

Medico-Legal and Scanning Electron Microscopic Study of Heart in Aluminium Phosphide Poisoning

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Abstract: Introduction: Scanning electron microscope is an analytical method in the field of forensic science, employed for primary information about unknown samples and materials. Heart is an extraordinary organ situated in thoracic cavity which along with the circulatory system, works to supply blood and oxygen to all parts of the body. Aluminium Phosphide (AIP) is a systemic lethal protoplasmic poison which has become one of the major ways of committing suicide in Northern India. Material and method: The analyzed sample of heart was collected and photographed before and after dissection of the heart during autopsy and autopsied samples of heart were painstakingly taken to be analyzed at Central Drug Research Institute, Lucknow. Victim's personal history, supportive treatment and hospital records were also noted. Result: The effect of AIP leads to a number of abnormalities on different organs, but the effect on heart is most prominent, which leads to fatalities in most of the cases especially during first 24 hours of consuming this poison. Conclusion: This is an original and pioneer work in the field of Forensics, as the electron microscopic studies at tissue and cellular level can be a road map to the field of research and clinical investigations as the antidote for this lethal poison is not available till date.

Keywords: Scanning Electron Microscope (SEM), AIP, CDRI, Myocardium, Round cell, GSR-Gun Shot Residues, PBR-Post Blast Residues

1. Background

A case of AIP poisoning was brought to autopsy room of Institute of Medical Sciences, BHU. The deceased, aged 21 years male had consumed AIP at 10.30 PM on previous day of examination as told by relatives and investigating officer. The hospital documents mentioned the time of admission to be 12.51 AM (2.21 hour after ingestion). The time of death was recorded to be 10 AM the following day that is the day of examination. Autopsied samples were collected at 1.55 PM. The time elapsed was about 15.5 hours after ingestion and 3.5 hours after death.

At this interval, the heart showed characteristic pallor area of infarction with swollen areas which became apparent due to swelling of its fibers. The bundles of muscles seemed separated and on cutting the ventricle at autopsy table, the affected muscle showed more coarsely fibrillar arrangement than the normal area.

2. Introduction

Scanning Electron Microscope is an analytical method in the field of forensic science, employed for primary information about unknown biological samples. The field encounters both materials of natural and vegetable origin. Routine exercise in forensic science deals with determination, description and comparisons of practically any phases of biological samples analyzed. The utility of SEM becomes evident in analyses of trace evidences present even in microns at the crime –scene which can be utilized by this technique. It can be used alone or in combination with other

supportive analytical equipment-EDS/WDS, EBSD, CL, XRF, etc. The process includes a defined protocol of sample preparation including steps of cutting, staining, dehydration and coating. [1] The sample preparation method varies according to different types of samples encountered: inorganic, organic, synthetic polymer, biological tissues etc. The image formation is produced by aiming a beam of electrons onto the specimen and studying the electron emission on a closed TV circuit. The beam of electrons emitted from a hot tungsten filament is focused by means of electromagnets onto the surface of the specimen and causes the emission of electrons from the upper layer of the surface of the specimen. By scanning this primary electron beam across the specimen's surface in synchronization with the cathode ray tube, it is possible to convert the emitted electrons into an image of the specimen. The major attractions of SEM are its high magnification, high resolution and great depth of focus resulting in stereoscopic picture of the specimen.[2] Its wide spread utility has led to day to day advancements in the technique to exploit its use in various fields. Focused Ion Beam (FIB) technology is gradually being employed as common analytical facilities. Transmission Detectors for standard SEM are becoming significant for morphological studies in Nano-fields. Combined systems SEM/FIB in forensic science allow by means of milling to remove materials, study of inner structure of gunshot residues and post-blast residues (GSR,PBR), Nanocomposites. The most crucial thing about SEM is attributed to the fact that the methods are practically non-destructive and after analysis, the evidential value is fully preserved. [1]

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Our present study focuses the effects of AIP with the help of SEM on Heart, which is a vital organ of the human body. Along with the circulatory system of which it is the most important part, it works to supply blood and oxygen to all parts of the body. This muscular organ is located in pericardial sac. Internally, it is divided into four chambers: two atria and two ventricles separated by valves. The heart wall is composed of connective tissue, endothelium and cardiac muscles, which enables it to contract and dilate to synchronize heartbeat. The heart wall consists of three layers:

Epicardium outermost protective layer assists in production of pericardial fluid.

