TO FIND OUT ELECTRO-CARDIOGRAPHIC CHANGES & ECHO CHANGES IN ALUMINIUM PHOSPHIDE AND ETHYLENE DIBROMIDE POISONING

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Abstract
Background & Method: The study was conducted in the Department of Medicine Shyam Shah Medical College and Associated Sanjay Gandhi Memorial Hospital, Rewa (M.P). History was followed by a careful clinical examination i.e. cardiovascular, respiratory and gastrointestinal and nervous system. Investigations had done included routine haematological examination, Biochemical analysis, urine examination, ECG, 2 D. Echo & Histopathological examination was done.

Result: ST-T changes were most common finding in Aluminium phosphide poisoning in relation to mortality. However hyperkalemia was the most ominous finding associated with 100% mortality, ECG finding in EDB was normal ECG. The most ominous finding was arrhythmia which was associated with 100% mortality. Survivors of ethylene dibromide poisoning echocardiography was normal in 11 (84.61%) followed by pericardial effusion in 2 (15.38%) patients.

Conclusion: Noteworthy finding was absence of correlation between cardiovascular involvement, histopathological changes and ECG findings. It was seen that even if ECG showed normal pattern there were significant histopathological changes in heart.

Keywords: electro-cardiographic, Aluminium phosphide, ethylene dibromide & poisoning.

Study Designed: Observational Study

Introduction
Poison is a substance (solid, liquid or gaseous), which if introduced in the living body, or brought into contact with any part thereof, will produce ill health or death, by its constitutional or local effects or both

Poisoning refers to the development of dose-related adverse effect following exposure to these substances (Linden & Burns 2001)[1]. A pesticide is usually defined as a chemical substance, biological agent, antimicrobial or disinfectant used against pests including insects, plant pathogens, weeds, molluscs, birds, mammals, fish, nematodes (roundworms) and microbes that compete with humans for food, destroy property, have a propensity for spreading or a vector for disease or simply a nuisance (Goel & Aggarwal 2007) [2].

The term pesticide has a broader connotation and also includes herbicides, rodenticides, fumigants, nematocides, algaeicides, ascaricides, molluscicides, disinfectants, defoliants and fungicides (Goel & Aggarwal 2007)[2]. Acute poisoning with pesticides is a global public health Problem and accounts for as many as 3 lack deaths, Worldwide every year. (Goel & Aggarwal, 2007) [2].

Poisoning is one of the commonest modes used for suicide (Aggrawal et al 1998)[3] reported their data from South Delhi (1974-93) which gives an idea that 36.08% of patients presented after poisoning followed by hanging in 33.30% & burns 19.46%.

The incidence of poisoning is rising in India. More than 50,000 people die poisoning every year. There has also been change in the pattern of poisoning seen in various areas. In north India. Aluminum phosphide and Organophosphorus poisoning have increased while those due to Barbiturate and Copper sulphate have declined. In South India plant poisoning is still commonly encountered (Aggrawal et al 1998)[3].

In India, the first case of Aluminium phosphide poisoning was reported in 1981 from MGM Medical College, Indore (Meena et al 1994)[4], Aluminium Phoshide poisoning
death first became public knowledge in 1983 when 37 cases were reported from Udaipur Medical College Hospital in one year. Then there was sort of epidemic of Aluminium Phosphide poisoning in northern India in 1987, considered even worse than the Bhopal gas tragedy of 1984. It may be the tip of an iceberg as large number of cases of Aluminium Phosphide does not come for treatment (Bajaj et al 1990)[5].
Material & Method

The study was conducted in the Department of Medicine Shyam Shah Medical College and Associated Sanjay Gandhi Memorial Hospital, Rewa (M.P). The study comprised of cases of poisoning being admitted in serious patient's ward of the department of medicine.

All patients were subjected to detailed clinical history including demographic data, intention of poisoning and precipitating factor. The amount and physical state of poison, time lag in hospitalization and symptoms present after the ingestion of poison were taken into consideration.

