl by her father using arsenic for the purpose of collecting money from a burial society, which was confirmed three weeks after her death (57).

Marie-Fortunée Lafarge (1816 - 1852) was convicted to for murdering her husband in 1840 (4-6).

A murder of a five-month-old infant, by her depressed mother who also killed herself, was reported by Foster (1841) (55).

According to another report in 1846, a mother killed two of her own children and poisoned a third to obtain a small amount of money from a burial society, in which all of them were members. She was convicted for murder and for collecting £1.5s. and £1.15s for the deaths of two of her children from the burial society (58).

Forensic toxicology and post-mortem

Arsenic was detected from a suspected overdose case seven years after his death in 1830. The author was surprised by the remarkable conservation of the body. He reported that the head hair had not undergone the least alteration, the trunk and extremities were in a state of perfect integrity, the chest was collapsed, and the heart and lungs molded into a fluid mass of black color without any smell (59).

Later in 1838, the cause of Widow Chevalier's death was confirmed to be arsenic poisoning-related three years after her death. Her exhumation also revealed that the body was in a state of remarkable preservation or as the author put it "in a state of complete mummified" (60). Similarly, an exhumed body observed to be sufficiently preserved 141 days after death, making it possible for the witnesses to identify the body (61). Rayner (1838) reported confirmation of an arsenic-involved murder three weeks after her death. In this occasion the body's relative decomposition was observed (57).

In 1834, Dr. Tranchina of Palermo, who published in the Sicilian Journal, *La Cerere*, suggested injecting an arsenical solution into the blood vessels shortly after death to preserve the body for teaching anatomy. His innovation was also reported in Calcutta Quarterly Journal in the same year (62). Later John Snow questioned the process of preserving bodies for dissection by injection of arsenic on safety grounds for the teachers and students (63).

Looking for arsenic in the ground where a body was exhumed was discussed in a court case in 1840 (64).

Occupational overdoses

Kerr (1875) reported the first occupational case of chronic arsenic poisoning in a weaver who used to bite the arsenic-contaminated ends of cotton before knotting (65).

Environmental toxicities

Environmental toxicity related to arsenic was reported by the middle and late 19th century. Potential exposure to arsenic was reported from contact with candles by Hunt (1837), fly-papers by Ord (1878), wallpapers, clothing and furniture by Brunton (1883), wallpaper by Lyon (1886), paper and fabrics in 1889 by Robinson (1891), and cigarettes by Murrell (1896) (66-72).

Coathupe (1838) in a span of thirty years reported several families that suffered from arsenic exposure off-site of a manufacturer of colored paper by mineral substances, where arsenic transported to the soil and water (73).

The Academy of Science's investigation on bone and soft tissues contradicted the belief that arsenic naturally exists in the body (44).

Cattell (1847) introduced a self-detective arsenic test to prevent homicide by arsenic (47).

Bateman (1857) reported that living in rooms papered with a certain green paper (arsenate of copper) resulted in arsenic poisoning. He also discussed the tendency of infants and children who carried items in their mouth as being more prone to toxicity in unsuspected cases (74).

Morris (1880) reported an interesting study, in which fifteen hundred circulars to the fellows of the Medical Society of London were contacted seeking potential cases of arsenic poisoning. They received 224 replies including 54 positive responses which consisted of 100 potential exposed cases. Amongst them, thirty-five cases referred with clinical symptoms (75).

Stevenson (1883) developed the theory of putrefying organic substances as sources of environmental arsenic poisoning (76).

By 1883, over 100 cases of environmental arsenic poisoning were presented to the Medical Society of London on the basis of similarity of symptoms to arsenic toxicity and other causes being ruled out, similarity of symptoms to arsenic toxicity and disappearance of the symptoms upon removal of the source (68).

Brunton (1883) suggested a form of legislative interference to control the arsenic problem (68).

In a scientifically orchestrated attempt to inform authorities and the public on the potential dangers related to using arsenical wall papers, availability of these papers fell from 47% in 1884 to 33% in 1886 in the state of Massachusetts, despite the fact that the proposed legislation was not passed (77).

Legislation for Prevention

A scientific committee was formed in 1849 in the UK to prepare a petition for Parliament under the title of "Prevention of the Indiscriminate Sale of Arsenic" to prevent secret poisoning (78). During this process many scientific rationales were presented including, even, to mislabel another chemical as arsenic to control the outcome of potential ill intentions for self-harm or suicide. The same author was opposed to the idea of coloring arsenic for this purpose (79). Cattell (1850) suggested to prohibit the sale of arsenic without the adoption of some precautionary measure (i.e. without rendering it self-detective) (80). Eventually, a Bill entitled "An Act to Regulate the Sale of Arsenic" was enacted in 1851 by Parliament including the following five effective items; 1. On every sale of Arsenic particulars of sale to be entered by seller, 2. Restrictions as to sale of Arsenic under Ten Pounds' weight.- Provision for coloring Arsenic, 3) A penalty of maximum 20 pounds for offending against this act, 4) Act not to prevent sale of Arsenic in Medicine under a medical Prescription, 5) "Arsenic" to include arsenious compounds (81). Additional amendments were suggested in later decades (68).

Three decades later, a pioneer attempt to limit *"arsenical wall papers*" was not passed in the State of Massachusetts; however, this scientifically orchestrated attempt on informing the authorities and the public for potential dangers related to the use of arsenical wall papers was fruitful and the availability of these papers fall from 47% in 1884 to 33% in 1886 (77).