Myocardium: thickest, middle muscular layer composed of cardiac muscle fibers, which enable heart contractions, thickness varies in different parts of heart.

Endocardium: thin inner layer, lines inner heart chambers, covers heart valves and continuous with the endothelium of large blood vessels. In atria, it consists of smooth muscles as well as elastic fibers. [3]

Earlier used as a grain fumigant for bulk preservation of wheat, AIP led to isolated fatal inhalation exposure to Phosphine in stray reports of accidental AIP toxicity. [4] It is used as pesticide, insecticide and rodenticide. It is available as dark brown or grayish green tablets of 3gm of 20 mm diameter and thickness 5 mm. Tablets are composed of pure AIP (the active ingredient which releases PH_3 gas), Urea and NH_4CO_3 (which in turn releases CO_2 and NH_3 and prevents self-ignition of PH_3 gas). It is generally available by the names Celphos, Alphos, Quickphos or Bidphos. (5) Each tablet has a capacity to liberate 1 gm PH_3 . On coming in contact with moisture, AIP liberates PH_3 . Phosphine is a systemic poison and affects all organs of the body. The chemical reaction is accelerated by the presence of Hydrochloric acid in the stomach. AIP has garlicky odor. It is widely used as a grain preservative. Phosphite and Hypophosphite of Al which are non-toxic are left in the grains [6]. It has become a common method of committing suicide in Northern India due to its easy availability, sometimes Organophosphorus being the more common one. The incidence of the poisoning has been increasing steadily and is now the commonest mode of suicide in the agricultural community in northern India (Ranga et al., 2004). Overall mortality in cases of Aluminium Phosphide poisoning varies between 70–100%. The rate of mortality in India from poisoning varies from 15-35% whereas in developed countries it is 1-2% Toxicity rating of AIP is 4 i.e. very toxic as its fatal dose is 0.5 gm [7].

Studies show that Suicide was the most common mode of poisoning deaths, with overall mortality varying between 70–100%. Mortality is higher in those who consume more than two tablets and none of the patients survived who had ingested more than 3 tablets (Siwach et al., 1988; Singh et al., 1985; Raman et al. 1985)[8][9][10]. According to Karamjit et al (2003)[11]. The pattern of poisoning varied in urban and rural areas, with a higher incidence of poisoning deaths in rural (64%) than in urban areas (36%). Aluminium phosphide was the most common poison consumed, being

responsible for 50% of deaths, followed by insecticides 24% (Gupta et al., 2006)[12]. Poisoning deaths increased from 19% in 1996 to 24% in 2005. The age group most commonly affected was 16–25 years (49%). The male to female ratio was 1.9:1.0 and the rural to urban ratio was 1.5:1.0 (Gupta et al., 2006) [12].

Toxicokinetics

Phosphine can readily be absorbed by the lungs and GI tract. The Phosphine gas released in the stomach is responsible for early signs and symptoms after getting absorbed in circulation. Some of the AIP absorbed and metabolized in liver with slow release of Phosphine causes delayed toxicity [13].

Toxicodynamics

Most of the toxic effects owe to the release of Phosphine, which is a protoplasmic poison that interferes with the function of cellular enzymes and proteins. The mechanism of its toxicity is electron transfer blockage and non-competitive blockage of cytochrome C oxidase. It further inhibits oxidative phosphorylation and in turn cellular respiration and activation of peroxide radicals. Phosphine can inhibit catalase and deplete Glutathione, which may result in cell wall and respiratory canal dysfunction as well [13].

Studies have revealed that ingestion leads to superoxide dismutase activity and low catalase levels that result in increased formation of free radicals and accelerated lipid peroxidation, which in turn results in damage to cellular membrane, disruption of ionic barrier, nucleic acid damage and cell death. Focal myocardial necrosis and changes in membrane action potential occurs as a result of altered permeability to Sodium, Magnesium and calcium, which manifest as various forms of ECG changes and arrhythmias [14]. The repercussions of consuming AIP are most evident on Heart, which becomes the most lethal cause of death in first 24 hours.