History was followed by a careful clinical examination i.e. cardiovascular, respiratory and gastrointestinal and nervous system. Investigations had done included routine hematological examination, biochemical analysis, urine examination, ECG, 2D. Echo & histopathological examination was done.

Criteria for selection of cases

1. Definite reliable history. 2. Presentation of remaining stuff/container by the relatives.
3. Typical clinical picture. 4. Garlic like odors imparted to breath in case of Aluminium phosphide poisoning.

Exclusion criteria

Patients having history of liver disease, renal diseases

A detailed clinical history was recorded which included demographic data, intention of poisoning and precipitating factors. In suicidal cases, amount and physical state of poison, time lags in hospitalization and symptoms after poisoning, Careful clinical examination was done which included general examination, cardiovascular, respiratory, and gastrointestinal and nervous system.

Investigation Included hematological examination, urine examination, blood sugar, blood urea, serum creatinine, serum bilirubin SGOT, SGPT, ECG, 2-D Echo and histopathological examination of heart in those patients who did not survive.

1. Haematological Examination: Total Leucocytes Count: It was done by standard technique using Adam standard micropipette for dilution and improved Neubour's Chamber for counting.

Differential leucocytes Count: It was done after staining the smear with Leishman's Stain. • Haemoglobin Estimation: It was done by Sahli's haemoglobinometer using acid hematin method.

2. Electrocardiography: Immediately after admission conventional 12 leads ECGS were recorded with the help of single channel electrographic machines. ECG recorded at paper speed of 25 mm/sec with 10 mm standard.

3. Echocardiography: Performed in case of Aluminium phosphide poisoning & Ethylene dibromide who survived after first 24 to 48 of admission in SGMH.

Results

Table 1: Relation of ECG changes recorded at the time of hospitalization to mortality in Aluminium phosphide poisoning

<table>
<thead>
<tr>
<th>S.NO</th>
<th>ECG Changes</th>
<th>Total no. cases</th>
<th>Mortality</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>ST. T Changes</td>
<td>43</td>
<td>28(65.11)</td>
</tr>
<tr>
<td>2</td>
<td>With in normal limit</td>
<td>36</td>
<td>10(27.77)</td>
</tr>
<tr>
<td>3</td>
<td>Sinus Tachycardia</td>
<td>11</td>
<td>02(18.18)</td>
</tr>
<tr>
<td>4</td>
<td>Sinus Bradycardia</td>
<td>04</td>
<td>03(75.00)</td>
</tr>
<tr>
<td>5</td>
<td>Atrial fibrillation</td>
<td>05</td>
<td>03(60.00)</td>
</tr>
<tr>
<td>6</td>
<td>RBBB</td>
<td>02</td>
<td>01(50.00)</td>
</tr>
<tr>
<td>7</td>
<td>Hyperkalemia</td>
<td>01</td>
<td>01(100)</td>
</tr>
<tr>
<td>Total</td>
<td>92</td>
<td>41</td>
<td></td>
</tr>
</tbody>
</table>

Total P=<0.0079, statistically significant

The above table shows that ST-T changes were most common finding in Aluminium phosphide poisoning in relation to mortality. However hyperkalemia was the most ominous finding associated with 100% mortality.

Table 2: Relation of ECG changes recorded at the time of hospitalization to mortality in Ethylene dibromide poisoning

<table>
<thead>
<tr>
<th>S.No.</th>
<th>ECG Changes</th>
<th>Total no.</th>
<th>Mortality %</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>With in normal limit</td>
<td>15</td>
<td>03(20.00)</td>
</tr>
<tr>
<td>2</td>
<td>ST-T Changes</td>
<td>03</td>
<td>01(33.33)</td>
</tr>
<tr>
<td>3</td>
<td>Atrial fibrillation</td>
<td>01</td>
<td>01(100)</td>
</tr>
<tr>
<td>Total</td>
<td>18</td>
<td>04</td>
<td></td>
</tr>
</tbody>
</table>

P = 0.2034 statistically non significant. The above table shows that most common ECG finding in EDB was normal ECG. The most ominous finding was arrhythmia which was associated with 100% mortality.