CLINICAL FINDINGS

Acute poisoning

Murray (1838) reported vomiting with excessively acute pain on pressure over the epigastrium in an intentional ingestion of 15 grains (1 grain is equal to 0.065 gram) of white arsenic (arsenious acid) (54). Foster (1841) reported a homicidal case of a five-month-old infant, in which great agony with severe bilious vomiting, convulsions, cold extremities, swollen and tense abdomen who died soon after consumption (55). Another accidental overdosed child was presented with restless, hot skin and a pulse of 140 with no

pain, cold extremities, livid lips, sunk eyes, fixed and dilated pupils, feeble respiration accompanied with sighing who later expired (52).

Incessant vomiting, agonizing pain in the stomach and bowels, headache with high febrile excitement and full bounding pulse, passing some bloody stool [which perceived to purge them], and convulsion were also the dominant manifestation of four food related poisonings including one death by Ewen (1842) (50). Hedley (1843) reported vomiting, pain in the stomach with slight tenderness, sense of constriction in the throat, feeble pulse and clean tongue (61). Allison (1845) reported violent spasmodic pain, vomiting, sensation of burning heat in the throat and stomach, feeble pulse and cold skin (56).

In severe cases, death was reported to happen in a matter of hours (55,82).

Sub-acute and chronic poisoning

Coathupe (1838) reported heaache, constant colic and premature deaths in cases that were continuously exposed to off-site industrial transported arsenic over several decades (73).

Dry and nearly black tongue, difficulty in swallowing, delirious, and being incapable of responding unless aroused was reported in a case where arsenic was used for medical purposes for several weeks (53).

Jones (1843) reported clinical findings of local application of sulphur "the liquor arsenicalis", resulting in obstinate diarrhoea for six weeks followed by frequent griping pain in the bowels with almost constant desire to act, considerable tenderness over the whole abdomen, constant pain and nausea and frequent vomiting after taking food, cool and dry skin, thirst tongue, sense of constriction of the throat, and a copious flow of saliva and faeces, a mucupurulent secretion mixed with specks of blood, pain and tenderness down the spine, frequent muscular tremors, crampy feeling of the lower extremities, with partial loss of motion and sensation (20).

Kerr (1875) reported chronic arsenic poisoning in a weaver who used to bite the ends of cotton before knotting, which led to loss of appetite, nausea, colicky pains, constipations, headache, emaciation, sleeplessness, strangury, inflammation of the conjunctivae, with intolerance of light was reported (65).

In an epidemiologic study Morris (1880) reported that amongst 100 potential arsenic exposed cases, mischief occurred in thirty-five subjects including severe depression in 16 cases, conjunctivitis in 19, and cough, asthma in 9 cases, and followed by a few cases of external irritation such as eczema from stockings, gloves and artificial flowers, and conjunctivitis from tulle dresses etc (75).

Roots of high dose exposure

Majority of the reported cases were exposed to arsenic via ingestion (food and water) (48-51). Dermal exposure was also reported in a case with psoriasis (20). In 1837 a Latin reprint of a report on two cases of vaginal exposure to arsenic from de Rebus, Leipsic, 1793, tom. 35 and in Metzger p. 390 were published, one of these cases died as a result of poisoning (83).

Treatments

In the 19th century, treatment of arsenic poisoning was expanded to using antidotes. Coxe (1806) discussed applying milk, white eggs, water and opium in treating arsenic poisoned cases (84).

Induction of vomitting was suggested by Wright (1829) by tickling the throat with a feather and drenching the throat with warm water, and later by administering sulphate of in 1837 (33,37).

Chambers (1842) successfully treated a child with arsenic poisoning with three drachms (one drachma is equal to 4.3 grams) of oxide of Iron (85). In addition, soft sugar, theriaca, water and milk were administered followed by tepid milk and flour boiled into a thick mucilage, castor oil, compound tincture of cardamoms and peppermint-water (85).

In cases of poisoning in children May (1845) discussed applying finger and administering ipecacuanha wine to encourage vomiting in arsenic-intoxicated children (52).

Hydro-oxide of iron was introduced by MM. Bunsen and Berthold of Gottingen (1834), as an antidote to arsenic acid based on animal experience (86). Although its efficacy was questioned in other animal studies in the same year (87). Later the efficacy of hydrated peroxide of iron was successfully tested in pigs, and in two human cases who drank contaminated wine in 1840 (88,89).

Ewen (1842) reported two patients that were bled, given castor oil, effervescence saline medicine and adopted a strictly antiphlogistic regimen (50).

Murray (1838) successfully treated arsenic poisoning with tritoxide of iron (54). Hydrated sesquioxide of iron was successfully employed in treatment of nine poisoning cases within one family (90).

Jones (1843) presented a case that received warm bath, farinaceous diet with a little chicken or veal broth, linseed tea, barley-water, liquor potass and decoction of bark and treated successfully (20). Allison (1845) reported using stomach-pump, castor oil, magnesia, and heaped table-spoonful of hydrated peroxide of iron mixed in water, cool-enough gruel containing magnesia injected into the stomach. In this occasion, stomach pump was evaluated to be useful when iron was administered, as she was vomiting (56).

Based on a theoretical and experimental approach, Thomas Cattell (1850) suggested that ferrate of potassa could be a potential antidote similar to dry peroxide of iron (80). Further in 1875, tobacco (Steinmetz), laudanum (Churchill) and antimony were suggested as antidotes to arsenic poisoning (91-93).

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