Changes in Heart muscles in AIP poisoning:

On autopsy, Heart sometimes appears to be normal on gross examination.

On dissection- hemorrhages are present, generally at delayed intervals.

Pathological changes:

Direct toxic effect of AIP on myocardium is hypomagnesemia brought on by focal myocardial damage, leading to arrhythmias. Hypotension and shock within 3-6 hours of ingestion of AIP. In persons, who survive the poisoning, the cardiotoxicity and hypoxia disappear within 5-7 days, due to excretion of PH_3 and restoration of normal cellular metabolism. The toxic myocarditis is responsible for varied fatal ECG changes. These symptoms are marked generally after 6-24 hours of ingestion among non-survivors. It leads to Ventricular ectopic beats, conduction disturbances, Left Bundle Branch Block, ventricular fibrillation, aberrant conduction and Idio-ventricular rhythm terminally leading to asystole. Death in first 24 hours appears to be due to cardiogenic shock as evidenced by ECG abnormality while survivors show complete ECG recovery. The effect of poisoning is due to some reversible factors

leading to disturbance in the permeability of Sodium, Potassium, Calcium and Magnesium leading to changes in trans-membrane action potential due to focal myocardial involvement and subsequent myocardial necrosis [15].

3. Material and Methods

The sample of heart was collected from the mentioned case brought to Autopsy room of IMS, BHU. The brief history of previous medications and substance abuse was noted which could interfere the study. The collected sample was preserved in 10% formaldehyde and refrigerated at 4⁰ C in departmental laboratory. Formalin fixed samples were taken for SEM analysis to CDRI (Central Drug Research Laboratory), Lucknow. They were treated there with 1% Osmium-tetraoxide for 1 hour. Traces of Osmium-tetraoxide were removed by washing the samples with PBS buffer. The samples were dehydrated through graded series of Alcohol (30%, 50%, 70%, 90% and finally by absolute alcohol i.e. 99.5%) and later subjected to Critical Point Dehydration. Samples were mounted over Aluminium stubs and coated with film of Gold-Palladium using sputter coat unit. Processed samples were examined under SEM for imaging.

Control sample (images) – It was taken from online available literature on Electron Microscopic studies of Heart.

14 photomicrographs were altogether taken, out of those 14 images, 5 images of single heart sample were chosen and viewed from different angles, focusing a particular area at different magnifications (varying from 1500X, 3000x, 6000x compared to normal).

4. Result

Figures 1-6 are the images of heart which were photographed before and after dissection of the heart during autopsy procedure in the mortuary. Figure 7-10 was used as control samples (images) in our study to compare normal myocardial tissue with pathological heart tissue samples (heart affected by ALP/Aluminium phosphide) as no study and documented literature is available till date on effect of ALP over cardiac tissue visualized by scanning electron microscope. So we have taken real as well as virtual images of normal heart tissue from Google scholar images available online.

Autopsy findings of heart in case of aluminium phosphide poisoning



Figure 1: Gross microscopic findings of heart (Naked eye appearance)

Image 1: Gross or macroscopic findings of heart (Naked eye appearance)



Figure 2: Showing generalized congestion of myocardium along with focal Myocardial Infarction patches and epicardial hemorrhagic spots over left atrial surface

Image 2: Hematoma formation over the root of ascending aorta along with hemorrhagic spot over epicardial surface.



Figure 3: Hematoma formation over root of ascending aorta along with hemorrhagic spot over epicardial surface.

Image 3: Showing generalized congestion of myocardium along with focal Myocardial Infarction patches and Epicardial hemorrhagic spots over left atrial surface



Figure 4: Sub-endocardial hemorrhagic area visible over right atrium. Edema of myocardium is also evident.

Image 4: Sub-endocardial haemorrhagic area visible over right atrium, edema of myocardium is also evident



Figure 5: The interventricular septum of dissected human heart showing disarray of myocardial fibers along with haemorrhagic spots and hematoma formation over left atrial surface.

