FETAL DEATHS AND PARENTAL EXPOSURE TO CHEMICAL WARFARE AGENTS

HAMID POUR-JAFARI

From the Dept. of Genetics, Hamadan University of Medical Sciences, Hamadan, Islamic Republic of Iran.

ABSTRACT

In the present work we studied the incidence of fetal deaths among conceptions whose fathers or both parents were survivors of chemical warfare attacks by Iraq. We showed that there was significant correlation between frequency of fetal deaths among conceptions which eventuated after exposure of the parents to gas bombs compared to the frequency of fetal deaths among the control group. This work shows that some lethal mutations probably result from the chemical warfare agents.

MJIRI, Vol. 6, No. 2, 87-88, 1992

INTRODUCTION

The use of inorganic chemicals to control insects possibly dates back to historic Greece and Rome. Homer mentioned the fumigant value of burning sulfur, and Pliny the Elder advocated the insecticidal use of arsenic and referred to the use of soda and olive for the seed treatment of legumes.1

The first large-scale use of chemical warfare agents occurred on April 22, 1915.2 During World War I it was noted that mustard gas poisoning caused bone marrow aplasia, dissolution of lymphoid tissue, and gastrointestinal ulceration.3

Despite the Geneva international protocol of 1925 for the inhibition of the use in war of both chemical and bacteriological warfare agents, the frequent use of chemical warfare by the Iraqi regime against both their own people and the Iranian nation has resulted in increased concern, unfortunately without any practical positive action regarding their application.4

In 1990, we interviewed more than one thousand male survivors of chemical attacks during the Iran-Iraq conflict. We selected them randomly, and the results of 807 of these interviews which were more complete were studied. All of these 807 males were married, at the moment of exposure had no special clothing, and some had used a gas mask only for a few moments.

The aim of our study was to detect any relation between fetal deaths and parental exposure to chemical gas bombs.

MATERIAL AND METHODS

As mentioned formerly, we interviewed more than one thousand men who were married and in different locations. 387 (48.0%) were civilian, 237 (29.7%) Revolution Guards, 151 (18.9%) volunteer forces and 27 (3.4%) military. We could not state whether the same gases had been used, as no analysis was performed on our samples. There was no doubt however, that they were injured with chemical war agents, and their names were in the lists of chemical victims in the official reports. The victims reported that the most frequent chemical agent used by Iraq in their chemical attacks was mustard gas. 75.5% of those exposed stated that the type of poison was pure mustard gas or mixed with other gases, e.g; nerve gas. 57.6% of them said that after injury, they had been confined to bed, and 85.9% of victims were distressed by their injury at the moment of our interview.

They were 807 men aged between 18 and 85 years old (mean age 33.53 years). About one-third of them (25.4%) said that their wives had been exposed also. Age of their wives was from 15 to 63 years old (mean 28.98 years). The number of their pregnancies was between 0.00 and more than 10 (.1%). The time interval between exposures and the time of interview was from 3.13 to 3.82 years.

Based on our information, derived from questionnaires, there were 2275 pregnancies and these were divided into two groups: 1) 1728 conceptions which
Fetal Deaths and Chemical Warfare Agents

occurred when the parents were not poisoned (control group), conceptions which occurred after exposure of the parents to gas bombs. We compared the incidence of fetal deaths (abortion and stillbirth) in these two groups of pregnancies.

RESULTS

Table I shows the incidence of fetal deaths in the two groups of conceptions (males & females). Group I is the control group.

Table I. The incidence of fetal deaths in two groups of conceptions

<table>
<thead>
<tr>
<th>Group I (Control)</th>
<th>Group II</th>
</tr>
</thead>
<tbody>
<tr>
<td>Event Count (Fetal deaths)</td>
<td>30</td>
</tr>
<tr>
<td>Interval Length (Total Pregnancies)</td>
<td>1728</td>
</tr>
<tr>
<td>Rate Estimate</td>
<td>0.017361</td>
</tr>
</tbody>
</table>

Rate ratio = 2.73784  P value = 8.86734E-5
Test statistic z = 3.91976  Significant (p< 0.0001). |

Table II shows the incidence of fetal deaths in two groups of male pregnancies. Group I is the control group.

Table II. The incidence of fetal deaths in male conceptions

<table>
<thead>
<tr>
<th>Group I (Control)</th>
<th>Group II</th>
</tr>
</thead>
<tbody>
<tr>
<td>Event Count (Fetal deaths)</td>
<td>23</td>
</tr>
<tr>
<td>Interval Length (Total Pregnancies)</td>
<td>958</td>
</tr>
<tr>
<td>Rate Estimate</td>
<td>0.0240084</td>
</tr>
</tbody>
</table>

Rate ratio = 1.65505  Test statistic z = 1.42985
P value = 0.152759.

Table III shows the incidence of fetal deaths in two groups of female conceptions. Group I is the control group.

Table III. The incidence of fetal deaths in female conceptions

<table>
<thead>
<tr>
<th></th>
<th>Group I (Control)</th>
<th>Group II</th>
</tr>
</thead>
<tbody>
<tr>
<td>Event Count (Fetal deaths)</td>
<td>7</td>
<td>14</td>
</tr>
<tr>
<td>Interval Length (Total Pregnancies)</td>
<td>770</td>
<td>245</td>
</tr>
<tr>
<td>Rate Estimate</td>
<td>9.09091 E-3</td>
<td>0.0571429</td>
</tr>
</tbody>
</table>

On the basis of the results stated in Table I, fetal deaths among progenies of exposed parents is higher than the control group such that the difference is significant (P< 0.00009).

As Table II shows, the difference between fetal deaths among group I and group II male conceptions were slightly different but not significant.

Table III shows the incidence of fetal deaths among female conceptions in the two groups. As we can see, the rate ratio is 6.28571, meaning there is a significant positive correlation between the results of the two groups.

Although we did not consider factors which can change, the incidence of fetal deaths, the high rate of fetal deaths among the progenies of exposed victims strongly suggest that unfortunately the parental exposure might be involved as a mutagen, increasing the incidence of lethal germinal mutations.

ACKNOWLEDGMENTS

The author wishes to thank F. Azizi, M.D., Jalali, M.D., Mr. Mahjob, M.S., Mr. Mani, M.S., M. Sadjjadi, Ph. D., and Mr. Kohansali, M.S., for their sincere help. Finally I have to greatly thank my students for their assistance.

REFERENCES

3. Lari MM: Nitrogen mustard. Abstracts of First International Medical Congress on Chemical Warfare Agents in Iran, Mashhad University of Medical Sciences, Mashhad, Iran, article No. 48, 1988.
4. Al- Saadi Z: Human protection against poisoning by military chemical weapons particularly organophosphates. Abstracts of First International Medical Congress on Chemical Warfare Agents in Iran, Mashhad University of Medical Sciences, Mashhad, Iran, article No. 49, 1988.