Table 3: Relation of 2-D ECHO changes in Survivors of Aluminium phosphide

<table>
<thead>
<tr>
<th>S.No.</th>
<th>2-D ECHO changes</th>
<th>Aluminium phosphide %</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>With in normal limit</td>
<td>45 (90.00)</td>
</tr>
<tr>
<td>2</td>
<td>Myocarditis</td>
<td>03 (6.00)</td>
</tr>
<tr>
<td>3</td>
<td>Diastolic Dysfunction</td>
<td>02 (4.00)</td>
</tr>
<tr>
<td>Total no. of Survivor's</td>
<td>50</td>
<td></td>
</tr>
</tbody>
</table>

P=0.052 statistically significant

Note:- 2-D Echo could be done only in survivor's of Aluminium phosphide poisoning. The above table shows that in survivors of aluminum phosphide poisoning 2-D echocardiography was normal in 45 (90.00%) followed by myocarditis in 3 (6.00%) patients.
Table 4: Relation of 2-D ECHO changes in Survivors of Ethylene dibromide poisoning S.NO 2-D ECHO changes

<table>
<thead>
<tr>
<th>S. No.</th>
<th>2-D ECHO changes</th>
<th>Ethylene dibromide %</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>With in normal limit</td>
<td>11 (84.61)</td>
</tr>
<tr>
<td>2</td>
<td>Pericardial effusion</td>
<td>02 (15.38)</td>
</tr>
<tr>
<td>Total no. of Survivor’s</td>
<td>13 -</td>
<td></td>
</tr>
</tbody>
</table>

P=> 0.05 statistically non significant

Note:-2-D Echo could be done only in survivor's of Ethylene dibromide poisoning. The above table shows that in survivors of ethylene dibromide poisoning echocardiography was normal in 11 (84.61%) followed by pericardial effusion in 2 (15.38%) patients.

Discussion
In present study of aluminium phosphide poisoning normal ECG found in 39.13% cases. Chugh et al (1989)[6] found normal ECG in Aluminium phosphide poisoning in 58.3% cases. Katira et al (1990) observed normal ECG in 18.27% cases. Gupta et al (1995)[7] noted normal ECG in 20%. Mortality associated in present study with normal ECG finding in Aluminium phosphide poisoning was 27.777%

In present study of aluminium phosphide poisoning ECG changes seen most frequently were ST-T changes in 46.73% cases. Gupta et al (2002) observed in their study on aluminium phosphide poisoning found ST-T change to be common finding in 50% of cases. Chugh et al (1989)[6] noted ECG abnormalities in 80% cases (with ST-T changes in 40%) of aluminium phosphide poisoning. Jaiswal et al (2009)[8] reported that commonest ECG finding in aluminium phosphide poisoning was STT changes. ST-T changes in Aluminium phosphide poisoning were also observed by Trivedi et al (1992)[9] in 30% cases and Gupta et al (1995) in 40% cases.

ST-T changes are probably due to focal damage of myocardium by the poison. ECG changes in aluminium phosphide poisoning reflecting the deleterious effect of poisoning on the myocardium (toxic chemical myocarditis) resulting in focal myocardial damage, as the pattern of ST T wave changes is not uniform and do not fit into distribution areas of any coronary artery pattern.

In present study of aluminium phosphate poisoning sinus tachycardia was seen in 11.95% with mortality of 18.18%. Sinus tachycardia in Aluminium phosphide poisoning was the prominent supra ventricular arrhythmia (17.5%) in study by Katira et al (1990)[10].


Conclusion
Noteworthy finding was absence of correlation between cardiovascular involvement, histopathological changes and ECG findings. It was seen that even if ECG showed normal pattern there were significant histopathological changes in heart.

References