Image 5: The interventricular septum of dissected human heart showing disarray of myocardial fibers along with haemorrhagic spots and hematoma formation over left atrial surface.



Figure 6: Dissected heart showing all the four chambers along with chordate tendinae attached with papillary muscles. The myocardium shows intense congestion and edema of muscle fibers.

Image 6: Dissected heart showing all the four chambers along with chordate tendinae attached with papillary muscles. The myocardium shows intense congestion and edema of muscle fibers.

Control Sem images of heart (taken from online sources)



Figure 7: Showing Electro Microscopic colored photograph of normal myocardium.

Image 7: Showing Electron Microscopic colored photograph of normal myocardium



Figure 8: Showing actual photomicrograph of normal myocardial cells.

Image 8: Showing actual photo micrograph of normal myocardial cells



Figure 9: False colored Scanning Electron Micrograph of an aggregation of RBCs due to sub-endocardial haemorrhages.

Image 9: False colored Scanning Electron Micrograph of an aggregation of RBCs due to sub-endocardial hemorrhages

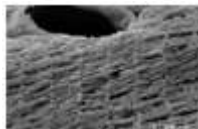


Figure 10: Cryocytogram of separated striated heart muscle fibers

Image 10: Cryocytogram of separated striated heart muscle fibers

Scanning electron microscopic findings of heart

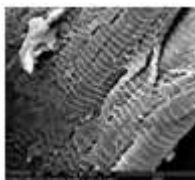


Figure 11: Scanning Electron photomicrograph of heart at 6000X showing rupture and separation of striated muscle fibers at places along with separation of intercalated discs. The picture also depicts the areas of focal myocardial necrosis. Areas of rhexis (can also be seen in cases of coagulative myonecrosis) occurring due to coagulation of hypercontracted sarcomeres.

Image11 Scanning Electron photomicrograph of heart at 6000X showing rupture and separation of striated muscle fibers at places along with separation of intercalated discs

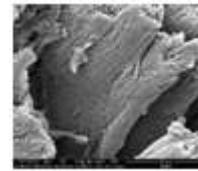


Figure 12: Scanning Electron photomicrograph at 3000X showing swollen myocardial fibers with separation myofibrils as well as intercalated discs areas of focal myocardial necrosis and myocytolysis.

Image 12: Scanning Electron photomicrograph at 3000X showing swollen myocardial fibers with separation myofibrils as well as intercalated discs areas of focal myocardial necrosis and myocytolysis

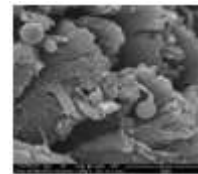


Figure 13: SEM Photomicrograph at 3000X shows soft and flabby myocardium with cytoplasmic vacuolation (also known as round cells).The features of toxic myocarditis and focal myocardial necrosis are also evident.

Image 13 Photomicrograph at 3000X shows soft and flabby myocardium with cytoplasmic vacuolation



Figure 14: Scanning Electron photomicrograph of swollen myocardial fibers with areas of myocardial separation with the gaps showing infiltration of round cells with Myocardial fibers with entangled and separated myofibrils at places are evident along with granularity of cytoplasm and eosinophilia of muscle cytoplasm.

Image 14 Scanning Electron photomicrograph at 3000X of swollen myocardial fibers with areas of myocardial separation with the gaps showing infiltration of round cells



Figure 15: Photomicrograph at showing swollen and separated (spindle shaped at few places) myocardium with round cell infiltration and separated and prominent myofibrils with focal necrosis. Beaded appearance of granulated cytoplasm is visible and appreciable photomicrograph of separated myofibrils along with edema of muscle fibers and characteristic branching pattern of striated (heart) muscle fibers.

Image 15 Photomicrograph at 1500X showing swollen and separated (spindle shaped at few places) myocardium with round cell infiltration and separated and prominent myofibrils with focal necrosis

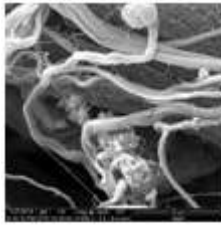


Figure 16: Photomicrograph at 6000X showing well separated myofibrils, at places branched / accumulated and entangled striated muscle fibers, also showing few single and separated myofibrils along with granularity of cytoplasm of Polymorphonuclear Cells infiltration of fibers. Also showing areas of myocytolysis with vacuolisation of cytoplasm. The muscles are showing coarse and beaded (round cells) and areas of focal necrosis.

Image 16 Photomicrograph at 6000X showing well separated myofibrils, at places branched / accumulated and entangled striated muscle fibers.

5. Discussion

Figure 1 – In fig 1 we have observed and studied the details of gross (morphological appearance of heart). The heart is an extraordinary organ. It is about the size of a clenched fist, weighs about 10.5 ounces and is shaped like a cone. Along with the circulatory system, the heart works to supply blood and oxygen to all parts of the body. The heart is located in the chest cavity just posterior to the breastbone, between the lungs, and superior to the diaphragm. It is surrounded by a fluid filled sac called the pericardium, which serves to protect this vital organ.

The heart wall is composed of connective tissue, endothelium, and cardiac muscle. It is the cardiac muscle that enables the heart to contract and allows for the synchronization of the heartbeat. The heart wall is divided into three layers: epicardium, myocardium, and endocardium. [3]

Figure 2- In fig 2, we have observed generalized congestion of myocardium along with focal myocardial infarction patches and epicardial hemorrhagic spots over left atrial surface. According to textbook of forensic medicine and toxicology, by Anil Agrawal, the author observed that in histopathological study of heart – congestion, edema and hemorrhagic myocardial lesions were observed [5].

Figure 3- In fig 3, we have observed hematoma formation root of ascending aorta along with hemorrhagic spots over epicardial surface. According to textbook of forensic medicine and toxicology, by Anil Agrawal, the author observed that heart becomes soft and flabby, may show epicardial hemorrhages especially in survival of more than a day [5].

Figure 4- in fig 4, we have observed, Sub-endocardial haemorrhagic area visible over right atrium while edema of myocardium was also evident. Post-mortem findings showed cardiac failure, severe and persistent hypotension, heart congestion and sub-endocardial infarction (Hashemi-Domemeh et al, 2016) [13].

Figure 5- In fig 5, we have observed that, the interventricular septum of dissected human heart showed disarray of myocardial fibers along with haemorrhagic spots and hematoma formation over left atrial surface. According to textbook of forensic medicine and toxicology, by Anil Agrawal, the author observed that in cases of AIP poisoning in the heart, there were areas of myocytolysis and degeneration of muscle fibers. [5]. Post-mortem findings showed pericarditis, cardiac fibers detachment due to edema, fiber destruction (Hashemi-Domemeh et al, 2016) [13].

Figure 6- In fig 6 observed that dissected heart showed all the four chambers along with chordae tendineae attached with papillary muscles. The myocardium showed intense congestion and edema of muscle fibers. According to textbook of forensic medicine and toxicology, by Anil Agrawal, the author observed that in post-mortem histopathological findings, heart showed congestion, edema, fragmentation of fibers, focal necrosis. [5] According to Modern Medical Toxicology by V V Pillay, the author observed features of toxic myocarditis with fibrillary necrosis [7].

Figure 7-10 - was used as control samples (images) in our study to compare normal myocardial tissue with pathological heart tissue samples (heart affected by AIP/aluminium phosphide) as no study and documented literature is available till date on effect of AIP over cardiac tissue visualized by scanning electron microscope. So we have taken real as well as virtual images of normal heart tissue from google scholar images available online [16].

Figure 11- In fig 11 we have observed, Scanning Electron photomicrograph of heart at 6000X which showed rupture and separation of striated muscle fibers at places along with separation of intercalated discs. The picture also depicted the areas of focal myocardial necrosis. Areas of rhexis (can also be seen in cases of coagulative myonecrosis) occurring due to coagulation of hypercontracted sarcomeres. According to diFiore's Atlas of Histology, a high magnification photomicrograph illustrates a section of the cardiac muscles cut in the longitudinal plane. The cardiac muscle fibers exhibit cross-striations, branching and a single central nucleus. The dark staining intercalated discs connect individual cardiac muscle fibers. Small myofibrils are visible within each cardiac muscle fibers. Delicate strands of connective tissue fibers surround the individual cardiac muscle fibers.[17]

Post-mortem findings showed cardiac failure, severe and persistent hypotension, heart congestion, sub endocardial infarction, pericarditis, cardiac fibers detachment due to edema, fibre destruction (Hashemi-Domemeh et al, 2016).[13]

Within early hours (12-14), in Robbin's Basic pathology, it is illustrated that Electron microscopic study revealed relaxation of myofibrils, glycogen loss and mitochondrial swelling (which is reversible phenomenon) [18]

In coagulative myocytolysis, irreversible hypercontraction of myocardial muscle fibers (tetanic death), which occurred in early hours of myocardial damage and this phenomenon is known as rhexis, which takes place at myofibrils level, that is anomalous irregular cross band formation.[19]

Figure 12- In fig 12 we observed Scanning Electron photomicrograph at 3000X which showed swollen myocardial fibers with separated myofibrils as well as intercalated discs, areas of focal myocardial necrosis and myocytolysis.

According to textbook of forensic medicine and toxicology, by Anil Agrawal, the author observed, that heart showed edema (swollen muscle fibers) with fragmentation of fibers, and degeneration of myocardial muscles along with areas of myocytolysis[5].

Cardiac fibers showed detachment due to edema and fibre destruction. (Hashemi-Domemeh et al, 2016) [13]

In colliquative myocytolysis, increasing edema and vacuolation were observed.[19]

Figure 13- In fig 13, we observed that Photomicrograph at 3000X showed soft and flabby myocardium with cytoplasmic vacuolation (also known as round cells).The features of toxic myocarditis and focal myocardial necrosis were also evident.

According to textbook of forensic medicine and toxicology, by Anil Agrawal, the author observed, that histopathology of heart showed epicardial hemorrhages, especially in survival of more than one day. The heart was soft and flabby. The heart showed areas of focal myocardial necrosis along with areas of myocyte vacuolation (round cell infiltration).[5]

Study conducted by Ashok Kumar Jain et al on Autopsy findings of Aluminium phosphide poisoning showed, Cardiac muscles with congestion, focal myocardial necrosis, and round cell infiltration.[9]

Figure 14- In fig 14, we observed Scanning Electron photomicrograph of swollen myocardial fibers with areas of myocardial separation with the gaps showing infiltration of round cells with Myocardial fibers with entangled and separated myofibrils at places are evident along with granularity of cytoplasm and eosinophilia of muscle cytoplasm.

Within early hours (12-14), in Robbin's Basic pathology, it is illustrated that Electron microscopic study revealed sarcolemmal disruption and mitochondrial amorphous densities (irreversible injury).[7]

Post-mortem findings of heart showed, cardiac fibers detachment due to edema and fibers destruction which appeared as granularity of the cytoplasm consists of

mitochondrial amorphous densities, neutrophilic infiltration and eosinophilia (Hashemi-Domemeh et al, 2016) [3]

Figure 15- in fig 15 we observed Photomicrograph at showing swollen and separated (spindle shaped at few places) myocardium with round cell infiltration and separated and prominent myofibrils with focal necrosis. Beaded appearance of granulated cytoplasm is visible and appreciable photomicrograph of separated myofibrils along with edema of muscle fibers and characteristic branching pattern of striated (heart) muscle fibers.

According to textbook of forensic medicine and toxicology, by Anil Agrawal, the author observed, the histopathology of heart showed congestion, edema and fragmentation of fibers. Myocardial muscle showed edema along with myocyte vacuolation, areas of myocardial necrosis and degeneration.[2]

According to textbook of forensic medicine and toxicology, by Krishan Vij, the author observed that, side by side swelling of cardiac muscle fibers and granularity of the cytoplasm. Loss of integrity of the sarcolemma leads to release of intra-cellular proteins into the extracellular spaces from the myocytes which has contributed in granularity of the cytoplasm, muscle showed more coarsely fibrillary arrangements[10] leading to cross band formation In coagulative myocytolysis, irreversible hypercontraction of myocardial muscle fibers (tetanic death), which occurred in early hours of myocardial damage and this phenomenon is known as rhexis, which takes place at myofibrils level, that is anomalous irregular cross band formation[8]

Study conducted by Ashok Kumar Jain et al on Autopsy findings of Aluminium phosphide poisoning on heart showed, congestion, focal myocardial necrosis and round cell infiltration [20].

Figure 16: In fig 16 we observed Photomicrograph at 6000X showing well separated myofibrils, at places branched / accumulated and entangled striated muscle fibers. Also showing few single and separated myofibrils along with granularity of cytoplasm of Polymorpho Nuclear Cells infiltration of fibers, also showing areas of myocytolysis with vacuolation of cytoplasm .The muscles are showing coarse and beaded (round cells) and areas of focal necrosis

According to textbook of forensic medicine and toxicology, by Anil Agrawal, the author observed, the heart showed degeneration in myocardial fibers [5]

In cardiac findings of Aluminium phosphide poisoning cases during post-mortem: cardiac fiber detachment due to edema and fiber destruction was evident (Hashemi-Domemeh et al, 2016) [13]

In coagulative myocytolysis, irreversible hypercontraction of myocardial muscle fibers (tetanic death), which occurred in early hours of myocardial damage and this phenomenon is known as rhexis[19]

According to textbook of forensic medicine and toxicology, by Krishan Vij, the author observed in post-mortem

demonstration of post myocardial infarction within 24 hours, swelling of cardiac bundles with fibrillary and coarsely arranged (entangled) myofibrils, eosinophilia or hyperchromasia was accentuated by placing green filter in light pathway of microscope using UV Lights. The myofibrils showed side by side swelling of the myofibrils (edema), granularity of the cytoplasm with deeply eosinophilic myocytes, coagulation necrosis of the muscle fibers and variable appearance of polymorphonuclear leukocytes.[21].

6. Conclusion

Prior to 1980, Aluminium phosphide poisoning was virtually unreported in India. Today, it has become the leading cause of suicidal and sometimes accidental death in northern Indian states since AIP has not built its in-roads into the agricultural sector, southern states have so far not been significantly affected. There are ominous indications of gradual rise in the number of cases being reported.

The purpose of selecting my study on this topic is that till no reference data is available for histopathological studies. Also the effect of AIP on heart is most prominent and the most evident cause of death in first 24 hours. Because of this fact, the fatality rate increases as the vital organ like heart cannot resist the fatal repercussions following the consumption of this poison. Most of the patients who come into contact, even accidentally, succumb to its toxicity because of the gap between ingestion and initiation of treatment, because of its fatal period which is as low as 1-3 hours and range up to 3-4 days. The cases of survival in Aluminium Phosphide poisoning are rare. Also, the signs and symptoms mild poisoning are similar to those of upper respiratory infections. A study on histopathological findings can reveal the consequences of consuming the poison at tissue and cellular level. Such study can be utilized for the purpose of finding its antidote which is not available till date. This can provide a guideline for the line of action for clinical purposes where the treatment is based on prognosis in such cases.

The IPC sections relevant to poisoning-

Section 284-it deals with negligent conduct in relation to poisons with a punishment of fine of Rs 1000 and imprisonment of up to 6 months

Section 328- It deals specifically with poisons. It states that"*whoever administers to any person, any poison or any stupefying, intoxicating, or unwholesome drug with intent to cause hurt to such persons shall be punished with imprisonment up to 10 years and shall also be liable to fine.*"

Section 309- Previously there was punishment for attempt to suicide which was imprisonment which may extend to one year with or without fine, now according to mental health act 2017, chapter XVI, section 115, notwithstanding anything contained in section 309 of the IPC, any person who attempts to commit suicide shall be presumed unless proved otherwise to have severe stress and shall not be tried under the said code.

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Conflict of Interest

Nil

Source of Funding

This research was not financially supported by any funding agencies.

Ethical Clearance

The present study was approved by "Institutional Ethical Committee" of Institute of Medical Sciences, Banaras Hindu University, Varanasi. All the information has been taken under consideration of medical ethical committee.

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